REPUBLIC OF KENYA

MINISTRY OF HEALTH

# Accelerating Progress Towards Universal Health Coverage

## Clinical Guidelines for Management and Referral of Common Conditions at Level:

**Level 4: Primary Care Hospitals**
**Level 5: Secondary Hospitals**
**Level 6: Tertiary Hospitals – National Hospitals**
**National Teaching and Refferal Hospitals**

## Volume 3

2024

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# List of Abbreviations

| | |
|---|---|
| ACT | Artemesinin combination treatment |
| AIDS | Acquired immune deficiency syndrome |
| APGAR | Appearance, pulse, grimace, activity, aspiration |
| ART | Antiretroviral therapy |
| ATLS | Advanced trauma life support |
| ARV | Antiretroviral drug |
| BCC | Behaviour change communication |
| CBO | Community-based organization |
| CHEW | Community health extension worker |
| CHW | Community health worker |
| CRHS | Child and Reproductive Health Services |
| CSHP | Comprehensive school health programme |
| CSOM | Chronic suppurative otitis media |
| DCT | Diagnostic counselling and testing |
| DEH | Division of Environmental Health |
| DHMT | Division of Leprosy, Tuberculosis and Lung Diseases |
| DLTLD | District Medical Officer of Health |
| DOMC | Division of Malaria Control |
| DON | Department of Nursing |
| DOTS | Directly observed therapy, short course |
| FP | Family planning |
| GOK | Government of Kenya |
| GORD | Gastro-oesophageal reflux disease |
| GUD | Genital ulcer disease |
| GYN | Gynaecology |
| HAART | Highly active anti-retroviral therapy |
| HAPAC | HIV/AIDS Prevention and Care Project |
| HFA | Health For All |
| HIV | Human immunodeficiency virus |
| HSV | Herpes simplex virus |
| IEC | Information, education and communication |
| INH | Isoniazid |
| ITN | Insecticide treated net |
| IUCD | Intrauterine contraceptive device |
| IUFD | Intrauterine foetal death |
| JRA | Juvenile rheumatoid arthritis |
| KCCT | Kaolin cephalin clotting time |
| KEPH | Kenya Essential Package for Health |
| KMC | Kangaroo mother care |
| KOH | Potassium hydroxide solution |
| LBW | Low birth weight |
| MDGs | Millennium Development Goals |
| MDR-TB | Multiple drug resistant TB |
| MOA | Ministry of Agriculture |

| | |
|---|---|
| MOEST | Ministry of Education, Science and Technology |
| MOH | Ministry of Health |
| MOPW | Ministry of Public Works |
| MOU | Memorandum of understanding |
| MOWI | Ministry of Water and Irrigation |
| NASCOP | National AIDS/STD Control Programme |
| NCD | Non-communicable disease |
| NGO | Non-government organization |
| NHSSPII | Second National Health Sector Strategic Plan 2005–2010 |
| OB | Obstetrics |
| PEP | Post-exposure prophylaxis |
| PHC | Primary health care |
| PID | Pelvic inflammatory disease |
| PLWHA | Person/people living with HIV/AIDS |
| PMTCT | Prevention of mother to child transmission (of HIV) |
| POP | Plaster of paris |
| PSC | Patient support centre |
| PTI | Prothrombin Time Index |
| SFP | School feeding programme |
| SHN | School health and nutrition |
| SHP | School health programme |
| STI | Sexually transmitted infections |
| TAH | Total abdominal hysterectomy |
| TB | Tuberculosis |
| TOF | Tracheoesophageal fistula |
| TT2 | Tetanus toxoid |
| TURP | Transurethral resection of the prostate |
| UNESCO | United Nations Educational, Scientific and Cultural Organization |
| UNICEF | United Nations Children's Fund |
| UTI | Urinary tract infection |
| VCT | Voluntary counselling and testing |
| WHO | World Health Organization |

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# Preface

These guidelines summarize current medical knowledge, weigh the benefits and harms of diagnostic procedures and treatments, and give specific recommendations based on credible information. In addition, these guidelines aim at supporting the clinical decisions of health care professionals on interventions for specific clinical conditions, discouraging inappropriate practices and improving coordination between different providers. This updated edition of the guidelines takes cognizance of the Kenya Essential Package for Health (KEPH), emphasizing distinct levels of care – including the community – to be provided to defined cohorts of the human life cycle. Specifically, the guidelines have been updated in relation to the following:

- Defining care protocols by the service delivery level, recognizing that the skills and facilities for care differ at the different levels of health care.
- Providing greater elaboration of the identification and preparation for referral of clients in case the presenting condition or state doesn't allow for management at the level where the client has presented.
- Updating management protocols to address current conditions and potential threats to the health of Kenyans.
- Including a process for monitoring and reviewing the guidelines.

For ease of reference and use, the guidelines are presented in 3 volumes:
Volume 1: Management Guidelines for Level 1 (Community)
Volume 2: Management Guidelines for Levels 2 and 3 (Primary Care)
Volume 3: Management Guidelines for Levels 4–6 (Hospitals).

The healthcare sector hopes that these guidelines will serve the users well as a guide for the appropriate care expected to be delivered at each level in the health system, thus facilitating the realization of Universal Health Coverage (UHC). Any information that could improve the management protocols is welcome and can be provided directly to the Office of the Director General for Health.

This collaborative effort has brought together health workers from all sectors - our universities, private and government facilities. We look forward to health workers using these guidelines to improve the quality of care given to all Kenyans as we strive towards a healthy and productive nation

**Dr Patrick Amoth, EBS**
**Director General for Health**

# Acknowledgment

These Clinical Guidelines for the management and referral of common clinical conditions have been developed through the contribution of many individuals and institutions committed to improving health outcomes. In particular, we are grateful to the Heads of Directorates, Departments, Divisions and programmes, and the County Health Management Teams that provided support.

The Ministry of Health wishes to thank all the contributing authors, led by the National Medicines and Therapeutics Committee (NMTC) and the Technical Working Group (TWG), for their expertise and time in writing these guidelines.

I take this opportunity to appreciate the effort of the secretariat and the team of experts indicated in the list of contributors.
I acknowledge the tremendous support the World Health Organization (WHO) provided towards finalizing these guidelines.

Finally, the Ministry would like to thank all those we have not enumerated who were either consulted during the development and review of the clinical guidelines or who contributed to this process in one way or another. This work would not have been possible without their contributions, and we are greatly indebted.

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# Introduction

The main objective of the health sector in Kenya is to prevent ill health. However, when this objective is not met, the medical and social implications of the resulting ill health are addressed. Clinical management relates to this objective by ensuring efficient and effective management of the impact of ill health. Clinical management complements public health services by ensuring that a specified quality of essential medical care is made available as needed, when needed, and in appropriate amounts**.**

## Rationale for Revision of Clinical Guidelines

The last time clinical guidelines in the health sector were revised was in 2002. Back then, the guidelines defined management approaches for the key conditions expected to affect the Kenyan population. However, the guidelines in 2002 had some weaknesses, including the following:

- The health sector lacked a clear, comprehensive, evidence-based approach to service delivery. Such an approach is crucial as it provides the overall guidance for the services the sector intends to provide, plus the process for delivering the services.
- The mechanism for monitoring and updating the clinical guidelines was not clear. As a result, new management protocols that have come up since the guidelines were developed have not been incorporated. Examples of these protocols include such as for avian influenza, management of multi-drug resistant tuberculosis (MDR/XDR TB), use of artemisinin combination treatment (ACT) for management of malaria, use of anti-retroviral drugs (ARVs) in HIV management, non-communicable diseases, and injuries/violence management, among others.
- Guidelines for the preparation and management of clients for referral were not included.

Besides these innate shortcomings, the clinical guidelines predated the approach to service delivery grounded in the framework of 6 life-cycle cohorts and 6 levels of care, as set out in the Kenya Health Sector Strategic Plan (KHSSP – 2018–2023). Thus, the 2002 guidelines did not consider the new approach that calls for different capacities and functions at the different service levels in the country. Significantly, there was no guidance on the management of services at community level, and the lack of a referral framework is a drawback that has become more apparent as the care-level approach has become institutionalized. The current updated guidelines attempt to address these shortcomings. In addition, they are aligned with the comprehensive, multilevel service delivery approach defined by the Essential Package for Health (KEPH**).**

# Comprehensive Service Delivery Approach

The review of the Kenya Health Sector Strategic Plan (KHSSP 2018-2023) highlighted stagnating or downward trends in health indicators, especially in key maternal, newborn, and child health areas. To respond to this worrying trend, the health sector in Kenya initiated an accelerated reform process to halt and reverse this trend.

The reform process is enshrined in Kenya Health Policy (2014-2030) which states the midterm goal of the health sector as "To reduce health inequalities and reverse the downward trends in health-related outcome and impact indicators". The defined strategic objectives of the plan are to:

- Increase equitable access to health services.
- Improve the quality and responsiveness of services in the sector.
- Improve the efficiency and effectiveness of service delivery.
- Foster partnerships in improving health and delivering services; and
- Improve financing of the health sector.

As part of the reform process, the sector elaborated clear operational approaches to achieve its strategic objectives and health service norms and standards.[3] Investment plans now guide multi-year investment priorities for different key areas of the sector .[4] The comprehensive service delivery approach is one of these operational approaches (refer to Figure A).

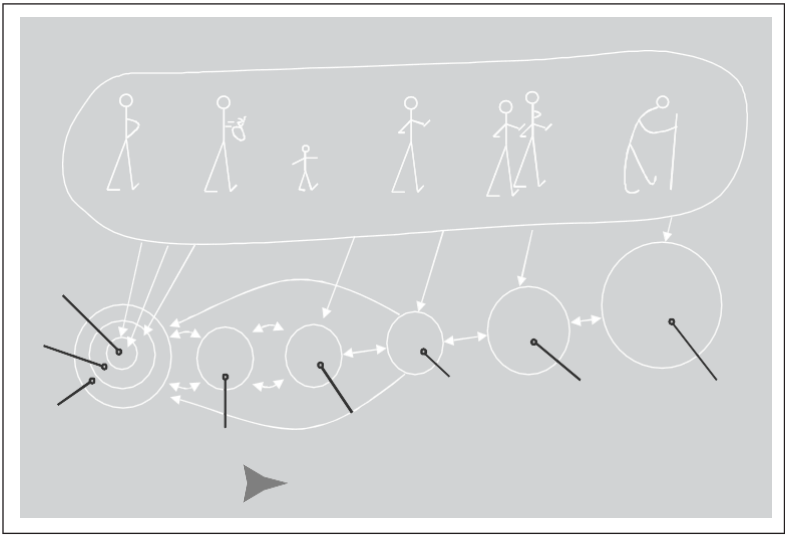
A comprehensive service delivery approach is based on the provision of guidance – at community, dispensary/health centre, and hospital levels of care – on services to be provided, service standards to be attained, service inputs (human resource, infrastructure, equipment) to be applied, and cross-linkages of services. This comprehensive approach guides the investment priorities for service delivery at the administrative level and the form and content of clinical management.

The services for each level of care are defined in the Kenya Essential Package for Health (KEPH). A particular focus of the package is the community level.[5] The service linkages are defined in the Sector's Referral Strategy. These documents describe the overall strategic approach for the sector and are further elaborated.

# The Kenya Essential Package for Health

KEPH is a life-cohort-based approach to the delivery of health care services. Its main focus is to define the priority services that will ensure a healthy population at 6 distinct levels of the health system–from the community level up to tertiary

**Figure B: The KEPH system**



hospitals – for each of 6 defined life cohorts. As a result, it describes, in a comprehensive manner, the services the sector is to prioritize to maintain health at all the different stages of life.

The diagram in Figure B illustrates the 6 life-cycle cohorts defined by KEPH: pregnancy and the newborn (up to 2 weeks); early childhood (to 5 years); late childhood (6–12 years); adolescence and youth (13–24 years); adulthood (25–59 years); and the elderly (60+ years). The diagram also illustrates the linkages of the 6 levels of care that KEPH defines:

- Level 1: Community: Village/households/families/individuals
- Level 2: Dispensaries/clinics
- Level 3: Health centres, maternities, nursing homes
- Level 4: Primary hospitals – County and subcounty hospitals
- Level 5: Secondary hospitals – Regional referral hospitals
- Level 6: Tertiary hospitals – National hospitals

The expected services to be provided are described in Table A. The KEPH has the following key characteristics:

- It emphasizes health (rather than disease), rights (rather than needs), and community empowerment to exercise their rights.
- It identifies and redefines 6 distinct functional levels of care. The community level is the first level of care, where significant decisions are made, and interventions that have an immediate impact are done. The focus at the community level is on the promotion of family practices that preserve and promote health.

**Table A KEPH Strategic Interventions, by Level and Life-Cycle Cohort**

| Level 1 (Community) | Level 2 (Dispensary/ clinic) | Level 3 (Health Centre) | Level 4 (Primary/county/ Subcounty hospital | Level 5 (Secondary/ regional referral hospital) | Level 6 (Tertiary/ national hospital) |
|---|---|---|---|---|---|
| ***Cohort 1: Pregnancy, delivery and newborn (to 2 weeks)*** | | | | | |
| Equip targeted communities with current knowledge and facilitate appropriate practices and attitudes leading to safe pregnancy and delivery of a healthy newborn | Ensure that health facilities are equipped to provide very basic ANC and refer all deliveries (regardless of risk analysis) | a) Ensure health centres are equipped to provide basic essential obstetric care<br>b) Enhance health systems to support the delivery of quality obstetric and newborn care<br>c) Establish a functional, supportive supervision system<br>d) Develop outreach programmes to serve "hard-to-reach" populations | Ensure that facilities are equipped to provide essential comprehensive obstetric care | Ensure that facilities are equipped to provide essential obstetric care | Ensure provision of facilities to manage mothers and newborns referred from lower levels adequately |
| ***Cohort 2: Early childhood (0–5 years)*** | | | | | |
| Equip the community and health care providers with knowledge about the prevention of common childhood diseases and disabilities; and facilitate appropriate practices and attitudes leading to healthy child growth and development | a) Develop an outreach programme for 'hard to reach populations.<br>b)Strengthen promotion/ prevention of common childhood illnesses/disabilities<br>c)Strengthen case management and surveillance of common childhood illness<br>d)Establish supportive supervision systems to ensure quality assurance | a) Strengthen prevention of common childhood illnesses, and disabilities.<br>b)Strengthen case. management & surveillance of common childhood illnesses.<br>c) Enhance the health systems support for the delivery of quality child health services<br>d) Establish a functional supportive supervision system to ensure quality assurance<br>e) Develop outreach programmes to serve the | "hard-to-reach" populations | Ensure availability of facilities, diagnose and appropriately manage sick children | Recognize and appropriately manage a sick child |

Ensure provision of facilities to adequately manage children referred from lower levels

**Table A** ***continued***

| Level 1 (Community) | Level 2 (Dispensary/clinic) | Level 3 (Health centre) | Level 4 (Primary/county/ subcounty hospital) | Level 5 (Secondary/ regional referral hospital) | Level 6 (Tertiary/ national hospital) |
|---|---|---|---|---|---|
| ***Cohort3: Late childhood 6–12 years)*** | | | | | |
| Equip the child with relevant knowledge And skills that promote<br><br>a healthy lifestyle, including psychosocial development | a) Develop an outreach programme to serve hard to-reach populations<br><br>b)Strengthen the promotion and prevention of common illnesses, impairments, and disabilities in late childhood.<br>c)Strengthen the case management and surveillance of common late childhood illnesses<br>d)Establish a functional supportive supervision system to ensure quality assurance | Facilitate and support caregivers and community in the<br><br>provision of a safe environment for child survival, growth and development | a)Ensure that the health regional team is able to recognize and appropriately<br><br>manage a sick child and. where necessary refer.<br>b)Facilitate rehabilitative care for disabilities and integration of children with disabilities | Strengthen hospitals to diagnose and manage compli-<br><br>cated childhood medical and surgical conditions | Ensure the provision of facilities to adequately manage children referred<br><br>from lower levels. |
| ***Cohort4: Adolescence and youth (13–24 years)*** | | | | | |
| Equip the youth with knowledge and life skills, and facilitate creation of a supportive environment to enhance adoption of healthy lifestyles for themselves and the community | Create an enabling environment for young people that discourages harmful practices, encourages psycho- social development, and prevents disease and injuries | Create an enabling environment for young people that discourages harmful practices and prevents disease and injuries | a) Ensure availability and access to quality youth-friendly services to encourage appropriate care seeking amongst the youth<br>b) Ensure provision of rehabilitative services for substance abusers | a) Ensure provision of comprehensive rehabilitative services for youth drug abusers<br>b) Ensure access to quality youth-friendly referral services for management of complicated medical and surgical conditions | Ensure provision of facilities to adequately manage youth referred from lower levels |

*Continued*

**Table A** ***continued***

| Level 1 (Community) | Level 2 (Dispensary/clinic) | Level 3 (Health centre) | Level 4 (Primary/ county/ subcounty hospital) | Level 5 (Secondary/regional referral hospital) | Level 6 (Tertiary/ national hospital) |
|---|---|---|---|---|---|
| ***Cohort 5: Adulthood (25–59years)*** | | | | | |
| Equip adults with knowledge and skills to facilitate creation of a supportive environment to enhance adoption of healthy lifestyles for themselves and the village | Provide information on and Encourage utilization of recommended services for disease/injury prevention and facilitate creation of supportive environment to enhance adoption of healthy lifestyles | Equip health facilities with staff who can conduct. general medical and reproductive care assessment, disease/. injury prevention and refer. complicated cases to the county hospital | Ensure accessibility to quality curative services for adults with acute and chronic conditions | Ensure access to quality services for the diagnosis and management of complicated and surgical | Ensure provision of facilities to adequately manage seriously ill adults referred from lower levels |
| ***Cohort6: Elderly (60+years)*** | | | | | |
| Equip the elderly persons, the community and health care providers with relevant knowledge on common old age diseases, impairments, and disabilities in old age; and how to improve quality of life and enhance longevity | a) Provide information on and encourage utilization of recommended services for disease/injury prevention<br>b) Refer complex cases to health centre | a) Provide information on and encourage utilization of recommended services for disease/injury prevention<br>b) Refer cases to county hospital | a) Ensure early recognition and appropriate management of acute and chronic illnesses/injury as per recommended guidelines.<br>b) Provide appropriate comprehensive and special rehabilitation to older persons with chronic illnesses and disabilities at all levels | Ensure provision of facilities for the diagnosis and management of severe illnesses in old age | Ensure provision of facilities to adequately manage seriously ill older persons referred from lower level |

- Its overall thrust is revitalizing health promotion and preventive care at the first 3 KEPH levels.
- It defines health needs at each level of human development – from birth to old age–and identifies comprehensive and cost-effective interventions required at each stage of the human life cycle.
- It recognizes the packages of health care services per level of care to be rendered by public and private health service providers.

KEPH is expected to improve the quality of services at levels 1–4 so that clients have confidence in these levels of care. This will result in increased client utilization of the lower-level health facilities. KEPH is also expected to improve the networking of providers and facilities at the different levels of the health system, thereby ensuring continuity of care for those who need the services provided at the higher levels of the system.

## Sector Norms and Standards

Norms and standards defined to guide the provision of KEPH services are a statement of the human resource, infrastructure, equipment, and financing inputs. These services are necessary to ensure efficient and effective delivery of health care services to the Kenyan population. Service delivery standards relate to the expectations of each level of care concerning service delivery and the types of human resources needed to provide these expectations. Service delivery norms define the quantities of these resource inputs required to efficiently, effectively, and sustainably offer the service delivery package. These norms and standards are defined based on the following principles:

- ***Units of service delivery:*** The focus is on the function, as opposed to the physical level, as a higher-level facility may provide the function.
- ***Equity in access and utilization:*** All inhabitants of the country and its respective districts have an equal right to access health services and use them equally for equal needs.
- ***Relevance and acceptability:*** Healthcare must be rooted in the communities' cultural and social reality and include user satisfaction in the healthcare delivery equation.
- ***Continuity of care:*** Care should be viewed in a continuum, from the start of the illness or the risk episode until its resolution, irrespective of the level at which care is sought. This means that a functional referral and counter-referral system should exist to ensure services are available.
- ***Integration of care:*** Every contact is used to ensure that a comprehensive set of defined services is available.
- ***A comprehensive/holistic approach:*** Health services must consider all the dimensions of the persons and their environment and maintain a permanent interaction and dialogue with clients.

- ***The involvement of individuals, households, and communities:*** *Involvement is expressed* in people taking up responsibility for their health; it provides them with a sense of ownership of all they undertake relating to their health

## Process of Elaborating the Clinical Management Guidelines

The current revision of clinical management guidelines has been carried out through an extensive 3-year consultative process between 2018 to 2022. The process has been coordinated by the top management in Government in the Ministry of Health through the office of Director General

Technical coordination of the revisions was structured around the key disciplines of Medicine, Surgery, Obstetrics/Gynaecology, and Paediatrics. A lead technical specialist from each of these areas was in charge of coordinating the internal consultation process in each of these areas. In addition, specialists in pharmacy reviewed and guided the definition of the medicines and medical products included in the management protocols, ensuring that these protocols are harmonized with the Essential Medicines List.

Four stakeholder consultations were held over the 3 years to ensure that the management protocols being defined were in line with the overall policy direction from the programme and Ministry levels and that their implementation was feasible. This involved management and technical specialists from the public and non-public sectors in each respective area.

## Description of the Revised Clinical Management Guidelines

In line with the process described above, this new addition to the clinical management guidelines is based on the latest orientation for each condition expected to afflict the population in Kenya. These are both for conditions in existence and conditions recognized as threats to the population.

Management descriptions are comprehensive, based on the expected capacity at each level of care. Descriptions of each condition are set out in terms of how it presents, physical and laboratory investigations for diagnosis, and the appropriate management, including when a referral has to be made.

**The referral management includes:**

- Identifying signs during client management that indicate referral should be considered.
- Preparing the client for a referral.
- Arranging the required logistics for a referral at the referring and receiving facility, plus during transport.
- Ensuring the receipt and emergency management of the client who has been referred.
- Managing the referred client by the referring facility when they return.

**For relevance, alignment with the service delivery approach, and ease of use, the guidelines are presented in 3volumes representing the major levels of care:**

- Volume I: Clinical Management and Referral Guidelines for Community Care– Corresponding to level 1 of the health care system
- Volume II: Clinical Management and Referral Guidelines for Primary Care– Corresponding to levels 2 and 3 of the health care system
- Volume III: Clinical Management and Referral Guidelines for Hospital Care – Corresponding to levels 4–6 of the health care system

# Referral Strategy

Categorizing KEPH into the 6 levels of care is primarily meant to rationalize the delivery of health services within the health system for efficiency in using existing resources. However, the implication is that a client in the health service delivery unit may have direct access to or be unable to adequately manage their health care needs. The referral system is intended to address this shortcoming. A referral system is defined as a mechanism to enable clients' health needs to be comprehensively managed using resources beyond those available where they access care. It is based on the premise that while capacity for health service delivery has to be rationalized around different levels of care, the services received by clients should not be determined only by the services available where they access care but rather by the full scope of care the health system can provide in the country.

An effective referral chain provides the linkages needed across the different health system levels – from level 1 (community) to level 6 (national hospitals). These linkages ensure that a given healthcare need of a client can be addressed irrespective of the level of the health system at which the client first physically accesses care. The referral system can thus be likened to an "elevator/lift" in a multistorey building: facilitating forwards and backward management of clients across different floors (levels of care).

The referral strategy thus guides the sector in building an effective referral system that responds to the needs of rural and poor populations, thereby contributing to the realization of Vision 2030 and the Sustainable Development Goals.

The community level consists of household caregivers, community health promoters (CHP) and community health assistants (CHAs) who are linked to a primary healthcare facility for referral. These providers are trained to identify illness, determine its severity and provide prompt management or referrals, when they cannot treat or if there is need for a continuum of care at higher-level health facilities. CHPs must refrain from carrying out procedures beyond their level of proficiency as guided by their training,

# The Process of Physical Referral

## Critical Inputs to Have at the Facility to Expedite Referral

| Input | Category | Type of input |
|---|---|---|
| | Description of needs Description | Number |
| Equipment | Emergency tray | |
| Emergency room | 4x4 ambulance | |
| Motorized bicycle | | |
| Staff | | |
| Supplies | Referral forms | 3-month supply |

# Referral Instruments

***1.*** ***Preparation of a client for referral***

1.1 Referral for a pregnant mother
1.2 Referral of a child with a medical problem
1.3 Referral for a child with a surgical problem
1.4 Referral for an adolescent, adult, or elderly patient for a medical problem
1.5 Referral for an adolescent, adult, or elderly patient for a surgical problem

***2.*** ***Handling of a client during referral***

2.1 Referral for a pregnant mother
2.2 Referral of a child with a medical problem
2.3 Referral for a child with a surgical problem
2.4 Referral for an adolescent, adult, or elderly patient for a medical problem
2.5 Referral for an adolescent, adult, or elderly patient for a surgical problem

***3.*** ***Receipt and emergency management of a client who has been referred***

3.1 Referral for a pregnant mother
3.2 Referral of a child with a medical problem
3.3 Referral for a child with a surgical problem
3.4 Referral for an adolescent, adult or elderly patient for a medical problem
3.5 Referral for an adolescent, adult or elderly patient for a surgical problem

***4.*** ***Follow up of a client who has been referred back***

4.1 Referral for a pregnant mother
4.2 Referral of a child with a medical problem
4.3 Referral for a child with a surgical problem
4.4 Referral for an adolescent, adult, or elderly patient for a medical problem
4.5 Referral for an adolescent, adult, or elderly patient for a surgical problem

# PART I: Internal Medicine and Related Disciplines

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# 1. Acute Injuries,Trauma,and Selected Emergencies

## 1.1 Anaphylaxis



Anaphylaxis occurs as allergic reaction to allergens facilitated by mediators in a sensitized individual. Allergens may be drugs, food, sera, stings, and intravascular contrast media.

**Clinical Features**
Include pruritus, urticaria, respiratory distress (due to laryngeal oedema, bronchospasm), and hypotension.

**Management**

Follow the ABC algorithm

- **Airway protection:** use of airway devices like the oral pharyngeal airway, laryngeal mask airway (LMA) and administration of oxygen using non-rebreather mask at 10l/min
- **Breathing:** make sure the patient is breathing if not ventilate the patient through bag-mask ventilation or artificial ventilation.
- **Circulation:** establish IV access and give fluids, check the blood pressure give s/c adrenaline 0.5mg after every 15 minutes up to 3 doses.
- Avoid offending agents.
- Nebulized with bronchodilators, e.g., salbutamol and ipratropium bromide 0.5mg ***OR*** aminophylline 6mg/kg IV over 20 minutes if there is wheezing and nebulization not possible.
- Antihistamine:
  - Chlorpheniramine10mg IV slowly. IM/SC then continued 10mg 8 hourly for 24–48 hours (children 0.1mg/kg)
  - 100mg IV is of secondary value but useful to prevent delayed recurrences
- Patients with mild to moderate reaction, e.g., urticaria or mild bronchospasm, should be observed for at least 6 hours because attacks may recur after full recovery.

**Admission**
Severe reactions, e.g., hypotension, severe bronchospasm (especially with orally ingested antigens). Severe reactions require intravenous fluid replacement with normal saline and close monitoring especially of BP and urinary output.

## 1.2 Cardiac Arrest

Cardiac arrest occurs due to asystole, ventricular fibrillation, and cardiovascular collapse in extreme arterial hypotension. There is absence of heart sounds and of carotid and femoral pulses. There may be associated apnoea and cyanosis.

— Cessation of circulation requires immediate treatment.

Optimal chances of survival are achieved with initiation of cardiopulmonary resuscitation within 4 minutes of the arrest, and when advanced cardiac life support including intubation, intravenous medications, and defibrillation is initiated within 8 minutes.

**Management**

**ABCD**

***Airway:***

- Open secure and maintain patent airway, use of airway devices like oral pharyngeal airway and laryngeal mask airway.
- Aspirate vomitus and secretions or remove with gloved fingers or handkerchief.

***Breathing – ventilation***

- Use bag mask ventilation even without oxygen, and if oxygen is available connect at 15l/min.

***Circulation:***

- Establish IV access
- **Cardiac massage:** Carry out external cardiac massage (compressions) by applying appropriate pressure over the sternum. Five breaths should be interposed between every 4 to 5 cardiac compressions.
- **Defibrillation:** Use standard defibrillators delivering 150–200J. Automated fibrillation is commonly used nowadays.

***Drugs***

- Intravenous adrenaline 1mg bolus, repeated every 3 to 5 minutes,***OR***
- Vasopressin 40IU by intravenous push, OR amiodarone 300mg in 5% dextrose administered over 30 minutes.
- Thorough investigation and treatment of the underlying causes should be undertaken.

***Admit***

- Defibrillation:
  - Standard defibrillators delivering 200–360J.
  - Biphasic defibrillators delivering 150–200J.
  - Intravenous epinephrine 1mg push, repeated every 3 to 5 minutes, ***OR*** vasopressin 40IU by intravenous push, ***OR*** amiodarone 300mg in 5% dextrose run over 30 min.
- Thorough investigation and treatment of the underlying cause should be undertaken.

**Table 1.1: Other Selected Emergencies**

| | Emergency | Management |
|---|---|---|
| 1. | Acute asthmatic attack | ABC, oxygen, treat underlying cause<br>Nebulize with salbutamol/ipatroprium bromide 3 doses every 20 min for 1 hr, give hydrocortisone 2mg/kg max 200mg.<br>If deteriorating give magnesium 2mg in 5% dextrose over 20 min. Consider intubation if there is respiratory failure and admit ICU/HDU |
| 2. | Acute coronary syndrome | Monitor, ABC, connect to the defibrillator, prepare to provide CPR and thrombolysis.Obtain and review 12 lead ECG, check vital signs, connect to oxygen at 10l/min. Brief history, examination, time of symptom onset. Establish IV access, take bloods for UECs and cardiac markers (Troponin I). Give aspirin 300mg stat (except in the presence of active PUD). Clopidogrel 300mg stat if less than 75 years then 75mg in combination with 75mg aspirin for one year, if more than 75 years avoid the loading dose. Give nitroglycerin sublingual spry 0.4 mg every 5 minutes max 3 doses. Do not give if blood pressure is lower then 90mmHG. Give pain reliever like fentanyl or morphine. Continue with close monitoring in acute HDU/ICU. |
| 3. | Pulmonary embolism | Monitor, ABC, be prepared to provide CPR, and thrombolysis. Obtain/review 12 lead ECG, vital signs, start oxygen 10l/min, establish IV access, take blood samples for FBC, UECs, D-dimers (done only when the probability of PE is low to rule out PE) if high probability do a CTPA to confirm the diagnosis , troponin I (to rule out myocardial infarction), if confirmed begin anticoagulation therapy and refer to ICU for possible thrombolysis. |
| 4. | Hypertensive emergency<br><br>Defined as a systolic BP > 180 mmHg or a diastolic BP>120 mmHg with the presence of acute target organ damage | Monitor, support ABC, check vital signs. Perform brief targeted history, and perform a physical examination. Obtain/review 12 lead ECG, blood samples for FBC, UECs, TSH, urinalysis, PDT (females in reproductive age when necessary). Commence labetalol 0.3-1.0mg/kg up to maximum dose of 20mg every 10 min. Also can give as an infusion @0.4-1.0mg/kg/hr. (alternatives: hydrallaine).<br><br>The management and BP targets depend on the specific target organ damage) (acute ischemic/hemorrhagic stroke, subarachnoid hemorrahge, aortic dissection, acute coronary syndrome, pre-ecclampsia/ecclampsia). |
| 5. | Stroke | Monitor, support ABC check, check glucose levels and if lower than 3.3mmol/l give bolus of 50mls 50% dextrose and maintain the glucose level at 7.7 mmol/l. Perform a brief targeted physical examination. Blood works, obtain a non-contrast brain CT scan – if there is haemorrhage consult a neurosurgeon. If no bleeding consult a neurologist and commence on aspirin 300mg stat then 75mg od lifelong, and high dose statin (e.g., atorvastatin 80mg for 4-6 weeks, then lower the dose to 20mg lifelong). |
| 6. | Seizures | Monitor, support ABC check, start on oxygen at 10l/min. Position on a soft mattress and pillow on left lateral position, open the airway with head tilt manoevere and maintain this position till the patient is awake. DO NOT RESTRAIN A SEIZING PATIENT. Establish an IV access. If know epileptic give phenytoin 20mg/kg or sodium valporate 20mg/kg if on |

| | Emergency | Management |
|---|---|---|
| | | ARVs. If pregnant give IV magnesium sulphate 4mg diluted in 100mls n/saline and continue the infusion at 2mg/hr. If still seizing give midazolam 0.1mg/kg after 10 min from the last dose, phenytoin 20mg/kg in normal saline STRICTLY OVER 30 MINUTES or sodium valporate for patients on ARVs. If still seizing, intubate and ventilate and admit to ICU and do a CT brain. |
| 7. | Bites (human and animal) | If rabies is suspected, scrub the wound with soap and water for at least 15 minutes then rinse and apply disinfectant eg povidone as soon as after the exposure. Give amoxillin/clavunate 1gm bd for 5to 7 days OR clindamycin 600mg PO/QID/600mg IV TDS OR azithromycin 500mg PO daily for 3 days. DO NOT SUTURE ANIMAL OR HUMAN BITE. The wound should be irrigated copiously, dressed and left to drain, examined daily to detect any signs of sepsis. Always administer tetanus toxoid to all bites. Rabies immunoglobulin (RIG) 20IU/kg is given once and can provide passive immunization. Rabies vaccine should be administered to all suspected rabies cases. The recommended site is the right deltoid (arm) in adults OR anterolateral thigh (children) intramuscularly (IM) (1 ml). Rabies vaccine should NOT be administered in the gluteal muscle. The regime for vaccine administration (days 0,3,7,14- a fifth dose is recommended for immunocompromised cases between 21 and 28 days. Zagreb regimen (days 0,7,21) |
| 8. | Hypglycaemia | This is when blood glucose level is less than 3.9 mmol/l. Monitor and support ABC. Check vital signs BP, PR, RR, T, SPO2. Start oxygen if the SPO2 is less than 94%. If able to take orally with mild hypoglycemia (≥3.0-3.9mmol/l); Treat with oral glucose OR give a sugary snack/drink repeat RBS after 15 minutes, if <3.9mmol/l give another sugary snack/drink, if ≥5mmol/l, give a 15mg of a simple carbodrate meal (rice, white bread, ugali). If not able to take orally establish IV access and give 50mls 50% dextrose, recheck blood glucose in 2 minutes, if <5mmol/l, repeat the step, followed by a 10% dextrose infusion, if ≥5mmol/l and patient alert and able to take orally, give a carbohydrate meal. If no IV access give SC or IM glucagon 1mg stat. If symptoms have resolved and the glucose level is above 3.3mmol/l continue with food dextro-saline at 110mls/hr. Treat underlying cause. Maintain glucose level at 4.4 mmo/l. Consider thiamine 100mg IV for malnourished and alcoholic patients followed by 100mg orally BD for 6 months. If symptoms haven't resolved continue with dextrosaline at 110mls/hr. |
| 9. | Diabetic ketoacidosis (DKA) | DKA-common in type 1 diabetes, can occur in type 2 diabetes). **Hyperglycemia** (RBS ≥11.1mmol/l, rarely may present with normal blood glucose (Euglycemic ketoacidosis), **Ketosis** (serum ketones ≥ 3mmol/l) OR urine ketones (≥3+) and **metabolic acidosis** (PH <7.3 and Bicarbonate (HCO3) <15mmol/l).<br><br>**Management**: ABC, take a focused history, identify the precipitating causes (missed insulin, infection, surgery, MI), Assess the degree of dehydration (BP, capillary refill time, skin turgor), Assess GCS. Treat any underlying infection. |

| | Emergency | Management |
|---|---|---|
| | | **Tests:** RBS (monitor hourly), urinalysis, serum ketones, TBC, UECR, LFTs, Arterial blood analysis (ABGs), blood/urine cultures, CXR, ECG).<br><br>**FLUID PROTOCOL:** (approximate fluid deficit 6 liters).<br>Normal saline 0.9%, 10ml/kg over one hour, repeat. Calculate the fluid requirements, correct fluid deficit over 24-48hours, alternate with 5% dextrose and half-normal saline (0.45%) when sugar is about 13.9mmol/l). Success in fluid therapy is reflected by improvement in haemodynamic and hydration status and PH values. A satisfactory urine output of 1-2mls/kg and clinical progress.<br><br>**POTASSIUM PROTOCOL:** (estimated potassium deficit; 3-6mEq**)** DO NOT give potassium if patient is anuric or potassium is more than 5.3 mmo/l. Check potassium levels hourly. If K+ is <3.3 mmo/l add 20-30 mmol or mEq of potassium chloride(KCL) in 1 litre of normal saline/hr. Withhold insulin and continue with cardiac monitoring with ECG until potassium is at a range of 4.4-5.3 mmo/l.<br><br>If K+ is 3.3-5.3; Give 20-30 mEq K in each L of IVF to keep serum K 4-5 mEq/l.<br><br>**INSULIN PROTOCOL –** DO NOT give insulin until you have the potassium level above 3.4 mmol/l. Give soluble/ regular/short-acting insulin 0.05-0.1 units/kg/hr infusion starting 1 hour after fluid resuscitation (An insulin pump should ideally be used. When not available alternative to insulin protocol:<br><br>Intramuscular insulin: if RBS >22.2mmol/l give 10 units of soluble insulin every 2 hours intramuscularly (IM), if RBS is 13.9-22.2mmol/l, give 5 units of soluble insulin IM every 2 hours, when RBS is <13.9mmol/l with no ketonuria ans patient is well hydrated, can switch to subcutaneous (SC) soluble insulin).<br><br>**Monitoring:**<br>● One hourly RBS, fluid input and output<br>● Two hourly: electrolytes<br>● ECG for T wave changes<br>● Hourly neurological deterioration warning signs (headaches, slowing heart rate, irritability, confusion, GCS, incontinence, specific neurological signs). Exclude hypoglycemia or cerebral edema<br><br>**Cerebral edema management**<br>● Give mannitol 0.5-1gm/kg OR 3% hypertonic saline.<br>● Adjust IV fluids to maintain normal BP but avoid over hydration.<br>● ICU management should be considered.<br>● After stabilizing the patient, consider cranial imaging with<br>● CT scan).<br>If clinical well, ketoacidosis resolved and tolerating oral fluids, transit to subcutaneous (SC) insulin and stop the insulin infusion. Calculate the total daily dose (TDD) of IV insulin from the mean hourly dose for the prior 6 hrs (use 50% as |

| | Emergency | Management |
|---|---|---|
| | | basal insulin and the remaining 50% as bolus insulin to be given with meals. Can also convert to premix insulin |
| 10. | Hyperosmolar hyperglycaemic state (HHS) | Differs from DKA in the following aspects:<br>● Only in type 2 diabetes either known or newly diagnosed.<br>● Marked hyperglycemia (>33.3mmol//540mg/Dl),<br>● Marked dehydration (approximate fluid deficit of 10 liters).<br>● Ketonuria or ketonemia slight or absent<br>● PH> 7.3<br>● HCO3 >18mmol/l<br>● serum osmolality (mOsm/kg) >320<br>● Higher mortality rates than DKA<br><br>**Precipitating factors**<br>infections, diuretic therapies, drinking glucose-rich drinks, MI/stroke.<br><br>**Management**<br>● Initial treatment is the same as DKA.<br>● Insulin requirements are lower than in DKA.<br>● Anticoagulation with heparin should be considered if no contraindications. |

# 1.3 Shock

Shock is a state of circulatory failure characterized by globally impaired tissue perfusion that is insufficient for the body's needs.

## 1.3.1 TYPES OF SHOCK

### HYPOVOLAEMIC SHOCK

This type of shock is due to loss of intravascular fluid volume. It results from blood and/or fluid loss and is due to decreased circulating blood volume leading to decreased diastolic filling pressure and volumes.

**Causes**

- Traumatic haemorrhage
- Non-traumatic haemorrhage (eg. Gastrointestinal bleeding, postpartum haemorrhage, epistaxis)
- Severe burns:
  - Rapid plasma loss from damaged tissues when over 25% BSA is burnt
  - Endotoxaemia makes matters worse
- Dehydration
- Vomiting and diarrhoea (cholera and enterocolitis)
- Septicaemia
- Intestinal obstruction (mechanical or paralytic ileus

**Clinical Features**

The patient becomes cold, clammy, drowsy, and tachypnoeic. There is cold sweat and restlessness, and blood pressure may become unrecordable. The skin is pale and cold with collapsed peripheral veins, with a tachycardia. The urinary output is an indicator of renal blood flow and will fall significantly.
Temperature is subnormal (less than 35$^{o}$C).

**Investigations**

- Hb and PCV
- Urea, electrolytes, creatinine
- Blood sugar
- Group and cross-match blood
- Blood gas analysis If possible
- Blood cultures

**Management**

Once shock is suspected, the medical staff taking care of the patient should initiate appropriate and coordinated emergency management:

- Treat the primary problem, e.g., control haemorrhage, endotoxaemia, etc.
- Secure a large intravenous line; do a cut-down if there is no accessible peripheral line.
- Central venous pressure line is preferable if available.

- Start infusion of isotonic saline (normal saline) or run 2 litres fast in an adult.
- Group and cross-match blood before you give plasma expanders (dextran 70,etc.).
- Transfuse incase the shock is due to blood loss.

**If shock is due to vomiting or diarrhoea, replace continuing fluid loss:**

- ***Adults:*** 1 litre 6 hourly Hartmann's solution or even normal saline.
- ***Continue with IV fluids*** until shock is reversed and cause treated.

- Closely monitor vital signs.
- Monitor urinary output. It's important to catheterize the patient in order to measure the urine output.
- Continue maintenance until shock is reversed and the cause is reversed.
- Undertake surgical intervention as soon as patient is stable (i.e., laparatomy for intestinal obstruction) and broad-spectrum antibiotics for sepsis and burns.

## SEPTIC SHOCK

### Clinical Features

This type of schock is due to systemic sepsis resulting in hypotension or multiple organ failure. Initially "warmshock":increased heart rate; diaphoresis; warmskin. Later "coldshock": decreased cardiac output; cool vasoconstricted skin.

### Complications

- Pulmonaryoedema
- Renal failure
- Disseminated intravascular coagulopathy (DIC)

### Investigations and Diagnosis at Levels 4–6

- Hb, WBC,platelets
- Urea and electrolytes,creatinine
- Blood sugar
- Culture and sensitivity (blood and body fluids)

### Management – General

Resuscitate with normal saline or dextran 70. Large volumes may be required but watch for heart failure. An CVP line is useful at levels 4 and above. In addition:

- Monitor pulse and BP hourly.
- Catheterize and monitor urine output hourly–if less than 20ml/hour after adequate fluid replacement then give frusemide 80mg IV STAT.
- Give oxygen via face mask.
- Determine and definitively treat cause.

### Management – Pharmacological

- Site-specific empiric antibiotic
  - **Pulmonary source**
    - *Commumity acquired pneumonia (CAP)*. Give Cefrtiazone 1gm IV once daily ***OR*** benzyl penicillin 4 mega units IV every 6 hours and macrolide (Azithromycin 500mg od for 3 days OR clarithromycin 500mg bd for 7-10 days), OR respiratory

fluoroquinolones (Levofloxacin 500mg OD 7-14 days OR 750mg OD for 5 days, Moxifloxacin 400mg OD for 10 days)

- *Hospital-Acquired or Ventilator associated pneumonia (HAP/VAP)*: cover for pseudomonas auroginosum and methicillin-resistant staphylococcus aureau (MRSA) mulitidrug therapy with vancomycin 500mg 6-Hourly OR 1gm 12-hourly OR Linezolid 600mg 12-hourly and piperacillin-tazobactam 4gm/500mg IV QID, ceftazidime 2gms 8-hourly, Meropenem 1gm 8-hourly. Duration of therapy 10-14 days.

– **CNS source**
  - Vancomycin 750-1000mg 12-hourly OR 10-15mg/kg IV 12-hourly and Cefriazone 2gm 12-hourly 10-14 days.
  - Ceftazidime 2gm 8-hourly 10-14 days.
  - Meropenem

– **Skin and soft tissue** (prompt surgical consultation for debridement or incision and drainage).
  - Necrotizing fascitis including Founier's gangrene: mulitdrug therapy with: Flucloxacillin (2g IV 6 hourly) and Vancomycin (for severely ill patients loading dose 20-35mg/kg then maintenance dose 15-20 mg/kg every 8-12 hours) OR Piperacillin-Tazobactam 3.375 mg IV every 6-8 hours + clindamycin 600-900mg IV every 8 hours + ciprofloxacin 400mg IV every 12 hours (For those with penicillin allergy, Clindamycin 600mg IV every 8 hours + (vancomycin 15mg/kg IV every 12 hours or Linezolid 600mg oral or IV every 12 hours) + (aztreonam 1-2g IV every 6-8 hours or gentamicin 3-5mg/kg/day IV in 3 divided doses or ciprofloxacin 400mg IV every 12 hours), OR Cefriazone 1-2g IV every 24 hours and Metronidazole 7.5mg/kg IV/Oral every 6 hours for 7-10 days
  - Non-purulent cellutitis: Vancomycin for severely ill patients loading dose 20-35mg/kg then maintenance dose 15-20 mg/kg every 8-12 hours and Piperacillin-Tazobactam 3.375 or 4.5g IV every 6 hours
  - Abscess: Flucloxacillin 2g IV 6 hourly then orally 500 mg 6 hourly.

– **Genitourinary source**
  - Acute pyelonephritis: cefriazone 1g IV every 24 hours, OR Ciprofloxacin 400mg IV every 12 hours for 10-14 days

– **Steriod therapy in septic shock**
  - Hydrocortisone 200mg 8 hourly for 24–48 hours.

– **Vassopressor** (dobutamine 0.5-1mcg/kg/min IV continuous infusion initially, then 2-20mcg/kg/min; not to exceded dopamine and adrenaline) as and where indicated.

♦ Commence oral medication once the required course of IV antibiotics is completed. Choice of antibiotics depends on the source of infection and culture and sensitivity results.

- Commence resuscitation measures immediately the patient is seen.
- Refer from level 4 to levels 5 and 6 if complicated, especially if urinary output starts falling, serum urea, creatinine and potassium start rising, or if there is evidence of any other organ failure despite attention to adequate hydration with brisk electrolyte balancing, and antimicrobial administration.

***~ Always anticipate the onset of disseminated intravascular coagulopathy.***

## 1.4 Stings and Bites

### 1.4.1 BEE STING

Bee stings cause sharp pain followed by intense itching. Signs subside within a few hours. In hypersensitive individuals, anaphylaxis may occur (see Section 1.1, anaphylaxis). Some patients may experience delayed reactions usually after 0–14 days.

### 1.4.2 BITE BY A SUSPECTED RABID ANIMAL (RABIES)

Any mammalian may carry rabies. Saliva from a rabid animal contains large numbers of the rabies virus, which is inoculated through a bite or any laceration or break in the skin.

**Symptoms**

Incubation period is 1–2 months. Initial symptoms include malaise, fever, headache while local symptoms at site of bite include itching and paraesthesia. Full blown illness manifests with encephalitis, which may be demonstrated by agitation or dumbness. There is also hydrophobia, which is a characteristic manifestation of the form of the disease with agitation, while paralytic manifestation of rabies is often missed.

**Diagnosis**

Based on high index of suspicion accompanied by clear history of stray animal bite or other physical findings and documentation of hydrophobia.Demonstration of basal ganglial lesions on MRI scans and autopsy findings help confirm the diagnosis.

***Management***

***Immediate local care:***

- Irrigate thoroughly with copious amounts of saline solution.
- Cleanse with a soap solution.
- Debride the wound(s).
- Administer antibiotic.
- Administer tetanus toxoid.
- Infiltrate the wound with rabies immunoglobulin.

**Indication for anti-rabies vaccine:**

- Bites from wild animals.
- Bites from UNPROVOKED domestic animal.
- Bites from a sick looking domestic animal, whether immunized or not.
- Severe injury (multiple or deep puncture wounds) or any bites on the head, face, neck, hands, or fingers.
- Laboratory findings of Negri bodies in the brain of the involved animal.
- Persons at high risk of exposure.

**Immunization**

- Pre-exposure prophylaxis should be offered to persons at high risk of exposure such as laboratory staff working with rabies virus, animal handlers and wildlife officers.
  - Three full intramuscular doses of 1ml on days 0,7,and 28 should be given in the deltoid area.
  - Post exposure prophylaxis of previously vaccinated persons: local treatment should always be given. Post exposure prophylaxis should consist of 2 booster doses either intradermally or intramuscularly on days 0 and 3 if they have received vaccination within the last 3 years. Otherwise full course of rabies vaccine.
- Post exposure prophylaxis:
  - Passive immunization: Human rabies immunoglobulin is given as a dose of 20IU/kg of body weight infiltrated around the wound and 20IU/kg givenIM in gluteal region followed by a course of rabies vaccine.
  - Intradermal schedule:1dose(0.1ml) should be given at each of two sites, either the forearm or the upper arm, on days 0, 3, and 7 and one dose at one site on days 30 and 90.
  - Intramuscular schedule: 1 dose (1ml) should be administered on days 0,3, 7, 14 and 28. All IM injections should be given into the deltoid region or in small children into the anterolateral area of the thigh muscle.

## 1.5 Poisoning

Poisoning can be acute or chronic. This guideline basically indicates for acute poisoning which is common. Acute poisoning is often life threatening and should always be treated as an emergency even if the immediate threat to life does not appear real. Refer to Table 1.1 for treatment summary

**Clinical Monitoring**

- Blood pressure measurement
- Urine output (1–2ml/kg/hour) catheterize
- Nasogastric suction in abdominal conditions
- Blood glucose levels
- Hb or PCV daily and correct appropriately

Treat renal complications appropriately, and more importantly treat the cause of the hypovolaemia top pre-empt these complications. Remember to consult in this very dire emergency.

**Prevention**

Public education about farm or household chemicals known to cause accidental, para-suicidal, or suicidal poisoning.

**Table 1.2: Clinical features and treatment of common acute poisonings**

| Substance | Clinical features | Recommended action |
|---|---|---|
| ***1. Household agents and industrial chemicals*** | | |
| Kerosene (paraffin) | Nausea, vomiting, cough, pulmonary irritation, difficulty in breathing; headaches, loss of consciousness | • Remove contaminated clothing;wash exposed skin with water and soap. Activated charcoal. Maintain airways and respiratory support<br>• DO NOT INDUCE VOMITING or perform gastric lavage |
| Carbon monoxide, e.g., car exhaust, charcoal jiko | Headache, dizziness, confusion, slurred speech, convulsions, coma; symptoms vary with per-centage ofcarboxyhaemoglobin | • 100%oxygen Hyperbaricoxygen |
| Corrosives,e.g., acids,alkalis hydrogen peroxide | Excruciating pain in the mouth, the pharynx,epigastric area; dysphagia,vomiting,haema-temsis; later develops laryn-geal oedema and obstruc-tion, oesophageal perforation;long-term; Stenosis of oesphagus | • Liberal water or milk orally<br>• Analgesic injection to relieve pain<br>• DO NOT INDUCE VOMITING DO NOT PERFORM LAVAGE |
| Methanol | Intoxication, drowsiness, muscle weakness, blurred vision, photophobia, papilloedema blindness, coma, cerebral oedema, cardio-respiratory depression,seizures,DEATH | • IV sodiumbicarbonate<br>• 10%Ethanolin 5–10% dextrose as oral or IV infusion<br>• Loading dose 0.7g/kg over 1 hour. Maintain at 0.1–0.2g/kg/hour upto ethanol level of 100mg/dl |
| ***2. Pharmaceuticals*** | | |
| Paracetamol | Nausea, vomiting, altered mental status, abdominal pain, evidence of liver failure (elevated liver transaminases ALT/AST) | Gastric lavage within 1 hour<br>• Activated charcoal<br>• Antidote |
| Chloroquin | Convulsions, cardiac arrhythmia, cardiac arrest | |
| Digoxin | Arrhythmias, ventricular fibrillation, anorexia,nausea, vomiting,confusion, amblyopia | |
| Iron tablets,e.g., FeSO4,vitamins withiron | Vomiting, abdominal pain, pallor, cyanosis, diarrhoea, shock | |
| Opiates, narcotics (drugs | Drowsiness, pinpoint pupils, shallow respiration, spasticity, respiratory failure | |

- Antidotalth erapywith N-acetylcysteine for up to 72hours
- Gastriclavage
- IV diazepam forconvulsions
- Refer if in coma

- Discontinue drug,administerpotassium
- Treatarrthymiaswithlidocaine ***OR*** phenytoin
- Antidigoxin FABfragments

- Emesis
- Gastriclavage
- Desferrioxamine1 gIV 15/kg/hourmax 80mg in 24hours
-

- Do not giveemetics

- Gastriclavage
- Activatedcharcoal
- Naloxone5 µg/kg IV to awaken and improverespiration
- IV fluids to supportcirculation

**Table 1.2, *continued***

| | | |
|---|---|---|
| Isoniazid | CNS stimulation, seizures,coma | • Emesis, gastriclavage<br>• Diazepam<br>• Pyridoxine(1mg for 1mg ingested up to 200mg)<br>• Sodium bicarbonate for acidosis |
| Warfarin | Generalized bleeding, with intracranial haemorrhage being most serious | • VitaminK 10mg IV STAT+OD for 5 days<br>• Transfuse fresh blood |
| ***3. Pesticides*** | | |
| Organo-phosphates,e.g., | Headaches, weakness,vomiting, colicky abdominal pain,profuse | • Decontaminate (see above).<br>• Remove contaminated clothing; wash |
| diazinon, dimethoate | cold sweating, hypersalivation, muscular twitching, | exposed skin with water and soap.DO NOT INDUCEVOMITING |
| | fasciculations, diarrhoea, tenesmus, convulsions, dyspnoea with bronchoconstriction,meiosis, bilateral crepitations | • IV atropine 2–4mg STAT, repeat after 10–20 min until full atropinization (pulse 100–120, dilated pupils) and maintain on SC atropine 4–6 hours x 24–48hours.<br>• Pralidoxime (PAM) 1–2g (children 30mg/kg) STAT, repeat 4 hourly,12–24 hours depending on response |
| Rodenticides, e.g., zinc phosphide | Severe abdominal pain, nausea, vomiting and diarrhoea; strong garlic smell; severe respiratory distress; myocardial injury | Supportive:<br>• Maintain airways<br>• Assist ventilation<br>• Observe for pulmonary oedema |
| Rodenticide (anticoagulant based) | Generalized bleeding, with intracranial haemorrhage being most serious | • Vit. K 10mg IVSTAT<br>• Transfuse fresh blood |
| Acaricides, e.g., Amitraz | Weakness, difficulty breathing, convulsions, coma. | • Remove contaminated clothing; wash exposed skin with water and soap. DO NOT INDUCE VOMITING<br>• IV sodium bicarbonate |
| Herbicides, e.g., Paraquat | Oral/pharyngeal inflammation, later multi-organ failure within | • Lethal dose as low as10ml<br>• Gastric lavage with 50–100 g |
| | hours or days depending on dose. Later interstitial pulmonary oedema and fibrosis.Multi-organ failure or pulmonary oedema invariably leads to death! | activated charcoal 4 hourly until patient improves |
| Organochlorines, e.g., DDT,aldrin, dieldrin | Excitement, tremors, convulsions | • IV diazepam for convulsions |
| ***4. Others*** | | |
| Lead: e.g., lead salts, solder, toys, paints, and | | painted surfaces |

| | | |
|---|---|---|
| with respiratory failure due to convulsions | • Gastric lavage if within 1hour<br>• Survivors by eye on d 48 hours almost invariably recover | TA) with close monitoring for renal function DMSA |
| Thirst, abdominal pain, vomiting, diarrhoea, encephalopathy following ingestion of suspicious substance | • Eliminate source of poisoning Chelation with Dimercaprol (BAL) Inj 4mg/kg and combined with calcium sodium edetate (ED | |

**Table 1.2,*continued***

| | | |
|---|---|---|
| | | (oral succimer)Treatment over longperiods (months toyears) |
| Mercury | Acute: gastroenteritis,vomiting, nephritis, anuria, delayed GI motility<br>Chronic: gingivitis, mental disturbances, neurodeficits, pneumonitis | • Gastriclavage<br>• Activatedcharcoal<br>• Penicillamine<br>• Haemodialysis for renalfailure<br>• Look out for GITperforation<br>• Lungs: supportivecare |

# 2. AIDS and Sexually Transmitted Infections

## 2.1 HIV/AIDS

HIV infection is caused by one of two related retroviruses i.e. HIV-1 and HIV-2 both which result in a wide range of clinical manifestations. Transmission requires contact with body fluids containing infected cells or plasma. The virus progressively destroys the body's immune functions leading to opportunistic infections and tumours. It is these opportunistic infections and tumours that give the manifestations of this disease.

### 2.1.1 HIV/AIDS IN KENYA

The first case of AIDS in Kenya was recorded in 1984. Afterwards, HIV infection spread very rapidly in the country and the magnitude and impact of HIV/AIDS is a major public health and development challenge. By 2007, an estimated 1.2 million Kenyans were HIV infected, with the countrywide prevalence at 5.1%. Manifestations of HIV infection vary from asymptomatic carrier states to severe debilitating and fatal disorders related to defective cell-mediated immunity.

### 2.1.2 HIV TRANSMISSION AND PREVENTION

Transmission requires contact with body fluids containing infected cells or plasma. HIV is present in blood, semen, vaginal secretions, breast milk, saliva, CSF and wound exudates. HIV is not transmitted by casual contact or even by the close nonsexual body contact that occurs at work, at school, or at home. Refer to Table 2.1 for a summary of transmission and prevention modes for HIV infection.

**Table 2.1: Modes of transmission and preventive measures for HIV infection**

| Mode of transmission | Preventive measures |
|---|---|
| Sexual intercourse:vaginal intercourse (majority of cases),anal oral sex | Practice abstinence<br>Avoid risky sex practices like casual and or multiple partners<br>Use condoms<br>Treat STIs promptly and effectively (STIs increase risk of HIV transmission) |
| Mother to baby: Inutero,during childbirth,breastfeeding(30–40% infant transmission rate). | Advise counselling andtesting<br>Give ARV(nevirapine) to both mother and infant.<br>Ensure that all blood is screened before transfusion. Arrange autologous transfusion where possible |
| Contaminated instruments:<br>Needles, skin piercing instruments circumcision, tattooing, etc., are sterile. | Ensure that sterile needles are used at all times<br>Ensure that instruments for earpiercing,<br>For needle drug addicts, do not share needles |

## 2.1.3 CLINICAL MANIFESTATIONS

The clinical manifestations of HIV vary from asymptomatic carrier states to severe debilitating and fatal disorders related to defective cell-mediated immunity. AIDS (acquired immune deficiency syndrome) is the end stage of the spectrum of disease and is characterized by life threatening opportunistic infections and neoplasms. The manifestations of HIV infection are many and present in all disciplines of medicine. Some of these are:

### *SKIN*

Dermatological manifestations are probably the commonest.
The diseases may be infective (bacterial, fungal, viral), reactive (eczema, hypersensitivities), or neoplastic. The most common ones are:

- **Herpes zoster (shingles):** This presents as vesicles and bullae distributed along one or two adjacent dermatomes and the rash does not cross the body's midline. Of young adults with Herpes zoster, an estimated 80% are positive for HIV infection. Herpes zoster occurs very early in the course of HIV infection. This provides an opportunity to provide intensive counselling to those affected by this disease. Can occur in eldelrly persons without HIV infection. The lesions usually heal and may leave a scar. Post herpetic neuralgia is a common complication.

**Management:**
**Antiviral:** Acyclovir shortens the length and severity of the illness. Should be taken as soon as the rash appears. Dose: Oral Acyclovir 800mg five time per day for 7-10days. For immunocompromised patients IV Acyclovir 10mg/kg/8hourly (adjust dose if in renal failure).
**Topical therapies:** Topical Acyclovir 5% Cream (Not recommended), Lidocaine ointment/lotion. Capsaicin. Ointment. Calamine lotion NOT RECOMMENDED. Potassium permanganate diluted to colour of nails (1:10,000) then apply 0.5% GV paint.

**Pain management:**
- Mild to moderate pain: paracetamol 1gm TID OR NSAIDS like Ibuprofen 400mg TID) with or without Codeine.
- For Moderate to Severe pain: Morphine with or without Gabapentin 900mg STAT OR Pregabalin 150mg STAT, Amitriptyline 25mg TID

**Post-Herpetic Neuralgia:** Carbamazepine 100mg BD OR 200mg OD or amitriptyline 25-50mg nocte.

- **Seborrhoeic dermatitis:** This is an eczematous skin condition usually affecting the scalp, central face (especially the naso-labial fold and eyebrows) and flexures of the limbs. The affected areas are erythematous and have greasy scales. Treatment is by use of steroids and tar preparations OR topical salicylic acid, Topical antifungal creams/shampoo (Terbinafine). Refer to dermatologist for severe cases or if no response to therapy.

- **Molluscum contagiosum:** This presents as umbilicated papules, usually around the genitals. They exude a whitish material (molluscum bodies) when pressed or cut. **Management:** Cryotherapy OR curettage OR laser therapy OT topical therapy.
- **Pruritic Purpura Eruption (PPE)**: HIV-associated pruritis. Small less than one centimenter, symmetrical, multiple, discrete papules or papules, commonly found on the extremities of HIV+ patients with CD4 count less than 100 **Treatment:** oral antihistamine (Chlorpheniramine 4mg 8-hourly OR cetrizine 10mg OD); Topical Steroids OR UVB light, antipruritic lotions, or creams. Pentoxifyline 400-800mg twice-thrice a day.
- **Herpes simplex virus (HSV) ulcers.** Are fluid-filled painful sores that normally appear within 7-10 days. Commonly found on the lips, tongue, roof of the mouth, or the gums. Can recur. **Treatment:** use antiseptic soaps or saline baths and topical acyclovir cream or systemic acyclovir tabs,800mg PO 5 hourly for 10 days. Antibiotics for secondary bacterial infections.
- **Psoriasis:** A chronic, inflammatory multisystem disease Multiple inheritance disease. Characterized by well-circumscribed silvery scaling papules and plaques of varying sizes. Found on the knees, elbow, trunk, or scalp. Tends to go through cycles, flaring for a few weeks or months, then subsiding for a while or going into remission. Condition exacerbated by HIV infection, obesity, stress, sunlight, and drugs (systemic steroids, alcohol, chloroquine). **Treatment**: No cure. Therapies include steroids (topical (triamcinolone acetonide 0.025-0.1%

cream, betamethasone 0.025-0.1% cream), systemic IM Triamcinolone - Use 10

mg/mL concentration only; multiple sites may be injected, separate by 1 cm or more, May repeat at weekly or less frequent intervals as necessary or intralesional keratolytic agents. Refer to Dermatologist for specialist care according to guidelines).

- **Kaposi's sarcoma:** This is a neoplasm of the vascular-forming cells. It presents as bluish black nodules or plaques on the skin or mucous membranes. It may also involve the lymphatics and other organs including the GIT and lungs.

- Involvement of the hard palate Is a poor prognostic sign. **Management:** Chemotherapy is the mainstay of management of Kaposi's sarcoma. Drugs commonly used
  - For localized and flat lesions, use Vincristine 1.4mg/$m^2$monthly.
  - For more extensive lesions, give combinations of Vincristine 1.4mg/$m^2$, Bleomycin 10 IU/$m^2$, and doxorubicin (25mg/$m^2$) monthly. Paclitaxel is a 2nd line; give either singly or in combination with doxorubicin. Refer to oncologists.
  - In addition
    - Administer antiretroviral therapy (ART) at the same time.
    - Apply radiotherapy or local ablative therapies localised disease.
    - Refer to an oncologist for specialist care.

## *GASTROINTESTINAL TRACT*

**Candidiasis**

Candidiasis is caused by yeast or fungus. Candida albicans is the commonest agent. Usually a normal inhabitant of mucosal surfaces but overgrows with increasing immune deficiency.

**Presentation**: Appears as white, milk-like, removable plaques on the oral mucosa. Oral thrush is a white coating on hard or soft palate and tongue; causes dysphagia if oesophagus involved; occurs in late disease.

**Management**

- **For localised oral candidiasis**: Nystatin100,000 units 4 times daily after food for 7 days.
- **For oro-pharyngeal candidiasis:** use systemic antifungal like Fluconazole 200mg STAT then 100mg OD for 14-21 days). In patients with suspected cryptococcal meningitis, avoid the fluconazole until it is confirmed and administer full dose for cryptococcocal infection. This avoids development of resistance by cryptoccocus). Alternatives: Itraconazole solution 200mg PO x 14-21 days OR Amphotericin B IV (avoid in renal failure and monitor UECR).
- **Apthous ulcers:** Small (<1cm) well defined painful shallow ulcers with elevated margins. Usually self-limiting within 2 weeks). They may be deep >1cm and persistent). The cause of aphthous ulcers is unknown. The differential diagnoses include herpes simplex virus, CMV and drug induced ulceration. Biopsy should be carried out if an ulcer fails to heal

after about 4 weeks. Treatment: Antiseptic mouth washes, Local anaesthetic preparation prior to meals, Corticosteroid preparations in oral gel. Secondary infection of ulcers is common requiring metronidazole + penicillin OR co-amoxiclav. For refractory cases: Oral prednisolone (40 mg per day for 1-2 weeks before tapering). Dapsone 100 mg /day.

- **Chronic Diarrhoea:** lasting for more than 2 weeks with at least 3 loose bowel motions per day. It is considered by WHO as an AIDS-defining illness. Causes include viral (HIV, CMV, adenovirus, Rotavirus), Protozoa (Giardia lamblia, entamoeba histolytica, microsporidia Cryptosporidum parvum Isospora bellli), Bacteria (Shigella flexneri, Salmonella enteritidis, Campylobacter jejuni), Mycobacterium avium complex (MAC)), Fungal (Candida albicans, Histoplasma). Parasitic: Strongyloides stercoralis; Drug-induced diarrhea (Protease Inhibitors or antibiotics), Malignancies (lymphoma) Tests: Stool microscopy, Stool C/S, CD4 count, Modified ZN-stain if Cryprosporidum or Isospora belli is suspected. Faecal fat to rule out malabsorption. Others: TBC, UECR, LFTs (serum albumin). Colonoscopy (CMV, Kaposi's sarcoma and lymphoma). **Treatment:** supportive care (fluids and electrolytes), nutritional support, anti-diarrheal drugs for non-bloody diarrhea (Loperamide 4 mg stat then 2 mg after every loose stool motion (up to 16mg/day). HAART. Isospora belli (Co-trimoxazole 960mg BD for 10 days, Entamoeba histolytica (Metronidazole 500mg TID for 10 days), Giardia lamblia (Metronidazole 250mg TID), Cryptosporidium parvum (no specific treatment, start HAART, nutritional support, antidiarrhea medications e.g., Loperamide 4mg stat then 2mg with every loose motion).

## *RESPIRATORY SYSTEM*

- Cough of more than one month's duration, with or without shortness of breath caused by lower respiratory tract infection.
- Pulmonary tuberculosis (PTB) cases have increased since the advent of the HIV/AIDS epidemic. The risk of reactions to anti-TB therapy is higher in HIV-positive patients.Thiacetazone (inThiazina) is to be avoided (see 7.3.3,TB).
- **PneumoCystis pneumonia (PCP):** caused by pneumocystis jiroveci as a reactivation of latent infection when CD4 count is less than 200cells/ml**.** Universal septrin prophylaxis has reduced its incidence**.** It is an AIDS-defining illness. If untreated, it is fatal. Presents with gradual onset of symptoms; dry cough/scanty sputum, shortness of breath, fever and chest pain. Generally, patient is sicklooking, in respiratory distress, tachypneic, tachycardic, may be cyanosed in severe cases, dehydrated and febrile. Chest exam often normal or may have crackles/rales. Dignosis is mostly clinical. Pulse oximetry low oxygen saturation <90%. **Investigations:** HIV/CD4 count, TBC, LDH levels. No role for sputum cultures for PCP. Bronchoscopy with Broncho-Alveolar Lavage (BAL) is invasive and rarely done. Imaging: CXR: may be normal, OR with bilateral, symmmetrical, reticular or granular opacities in a ''butterfly pattern''). May have pneumothorax. If CXR is normal, A CT Scan Chest (HRCT) should be considered which may demonstrates non-specific ground-glass opacities. **Treatment:** If no sulphur allergy, give high-doseTrimethoprim-Sulphamethoxazole (TMP-SMX) (Co-trimoxazole) orally for mild-moderate cases or IV for severe cases where available or use NG-Tu Dose calculation:

TMP 15–20 mg/kg/day OR SMX 75–100 mg/kg/day for 21 days. Prophylaxis dose of Cotrimoxazole should follow the treatment (960mg per day) lifelong.

- Desensitization can be attempted in with prior mild sulphur allergy. AVOID DESENSITIZATION in those with severe drug allergic reaction like SJS.

Alternative regimens to those with sulphur allergy:

- IV Pentamidine 4mg/kg/day IV/IM.
- Clindamycin 600-900 mg 8 hourly IV OR 300-450 mg 6 hourly PO PLUS primaquine 15-30mg/day for 21 days.
- trimethoprim 15-20mg/kg/day PLUS dapsone 100mg
- atovaquone suspension dose

*Adjunctive corticosteroids in PCP:* Recommended for patients with moderate -severe PCP (PaCO2< 70 mm Hg OR an alveolar-arterial oxygen gradient > 35 mm Hg).

Prednisolone dose for PCP: 40mg BD for 5 days, 40mg OD for 5 days and 20mg OD for 11 days (a total of 21 days).

**Additional reference:** National Manual for management of HIV-Related Opportunistic Infections and Conditions.

## *NEUROLOGICAL SYSTEM*

Neurological complications are common in PLHA and are associated with significant morbidity and mortality. They range from opportunistic infections (Cryptococcal meningitis, TB-meningitis, Cerebral toxoplasmosis, cerebral abscess (bacterial/tuberculous), **;** progressive multifocal leucoencephalopathy (PML); bacterial and viral meningitis), neoplasms (CNS lymphoma) HIV infection per se (HIV-Associated Dementia (HAD) or AIDS-Dementia Complex (ADC), peripheral neuropathies and myopathies), complications of drugs including antiretroviral therapy. **Clinically Features** headache, confusion, fever, vomiting, convulsions, gait disorders, visual disturbances, reduced conscious level, focal neurological deficit, cranial nerve palsies, involuntary body movement. **Investigations:** TBC, RBS, malaria test, UECR, LFTS, urine/blood culture, VDRL, Toxoplasmosis IgG antibodies, serum CRAG, CD4 count, viral load, Lumbar puncture should be done if no focal neurological deficit exists. Assess CSF microscopy (AAFBS, India Ink, gram stain), Cultures/senstivity, CSF CRAG. Imaging: CXR, CT Scan brain or MRI brain.

**Treatment:**

**Non-specific/supportive treatment:** admit patient: For seizures; commence anticonvulsant, Fever: antipyretic (e.g., paracetamol 1gm 6-8-hourly), tepid sponging if fever >40$^{o}$C). If unconscious, offer care as per the standard guideline for comatose patient paying attention to Airway, Nutrition (NG tube feeding), Catheterization, physiotherapy, VTE-prophylaxis with heparin (UFH: 5000units TID or LMWH where available.

**Specific treatment:**
**TB-Meningitis:** initiate anti-TBs as per the national clinical guidelines. Add adjunctive steroids to reduce inflammation on the brain surface and reduces intracranial pressures. Dexamethasone IV followed by oral 6-8 weeks.
**Cryptococcal meningitis:** caused by a yeast-like fungus, Cryptococcus neoformans affecting severely immunosuppressed HIV patients with CD4 count less tha 100cells/ml. Rarely can occur in non-HIV patients. It is transmitted via inhalation of the fungus leading to colonizaion of the airways. Untreated disease is usually fatal. CSF manometry should be performed to measure the CSF pressure. **Treatment:** 3 phases: Induction for weeks with **Amphotericin B at 0.7-1mg/kg/day** COMBINED WITH **Fluconazole 1200mg/day.** The induction phase targets to achieve sterlization of the CSF.

This is followed by 6-8 weeks Consolidation phase with **Fluconazole 800mg od**. Maintance phase: with Fluconazole 200mg continued until the immune system has reconstituted with CD4 count above 100cells/ml. (NB: Amphotericin B is nephrotoxic and also causes hypokalemia, hypomagnesium and phlebitis. UECR should be monitored, and patient should be well hydrated before administration of amphoterin B. Potassium should be supplemented with potassium chloride (KCL) in case of hypokalemia. In case of renal toxicity or where amphoterin B is not available, the alternative therapy is Fluconozole monotherapy at 1200mg/day for 10-12 weeks for the induction and consolidation phases, then 200mg/day for the maintainance phase as above. Supportive care: as per the guideline of comatose patient. Those with worsening headache or deterioration of GCS level, reduce the intracranial pressures by performing therapeutic Lumbar puncture 10-20mls/day until CSF pressure is below 200cm of Water by manometry. There is NO ROLE for acetazolamide for reduction of intracranial pressures. CSF Shunting may be indicated when repeated Lumbar punctures are not tolerated and the pressures are still high. HAART should be initiated when patient has stabilized if patient was not already on the the ARVs.This is after about 8 weeks. In cases where patient was already on the HAART and develops Cryptococcal meningitis, this may be a sign of treatment failure and the regimen should be revised as the cause of treatment failure is identified and addressed.

**Cerebral toxoplasmosis:** Caused by a protozoa Toxoplasma gondii in whom cats are the definitive hosts. Transmitted to humans via ingestion of oocysts from the infected cat's faeces or ingestion of tissue cysts in under-cooked meat. In PLHA, the CNS Toxoplasmosis usually as a result of reactivation of old latent infection in those with CD4 < 100 cells/$mm^3$. The greatest risk of disease is in those with CD4 < 50 cells/mm 3 while clinical disease is rare in patients with CD4 count > 200 cells/mm3. Other transmission pathways include Congenital toxoplasmosis, materno-fetal transmission, organ transplant or blood transfusion. In immune-competent persons, acute toxoplasmosis is usually asymptomatic. The incidence of CNS-toxoplasmosis has reduced due to the widespread use of Cotrimoxazole prophylaxis and ART by HIV positive individuals in Kenya. HIV infected patient with CNS toxoplasmosis may also present with extra-cerebral manifestations including pneumonitis or chorioretinitis (white cotton wool spots on the retina), hepatosplenomegaly and

generalized lymphadenopathy. Investigations: Toxoplasmosis IgG antibodies, CT/MRI Scan Brain: multiple ring enhancing lesions.

**Preferred regimen**
Pyrimethamine 200 mg loading dose, then 50 mg –75 mg/day AND Sulfadiazine 1000 mg to 1500mg P.O. 6 hourly AND Folinic acid (leucovorin) 10-20mg/day PO for 6 weeks. NB: Folate is not a substitute for Folinic acid. The dose of Folinic acid can be increased to >50 mg/day toreduce Pyrimethamine-associated haematological toxicity.
**Alternative regimen:** This is the first choice locally as the above preferred regimen is not always available. Cotrimoxazole: 5mg/kg/day of Trimethoprim OR 25mg/kg of Cotrimoxazole BD PO/IV per day for 6 weeks then continue with Cotrimoxazole PO at 960mg per day. Adjuvant therapy with steroid if cerebral edema is present (Dexamethasone 4mg PO/IV 6-hourly.

**Progressive Multiple Leukoencephalopathy (PML)**
PML is a severe opportunistic infection of the CNS caused by reactivation of latent childhood infection by JC polyomavirus. The JC virus destroys oligodendrocytes leading to multifocal areas of demyelination limited to the white matter, which causes neurologic dysfunction.
**Clinical Features:** of insidious onset with relatively rapidly progressive cognitive dysfunction, dementia, convulsions, ataxic gait, cranial nerve palsies, focal neurological deficits, coma. Fever is absent. Fatal in the absence of effective HAART. **Diagnosis**: Mostly clinical with compatible CT scan result (multifocal lesions in the white matter with no mass effect or enhancement with contrast). **Treatment::** No specific treatment for PML. Start HAART as soon as possible.

**Peripheral Neuropathy in HIV**
Affects peripheral nerves. Can present as mononeuropathy or polyneuropathy. **Causes**: HIV infection and can occur at any stage of HIV infection. Drugs e.g. Nucleoside analogues (ddI, d4T), Dapsone, Metronidazole, Isoniazid,). Others include Diabetes mellitus, vitamin B12 deficiency. Toxins e.g. alcohol. **Clinical Presentation:** pain or a burning sensation, numbness, tingling sensation or parasthesia or weakness. More common in the lower limbs. Symptoms usually start distally and progress proximally. May be bilateral or unilateral. Diagnosis: **Examination:** sensory loss, loss of ankle reflexes. Muscle weakness is mild. **Investigations:** HIV test; TBC with PBF (macrocytosis); VDRL, RBS, B12 and folic acid**.** .
**Management:** Stop the offending agent if known e.g. stavudine/didanosine) and substitute with another agent like tenofovir. Treat confirmed underlying causes like DM, B12 & folate deficiency. If on Isoniazid (INH), treat with Pyridoxine 200mg PO OD till end of TB therapy. Pain management with amitriptiline 25-50mg nocte monotherapy OR in combination with carbamazepine 100-200mg OD OR Gabapentin 600-900mg/day.

### *OPTHALMIC*

HIV related infections or manifestations like cytomegalovirus retinitis, toxoplasmosis infections of the eye, Herpes zoster affecting the ophthalmic nerve, Kaposi's sarcoma involving the conjunctivae.

**General Features**

- Fever, constant or recurrent.
- Unexplained weight loss of >10% of body weight.
- Chronic malaise or fatigue.
- Enlarged lymph nodes at 2 or more extra-inguinal sites for more than 3 months.

**Investigations**

- Rapid tests: 2 parallel tests with 2 different kits. A third kit can be used as tie breaker. Alternatively use a double ELISA.
- Estimation of viral load: PCR test for viral load in plasma.

- Test for immunocompetence:CD4/CD8T-lymphocyte count and total lymphocyte count.
- Not specific to HIV/AIDS:These depend on the presentation of the individual case,e.g.:
  - Diarrhoea:Stool for ova and cysts and C&S, endoscopy,and biopsy
  - Cough:Chest x-ray,sputum for AFB(acid fast bacilli) microscopy,culture and sensitivity. KOH (potassium hydroxide solution)
  - Fever: Malaria parasites, blood cultures, septic screen.

**~ Investigations should be ordered as clinical features indicate, since most HIV related diseases are treatable.**

— Routine screening for HIV should be encouraged through VCTs and DCT/ PITC to help people know their serostatus. This is useful because ARV therapy is now widely available. It is also hoped that people who know that they are HIV infected will take care not to transmit the infection to others. Testing is also done in health care facilities if there is strong clinical indication for HIV infection or AIDS. These individuals should also be counselled and informed consent obtained before testing unless it is under emergency situations.

## 2.1.4 HIV TESTING AND PATIENT EDUCATION

- Pre-test and post-test counselling:

- HIV test should not be done without first counselling the patient, unless under emergencysituations. This approach makes it easier to communicate the results and the patient/client is better suited to cope with the news. The results should be held in confidence.
- Both positive and negative results must be communicated in person by a health care provider. Post test counselling should be done prior to disclosure of results. Everyone should know:
- How HIV is transmitted (see Table 2.1 above)
- How one can avoid getting infected (see Table 2.1)

- That HIV cannot be transmitted by shaking hands or touching people with AIDS; sneezing or coughing; eating food, drinking water or sharing utensils; from infected insect bites; from using contaminated toilets or latrines.

♦ HIV-negative patients/clients need to know:
  - That one can be in the window period (i.e.,time between infection with HIV and development of detectable antibodies).
  - That a negative result does not mean that he/she cannot acquire HIV if exposed.

♦ HIV-positive patients need to know the following
  - They can transmit the infection to their sexual partner(s),baby in utero(if the patient is pregnant):
  - Their health can deteriorate faster if they acquire other infections,including STIs.
  - Their health can deteriorate faster if they have some lifestyles like excessive intake of alcohol, smoking, poor nutrition, and multiple sexual partners.
  - Condoms,as generally used, are roughly 70–80% effective in preventing acquisition and transmission of HIV and other STIs. Proper education on condom use can increase the effectiveness of the condom to 90%.
  - Pregnancy hastens the progression of disease and up to 40% of babies born to HIV infected mothers will acquire the infection. Contraceptive advice should be given. IUCDs (Coil) are known to predispose to PIDs and hence are discouraged.

## 2.1.5 STAGING OF HIV/AIDS

The World Health Organization (WHO) has developed a guide to the progression of HIV and AIDS through the various stages of the disease, as summarized in Table 2.2.

**Table 2.2: WHO classification of HIV and AIDS clinical stages– Adults and adolescents**

***Clinical Stage I – Asymptomatic***
- Persistent generalized lymphadenopathy

***Clinical Stage II – Early (mild disease)***
- Weight loss <10% body weight
- Minor skin infections
- Herpes zoster
- Recurrent upper respiratory infections

***Clinical Stage III – Intermediate (moderate)***
ú Weight loss >10% body weight, chronic diarrhoea, fever, oral candida, TB, severe bacterial infections

***Clinical Stage IV – Late (severe disease)***
- HIV wasting syndrome, CMV, Pneumocystis carinii pneumonia, toxoplasmosis
- Kaposi's sarcoma, HIV encephalopathy

## 2.1.6 MANAGEMENT OF HIV/AIDS

**General Management**

- Recommend a well-balanced diet, good rest, and exercise.
- Discourage excessive alcohol drinking and smoking.
- Pay prompt attention to any health problem.
- Provide social support by counselling patients/clients,to enable them to cope with the condition. With the patient's consent, involve other family members and use their, or the community's,social support system.
- Home-based care stems from understanding among the patient, the family/relatives, and the health workers.

**Pharmacological Management**

The main aim of anti-retroviral therapy (ART) is to suppress the viral load, achieve reconstruction of the immune system and hence improve quality of life. Refer to Table 2.3 for a summary of Kenya's standardized regimes for anti- retroviral drugs (ARVs) for adults and adolescents.

**Table 2.3:ARV standardized regimes In Kenya (adults and adolescents)**

***1st line:*** TDF/3TC/DTG
For pregnant women and those likely to get pregnant give TDF + 3TC + DTG
***2nd line:*** Replace TDF with ABC if impaired renal function; Replace DTG with EFV if not able to tolerate DTG

**Principles of Treatment**

- Ensure patient compliance through counselling and followup.
- Use combination therapy of 3–4drugs.
- Nutritional support is an important component of management.
- Antiretroviral treatment: So far no drug or herb has been shown to eliminate the virus from the body. Some drugs have been shown to slow the multiplication of the virus and thus improve quality of life and delay the progression of the disease.
- These drugs include:
  - Nucleoside analogues (reverse transcriptase inhibitors), e.g.,zidovudine.
  - Non-nucleoside reverse transcriptase inhibitors, e.g.,nevirapine.
  - Protease inhibitors, e.g.,indinavir.
  - Fusion inhibitors.

**Those to Be Given ARV Therapy**

All individuals with confirmed HIV infection are eligible for ART irrespective of CD4 cell levels, WHO clinical stage, age, pregnancy or breastfeeding status, co-infection status, risk group or any other criteria.

Laboratory Monitoring of HIV Patients on Treatment

The following laboratory monitoring should be done for patients with HIV on pharmacological treatment:

- Haemogram
- Liver function tests
- Serum amylase

- Renal function tests
- Blood and urine sugar
- Lipid profile
- CD4 lymphocyte sestimation
- Viral load estimation

**When to Change Drugs**

ARV drugs being used to treat patients with HIV should be changed under the following circumstances:

- When there is treatment failure.
- In the presence of unacceptable drug toxicity
- When there is drug intolerance by the patient.
- When there is non-adherence to drug administration requirements.
- When there is suboptimal treatment regime.
- When there are opportunistic infections and other manifestations.

**Management of Opportunistic Infections**

- Opportunistic infections respond to conventional treatment although they may require a longer treatment period or a higher dose than is necessary for HIV negative patients. Although the management of the specific infections is covered in the relevant chapters, a few of the conditions are discussed briefly below:
  - Pneumonia: Most pneumonia is due to Streptococcal infections. Use crystalline penicillin(or ampicillin) or a combination of cotrimoxazole and gentamycinin unresponsive cases.
  - Diarrhoea: Correct dehydration. Specific therapy depends on the causative organism. Combination of cotrimoxazole and metronidazole is often helpful, or chloramphenicol may be combined with metronidazole in an alternative treatment.
  - Oropharyngeal candidiasis: Apply 1% gentian violet paint TDS or nystatin oral drops or cream,***OR*** miconazole oral gel BD or tabs ketoconazole 3–6mg/kg/day in 2 doses for 7 days, ***OR*** fluconazole 200mg STAT then 100mg OD for 2 weeks.
  - Boils/furuncles:Cloxacillin 500 mg QID for 14 days ***OR*** erythromycin 500mg QID for 14 days ***OR*** topic bactroban.
  - Cryptococcal meningitis: Amphotericin B,0.7–1mg/kgdaily ***OR*** fluconazole 400mg daily for 6–10 weeks then 200mg OD for life.
  - Pneumocystis carinii pneumonia (PCP): Tabs prednisone 60mg daily and taper off over 3 weeks in addition to cotrimoxazole(TMP/SMX)IV15mgTMP/kg/day IV 6 or 8 hourly for 21 days or double strength cotrimoxazole 2 tablets 8 hourly for 21 days in addition to oral dapsone 100mg OD daily for 21 days.
  - Toxoplasmosis: Pyrethamine 25–100mg PO OD + folic acid 10–20mg QID + either sulphadiazine1–1.5mg 8 hourly ***OR*** clindnamycin 600–1,200 mg 8 hourly ***OR*** azithromycin 1,200–1,500mg every 24 hours

**CTX is safe after second trimester and azithromycin is safe in pregnancy since it is similar to erythromycin.**

**The Role of Admission for Patients on Management of HIV**

- Admission is preferred under the following circumstances:
  - For investigations, if diagnosis is uncertain and if such investigations are not possible in an outpatient setting.
  - There are opportunistic infections that cannot be effectively treated in an outpatient setting.

- Admission is discouraged under the following circumstances:
  - For terminally ill patients: Whenever possible, home- and community-based care is preferred for such patients, as the hospital offers little benefit. Efforts should be made to support the family in caring for the terminally ill patient.

**Treatment in Tuberculosis Patients**

**~ Avoid ARVs in intensive phase: D4T+3TC and EFV (800 mg per day).**

— ***NB:Protease inhibitors are contraindicated when rifampicin is used.***



## 2.1.7 PREVENTION OF MOTHER TO CHILD TRANSMISSION

HIV can be passed from an infected mother to her baby before birth, during delivery and or while breastfeeding. Studies show that 23–42% of babies born in developing countries are infected with HIV. Prevention of the transmission can be reduced further by using ARVs. For further details, refer to Part II, Section 28.10.1,prevention of mother to child transmission of HIV/AIDS.

## 2.1.5 PREVENTION OF HIV TRANSMISSION IN HEALTH FACILITIES

HIV does not spread through casual contact, hence patients with HIV infection may be nursed in open wards. Eating utensils need not be handled in a special way. However, health workers who handle HIV-contaminated blood or certain body fluids are at risk.

**Precautions in the health facility include:**

- Wear gloves and take care in all situations involving direct exposure to blood and other body fluids, e.g., wound dressings, surgery and other invasive procedures, vaginal deliveries, collection of laboratory specimens, handling soiled bedding.
- Decontaminate surfaces that have been soiled by blood or other body fluids with sodium hypochlorite 0.25% (e.g.,Jik).
- Soak instruments in glutaraldehyde solution.
- Wash hands and other contaminated parts of the body with soap and water.
- Soak in bleach (e.g.,Jik),for 30 minutes, all soiled bed linen and clothing before general washing.

**—After accidental contaminated needle prick injury, the following needs to be done:**

***Immediate measures:***

- Skin:
  - Decontaminate skin by washing thoroughly with soap.
  - Squeeze wound and let blood flow freely.
  - Apply iodine, methylated spirit, betadine, or other virucidal agents.
- Eye:
  - Rinse ***thoroughly*** with sterile saline, eye irrigant, and clean water splash.
- Mouth/nose:
  - Clean water rinse, flush.
  - Use oral disinfectants.
- Post exposure care:
  - Allay anxiety.
  - Discuss safer sex/third party risks.
  - Advise/conduct HIV pre- and post-test counselling.
- Testing:
  - Conduct baseline HIV screening atinjury.
  - Repeat 6 weeks, 3 months, and 6months.
  - Give post HIV exposure prophylaxis (Table 2.4).

**Table 2.4: Post HIV exposure prophylaxis**

| |
|---|
| TDF+3TC+DTG (or TDF+3TC+ATV/r) for women and adolescent girls of childbearing potential for 28 days |

**— Most opportunistic infections in HIV/AIDS are treatable. Patients respond well and are able to resume work**

## 2.2 Sexually Transmitted Infections (STIs)

These are communicable diseases that are usually transmitted through sexual contact. Other forms of transmission include vertically from mother to child in utero, during birth, or soon after birth; transfusion of contaminated blood; or via contaminated needles, syringes, specula, gloves, and skin piercing and cutting instruments. Injecting drug addicts who share needles are a high risk group. Clinical manifestations of these conditions depend on the offending organism and are numerous.

**Accurate diagnosis and effective treatment of STIs is an essential and cost-effective HIV/AIDS prevention strategy.**

**Management**

- Give full course of appropriate drug therapy (see Table 2.5).
- Treatcomplications.
- Follow up thepatient.
- Provide health education andcounselling.
- Manage the sexual contacts, including contact tracing, diagnosis, treatment, health education, and counselling.
- Manage complications accordingly.

**Follow the 4C's of STI management.**

**Patient Education**

- Avoid multiple or anonymous partners, prostitutes, or any other personwith **multiple sex partners**
- Use condoms correctly, e.g., avoid oil-**based lubricants, use a new condom for every sex act.**
- Avoid alcohol or drug abuse, which may lead to irresponsible sexual behaviour.

**THE 4 C's OF STI MANAGEMENT**

Each and every treatment of STI must include the 4 C's:

- Compliance with the fulldrug course & follow-up
- Counselling on safer sexual behaviour
- Condoms: Ensure properuse
- Contact tracing, partner treatment and notification

## 2.2.1 SYNDROMIC MANAGEMENT OF STI

Many healthcare facilities in developing countries including Kenya lack the equipment and enough trained personnel required for etiological diagnosis of STIs (using laboratory tests to identify the causative agent). Where no laboratories or point-of-care diagnostic tests are available, a syndrome-based approach to the management of STI patients is used.

Syndromic management approach ensures accessible, affordable, and effective management of individuals with STIs relies on the utilization of flowcharts (algorithms) for each STI syndrome. The flowcharts enable diagnoses of common STI syndromes, provision of current country-specific appropriate treatment, and advice on the management of sexual partners. The flowcharts were prepared based on local etiological and antimicrobial susceptibility data and the WHO treatment recommendations where local data are not available.

In Kenya, the treatment guidelines were developed and adopted **in 1990** and validated in **1995** by the Ministry of Health in collaboration with stakeholders based on the data from local studies. The treatments cover the major pathogens responsible for the syndromes in the specific geographical areas. The treatment protocols are reviewed periodically for two reasons: to ensure that the antimicrobial choices are still valid, and to monitor any antimicrobial resistance. The last one was done in 2015 when the Rapid Advice was developed. If reviews are not done, the syndromic approach would lose its scientific basis.

The specific syndromes, causes and presentation are summarized in table 2.5.

**Table 2. 5: Syndromic management of STIs**

| Syndrome | Symptoms | Signs | Causes | Treatment |
|---|---|---|---|---|
| **Genital ulcer disease (GUD)** | -Genital sore(s)<br><br>-Pain or itching. and until the infection clears.<br><br>-Small red bumps or white blisters. may appear a few days to a few weeks after infection. | Genital ulcer(s), swelling and tenderness in the genital area | Chancroid<br>Genital herpes<br>Syphilis | **For Non-vesicular GUD**<br>**First Line Preferred** Treatment: Benzathine penicillin 2.4 MU IM weekly for 3 weeks AND Azithromycin 2 gm PO stat: 4Cs OR **Second Line Preferred**: Ceftriaxone 1 gm IM stat AND Doxycycline 100 mg PO BD for 14 days: 4Cs.<br>**NB: Doxycycline** is contraindicated in pregnancy or allergy to penicillin Azithromycin 2 gm PO stat should be given instead of Benzathine penicillin or Ceftriaxone.<br>**For Vesicular Ulcers First Line Preferred**: Azithromycin 2 gm PO stat AND Acyclovir 400 mg PO TDS for 10 days: 4Cs OR<br>**Second Line Preferred**: Acyclovir 400 mg PO TDS for 5 days or 800 mg PO TDS for 2 days if there is a history of recurrent HSV 2: 4Cs |

| Syndrome | Symptoms | Signs | Causes | Treatment |
|---|---|---|---|---|
| **Urethral discharge**<br><br>**(Urethritis)** | - Dysuria (pain during urination)<br>- Frequent urination<br>- Irritation in the distal urethra or meatus<br>- Urethral discharge | - Urethral discharge (if necessary, ask the patient to milk the urethra)<br>- Meatal erythema | Chlamydia Gonorrhea Trichomoniasis Herpes simplex virus, Mycoplasma genitalium, Ureaplasma urealyticum | **First Line Preferred:** Cefixime 400 mg PO stat AND Azithromycin 1 gm PO stat: & Tinidazole 2 gm stat plus 4 Cs OR<br>**Second Line Preferred:** Ceftriaxone 500 mg IM stat AND Azithromycin 2 gm PO stat: 4 Cs |
| **Vaginal discharge**<br><br>**(Cervicitis)** | | Abnormal vaginal discharge, Cervical erythema, Strawberry cervix | Candidiasis Chlamydia Gonorrhea Trichomoniasis | **First Line Preferred:** Cefixime 400 mg PO stat AND Azithromycin 1gm PO stat: & Tinidazile 2 gm stat plus 4Cs OR<br>**Second Line Preferred:** Ceftriaxone 500 mg IM stat AND Azithromycin 1gm PO stat; OR Gentamicin 240 mg IM stat AND Azithromycin 1gm PO stat: & Tinidazole 2 gm stat plus 4Cs<br>**In case the client is pregnant;**<br>**First Line Preferred:** Cefixime 400 mg PO stat AND Azithromycin 1gm PO stat: 4Cs OR<br>**Second Line Preferred:** Ceftriaxone 500 mg IM stat AND Azithromycin 1gm PO stat: 4Cs |
| **Genital Ulcer Disease/Syndrome**. | Genital sores, rashes with fluid, itching, swollen lympnodes | Ulcers (erosive or pustular), Vesicles Papules, Inguinal lymphadenopathy | Herpes simplex virus 1 or 2<br><br>Syphilis, Chancroid, Granuloma inguinale & LGV | Treatment depends on the specific syndrome:<br>-Doxycycline 100 mg, orally, twice a day for 14 days or Erythromycin 500 mg, 4 times a day for 14 days plus Benzathine penicillin 2.4 million units by intramuscular injection, once weekly for 3 consecutive weeks<br>For late Syphilis, Doxycline and Erythromycin are given for 30 days |
| **Scrotal swelling** | Unilateral testicular pain/swelling, with or without urethral discharge Fever | Swelling, lymphadenopathy, erythema and edema of the overlying skin | Gonorrhoea, Trichomoniasis, Pseudomonas & Coliforms infection | -Doxycycline 100 mg BD x 14 days, Ciprofloxacin 500mg stat & Trimethoprim 200mg BD x 14 days |
| **Pelvic Inflammatory Disease** | Vaginal discharge abnormal odour Vaginal/vulvar pruritus Vulvovaginal Vaginal/vulvar erythema Dysuria | Vaginal discharge offensive vaginal odour, Vaginal/vulvar pruritus, Vaginal/vulvar & erythema Dysuria | Gonorrhoea, Bacterial vaginitis, Vulvovaginal candidiasis and Trichomoniasis | **First Line Preferred:** Cefixime 400 mg PO stat AND Doxcycline 100 mg PO BD for 14 days AND Metronidazole 400 mg PO TDS for 14 days: 4Cs OR<br>**Second Line Preferred:** Ceftriaxone 500 mg IM stat AND Doxcycline 100 mg PO BD for 14 days AND Metronidazole 400 mg PO TDS for 14 days: 4Cs |

| Syndrome | Symptoms | Signs | Causes | Treatment |
|---|---|---|---|---|
| | | | | OR Gentamicin 240 mg IM stat AND Doxycycline 100 mg PO BD for 14 days AND Metronidazole 400 mg PO TDS for 14 days: 4Cs |
| **Anorectal Syndromes (Proctitis, . Proctocolitis & Anal/Genital Lesions/growths)** | Anorectal pain Anal or genital rashes, lesions and masses | Mucopurulent rectal discharge | Gonorrhea, (LGV and non-LGV serovars) | Treatment depends on the specific cause identified: eg- **If primary syphilis, secondary syphilisor non-reactive RPR test** within the past 2 years: Benzathine penicillin G 2.4 million units IM stat Or Ceftriaxone 1gm IM daily for 8-10 days in case of penicillin allergy |
| **Ophthalmia neonatorum in newborns** | Thick whitish/yellowish eye discharge, pain | Red, discharging, painful and sticky eyes | Gonorrhoea, Chlamydia | 1% Tetracycline eye ointment TDS x 5-7 days, and for prevention: Instill into both eyes; **1% Silver Nitrate drops or 1% Tetracycline Eye drops, or 1% Chloramphenicol eye drops** & treat the mother & her partner for cervicitis. |

## 2.2.2 GONORRHOEA AND URETHRAL DISCHARGE

**Clinical Features**

Many people with gonorrhea have no symptoms but some will often experience a burning sensation during urination.Males may also experience:

- White, green, or yellow discharge from the penis.
- Pain or swelling in the testicles.
- Inflammation or swelling of the foreskin.

Females may also experience:

- Abnormal vaginal discharge and bleeding between periods.
- Rectal symptoms may also occur if a person has engaged in anal sex.
- Discharge per vagina.
- Itching around the anus.
- Soreness of the genitals.
- Bleeding per vagina.
- Pain during bowel movements (dyschezia)

If gonorrhea results from oral sex, the person may have a throat infection, but they might not notice any symptoms. if discharge or vaginal fluid containing this bacteria gets into the eyes, the patient may develop conjunctivitis.

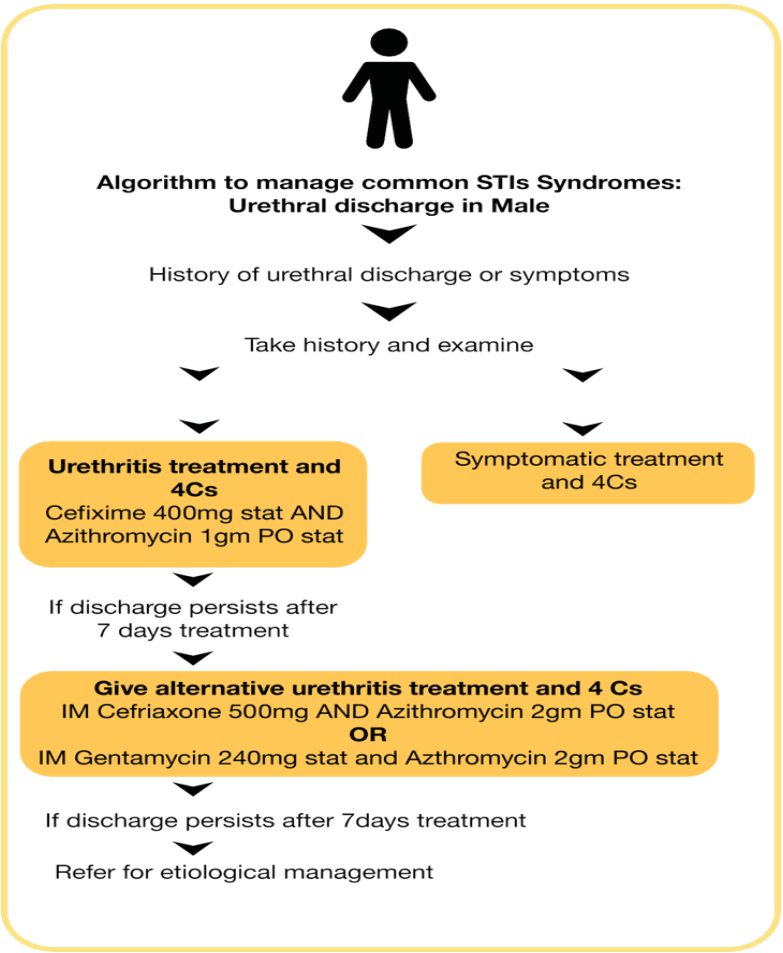
**Investigation**

- Diagnosis in males is usually clinical but if confirmation is required a urethral smear is done.

When attending to a patient who has been exposed to the infection from history and physical examination, the tests that ill help confirm the diagnosis include:

- Urine test – urinalysis and culture will help identify bacteria responsible
- Swab of discharge from affected area - eg throat, urethra, vagina or rectum for Gram stain and culture.
- Nucleic acid amplification test (NAAT) assay is the test of choice in well-established hospitals.

**Figure 2.1: Algorithm for the management of urethral discharge**




Source: Kenya National Guidelines for the Prevention and Control of Sexually Transmitted Infections, 2018

**Management**

- Anyone with gonorrhea needs prompt treatment to stop the infection from progressing. The treatment typically involves use of antibiotics that should be given as soon as possible
- The recommend drugs include; **a single dose of Ceftriaxone (Rocephin) 500mg Im and 1 gram of oral Azithromycin (Zithromax).** Other different antibiotics may be used in case of resistance or allergy to penicillins.
- However, Neisseria gonorrhoeae often develops resistance to nearly all the antibiotics that have traditionally been used to treat it.
- The patient should come for follow-up appointments and avoid sex until recommended treatment period specified by a healthcare provider.
- During pregnancy, it is essential to be more cautious as the infection can spread to the baby during delivery, so the newborn will usually need antibiotics as prophylaxis.
- Some newborns develop conjunctivitis, and the symptoms usually appear 2–4 days after birth and include red eyes, thick pus and swollen eyelid.

**Complications**

There are a number of severe complications of gonorrhea. For this reason, it is important to receive treatment as soon as possible.
In females, gonorrhea can lead to:

- PID
- Chronic pelvic pain
- Infertility
- Ectopic pregnancy, which is a medical emergency

Other complications in pregnancy and during delivery if not treated, includes an increased risk of preterm labor or stillbirth. Gonorrhea in the newborn, apart from eye infection, can lead to joint infection, loss of vision, or bacteremia, a life threatening blood infection.
In males, gonorrhea can lead to epididymitis, which can lead to loss of fertility. Untreated gonorrhea can lead to a disseminated gonococcal infection a life threatening condition. Some signs and symptoms include:

- dermatitis, which usually involves a rash or itchy, dry skin
- fever
- arthritis
- tendonitis

People with gonorrhea also have a higher risk of contracting or transmitting HIV. One reason is that either infection can cause open sores, which make it easier for viruses and bacteria to enter the body.

**Prevention**

Methods of preventing gonorrhea include:

- avoiding sexual activity if there is the possibility of infection
- using a barrier method of protection, such as condoms, during vaginal or anal intercourse
- using condoms or dental dams during oral intercourse

- only having sexual activity with a mutually monogamous partner who does not have the infection

## 2.2.3 GENITAL DISCHARGE IN THE FEMALE

Causes of vaginal discharge include Candida vulvovaginitis (monilia or thrush), trichomonas vaginitis, and bacterial vaginosis. Endocervical discharge can be caused by gonorrhoea, chlamydia trachomatis, and mycoplasma hominis. Refer to diagnostic chart in Figure 2.2.

### *CANDIDA VULVOVAGINITIS (MONILIA OR THRUSH)*

This is a common infection of the vulva and vagina caused by a fungus called Candida albicans. It is not always transmitted by sexual intercourse. Predisposing factors are diabetes mellitus, systemic antibiotics, pregnancy, hormonal oral or injectable contraceptives and decreased host immunity.

**Clinical Features**

Vaginal discharge is usually creamy and thick (curd like). Further, an associated itching, burning and soreness during micturition and sexual intercourse is felt. There is erythema, excoriation and fissures. Diagnosis is mainly clinical.

**Investigations**

Wet mount is prepared by putting a drop of the discharge on to a glass slide and adding a drop of saline or 10% potassium hydroxide (KOH) and covering with a cover slip. Examine under low-power microscope. Candida albicans is identified by pseudohyphae and spores.

**Management**

- Give clotrimazole pessaries 200mg OD for 3 days and clotrimazole cream.
- Give fluconazole 200mg STAT.
- Treat partner with fluconazole 200mg STAT and cotrimazole cream also.

**Prevention**

People who get recurrent infection should be given concurrent prophylactic treatment whenever broad-spectrum antibiotics are prescribed.

### *TRICHOMONAS VAGINITIS*

It is a common cause of vaginal discharge. Caused by Trichomonas vaginalis, a flagellated protozoan, and is mainly sexually transmitted

**Clinical Features**

Symptoms depend on the severity of the infection and include a frothy, greenish- yellow, foul-smelling discharge. Other features are vaginal soreness, dyspareunia and post-coital spotting. Infection usually involves the vulva, vagina, and cervix and may appear reddish and swollen. Diagnosis is mainly clinical.

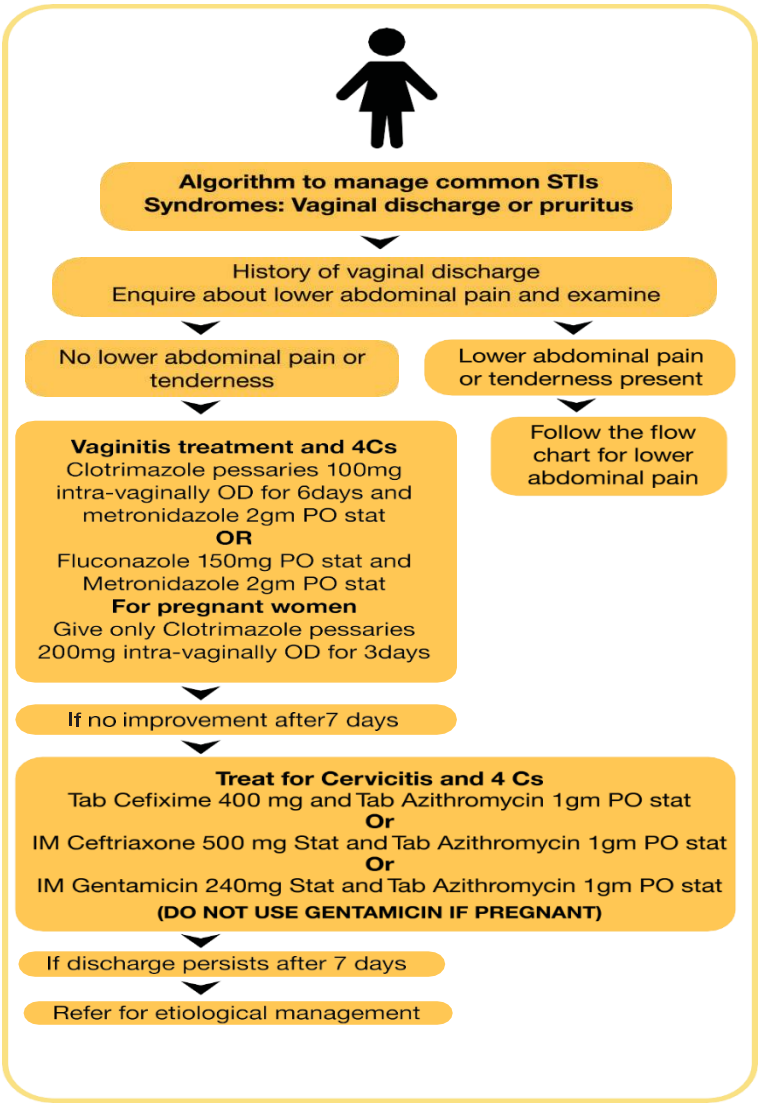
**Investigations**

- Wet mount preparation demonstrates flagellated protozoa.
- Trichomonas may also be noted on urine microscopy or papsmear.

**Management**

- Metronidazole 400mg TDS for 7days. The same dose for the male partner. (Alcohol consumption to be avoided during treatment with metronidazole.). Drug to be avoided during first trimester of pregnancy. If possible with hold treatment until third month of pregnancy.

**Figure 2.2: Algorithm for the management of vaginal discharge or pruritis**




Source: Kenya National Guideliens for the Prevention and Control of Sexually Transmitted iNfections, 2018

**BACTERIAL VAGINOSIS**

Bacterial vaginosis (BV) is an infection of the vagina resulting from a change in the normal balance of vaginal bacteria. BV is usually asymptomatic in many patients but it can lead to complications, especially in pregnancy. The common risk factors are; having more than one intimate partner or a new partner. The other contributing factors are; smoking, vaginal douche (Douching upsets the natural balance of bacteria. So can scented soaps, bubble baths, and vaginal deodorants). The causative organism is Gardeneralla vaginalis and lactobacillus species, Prevotella and Mobiluncus.

**Clinical Features**

Women with BV have no symptoms but a few may experience:

- Burning sensation during micturition
- Fishy odour after sex
- Itching
- Thin white, gray, or green discharge

**NB:** The discharge must be differentiated from a yeast infection which presents with a thick white discharge that is not foul smelling.

**Investigations**

- Wet mount preparation, which will show vaginal epithelial cells with adherent clusters of Gram-negative bacilli or coccobacilli (Clue Cells).
- Whiff-test in which a drop of discharge is mixed with a drop of potassium hydroxide,which gives a characteristic fishy odour.
- Measuring the vaginal pH or acidity level.

**Management**

- The mainstay is antibiotics **(Metronidazole 2gm stat, Clindamycin 300mg TDS x 5 days, or Tinidazole 2gm OD x 3 days)**.
- Topical treatment using a cream or gel to be inserted into the vagina can also be used. Most antibiotics should last for 5 to 7 days.
- Since BV can be spread through sex, do not engage in sex until complete taking the medicines. If your partner has another woman, they may want to see their doctor to find out if they need treatment.
- If a patient use an IUD and the infection recurs, the may change to a different type of family planning method.

**Complications**

Bacterial vaginosis has been been associated with a higher risk of other problems, including:

- Endometritis
- A premature or low birth weight baby
- Other STIs eg herpes genitalis, chlamydia, or gonorrhea, or HIV infection.
- Less success with fertility treatments like in vitro fertilization (IVF)
- Chronic PID - an infection of your uterus, fallopian tubes, and ovaries

**Prevention**

To lower your chances of getting BV, take these steps:

- Avoid douching.
- Limit your number of sex partners.
- Improved hygiene by wipe from front to back after visting the washrooms.
- Go for testing (even the partners)
- Plus 4Cs.

***CERVICITIS***

About one third of all women presenting with vaginal discharge have cervicitis. The commonest causes of endocervicitis are gonorrhoea, chlamydia, trichomonas, and herpes simplex virus.

**Clinical Features**

Cloudy-yellow vaginal discharge that is non-irritating, non-odorous, and mucoid. There may also be inter-menstrual or post-coital spotting or both. There may also be dyspareunia or pelvic discomfort or both. Cervical mucosa appears inflamed with focal haemorrhages. Cervix is friable and bleeds easily on touch. Vesicular herpetic lesions will be found on vulva, vagina, and cervix. Abdominal and bimanual pelvic examination should be done to rule out pelvic inflammatory disease (PID).

**Investigations**

- Wet mount preparation: Look for pus cells, trichomonas,and yeasts.
- Gram-stain of the discharge of endocervical swab (Neisseria gonorrhoea: shows Gram-negative intracellular diplococci).
- Culture for gonorrhoea or chlamydia if available.
- Pap smear after treatment.

**Management**

- See Figure 2.2, vaginal discharge flowchart.
- Norfloxacin 800mg STAT then 400mg BD for 7 days Ceftriaxone 250mg.
- Doxycycline 100mg BD.
- Metronidazole 2g STAT.

## TRICHOMONIASIS

Trichomoniasis is a sexually transmitted infection caused by a parasite called Trichomonas vaginalis (Tv) which is passed on through vaginal, oral, or anal sex. Trichomoniasis is highly curable if treatment is given promptly.
Without treatment, TV can lead to complications. It can affect a pregnancy, and it also appears to increase the risk of getting or transmitting HIV.

Trichomonas vaginalis can be transmitted during oral, anal, or vaginal sex or through genital touching. In females, is most likely to affect the lower genital tract. In males, it affects the urethra, the tube through which urine passes. Other parts of the body, such as the anus, hands, or mouth, cannot usually become infected.

**Risk factors**
The following are risk factors of trichomoniais:
- Female sex
- Having more than one sexual partner
- A previous history of other STIs
- Having unprotected sex

As the number of sexual partners that a person has increases, so does their risk of getting trichomoniais.

**Signs and symptoms:**
Symptoms may appear between 5 and 28 days after exposure, or they may appear later or not at all. When symptoms are present, they can affect males and females differently. Minor symptoms include irritation, but someone with a more severe case may have inflammation with discharge.
The usual symptoms in females include:
- frothy, foul-smelling vaginal discharge, which may be clear, white, gray, yellow, or green
- vaginal discharge with blood
- genital irritation
- discomfort during sex or when urinating
- swelling in the groin
- frequent urination
- lower abdominal pain

**Symptoms in males may include:**
- Urethral discharge from the penis
- itching in the genitals
- burning sensations after ejaculating or urinating
- frequent urination
- pain when urinating

**Complications**
Trichomoniasis can lead to several complications including:
- preterm delivery
- premature rupture of the membrane (PROM)
- low birth weight newborn
- infertility

A woman can sometimes pass on the infection to the newborn during delivery, but this is rare.

**Other complications:**

- Trichomoniasis may increase the risk of reproductive tract infections.
- Tv can increase the risk of getting HIV and other STIs, especially in females due to inflammation of the genito-urinary system, reduced immune response and hangs in the balance of vaginal flora, in females. These factors may lower a person's natural protection from the virus.

**Investigation**

To diagnose a trichomoniasis infection is confirmed through history taking and pelvic exam.Labortory tests include:

- Vaginal or penile discharge for microscopy and gram stain.
- Vaginal/urethral swab for a culture.
- Urinalysis and culture.

Regular follow-up appointments for 3 months are necessary especially for women.

Before any appointment, the subject should avoid using deodorant on the vulva, as this masks odor and can cause irritation. The doctor may also advise them to avoid vaginal intercourse or inserting any object, including tampons, into the vagina for 24–48 hours before hand A pap smear test does not check for trichonomiasis. If a person has a clear pap test, they may still have trichomoniasis or another STI.

Because Trichonoma vaginalis (TV) increases the risk of passing on HIV, people with HIV should also have a test at least once a year. If the result is positive prescribe treatment and discuss with the patient next action to be taken. Contact tracing should be done to all sex partners, as the also need testing and treatment

Information during follow up:

- Take the whole dose or course of treatment to stop the infection from coming back.
- Avoid sexual contact until the treatment is complete.
- Seek further advice if symptoms remain a few days after finishing a course of antibiotics.
- Also recommend having tests for other STIs.

**Treatment**

- This usually involves taking a single dose of an antibiotic by mouth. You may also prescribe a vaginal suppository or a cream to apply topically.
- The antibiotic medications recommended include; Metronidazole (Flagyl) 400mg TDS x 7 Days, and Tinidazole (Fasigyn) 2gm OD x 3 Days.
- Patients should avoid alcohol while taking metronidazole, as there may be an adverse reaction, which can lead to abdominal cramps, nausea, headaches, and flushing.
- If symptoms continue after taking the treatment, 2$^{nd}$ line drugs should be used after reevaluation.
- NB: Lactating mothers should not take Tinidazole while breastfeeding.

**Prevention**
To prevent infection or reinfection, any sexual partners should also be treated. The preventive measures include:
- ♦ limiting the number of sexual partners
- ♦ avoiding sex for 7–10 days after treatment
- ♦ limiting or avoiding the use of recreational drugs that can increase the risk of unsafe sex.

**CHLAMYDIA**
Chlamydia is a sexually transmitted infection caused by a bacterium called Chlamydia trachomatis. Usually chlamydia has no signs or symptoms and can lead to long-term complications if not diagnosed and treatment instituted early. Chlamydia can be passed on through genital contact. One can also get chlamydia if you come into contact with infected semen or vaginal fluid, or getting into the eyes. Chlamydia cannot be transmitted through kissing, hugging, sharing towels or using the same toilet as someone with the infection.



**Clinical features**
Many people with chlamydia do not experience any symptoms. If you do get symptoms, you may not notice them until several weeks after infection. Other people might not have any symptoms for several months.

The signs of chlamydia in women include:

- ♦ increase in vaginal discharge
- ♦ pain or burning when urinating (peeing)
- ♦ pain during sex and/or bleeding after sex
- ♦ pain in the lower stomach – especially when having sex
- ♦ bleeding between periods and/or heavier periods.

Signs of chlamydia in men include:

- ♦ white, cloudy or watery discharge from the penis
- ♦ pain or burning when urinating
- ♦ pain and/or swelling in the testicles.
- ♦ ycan also get chlamydia infection in your anus

**Investigations**
Tests for chlamydia include:
- ♦ Urine sample for analysis and culture
- ♦ Take wabs from the areas that are infected such as from the cervix or the vaginal wall in women, and the tip of the penis (urethra) for men.
- ♦ In case of anal or oral sex, you may have a swab taken from your anus or throat.

When there is history of having had unprotected sex, or isa patient is worried about STIs, test as soon as possible. However if the test is negative within two

weeks of having sex without protection, there is need to repeat the test later as the infection may not always be detectable in the early stages.

**Treatment**
We use Doxycycline 100mg BD x 14 days or Azithromycin 500mg OD x 3 to 6 days.
**Complications or long-term effects of untreated chlamydia**
If left untreated, chlamydia can lead to other, sometimes serious, health problems:

- **In women** untreated chlamydia causes pelvic inflammatory disease (PID). PID can cause pelvic pain, infertility (inability to get pregnant), and ectopic pregnancy (pregnancy outside the uterus) which can be life-threatening. PID can be treated with antibiotics.
- **In men** untreated chlamydia can cause swelling and pain in the testicles, and pain when urinating or during sex. Rarely, it can cause infertility in men.
- Chlamydia can also cause reactive arthritis in both women and men – inflammation of the joints, and in some people, the urethra and the eyes (conjunctivitis).

**Chlamydia and HIV**
If you have been diagnosed with chlamydia you should also test for HIV. Having chlamydia increases your risk of getting HIV, as it causes inflammation and sores that make it easier for HIV to enter the body.
In those living with HIV and not on treatment, having chlamydia can make them more likely to pass HIV particularly if they have sex without a condom. However, for those on treatment with HAART and have an undetectable viral load, they will not be able to pass HIV on – having chlamydia will not affect this.

If you're taking antiretrovirals, it's important to discuss with the doctor or health care provider how the chlamydia treatment may interact with HIV drugs.

**Prevention**
Using a new male or female condom or dental dam every time you have sex is the best way to protect against chlamydia.

Chlamydia can be passed on by sharing sex toys. Always cover sex toys with a new condom and wash them after use to reduce your risk of getting chlamydia and other STIs.

## *HEPATITIS B* INFECTION

It is caused by hepatitis B Virus (HBV) a DNA virus and is associated with significant morbidity and mortality. It is a common disease in Sub-Sahara Afria (SSA). In Kenya, the prevalence rate ranges between 8-30%.

**Risk factors for Hep B**

- Use injected drugs like diabetics or drug addicts.
- Sex workers and men who have sex with men (MSM).
- Frequent change and/or multiple sexual partners.
- Having close contact with someone who has chronic hepatitis B.
- Occupational exposure to the virus as with nurses, clinicians and laboratory staff.
- Haemodialysis

**Clinical Features**

Many people with hepatitis B do not have any symptoms. However, when present, symptoms may be noticed two or three months after infection, and they can last up to three months.

Therr are two typs of infections: acute and chronic

**Acute (or short-term) symptoms include:**

- flu-like symptoms, including tiredness, fever and aches and pains
- feeling and/or being sick
- loss of weight/appetite
- diarrhoea
- abdominal pain
- jaundice, meaning your skin and the whites of your eyes turn yellow
- dark urine
- pale faeces

Patients with acute infection after six months, such as babies, young children and people with a weakened immune system (eg in HIV co-infection) progress to develop chronic hepatitis B. This is when patients are at higher risk of liver failure, end satge liver disease, liver cirrhosis and cancer of the liver [hepatoma/hepatocellular carcinoma (HCC)].

**Investigations**

- Blood test for Hepatitis B-surface Antigen (HBsAg) will confirm the presence of the virus.
- Liver function Tests can help assess the extent of liver damage. The ALT can indicate if the disease ia active (ALT>2UNL).
- HIV test, VDRL, HCV.
- Abdominal/Liver ultrasound and Fibroscan (where available).
- Other tests: Hepatitis B e antigen test (HBeAg) and Hepatitis B DNA copies (to assess if the disease is active, Hep B-Core IgM/ or IgG, Anti-HBc IgG antibodies, HBsAg serum levels (quantification), Alpha-Fetal protein if HCC is suspected, Liver Biopsy for selected patients, coagulation profile.

It is also recommended for one to test for other STIs (HIV, syphilis) even for the recent sexual partner(s) so they can also get treated. Many cases of hepatitis B do not have any symptoms or signs but require to stop further transmission of the infection to others.

**Management**

**For Acute hepatitis B infection**

- There isn't any specific treatment for acute hepatitis B. However, most people recover within one to two months. Usually, patients manage symptoms at home with analgesics if necessary.
- Do regular follow up by blood tests and physical check-ups. Most people make a full recovery from acute hepatitis B.

Ninety percent (90%) of childhood infection (maternal-child transmission) are likely to be chronic infection. This calls for screening for all pregnant females for Hepatitis B infection. If found positive, the baby should receive Hep B immnunogloglobulins and Hep B vaccine to reduce their risk of getting infection. If the disease is acquired during adulthood, majority have an acute infection which resolves in 90% of the patients. 10% of patients infected in adulthood carries the risk of chronic hepatitis infection. The infection may remain latent and inactive, termed as carrier state. There is no evidence of liver inflammation (ALT normal, HBeAg negative, and Hep B DNA copies (less than 2000IU/ml) to assess the viral load.

HBV carriers do not require treatment but should be followed up as there is a 5-15% risk of reactivation and progression to chronic hepatitits B infection, liver cirrhosis or HCC.

Those with an active disease (Elevated ALT >2UNL, Elevated HBV DNA and HBeAg positive, may require treatment). They should be reviewed by medical specialists (physicians, paediatricians or where available gastroenterologist or liver specialists).

Before treatment is commenced, it is crucial to rule out HIV co-infection which is very common as the two diseases share the same transmission routes as well as being managed with shared antiviral drugs (Tenofovir, Lamuvidine and Emtricitabine).

**For Chronic hepatitis B infection**

**Goals of management: (i)** reduce risk of progression to chronic liver disease (CLD), (ii) prevent spreading of the disease in the population, (iii) prevent long-term complications (liver cirrhosis, end-stage liver disease), (iv)reduce mortality.

**Eligibility for treatment**

The following information is necessary to determine which patients qualifies for treatment:

- Alanine aminotransferase (ALT) level
- HBeAg status
- HBV DNA levels
- +/- Liver biopsy when indicated
- +/- HBsAg serum levels (quantification)

**Who to treat:**

- HBeAg positive with negative anti- HBe antibodies with HBV DNA copies >20,000 IU.ml)
- HBeAg negative with positive anti-HBe antibodies with HBV DNA copies >2000 IU/ml and ALT >2ULN.
- HBeAg positive and HBV DNA >20,000 and if:
    a. ALT more two-fold the upper limit of normal (>2ULN) (liver Biopsy not required) OR
    b. ALT 1-2 times the ULN (do Liver biopsy and treat if moderate-severe inflammation is reported.
    c. ALT normal in a patients aged >40 years
        i. Follow up with 3-6 monthly ALT. If 2 consecutive ALT levels are elevated >2 ULN the patient should be treated.
        ii. if they remain >1-2 ULN then do a liver biopsy. #NB: ALT should be done serially.
- Treatment should be given treatment to reduce the risk of permanent liver damage and liver cancer. Supportive and prophylactic treatment should continue for life.
- Without treatment, chronic hepatitis B can cause scarring of the liver (cirrhosis), which can cause the liver to stop working properly.
- A small number of people with cirrhosis develop liver cancer; a complication can lead to death.
- Liver transplant may be an option since there is no cure for cirrhosis.
- If HIV test is negative.

**Treatment of Chronic Hep B without HIV co-infection**
**First line therapy**

- Pegylated Interferon alpha-2a OR 2b for 48 weeks

(not indicated in those with decompensated liver failure, those on chemotherapy, immunocompromised patients, pregnant females, psychiatric/cardiovascular or thyroid diseases).
Pegylated interferon alpha 2a 180ug SC weekly.
Pegylated interferon alpha 2b 15ug/kg SC weekly.

Second-line therapy (if Peg-interferon is unavailable OR contraindicated:

- Tenofovir (a nucleotide analogue): 300mg PO daily possibly for life. Should be avoided in renal insufficiency.
- Entecavir (a nucleoside inhibitor): 0.5mg PO OD (dose adjusted in renal failure).

**Treatment of Chronic Hep B with HIV co-infection**

Use antivirals (NRTIs) targetting both HIV and HBV

- Tenofovir (TDF) PLUS Emtricitabine (FTC) and *NNRTI/Pi* OR
- Tenofovir (TDF) PLUS Lamivudine (3TC) and NNRTI/Pi

**Hepatitis B and HIV**

- Vaccines for hepatitis B are routinely offered to infants. Adults at a higher risk of getting hepatitis B may also be offered the vaccine.
- Hepatitis B does not always cause symptoms and can pass in a few months without treatment (acute infection). People can also have a lifelong infection (chronic), and without appropriate treatment and care, it can become more serious and lead to liver damage or death.

**Prevention**

It can be achieved by:

- Not sharing of needles and syringes or other items that may be contaminated with blood, such as razor blades, toothbrushes and manicure tools (even old or dried blood can contain the virus).
- Avoid tattoos, body piercings or acupuncture in professional settings, and ensure new, sterile needles are used. Vaccinate those who may be at an increased risk of infection.

## 2.2.4 DYSURIA IN THE FEMALE

It can result from urinary tract infection, vaginitis,or cervicitis. See relevant sections of manual for clinical features, investigations, and management. Gonorrhoea should be considered for patients at high risk for STIs.

## 2.2.5 LOWER ABDOMINAL PAIN IN THE FEMALE

**Clinical Features**

This is often due to pelvic inflammatory disease (PID – see Part IV, Section 57.7). Must be differentiated from urinary tract infection, ectopic pregnancy, threatened abortion, appendicitis, and other causes of acute abdomen

**~ An abdominal and pelvic examination must be done on all cases of lower abdominal pain in women.**

**Management**

- See Figure 2.3 ,Table 2.5 and relevant sections of manual.

## 2.2.6 GENITAL ULCER DISEASE

This condition can present with a variety of features and have a variety of probable causes, from primary syphilis chancre to Herpes to Granuloma inguinale. A thorough physical examination is required**.**

**Clinical Features**

Refer to Table 2.6 for a summary of the various presentations of this condition, along with the probable causes and diagnoses.

**Management**
See Table 2.5 above and Figure 2.2.

## 2.2.7 BUBOES OR SWOLLEN INGUINAL GLANDS

Buboes are enlarged lymph nodes in the groin. They may be associated with an ulcer in the genital area or on the lower limbs. Refer to genital ulcer disease.

**Clinical Features**

- Lymphogranuloma venerium: Several nodesmatted together on one or both sides, usually without suppuration.
- Chancroid tenderfluctuant bubo that suppurates, leaving an undermined inguinal ulcer that should be aspirated before suppuration.

**Investigations**
Serology for syphilis should always be performed.

## 2.2.8 GENITAL WARTS

**Clinical Features**

- Condylomaacuminatum (Humanpapillomavirus): Cauliflower-likewarts. May be single ormultipleon the vulva,vagina,perineal area,penis,urethra and sub-prepucial. Vaginal discharge, pain, and bleeding on coitus or touch may occur.
- Molluscum contagiosum (Pox group virus): Umbilicated multiple papules with whitish,cheesy material being expressed when squeezed. Secondary infection and spread to other sites may occur.

— *Secondary syphilis should be ruled out when evaluating genital venereal warts.*

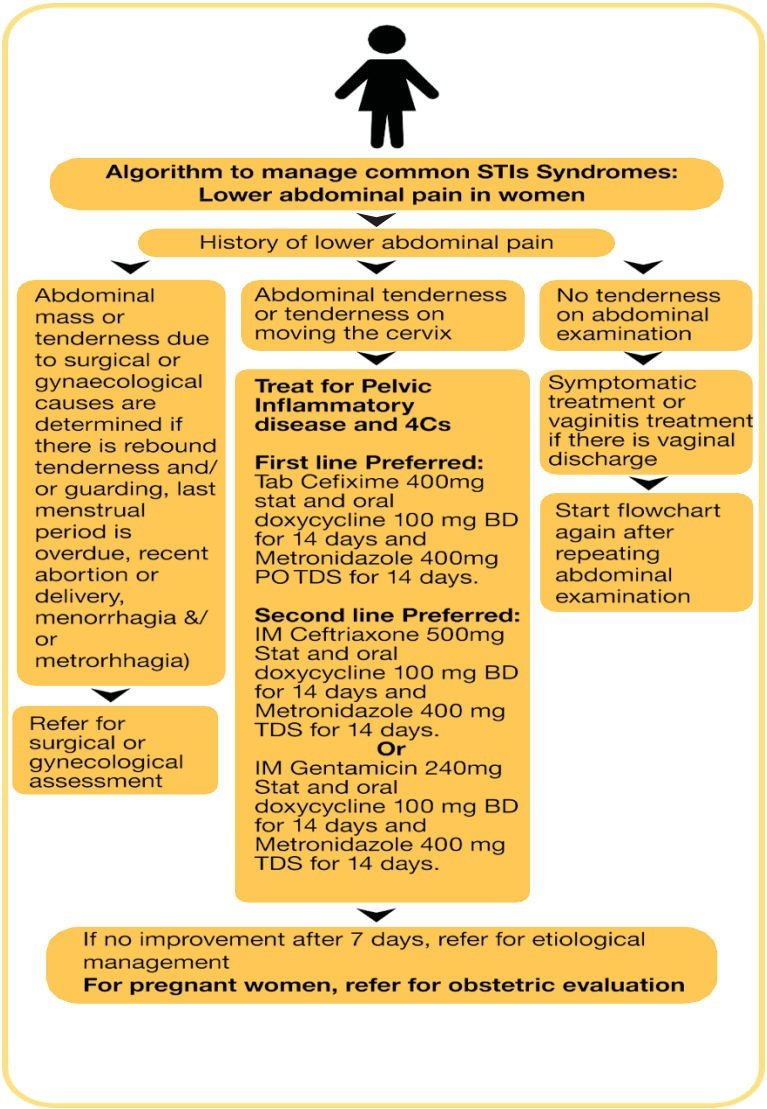
**Management**

- Carefully apply podophyllin25% intincture of benzoin to each wart, protecting the normal surrounding skin with petroleum jelly. Wash off the podophyllin thoroughly 1–4 hours later.
- Repeat 1–2 times weekly. If there is no regression after 4 applications,use alternative treatment given below or refer:
  - Alternative treatments:Podophyllotoxin 0.5% electrosurgery, cryotherapy,5-Fluorouracil, surgical removal, silver nitrate pencil application.
  - In pregnancy: Podophyllin should not be used during pregnancy, not in vaginal,cervical,internalurethral,anal,or oralwarts. Alternative regimens may be used, except 5-Fluorouracil and podophyllotoxin.

**Table 2.6:Genital ulcer disease clinical features and probable diagnosis & cause**

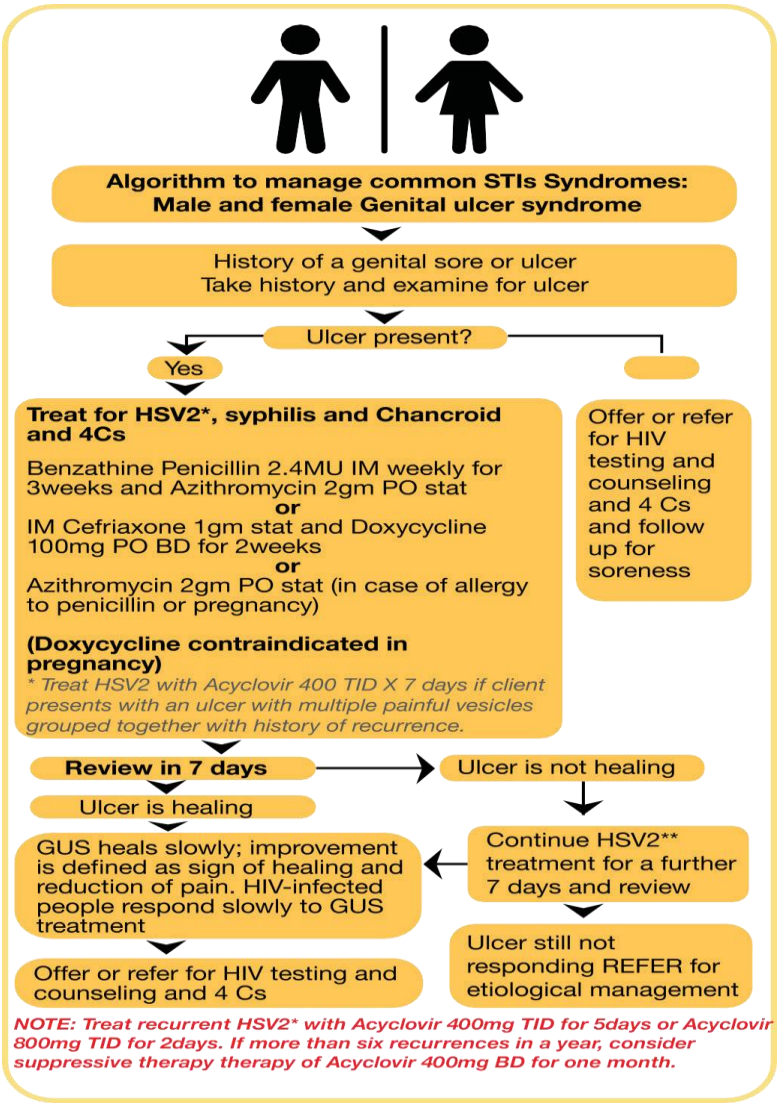
| Clinical features | Probable diagnosis & cause |
|---|---|
| · Single, painless, relatively clean ulcers without pus<br>· Incubation period up to3 weeks<br>· Painless lymphadenopathy | **Primary syphilis chancre**<br>T.pallidum |
| · Multiple, soft, deep, tender ulcers with profusepus<br>· Incubation period1 week<br>· Very painful lymphadenopathy, which can be fluctuant<br>· Disfiguration of thegenitalia<br>· Secondaryinfection | **Chancroid**<br>H.ducreyi |
| · Multiple shallow and tender ulcers<br>· May start as vesicles grouped together. Itchy<br>· Incubation period 1week<br>· Tender lymphadenopathy, may be recurrent, rarely suppurative | **Herpesgenitalis**<br>H. simplex |
| · Single, small and transientulcers<br>· Incubation period 1–2 weeks<br>· Lymphadenopathy; several glands may be matted together<br>· Fistula and stricture formation | **Lymphogranulomavenerium (LGV)**<br>C. trachomatis |
| · Large,beef ulcers<br>· Variable incubation period<br>· None or rarely lymphadenopathy | **Granulomainguinale**<br>Calymmatobacterium granulomatis (Donovanbacilli) |

**Figure 2.3: Algorithm for the management of lower abdominal pain in women**




Source: Kenya National Guideliens for the Prevention and Control of Sexually Transmitted iNfections, 2018

**Figure 2.4: Algorithm for the management of genital ulcer disease (GUD)**




Source: Kenya National Guideliens for the Prevention and Control of Sexually Transmitted iNfections, 2018

# 3. Cardiovascular Diseases

These are diseases and disorders of the heart and blood vessels. They include hypertension, coronary artery disease/ischaemic heart disease, rheumatic fever/rheumatic heart disease, infective endocarditis, congenital heart disease and deep venous thrombosis (DVT).

## 3.1 Venous Thromboembolism (VTE)

Comprises of DVT and PE

## 3.2 Deep Vein Thrombosis

The commonest site for DVT is the calf of the lower limbs followed by the pelvis. Commonly involves on limb (unilateral), can also be bilateral.

**Risk factors include** immobilisation, oral contraceptive pills, pregnancy, pueperium, rrecent surgery, trauma, fracture, active cancer, HIV infection, prior history of DVT, heart failure, nephrotic syndrome amongst others. The risk factors are modelled from the Virchow's Triad of vascular injury, stasis, and hypercoagulable state.

The possible risk of the DVT should be sought as it guides on the duration on therapy, prognosis and preventive measures.

The commonest site for DVT is the calf of the lower limbs followed by the pelvis (See also Section 58.16.2, DVT in pregnancy.)

**Clinical Features**
There is pain usually of sudden onset with warmth on palpation and local swelling with tenderness, and an extremity diameter of 2cm or greater than the opposite limb from some fixed point. In DVT related to pregnancy and its complications as risk factors, the left lower limb is involved in over 80% of cases. Diagnosis is mainly clinical. Use Well's score to assess the probability of DVT especially in resource limited set up.

**Investigations**
- Prothrombin time index (PTI)
- International Normalized Ratio (INR)
- Activated partial thromboplastin time (APTT)
- TBC (WBC, platelets)
- Urea, electrolytes and creatinine
- LFTs
- PDT
- Urinalysis
- Doppler ultrasound scan
- Confirmatory tests (venography)

- D-Dimers (done only when the probability of DVT is low as a rule out test). If the probability is high, D-DIMERS should not be done routinely.
- Doppler ultrasound scan (venous compression sonography is the imaging modality of choice. Failure to compress the vein is diagnostic of DVT).
- Alternative imaging: CT scan, MRI scan or Contrast venography. Reserved for imaging areas that are not accessible by doppler ultrasound (e.g., pelvic veins or subclavian veins).



**Management – General**

DVT can be managed as inpatient or outpatient.

Outpatient management can be considered in stable patients who are on either LMWH or Fondaparinux as the initial therapies for the first 5 days.

If patient is on UFH as the initial parenteral anticoagulant, admission is advised.

- Controlpain.
- Promote venous drainage:
  - Bed rest
  - Elevation of involved limb
  - Place the foot of the bed in a slightly elevated position (Trendelenburg's)
- Apply warm packs around involved limb.
- Encourage limited extension and flexion of involved limb.
- Encourage early ambulation as soon as pain and inflammation have begun to resolve.

**Management – Pharmacological**

**A. Parenteral anticoagulants**

**Unfractionated Heparin (UFH):** 80 IU/kg by bolus IV injection then 18 IU/kg/hour best by continuous intravenous infusion for 2–5 days. Adjust dose to achieve a PTT that is 1.5 to 2.0 times the control. **Antidote for heparin:** if patient is bleeding, stop heparin immediately and administer Protamine sulphate 1-1.5 mg per 100 USP units of heparin, (dose should not exceed 50 mg). Monitor APTT after 5-15 min then in 2-8 hr. Protamine sulphate is given as a slow IV infusion over 10 minutes.

- **Low molecular weight heparin** (LMWH): Does not require monitoring. Dose according to weight. Reduce dose if eGFR <30ml/min/1.73m2). The anticoagulant of choice in those with active cancer.
  - enoxaparin sodium 1mg/kg 12 hourly SC or
  - dalteparin 200 IU/kg /day SC
  - Tinzaparin: 175 Units/kg SC OD

Antidotes: Protamine sulphate adminstered at 1mg for every 100units of Dalteparin OR Tinzaparin. For Enoxaparin overdose, administer protamine sulphate at 1mg per each mg of enoxaparin and repeat after 4hours if patient is still bleeding at half the dose (0.5mg per mg of enoxaparin).

**Note:** Protamine sulphate is given as a slow IV infusion over 10 minutes.

- **Fondaparinux:** A synthetic pentasaccharide. Administered according to weight. Does not require monitoring.
  - Body Weight <50mg: 5mg SC OD for 5 days.
  - Body weight 50-100kg: 7.5mg SC OD for 5 days.
  - Body weight >100kg: 10mg SC OD for 5 days.

NO ANTIDOTE for Fondaparinux overdose. Stop the therapy incase of bleeding.

**B. Oral anti-coagulants**

- **Warfarin**: A vitamin-K antagonist (VKA). No dose adjustment is required in renal failure. Routine monitoring is required with INR. Avoid giving warfarin monotherapy in acute DVT (slow onset action upto 72 hours, and initially, warfarin is pro-coagulant with reduction of coagulation factors; protein C, S and anti-thrombin III).

  Warfarin therapy is started on the first day with 10mg OD for 2 days and subsequent doses are adjusted until the INR is 2 to 3 times the control for two consecutive days, then discontinue heparin. The required dose varies between 2mg and 15mg OD.
  - For calf vein thrombosis, warfarin is given for 6 weeks.
  - For proximal vein thrombosis, warfarin is given for 3–6 months.

  Warfarin interacts with many drugs like aspirin, NSAIDs, alcohol, erythromycin, metronidazole, sulfonamides, tetracyclines, omeprazole, rifampicin etc. All enhance warfarin's activity, therefore closely monitor the patient's INR. It also interacts with some foods. Dietary advise should be offered.

**Antidote for Warfarin Toxicity**

For any major bleed (e.g., ICH, GI bleeding),

- Recurrent thrombosi
- Stop warfarin. Consult a Physician.
- Give Fresh Frozen Plasma (FFP) (15mL/kg) and
- Vitamin K 5–10mg IV slowly.
- Where available, give prothrombin complex concentrate 50units/kg.
- Watch out for the following complications:
  - Recurrent thrombosis
  - Pulmonary embolism *(see under Pulmonary embolism below)*
  - If present, start Unfractionated heparin 6,000 IU as a STAT dose; aim at 24,000–30,000IU in the first 24 hours by continuous infusion. The rest will be guided by an APTT, which should be 2 times the control. Where available LMWH (dalteparin and enoxaparin). Dalteparin the dose is 80mg/day SC for those<60kg twice a day. For the elderly the dose should not exceed 40mg/day.

**Direct Oral Anticoagulants (DOACS)**

Commonly referred as novel/new anti-coagulants (NOACs). They are fixed doses with no need for monitoring.

They include:

- Factor Xa inhibitors:
  - Rivaroxaban 15mg BD for 21 days (3 weeks) followed by 20mg OD
  - Apixaban

- Edoxaban
- Antidote for Rivaroxab and Apixaban: Administer **Arizapine** Dose. This is a universal antidote which can be given also in Dabigatran overdose.

♦ Direct Thrombin inhibitors:
- Dabigatran: 150mg BD started after 5-10 days of heparin therapy
- Antidote: idarucizumab (a human monoclonal antibody fragment (Fab). In case of Dabigatran toxicity, stop the drug, give idarucizumab at 5gm IV (2 vials of 2.5/50mls) OR Arizapine (dose) (Are these antidotes available in Kenya?)

**Duration of Anti-coagulation**

♦ Those with reversible or transient causes OR first time unprovoked VTE anticoagulate for at least 3 months.
♦ Those with unreversible risk or recurrent unprovoked VTE should continue anticoagulation indefinitely and reviewed to review the risk vs benefit of long-term anticoagulation.



## 3.3 Pulmonary Embolism

Pulmonary embolism (PE) commonly occurs when a thrombus from distal of proximal veins travels to the pulmonary circulation occluding blood leading to pulmonary infarction. Rarely it may occur from the embolization of other materials into the pulmonary circulation such as air, fat, or tumor cells. It carries a significant morbidity and mortality if undiagnosed and not treated. Acute PE is associated with right ventricular (RV) dysfunction, leading to arrhythmia, haemodynamic collapse, shock and death. It can be acute or chronic, symptomatic, OR asymptomatic, provoked or unprovoked, small, medium or massive in size.

**Risk factors for PE:** similar to risk factors for DVT.

**Clinical Features:** variable from being asymptomatic to sudden death depending on the size ofthe thrombus. Most of the symptoms and signs of PE are nonspecific and include tachycardia, dyspnea, pleuritic chest pain, epigastric pain, hypoxemia, and shock (DDX: acute MI, Acute decompensated HF, or pneumonia).

**Diagnosis:** Assess the clinical probability of PE using validated criteri like the Well's score.
**Investigation:** In low probability: Do D-Dimers (if negative, this rules out PE as the cause of the symptoms. If probability is high OR critically ill patients, DO NOT do D-dimers. instead request for CT Pulmonary Angiogram (CTPA). Other tests: ECG: RBBB, RV strain pattern, RAD, S1Q3T3 pattern, CXR, BNP profile.

**Management: supportive:** Oxygen therapy, pain control. Definitive therapy: Anticoagulation as discussed above under DVT.

Other therapies:

- Thrombolytic therapy
- Thrombectomy
- Inferior venacavalfilters

**VTE Prophylaxis**

- Recommended whereVTE is likely to occur, e.g., hip operations and prolonged immobilization.
- **Pharmacological prophylaxis**
  - Heparin 5,000units/SC BD until the condition is treated.
  - LMWH: Enoxaparin 40mg/day SC can be given.
  - Fondaparinux 2.5mg SC OD

- **Mechanical prophylaxis**

Use of graduated compression stockings which provide a pressure of about 14-15mmhg at the calf. Advise the patients on the following

- The leg(s) should be measured to get the correct size foe stocking. If the size is too tight, may impede blood circulation, too loose, non-effective.
- The stocking preferably be worn daily both day and night and only removed for while bathing and inspecting the skin.
- The stocking use should be stopped in the presence of observation of blistering, skin discoloration or pain or discomfort.

## 3.4 Heart Failure

Heart failure occurs when the heart is unable to supply output that is sufficient for the metabolic needs of the tissues, in face of adequate venous return.

**Common causes of heart failure** are hypertension, valvular heart disease, ischaemic heart disease, anaemia, and pulmonary thromboembolism.

**Clinical Features:** Tachycardia, gallop rhythm, raised JVP, dependent oedema, tender hepatomegaly, orthopnoea, fatigue, exercise intolerance, and basal crepitations. Common precipitating factors of heart failure in cardiac patients must be considered in treatment of acutely ill patients: poor compliance with drug therapy; increased metabolic demands, e.g., pregnancy, anaemia; progression of underlying disease, e.g., recurrent myocardial infarction, uncontrolled hypertension; cardiac arrhythmias; pulmonary embolism; infective endocarditis; infection, e.g., pneumonia.

**Investigations**

**Laboratory tests:**

- Haemogram – To rule out anaemia, infection
- Urea and electrolytes, creatine (Na+ may be reduced due to dilutional hyponatremia, K may be low/normal/high), Creatinine and BUN may be elevation in cardiorenal failure.

- NTPro-BNP or BNP profiile (highly specific for HF)
- Urinalysis (proteinuria r/o renal causes e.g., nephrotic syndrome).
- Others: RBS/FBS/HBA1c, Lipid profile, LFTS, TSH or TFTs, troponin, PDT.

I**maging**

- ***Chest x-ray:*** May show cardiac enlargement as well as evidence of other cardiac or pulmonary lesions.
- Electrocardiogram (ECG): arrythmias, feautures of ischemic heart disease.
- Echocardiogram (to diagnose underlying heart condition, estmate the ejection fraction (EF).
- Others: Cardiac MRI or CT (requires specialist approval).

**Management – General**

- Restrict physical activities.
- Order bed rest in cardiac position.
- Give oxygen by mask for cyanosed patients.
- Restrict salt intake, control fluid intake, and measure urine output.
- Measurement weight daily.

**Management – Pharmacological**

## 3.4.1 ACUTE DECOMPENSATED HEART FAILURE (ADHF)

Admit hospital/HDU/CCU

Carries high mortality. Patient presents with acute respiratory distress due to cardiogenic pulmonary edema. Most patients have underlying chronic heart failure with certain factors precipitating an acute decompensation (e.g., non-adherence to drugs, infection, fluid overload, Acute MI, PE, arrythmias).

**Management:** ABCs (Airway assessment, supplemental oxygen (SPO2<90%) by use of non-rebreather face mask with high-flow oxygen at 100% (unless there is COPD). Oxygen should not be given if there is no evidence of hypoxemia as it may cause vasoconstriction and reduction of cardiac output, Mechanical ventilation where indicated. Establish IV access. Nurse in a propped-up position, Catheter insertion and flluid output assessment. VTE

prophylaxis therapy with LMWH/UFH. Assess the vital signs (BP: Hypotension or hypertension).

**Definitive management**

- Diuresis:
  - Vasodilator therapy:
    - Sodium Nitroprusside
    - IV nitroglycerine
    - Nesiritide:
  -
- **Inotropic therapy:** Enhance cardiac contractility and increase cardiac output. 3 types:
  - Beta adrenergic agonists: Dopamine, Dobutamine, Adrenaline/Epinephrine, Noradrenaline/Norepinephrine
  - Phosphodiesterase 3 inhibitor: Milrinon
  - Calcium Sensitizer: Levosimendan

Table 3.1 summarises the drugs used in acute decompensated heart failure.

**Note**: Beta blockers should be avoided in acute heart decompensated heart failure. They should be initiated once edema has subsided.

**Table 3.1: Drugs used in acute decompensated heart failure**

| Drug | Mechanism of Action | Dosages | Remarks |
|---|---|---|---|
| **I. LOOP DIURETICS:** | | | |
| 1. Furosemide<br><br>2. Torasemide<br><br>3. Bumetamide | Diuresis | 20-40mg IV BD/TDS<br>Max dose: 240mg/day<br><br>10-20mg IV BD<br>Max dose: 200mg/day<br><br>1-2mg IV BD<br>Max dose 8mg/day | ✓ Should be initiated promptly.<br>✓ Doses should be individualized.<br>✓ Those who have chronically been on loop diuretics may require initial higher doses (2.5times their usual daily doses).<br>✓ Intravenous administration is preferred.<br>✓ Avoid in severe hypotension and cardiogenic shock. |
| II. **VASODILATORS:** those with hypertension or normotensive. Avoid if hypotensive | | | |
| Sodium nitroprusside | Arterial vasodilator | 0.3-10mcg/kg/minute | Protect from sunlight. Taper dose before stopping to avoid rebound hypertension. Risk of cyanide toxicity |
| IV Nitroglycerine | Venodilator | 30-60mcg/minute | Mix 50mg with 250ml of D5W. |

| | | | |
|---|---|---|---|
| Nesiritide | A human recombinant Brain Natriuretic Peptide (BNP) analogue | 2mcg/kg IV bolus over one minute STAT followed by an IV infusion at 0.01mcg/kg/minute. | Used as second-line due to high cost. |
| **III.** | **INOTROPIC THERAPY:** increase cardiac contractility and increase cardiac output. | | |
| **i). Beta-adrenergic agonists:** *Dopamine, Dobutamine, Adrenaline/Epinephrine, Noradrenaline/Noradrenaline* | | | |
| Dopamine | Positive inotropic and chronotropic effects | 3-5mcg/kg/minute | Used short term |
| Dobutamine | Mainly inotropic effects | 5-10mcg/kg/minute | Dobutamine: a synthetic analogue of dopamine |
| Adrenaline/Epinephrine | Inotropic and vasoconstriction | 0.05-0.5mcg/kg/minute | Used in cardiac arrest<br>Avoid in cardiogenic shock |
| Noradrenaline/ Norepinephrine | | 0.01–0.03 µg/kg/min upto 1 µg/kg/min, | |
| **ii). Phosphodiesterase 3 inhibitor:** *Milrinone* | | | |
| Milrinone | Increase cAMP and Intracellular calcium.<br>Improves cardiac contractility (inotropy) and cardiac relaxation (lusitropy) | 25-50mcg/kg IV bolus over 10minutes, then 0.375-0.75mcg/kg/min IV OR<br>IV bolus: 25-75mcg/kg | For chronic HF under beta-blockade who present with AHF or cardiogenic shock<br>Adjust dose in renal failure<br>Avoid in AKI<br>Can cause tachyarrythmias, headache, hypotension |
| **iii). Calcium sensitizers:** *Levosimendan* | | | |
| Levosimendan | Phosphodiesterase 3 inhibition<br>Act on troponin C | 0.05-0.20mcg/kg/minute | Side effects: hypotension, tachyarrythmias, headaches |

## 3.4.2 MANAGEMENT OF CHRONIC HEART FAILURE

**Loop diuretics:** First line diuretic in fluid overload state: Frusemide 20–240mg/day IV OR PO use higher doses in patients who were already on it. Torasemide: 5-40mg/day.

**Thiazide OR Thiazide-like diuretics** may be added to the loop diuretics when necessary. Metolazone: 2.5-10mg/day PO.

**RAS-Inibitors (ACE-I** *OR* **ARBs)**. NB: The two should NOT be combined. They may cause hyperkalemia and angioedema. ACE-I may cause a dry cough.
Examples of CE-I: Enalapril, Captopril, Lisinopril, Ramipril, Perindopril, Trandolapril *(see the dosages under Hypertension* OR draw table
Examples of ARBs: Losartan, Telmisartan, Irbesartan, Olmesartan, Candesartan *(see doages under hypertension).*

**Cardioselective beta-Blockers.** Include: Bisoprolol, Metoprolol, nebivolol and carvedilol. Should NOT be initiated in acute decompensated HF with edema. Start once edema has subsided/resolved, start at a low dose and increase dose gradually to target doses. If patient was already on the beta-blocker, reduce dose then titrate upwards once edema resolves.

**Aldosterone Antagonists:** added to those with class III or IV HF. There are two drugs in this category: Spironolactone (25-50mg PO/day) OR Eplerenone 25-50mg/day PO. When ascites is present, higher doses are required for diuresis (see under Ascites treatment).

**Sodium Glucose-Linked Transporter (SGLT)-2 Inhibitors:** Approved for HF treatment for both HFrEF and HFpEF.with or without diabetes. Dapagliflozin 10mg OD PO, Empagliflozin 10-25mg/day PO.
**Angiotensin Receptos Neprilysin Inhibitors (ARNIs)**: Alternatives to RAS-Inhibitors. Indicated for chronic HF (Class II/III/IV) in patients who are able to tolerate RAS-I. Sacubitril PLUS Valsartan: Dose 24/26 (50mg); (49/51 (100mg) to 97/103 (200mg PO BD.

**Vasodilators:** Can be used as alternatives in that intolerant to RAS-I or as add-on therapies for those with advanced HF and uncontrolled hypertension. Indicated for black patients. Available in a fixed Dose Combination or given separately. Hydrallazine 25-175mg/day in 3 divided doses PO PLUS (Isosorbide dinitrate 20-90mg PO in 3 divided doses PO.

**Ivabradine:** A specific and selective inhibitor of cardiac pacemaker current ($I_f$). reduces heart rate in a dose dependent. Indicated in those with HFrEF (<35%), with a sinus rhythm and HR of ≥70 bpm. Dose 5-7.5mg BD PO.
**Digoxin:** A cardiac glycoside. Exerts a positive inotropic effect on the heart. Indicated in systolic HF (HFrEF) with Atrial Fibrillation. It reduces hospitalisation due to HF. Dose 0.0625mg-0.250mg OD PO. **Digoxin toxicity** is a life-threatening condition that occurs on high doses or normal doses in elderly patient with compromised renal function. Hypokalemia and concomitant use of some drugs

like quiniidine, amiadarone and verapamil may increase the risk of digoxin toxicity. Patients presents with nausea, vomiting, diarrhea, abdominal pains, visual disturbances, lethargy, confusion and arrythmias. Management of digoxin toxicity: stop the drug, give activated charcoal. Digoxin-specific antibody fragments may be indicated in severe cases (Consult a cardiologist).

### Other drugs in Heart Failure

**Anticoagulants** (E.g., Warfarin) in patients with HFrEF \9EF<40%) in the absence of contraindication.
**Statin therapy** and Antiplatelet therapy indicated if IHD is present.
IDentify and treat the underlying cause of HF and precipatiating factors accordingly.
**Non-pharmacological Therapy in HF**
**Cardiiac Resynchronisation Therapy (CRT)**. Indicated in those with refractory advanced HF (HFrEF EF <35%) with QRS >140ms not responding to medial therapy. Involves insertion of cardiac devices [(Biventricular pacing with or without implantable cardioverter debrillator (ICD)]. Consult the cardiologist.

## 3.5 Cor-Pulmonale:

Refers to right sided heart dysfuction as a result of chronic lung pathology (e.g., COPD, lung fibrosis, ILD).
Presents predominatly with feature of right sided HF (edema, elevated JVP, tender hepatomegally. Ascites may be present. Patent do not exhibit any orthopnea or PND. They are seen lying confortably lying flat in bed depite the edema.

**Investigations:** ECG: RV strain, **ECHO:** RV Hypertrophy, RA Enlargement, Tricuspid Regurgitation (TR) Pulmonary Hypertension,
**Management**: Loop diuretics (Furosemide OR Torasemide). Avoid RAS-Inhibitors. Anticoagulation may be indicated. Treat the underlying lung pathology.

## 3.6 Acute Myocardial Infarction (AMI)/Acute Coronary Syndrome (ACS)

AMI or ACS is caused by the complete or partial occlusion of a coronary artery resulting in myocardial ischemia and/or infarction. It requires prompt recognition and hospitalization for extensive care management. It comprises of three entities: (I) STEMI (ii) NSTEMI and (iii) Unstable angina.

**Clinical Features**
Chest pain: Severe, retrosternal/epigastric crushing or burning or discomfort. Discomfort radiates to neck and down the inner part of the left arm lasting at least 20 minutes to 7 hours. Occurs at rest and is associated with pallor, sweating, arrhythmias, pulmonary oedema, and hypotension. May also occur with physical activity, acute dyspnea/breathlessness and diaphoresis may occur with or without the chest pain (silent MI). others: nausea, vomiting, unexpalined fatigue. The patient

may present with cardiac arrest, arrythmias, cardiogenic shock or acute decompensated heart failure.

It is important to recognise the differential diagnsosis of ACS (Pulmonary embolism (PE), aortic dissection, esophageal rupture, pneumothorax, pneumonia, pericarditis, GI causes (gastritis/GERD) or musculoskeletal causes like costohondritis/cervical radiculopathy, herpes zooster infection).

**Diagnosis:** Timely diagnosis of ACS is of essence as 'time is muscle.' A 12-lead ECG should be done and interpreted within 10 minutes of presentation. It is may initially be normal and non-diagnostic. It should be repeated 10-15 minutes intervals within the first one hour. Look for ST-elevation (STEMI) or ST-depression or new T-wave inversion (NSTEMI), new LBBB, Q-waves. inferior STEMI, right-sided ECG leads should be obtained to screen for ST elevation suggestive of right ventricular (RV) infarction.

Biomarkers for cardiac necrosis; Highly sensitive Troponin I is the most sensitive and specific cardiac biomarker. Trononin T is an alternative cardiac biomarker. It the markers are within normal; a repeat test may be done after 4 hours. Other blood tests: TBC, lipid profile, UECR, or ABGs. Imaging: ECHO: Look for regional wall motion dyskinesia. CXR (preferably portable) to rule out respiratory causes,

**Management:**
Consult a multidisciplinary team physician/cardiologist/emergency medicine physician, critical care nurses)
Perform a focused physical examination

- Support and maintain vital functions (ABC)
- Give cardio-pulmonary resuscitation (CPR).
- Administer 100%oxygen when SPO2 <90%.
- IV fluids with normal saline (if hypotensive) with inferior MI or RV infarction. Caution should be exercised when giving fluids due to worsening of HF.
- Anti-ischemic therapy
    - Sublingual or IV or PO or Spray nitroglycerine 0.4mg every 5 minutes for 3 doses. (avoid nitrate if hypotensive SBP <90mmhg, bradycardia <50bpm, or tachycardic >100bpm) or when RV infarction is suspected. Concomittent use with PDE-5 inhibitors like sildenafil/tadalafil is contraindicated).
    - Cardioselective beta blockers. They reduce oxygen demad by reducing heart rate, cardiac contractility, and BP. They reduce the infarct size and early mortality in ACS. Caution is required. Bisoprolol 2.5-10mg PO OR Metoprolol 2.5-5mg IV every 5 minutes to a maximum of 15mg, then covert to oral metoprolol 25mg PO OD.
- Alleviate pain and anxiety:
    - Morphine is the analgesic of choice in ACS. May reduce absorption of antiplatelet.
        - 10–15mg 2.5-5mg (0.005/kg) IM ***OR*** IV increase by 2-8mg every 5-15 minutes until a maximum dose of 20mg. (10mg morphine must be diluted with normal saline or water for injection).

- Fentanyl: more potent than morphine. No effect on absorption of antiplatelet. So, a good alternative analgesic in ACS.
  25-100 mcg IV (1 mcg/kg) may repeat ½dose every 5 minutes until maximum of 3 mcg/kg

- Statin therapy at high dose (e.g., Atorvastatin 80mg or Rosuvastatin 40mg)
- AVOID the following drugs in ACS due to their deleterious effects:
  - NSAIDS
  - Steroids

**For STEMI:**

Admit patient in ICU or HDU for close monitoring

- Aspirin 300mg PO (chewable) STAT then 75mg PO OD.
- Clopidogrel 300mg PO STAT then 75mg PO OD OR Prasugrel 60mg OR Ticagrelor 180mg .

- LMWH (e.g., Enoxaparin 1mg/kg) SC STAT for one week. OR Fondaparinux 2.5mg SC.
- Cardioselective betablockers (IV) .
- ECG monitoring
- Oxygen therapy if saturations are less than 92%

**Reperfusion Strategies in STEMI**

- **Thrombolytic therapy** within 30 minutes of presentation ''Door-to-needle time''.
- Anti-thrombotic/fibrinlytic agents
  - Streptokinase: 1.5 million units' IV infusion over 30-60 minutes.
  - Alteplase (tPA): 15mg IV bolus OR 0.75mg/kg IV infusion over 30minutes (max 50mg) then 0.5mg/kg IV infusion for 60 minutes (upto 35mg).
  - Tenecteplase (TNK-tPA): 30-90mg IV single bolus 30-50mg depending on body weight.
  - Reteplase: 2c boluses of 10mg each 30 minutes apart.

**Contraindications for fibrinolysis** (have a check list)

- Prior intracerebral hemorrhage (ICH) or stroke of unknown origin.
- Ischemic stroke 6 months prior.
- Brain injury/ brain tumors or Arterio-venous malformations (AVM).
- Recent major trauma/surgery/head injury one month prior.
- Bleeding disorder.
- Recent GI bleeding.
- Severe hypertension (BP>180/110mmhg).

**Percutaneous Coronary Intervention**

Involves inserting a stent or stents in a stenosed coronary artery to enable reestablishment of coronary blood flow for myocardial reperfusion. There are two approaches to PCI:

- **Primary PCI (1º PCI)**: Emergent intervention given to patients who have not recieved prior thombolytic therapy due to contraindication, late presentation beyond the window period for fibrinolysis (>12 hours). ''Door-to-Balloon'' time.

- **PCI after fibrinolysis therapy** done After fibrinolysis therapy, the patient should be transferred to a center with PCI capability in a Cath Lab): Catheterization Laboratory. Done by an Interventional cardiologist.

**Emergent Coronary artery bypass graft surgery (CABG) in ACS**

Indicated for reperfusion where:

- PCI is not feasible due to unfavorable vessel anatomy
- Involvement of multiple vessels.

**FOR NSTEMI and Unstable angina**

The management is similar as STEMI except that REPERFUSION therapy with THROMBOLYSIS is NOT INDICATED.

**Management of Chronic stable IHD**

- **Dual antiplatelet therapy (DAT)** with junior aspirin (low dose aspirin) 75mg OD PO combined with Clopidogrel 75mg OD PO for one year. Clopidogrel is then stopped, and aspirin continued indefinitely. Clopidogrel can be used as an alternative antiplatelet in those with aspirin intolerance or contraindications.
- **Statins:** High dose statin (e.g., atorvastatin 80mg or rosuvastatin 40mm nocte should be continued. The duration is not yet well defined with some literature indicating upto 5 years. The dose can later be reduced and maintained indefinitely.
- Beta-blockers should be continued.
- Antianginal drugs: this can be added if angina persist despite beta blockade:
    - Long-acting nitrates: Isosorbide mononitrate 30-120mg/day.
    - Ranolazine 500mg-1000mg BD
    - Trimetazidine 35mg BD PO
    - Nicorandil
    - Non-Dihydropyridine CCBs
        - Verapmil 80-120mg 8-hourly
        - Diltiazem 180-360mg/day in 4 divided doses.
    - RAS-Inhibitors Post MI in those with left ventricular dysfuction/HF (ACE-I OR ARBs) seee section on HF.
    - Elective CABG may be performed in selected patients.

**Supportive care to prevent recurrent IHD**

- ALifestyle intervention: Smoking cessation, Graded exercise (expert advise is needed), alcohol moderation, stress management.
- Control comorbidities like hypertension and Diabetes (use therapies with proven CV benefit like SGLT-2 inhibitors or GLP-1 analogues).
- Regular patient follow up to ensure drug adherence.

## 3.7 Acute Rheumatic Fever

This is an acute, systemic connective tissue disease related to an immune reaction to untreated group A beta haemolytic streptococcus infection of the upper respiratory tract in children. The major complication of this disease is cardiac involvement, which can eventually lead to severe heart valve damage. This is the commonest cause of heart disease in Kenyan children. The initial attack of acute rheumatic fever occurs in most cases between the ages of 3 and 15 years.

**Clinical Features**

- Major criteria: Migrating polyarthritis, carditis(signs of cardiac failure, persistent tachycardia,pericardial rub,or heart murmurs), Sydenham's chorea, erythema marginatum, and subcutaneous nodules.
- Minor criteria: Past history of rheumatic fever, raised ESR, fever,arthralgia.
- Diagnosis: 2 major and 1 minor or 1 major and 2 minor manifestations.

**Investigations**

- Anti-streptolysin-0-titre (ASOT) – titre of 1:300
- Throat swab for B-haemolytic Streptococci group A for C&S
- ESR
- Chest x-ray – features of cardiomegaly
- Electrocardiography
- Echocardiography

**Management**

- Eradicate streptococcal infection from the throat:
  - Amoxicillin 250–500mg(children 25–50mg/kg in divided doses) TDS for 10 days
  - If allergic to penicillin or amoxicillin, erythromycin12.5mg/kg QDS for 10 days
- Control fever and inflammation: Aspirin: 75–100mg/kg/day in 4–6 divided doses.Treatment continued until fever and joint inflammation are controlled and then gradually reduced over a 2-week period.
- Treat failure if present (see Section 3.2, on heart failure).
- Treat chorea if present with haloperidol 25mcg/kg (0.025mg/kg)TDS.
- Admit for strict bed rest until symptoms resolve.

**Prevention**

- Avoid overcrowding.
- Early treatment of streptococcal sore throat with benzathine penicillin 1.2 megaunits STAT dose ***OR*** Phenoxymethylpenicillin125–250mg TDS for 10 days.

**Prophylaxis**

- If there has been previous acute rheumatic fever without carditis, give benzathine penicillin1.2 mega units monthly for 5 years or upto the age of 18 years, whichever is longer.***OR***
- Erythromycin 125–250mg BD for 5 years for those sensitive to penicillin.

- If there has been previous acute rheumatic fever with carditis give benzathine penicillin1.2 mega units ***OR*** Erythromycin125–250mg BD for those sensitive to penicillin for life.
- For patient education:
  - Emphasize need for follow up for prophylaxis.
  - Advise that rheumatic heart disease is a known complication.

## 3.8 Rheumatic Valvular Heart Disease

This is a complication of rheumatic fever. The main site of pathology is on the valves. There may be mitral stenosis, mixed mitral valve disease (both stenosis and incompetence), mitral incompetence, aortic stenosis and incompetence. Dyspnoea, palpitations, or heart murmurs may occur depending on the valvular lesion. Patients may be asymptomatic and may be discovered to have the lesion during routine examination or during periods of increased demand such as pregnancy or anaemia. Patients may also present with congestive cardiac failure.

**Investigations**
- Chest x-ray
- Electrocardiogram
- Echocardiogram
- Cardiac catheterization

Table 3.2 describes the various valvular lesions in RHD and their management.

**Table 3.2: Description of various valvular lesions in RHD and their management**

| Valve lesion | Description | Clinical presentation | Management |
|---|---|---|---|
| **Mitral stenosis**<br><br>*Mostly rheumatic. Presents 20-40 years after acute rheumatic fever. Can have non-rheumatic MS: valvular calcification with aging,* | Narrowing of the MV causing obstruction of blood flow from LA to LV.<br>The normal mitral valve (MV) area is 2-4cm$^2$<br><br>Mild MS: >1.5cm2,<br>Moderate MS: 1-1.5cm2,<br>Severe MS: <1cm2<br>May be isolated or combined with MR. | Depends of severity of stenosis.<br>Severe MS increases LA pressures and pulmonary hypertension.<br>**Symptoms:** Exertional dyspnea, fatigue, palpitations, hemoptysis, hoarseness of voice (compression of recurrent laryngeal nerve).<br>**Signs:** Afib, mitral facies, atrial fibrillation, loud first sound (S1), loud P2, opening snap, mid-diastolic murmur at the apical area (use the bell of the stethoscope as the murmur is of low intensity). | **Medical management:**<br>Infective Endocarditis prophylaxis in high-risk procedures (e.g., dental).<br>Rheumatic fever prophylaxis with monthly Benzathine penicillin *(see notes on RF prophylaxis*)<br>Anticoagulation to prevent stroke due to LA thrombus/ Afib.<br>Loop diuretics if congestion is present.<br>Beta blocker and/or NDHP-CCBs for tachyarrythmias.<br>Digoxin for Afib.<br>**Surgical management**<br>Percutaneous mitral balloon valvuloplasty (PMBV) OR<br>Mitral valve replacement (MVR) surgery |
| **Mitral Regurgitation** (MR)<br>*The most common Valve lesion.*<br>*Can also be functional where no valve lesion exists in the presence of cardiomegaly.* | Insufficiency of the MV causing backward flow of blood from the LV to the LA.<br>There is LV pressure overload leading to remodeling of the LV | **Symptoms:** Fatigue, exertional dyspnea. , orthopnea, PND, weight gain<br>**Signs:** wide-pulse pressure, displaced apex, Pansystolic murmur at the apex (radiating to the axilla), dependent edema, displaced apical impulse, and distended JVP. | **Medical management**<br>RAS-Inhibitors (ACE-I or ARBs).<br>Loop Diuretics<br>**Surgical management.**<br>Indications for surgery in MR: in the presence MV rupture, Infective Endocarditis, deterioration of LV function.<br>Involves MV repair or MV replacement |
| **Aortic Stenosis (AS)**<br><br>*Can be rheumatic, calcification with aging or congenital*<br>*Symptoms my begin 50-70 years.* | Narrowing of the aortic valve causing LV outflow obstruction.<br>Leading to increased pressure in the LV.<br>increased LV ejection time (LVET), decreased aortic | **Symptoms:** exertional dyspnea, syncope, angina, gradual decrease in exercise tolerance, symptoms of heart failure, GI bleeding in severe aortic stenosis due to angiodysplasia.<br>**Signs:** Slow rising pulse with late peaking (pulsus parvus et tardus), Palpable | **Medical therapy:** RAS-inhibitors (ACE-I or ARBs).<br><br>**Surgical management**<br>Balloon aortic valvuloplasty<br>Aortic Valve Replacement Surgery |

| Valve lesion | Description | Clinical presentation | Management |
|---|---|---|---|
| | pressure, and increased LV end-diastolic pressure. | carotid pulse, Single S2 sound, A mid-systolic ejection murmur, best heard over the right second intercostal space, with radiation into the right neck. | |
| **Aortic Regurgitation** (AR) | Insufficiency of the aortic valve due to incomplete closure of the aortic valve (AV) during diastole. This causes backward blood flow from the systemic circulation back into the L leading to LV volume overload and increased LV end-diastolic pressure. | **Symptoms:** dyspnea, orthopnea, PND (pulmonary congestion in advanced cases), angina, palpitations, head pounding. **Signs:** Wide pulse pressure systolic hypertension and decreased diastolic pressure (pulse pressure: Difference between the systolic and diastolic pressures), bounding pulses, apical heave (LVH) and systolic thrill, diastolic murmur best heard on the 3rd intercostal space on the left sternal border. **Others**: Austin Flint murmur, Corrigan sign (water-hammer pulse), de Musset sign, Duroziez sign, Becker sign, Quincke sign, Rosenbach sign, Traube sign (Pistol-shots, Mayne sign, Hill sign. | **Medical management** Stabilize patient in acute AR.<br><br>IV diuretics<br>IV sodium nitroprusside<br>inotropes:<br>Dopamine/dobutamine<br><br>NB: Avoid beta blockers<br><br>Chronic stable AR: No treatment required initially, follow up with ECHO 12-24 months.<br>Treat systemic hypertension preferably with RAS-I or NDP-CCBs.<br><br>**Surgical management**<br>Aortic valve replacement (AVR). |

**Management**

- Treat underlying complication, e.g., heart failure, pulmonary oedema.
- Continuous prophylaxis against recurrent rheumatic fever is indicated.
- Infective endocarditis prophylaxis is indicated.

**Prophylaxis**

- For rheumatic fever: All patients with a history of rheumatic fever should be given prophylaxis for recurrences, for life, with benzathine penicillin 1.2 mega units IM monthly,

***OR* amoxycyllin 125–250mg PO BD**
***OR* erythromycin 125–250mg PO BD**

- For infective endocarditis prophylaxis: In addition to rheumatic fever prophylaxis, the following should be done:

  - Before dental procedures patients should be given amoxicillin 3.0 g PO 2 hours before procedure and 1.5 g PO 6 hours after the initial dose.
  - If allergic to penicillin they should be given erythromycin 1g PO 2 hours before procedure, then half the dose 6 hours after the initial dose.
  - For lower gastrointestinal and genitourinary procedures patients should be given amoxicillin 2g IM 30 minutes before procedure and 6 hours after the initial dose + gentamicin 1.5mg/kg IM 30 minutes before procedure and 8 hours after the initial dose.

**Patient Education**

- Emphasize need for followup.
- Advise female patients on contraception.

**Complications**

- Congestive cardiac failure
- Pulmonary oedema
- Bacterial endocarditis

## 3.9 Hypertension

Hypertension is diagnosed when blood pressure (BP) reading is greater than 140/90 mmHg on 3 separate readings.

**Classification**

Primary Hypertension: Also known as essential hypertension. No obvious cause if found. Associated with certain risk factors like age, smoking, family history, lack of exercise. Comprises of majority of patients with hypertension. (85-90%).

**Secondary Hypertension**: Has an identifiable cause like kidney disease (chronic glomerulonephritis, PCKD), endocrine disorder (thyroid diease, Hyperaldosteronism, Cushing's Syndrome/disease, Phaechromocytoma, Acromegaly), Vascular disease (Renal Artery stenosis, Coarctation of aorta) or drugs (steroids, hormonla contraceptives) coccaine, (10-15%).

*Secondary Hypertension*: Has an identifiable cause like kidney disease, endocrine disorder or drugs.

**Clinical Features**

Majority of patients are asymptomatic. Occasionally patients may present with early morning occipital headaches, dizziness, or complication of hypertension, e.g., renal failure, stroke, and heart failure. Majority of patients have essential hypertension. Refer to Table 3.1 for classifications of hypertension.

**Table 3.3: Definition and classification of hypertension**

| Systolic (mmHg) | Diastolic (mmHg) | | |
|---|---|---|---|
| Optimal | <120 | and | <80 |
| Normal | 120-129 | and/or | 80-84 |
| High normal | 130-139 | and/or | 85-89 |
| Grade 1 hypertension | 140-159 | and/or | 90-99 |
| Grade 2 hypertension | 160-179 | and/or | 100-109 |
| Isolated systolic hypertension | >140 | and | <90 |

**Adapted from the ESH/ISH guidelines**
**NB: The class is determined by whichever the readings are highes**

**Investigations**

**Table 3.4: Essential package of investigations**

| Investigation | Rationale |
|---|---|
| Urianlysis | Evidence of kidney disease or diabetes |
| Blood glucose | Diagnosis of diabetes |
| Full blood count | Anaemia may indicate chronic kidney disease |
| Creatinine, electrolytes | Diagnosis of renal disease as a cuase of hypertension or complication<br>Electrolytes imbalance may suggest renal or hormonal anomaly |
| Lipid profile | Dyslipidaemis is a cardiovascular disease risk factor |
| Electrocardiography (ECG) | Identify cardiac anomalies such as enlargement, infarction, ventricular dysfunction, LVH etc |

Note: additional tests may be requested as needed as the health care team find necessary (e.g., TFTs and other tests for endocrine causes).

**Management**

Goal of therapy: Control Blood pressure to a target of < 140/90mmhg in order to reduce cardiovascular risk. This can be achieved by two ways;

- Lifestyle modification (no use of drugs): These include; healthy diets, avoidance of tobacco and alcohol use, adequate exercise, weight reduction. This advise should be emphasized in every clinic visit. If patient has stage 1 hypertension, drug therapy may be delayed for about 3months as the lifestyle intervention is being promoted and adhered to. This should be maintained even in other stages of hypertension. If BP is controlled to less 140/90mmhg, drug therapy is not required. Patient to be followed up regularly and encouraged to have out-of office BP measurements. Otherwise, drug therapy should be initiated.
- Pharmacological therapy: This involves use of various antihypertensive drugs as monotherapy or in combination as an add on the above lifestyle modification.

There are six major classes of antihypertensive agents:

**A:** Angiotensin Converting Enzyme Inhibitors (ACEIs) and Angiotensin receptor blockers (ARBs);

**B:** β-blockers (BBs);

**C:** Calcium Channel Blockers (CCBs);
**D:** Thiazide or thiazide-like diuretics;
**Z:** Others (sympatholytics, α adrenergic blockers, centrally acting drugs, alpha-2 agonists and direct arterial vasodilators).

For those with stage 1 hypertension, monotherapy is adequate to control, whereas grade 2 and above require combination therapy.
All the above drugs are effective in lowering the blood pressure. However, when choosing the best anti-hypertensive drug to start with, there are certain factors to consider;

*Availability, cost, side effects, comorbidities, complications, age and race.*

The tables below summarizes the compelling indications when choosing an anti-hypertensive drug.

**Table 3.5: Compelling indicators when choosing an antihypertensive**

| **Compelling indications (major improvement in outcome independent of blood pressure** | |
|---|---|
| Heart failure with reduced EF | RAS- inhibitor (ACE-I or ARB), beta blocker, diuretic, aldosterone antagonist* |
| Postmyocardial infarction | RAS- inhibitor (ACE-I or ARB), beta blocker, aldosterone antagonist |
| Proteinuric chronic kidney disease | RAS- inhibitor (ACE-I or ARB) |
| Angina pectoris | Beta blocker, calcium channel blocker |
| Atrial fibrillation rate control | Beta blocker, nondihydropyridine CCBs |
| Atrial flutter rate control | Beta blocker, nondihydropyridine CCBs |

**Table 3.6: Conditions in which use of certain anti-hypertensives would be preffered**

| Likely to have a favourable effect on symptoms in comorbid conditions | |
|---|---|
| Beningn prostatic hyperplasia | Alpha blocker |
| Essential tremor | Beta blocker (noncardioselective e.g., propranolol) |
| Hyperthyroidism | Beta blocker, |
| Migraine | Beta blocker, CCBs |
| Osteoporosis | Thiazide diuretic |
| Raynaund phenomenon | Dihydropyridine CCBs |

**Table 3.7: Drugs used in the treatment of hypertension and their possible side effects**

| CLASS | EXAMPLES | USUAL MONOTHERAPY | MAXIMUM DAILY DOSE | POSSIBLE SIDE EFFECTS |
|---|---|---|---|---|
| ***Long-acting CCB*** | Amlodipine | 5mg OD | 10mg OD | Oedema<br>Fatigue<br>Headache<br>Palpitations |
| | Felodipine | 5mg OD | 10mg OD | |
| | Nifedipine | Retard tabs: 10-20mg OD<br>LA tabs: 30mg OD | Retard tabs: 30mg BD<br>LA tabs: 90 mg OD | |
| | Cilnidipine | 5mg OD | 20mg OD | |
| ***Thiazide diuretics*** | Chlorthalidone | 25mg OD | 50mgOD | Hypokalaemia<br>Hyponatremia<br>Hyperuricemia<br>Hypocalciuria<br>Hyperglycemia<br>Rash<br>Dyslipidaemia |
| | Hydrochlorothiazide (HCTZ)<br>Bendroflumethiazide | 12.5mg OD | 25mg OD | |
| ***Thiazide-like diuretics*** | Indapamide | 2.5mg OD | 5mg OD | |
| | Metolazone | 2.5mg | 10mg | |
| ***ACE-Inhibitor*** | Captopril | 25-50mg BD or TDS | *50mg TDS* | Cough<br>Hyperkalaemia<br>Increased serum creatinine<br>Angioedema |
| | Enalapril | 5-20mg daily in 1 or 2 divided doses | *20mg daily in 1 or 2 divided doses* | |
| | Lisinopril | 10mg OD | *40mg OD* | |
| | Perindopril | 4mg or 5mg OD | *8mg OD or 10mg OD* | |
| | Ramipril<br>Trandolapril<br>Zofenop | 2.5mg OD | *10mg OD* | |
| ***Beta-blockers*** | Bisoprolol | 2.5mg OD | 20mg OD | Bradycardia<br>Bronnchospasms<br>Impotence<br>Fatigue<br>dizziness |
| | Carvedilol | 6.25mg BD | 25mg BD | |
| | Labetalol | 100mg BD | 400mg BD | |
| | Metoprolol succinate | 25mg OD | 100mg OD | |
| | Nebivolol | 5mg OD | 20mg OD | |
| ***ARB*** | Candesartan | 8mg OD | 32mg OD | As per ACE- I except for cough |
| | Irbesartan | 150 mg OD | 300mg OD | |
| | Losartan | 50mg OD | 100mg OD | |
| | Telmisartan | 40mg OD | 80mg OD | |
| | Valsartan | 80mg OD | 160mg OD | |
| | Olmesartan | 20mg OD | 80mg OD | |

**Note:** Fixed drug combinations (FDC) should be used where available due address the pill burden challenge.

***Mechanisms of Action***
***CCB: Calcium channel blocker (***inhibit Ca2+ entry into excitable cells causing vascular smooth muscle relaxation, CCBs decrease systemic vascular resistance, which lowers arterial blood pressure*)*
***ACE: Angiotensin converting enzyme*** (ACE inhibitors produce vasodilation by inhibiting the formation of angiotensin II. This vasoconstrictor is formed by the proteolytic action of renin (released by the kidneys) acting on circulating angiotensinogen to form angiotensin I).
***ARB: Angiotensin receptor blockers (***receptor antagonists that block type 1 angiotensin II ($AT_1$) receptors on bloods vessels and stimulate vascular smooth muscle contraction).

**Preferred drug combinations:**
- CCB *plus* Thiazide diuretic
- RAS-I *plus* Thiazide diuretic

ACE-I and ARBs should NOT be used in combination.

**Contraindications to various anti-hypertensives**

**RAS-inhibitors (ACE-I and ARBs):** Angioedema, Pregnancy, bilateral arterial renal stenosis, hyperkalemia

**Beta-blockers:** (non-selective beta-blocker e.g., propranolol): Asthma and COPD. Cardioselective beta-blockers can be used with caution. Also contraindicated in bradycardia or heart block.

**Methyldopa**: Liver disease.
**Thiazide diuretics:** Gout

If patient fails to respond to the regimens above, consider the following:
- Inadequate patient compliance.
- Inadequate doses.
- Drug antagonism, e.g., ephedrine raises blood pressure.
- Secondary forms of hypertension, e.g.,pheochromocytoma.

**Resistant hypertension:** When BPs remain >140mmhg despite being on maximum doses of atleast 3 anti-hypertensive drugs of which a thiazide-diuretic is included.

Table 3.8 lists causes of resistant hypertension as *Adapted from 2018 Kenya National Guidelines for Cardiovascular Diseases Management*

**Table 3.8: Causes of resistant hypertension**

| Category | Possible Causes | Intervention |
|---|---|---|
| **Non-adherence to therapy** | ✓ Instructions not understood<br>✓ Side effects<br>✓ Cost of medication and/or cost of attending at healthcare centre<br>✓ Lack of consistent and continuous primary care Inconvenient and chaotic dosing schedules<br>✓ Organic brain syndrome (e.g. memory deficit) | ✓ Adherence counselling<br>✓ Ensure family/social support mechanism for the patient<br>✓ Tailor dosing schedules to individual patients |
| **Volume Overload** | ✓ • Excess salt intake<br>✓ • Inadequate diuretic therapy • Progressive renal damage (nephrosclerosis) | ✓ Counsel on low salt diet, optimize diuretic therapy, refer as appropriate |
| **Associated conditions** | ✓ Smoking<br>✓ Increasing obesity<br>✓ Sleep apnoea<br>✓ Insulin resistance or hyperinsulinaemia<br>✓ Ethanol intake of more than 30 g (three standard drinks) daily Anxiety-induced hyperventilation or panic attacks<br>✓ Chronic pain<br>✓ Intense vasoconstriction (Raynaud' s phenomenon), arteritis | ✓ Manage the associated condition |
| **Identifiable causes of hypertension** | ✓ Chronic kidney disease<br>✓ Renovascular disease<br>✓ Primary aldosteronism<br>✓ Coarctation<br>✓ Cushing' s syndrome<br>✓ Phaeochromocytoma | ✓ Investigate and/or refer |
| **Pseudo-resistance** | ✓ ' Whitecoat hypertension' or office elevations<br>✓ Pseudohypertension in older patients<br>✓ Use of regular cuff in obese patients | ✓ Out of office BP measurement<br>✓ Ensure proper BP measurement technique |

| | | |
|---|---|---|
| **Drug-related causes** | ✓ Doses too low<br>✓ Wrong type of diuretic<br>✓ Inappropriate combinations<br>✓ Rapid inactivation (e.g. hydralazine) | ✓ Review treatment plan |
| **Drug actions** | ✓ Non-steroidal anti-inflammatory drugs (NSAIDs)<br>✓ Sympathomimetics: nasal decongestants, appetite | ✓ Remove offending drug, refer for specialist care |

**Patient Referral**
The following patients require specialized care and should be referred if the requisite expertise is not available at the facility of initial contact:

• All pregnant women
• Pre-existing diabetes or new onset diabetes.
• Heart failure
• BP >180 mmHg systolic and/or 110 mmHg diastolic BP
• Abnormal results on urine dipsticks or blood test
• Patients not reaching target BP of <140/90mmhg after a reasonable trial of anti-hypertensive therapy (4 weeks)
• Hypertensive patients aged ≤18 years.
• Suspected secondary hypertension
• Associated clinical condition: CAD, HF, CKD, CVA/TIA, PAD.
• Consider referral for patients aged ≥ 80 years with new onset hypertension.



## 3.10 Hypertensive Crisis

Sudden or sustained diastolic blood pressure of more than 120mm Hg with papilloedema, progressive decrease in renal function, and evidence of neurological dysfunction. Blood pressure should be controlled within 1 hour in order to prevent permanent damage and hypertensive emergencies. In both, the aim of treatment is to achieve diastolic BP of 100–110mm Hg.

However, rapid decrease of BP should be avoided to reduce the risk of cerebral hypoperfusion.

**Management**
There are two approaches to choose from in managing this condition, Approach A and Approach B, as shown in Table 3.9.

**Table 3.9:The approach in managing hypertensive crisis**

| Approach A | Approach B |
|---|---|
| Frusemide 40 IV+Hydralazine10mg IV every 15 minutes until desired effect or 50mg has been administered. The total dose may be repeated IM or IV after 6 hours ***OR*** sodium nitroprusside 0.25-10ug/kg/minute IV infusion | Nifedipine 20mg PO repeated after 1 hoour. |

- Following initial control of BP, switch to multiple oral therapy (hydrochlorothiazide+atenolol+hydralazine ***OR*** nifedipine ***OR*** methyldopa ***OR*** captopril).
- Admit those patients with severe hypertension or with hypertension crisis.
- Complications include congestive heart failure and renal failure. Refer to sections 3.2 heart failure, and 15.8, renal failure, for management guidelines.

**Patient Education**

Untreated hypertension has a high mortality rate due to: renal failure, stroke, coronary artery disease, and heart failure.

## 3.11 Cardiogenic Pulmonary Oedema

This is an acute medical emergency due to an increase in pulmonary capillary venous pressure leading to fluid in the alveoli. This is usually due to acute left ventricular failure. It may be acute or chronic.

**Clinical Features**

*Acute pulmonary edema:* shortness of breath of acute onset worse on exertion or lying in a recumbent position, a feeling of drowning/anxiety on lying flat, gasping for breath, sweating/diaphoresis, productive cough frothy blood tinged /pink sputum, chest pain, respiratory distress,

*Chronic pulmonary edema:* fatigue, shortness of breath on exertion, orthopnea, PND, weight gain, legs swelling.

Examination: confusion, agitation, irritability, cyanosis, cold extremities, orthopenic (prefers to sit upright, raised JVP, tachypnia, tachycardia, edema, rhonchi and crepitations, tender hepatomegaly.

**Investigations**

Chestx-ray reveals loss of distinct vascular margins, Kerley B lines and diffuse haziness of lung fields. ECHO: to rule out cardiac causes.

**Management – Pharmacological**
**This must be immediate:**
**ABCs**

- Prop up patient in bed.
- Give 100% oxygen 3.5–5L/min if patient is hypoxemic (SPO2 <90%)
- IV access and catheterization
- VTE prophylaxis
- Start IV Loop diuretics frusemide 40mg initial, repeat with higher dose every 20–30 minutes to 200mg maximum total dose. Others: Torasemide 10-200mg IV OR Bumetamide 1-8mg IV/day.
- Vasodilators: IV nitroglycerine 5-10mcg/minute titrated to a maximum dose of 200mcg/minute, IV Sodium nitroprusside 5-10mcg/minute (max: 400mcg/min).
- IV morphine: NOT RECOMMENDED in pulmonary edema due to increased risk of respiratory distress and incresed mortality.

**If not already on digoxin, digitalize except if due to myocardial infarction (see sections 3.2, heart failure, and 3.3, acute myocardial infarction).**

- IV aminophylline NOT RECOMMENDED risks outweigh the benefits.
- Restrict fluids/salt intake
- Monitor input/output
- Daily weight measurement
- Start on oral medication as soon as possible

**— Watch for respiratory depression.**

- Admit for
  - Management of all patients with pulmonary oedema.
  - Investigative procedures for underlying causes.
  - Management of underlying causes like hypertension.

# 4. Central Nervous System

## 4.1 Headache

Headache also known as cephalalgia, refers to diffuse pain in various parts of the head, with the pain not restricted to the area of distribution of a nerve**.** Headaches are due to activation of the primary afferent fibres that enervate cephalic blood vessels, chiefly meningeal or cerebral blood vessels. Headache is a common clinical presentation. Can be primary or secondary.

***Primary headaches:*** are due to over-activity of pain-sensitive structures in the brain. Triggered by certain factors like; fasting, alcohol, caffeine/caffeine withdrawal, stress, insomnia, somnolence, menstruation, fatigue, change in weather changes, head injury, exposure to bright lights, loud noises, smoke, and strong smells and foods etc. They form majority of the causes of headaches. They are benign often recurring. ***Examples of primary headaches*****:** Cluster headaches, migraines, and tension headaches.

***Secondary headaches:*** have identifiable causes with a close temporal relationship with the headache. These include head and neck trauma, vascular causes (e.g., non-traiumatic intracerebral hemorrhage), epileptic seizures, acute substance use or withdrawal and intracranial infections, assocated with. The headache resolves after treatment of the underlying cause.

**Approach to headaches**

**History taking:** SOCRATES acronym (Site, Onset, Character, Radiation, Associated symptoms, Time duration/course, Exacerbating or Relieving factors, Severity. Look for **'red-flags' for secondary headaches**: sudden onset (SAH), head after 50-years of age (tumor/temporal arteritis) increased frequency and severity of the headache (mass lesion/subdural hematoma), presence of underlying medical/surgical condition, presence of neurological signs and symptoms, papildema on fundoscopy, history of trauma to the head..

**Physical examination:**

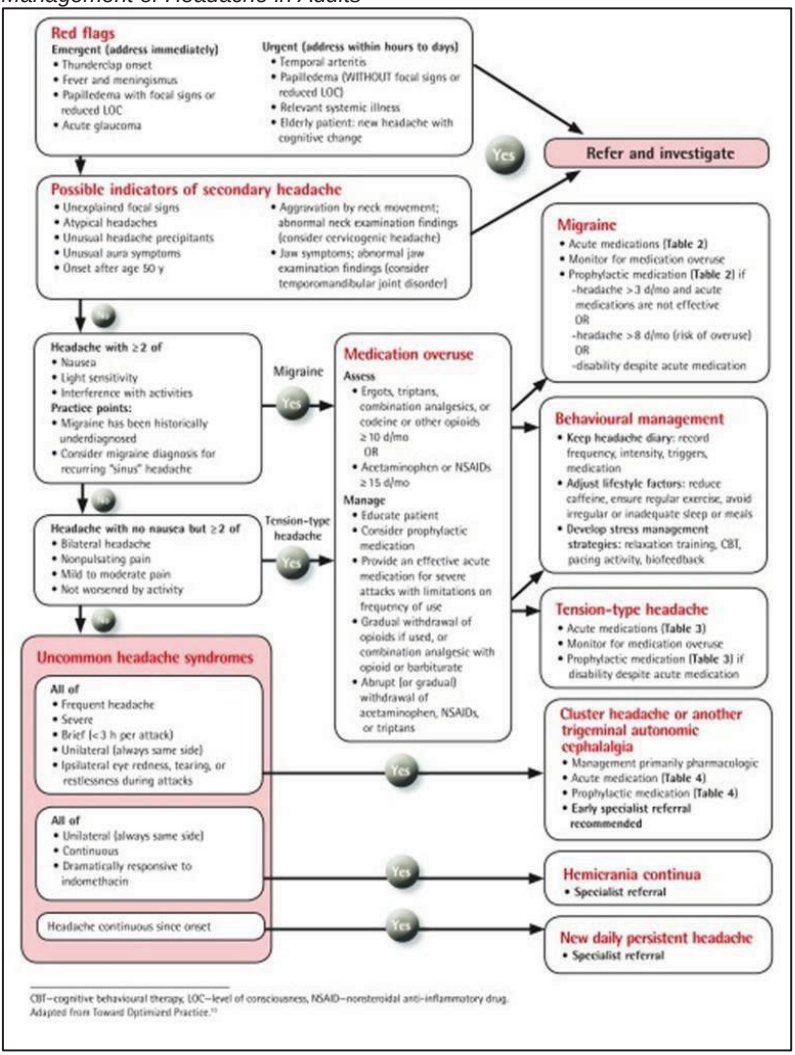
**Investigations:** General examination, Vital signs, fundoscopy, CV examination, neurological examination (signs of meningeal irritation), cranial nerves assessment, motor system examination,

Investigations: ESR (arteritis), TBC 9systemic infeection), Blood sugar (metabolic cause),, HIV, LP when necessary).

**Imaging:** Neuroimaging: Indicated in chronic headaches, neurological deficits. Do CT scan brain when cute hemorrhage, acute stroke OR head trauma) are suspected), otherwise, an MRI brain scan is preferred as it is more sensitive in diagnosing other conditions. Migraine headache no need for neuroimaging.

Figure 4.1 below represents the suggested Quick **reference algorithm from the** *Guideline for Primary Care Management of Headache in Adults as adapted from Becker, W. et al 92015).*

**Figure 4.1:** Quick **reference algorithm from the** *Guideline for Primary Care Management of Headache in Adults*



**Treatment**

- Can be non-pharmacological and pharmacological. The former includes behaviour and lifestyle modification such as:
  - Avoiding certain foods known to trigger migraine headaches.
  - Adopting consistent sleeping patterns.
  - Minimizing environmental stress.
  - Pharmacological treatment may involve administration of:
  - Analgesics like paracetamol.
  - Nonsteroidal anti-inflammatory drugs, ergotamine, and valproic acid for migraine.
  - Oxygen inhalation or sumatriptan and ergotamine tartrate for cluster headaches.
  - Nonsteroidal anti-inflammatory drugs and tricyclic antidepressants such as amitriptyline for tension headaches.
- Secondary headaches are treated with analgesics plus treatment of the underlying cause.

**Specific headaches** (Consult the Physician or Neurologist)

**Migraine headache:** Migraine headaches are common public heath problems affecting more women than men in a ratio of 3:1. They affect the quality of life of the patient and associated with a heavy economic burden.
Suspect migraines when: Headache is unilateral, pulsatile in a nature, one-day duration, associate with nausea and vomiting, very severe headaches. Can be triggered by; hormonal changes, fasting, certain foods (chocolate, baked foods with yeast, cultured dairy products like yoghurt, cheese, fruit juices, MSG-containing foods, salty foods), alcohol/ red wine, stress, sensory stimuli (lights/smell/noise).

**Acute Migraine headache:** should be started as early as possible to abort the symptoms They are abortive or symptomatic therapies.

**1st line:** Acetaminophen and NSAIDs for mild-moderate migraine attacks.

- Acetaminophen/paracetamol); 325mg 4-6hourly to a maximum of 4gm/day. Acetaminophen can be combined with aspirin or caffeine (acetaminophen 250mg+aspirin 250mg+caffeine 65mg 1-2 tablets/capsules 6-hourly (maximum 8 tablets/capsules per day).
- NSAIDS: **Ibuprofen** 200-800mg 4-6hourly (maximum dose 2.4gm/day), **Naproxen** 250-400mg 12-hourly (maximum 1gm/day); **Mefenamic acid** for acute menstrual migraine 500-1500mg/day; **Acetylsalicylic acid (ASA)** 1-3gm/day; **Ketoprofen** 100mg IM STAT; Ketorolac; 60mg IM STAT then every 15-30minutes to a maximum daily dose of 120mg at at most 5 days. Caution on gastritis can add a PPI like omeprazole. Avoid NSAIDs in heart failure.
- **Triptans a**re 1st line therapies for moderate-severe migraine or second line to mild-moderate migraine not responding to Acetaminophen or
- NSAIDS. They are Serotonin (5HT-1 receptor specific agonists). Examples of triptans:

- **Sumatriptan**: 6 mg SC, repeat after one hour; maximal dosage, 12mg/day OR 25-100 mg PO every two hours, maximal dosage: 200mg/day. Also available as an intranasal spray.
- **Zolmitriptan**: 2.5-5mg PO 2-hourly (maximum dose 10mg/day).
- **Rizatriptan:** 5-20mg 2-hourly (maximum 30mg/day).
- **Naratriptan**: 1-2.5mg 4-hourly (maximum : 5mg/day).

**Note:** Triptans are CONTRAINDICATED in ischemic vascular conditions, vasospastic coronary disease, uncontrolled hypertension, or other significant cardiovascular disease.

**Ergotamine and Ergotamine-derivatives:** a 5-hydroxytryptamine (5-$HT_1$) non-selective agonist. Currently **less favorable for migraine** headaches due to their adverse effects including medication-overuse headaches, increasing the frequency of headaches and ergot poisoning.

- **Ergotamine:** 1-2mg PO every hour to a maximum of 3 doses in a day.
- **Ergotamine + Caffeine:** (Ergotamine 1gm, Caffeine 100mg): 2 tablets STAT then half-hourly to a maximum of 6 tablets in an attack.
- **Dihydroergotamine (DHE):** 0.5-1mg STAT IM or SC, repeat every one hour to a maximum of 3mg.

**Anti-emetics:** Dopamine receptor antagonists

- **Metoclopamide:** 10mg IV/IM or PO 20-30 minutes before.
- **Prochloperazine** 25mg IM/PO/suppository

**Narcotic/opioid analgesics:** Their use should be limited due their potential for abuse and rebound headaches

**Intranasal Lidocaine/Xylocaine:** 4% administered into the nostrils.

**CGRP antagonists: Oral Gepants**: short-acting

- Rimegepant
- Urogepant
- Atogrogepant
- Ubrogepant

**Migraine headaches Prophylaxis**

These are drugs taken to prevent migraine headaches by reducing the frequency, severity, and headache-related distress while improving improve quality of life of migrainers and prevent the progression to chronic migraines.

**Indications for prophylaxis** therapies for migraines include:

- ≥ 4 attacks in a month,
- ≥ 8 headache days a month,
- debilitating headaches, and
- medication-overuse headaches.
- When acute/abortive therapies are not tolerated or contraindicated.
- Patient's preference for fewer attacks.

**Preventive therapies:**

- **Beta-blockers:** Propranolol (40-320mg/day PO), Metoprolol (37.5-200mg/day PO, Timolol (20-30mg/day PO).
- **Anti-convulsants:** Topiramate 25-100mg/day PO, Divalproex (250-1000mg BD PO Sodium Valproate (Valproic acid): 250-500mg BD PO.
- **Tricyclic antidepressants (TCAs)**: Amitriptylline (25-150mg/day PO).
- **SSRIs:** Venlaxafine 12.5-150mg PO OD (miraine with vertigo)**.**
- **Calcitonin gene-related peptide (CGRP) antagonists:** long acting CGRP is neuropeptide released from the trigeminal ganglia cells that sensitizes the peripheral nerves and cause dilation of the cerebral and dural blood vessels. CGRP antagonists reduces the vasodilation.
  Anti-CGRP Monoclonal antibodies: FDA Approved:
  a) **Erenumab**: Montly SC injection,
  b) **Fremanezumab:** 1-3 monthly injection
  c) **Galcanezumab:** monthly injection
  **d) Eptinezumab:** 3-monthly injection

Discontinuation of migraine prophylaxis therapy if headaches are controlled for at least 6-12 months, consider slowly tapering and discontinuing therapy.

- **Nonpharmacologic therapies** (alternative therapies
  - Relaxation training,
  - Thermal biofeedback combined with relaxation training,
  - Electromyographic feedback, and
  - Cognitive behavior therapy (CBT)
  - Acupunture, massage
  - Keep headache diaries, avoid triggers

Table 4.1 below summarizes the chronic headache syndromes.

**Table 4.1: Summary of chronic headache syndromes**

| Migraine Headache | Tension Headache | Cluster Headache |
|---|---|---|
| *At least 2 of these features:*<br>Unilateral location<br>Throbbing character<br>Worsening pain with routine activity<br>Moderate to severe intensity<br><br>*At least 1 of these features*<br>Nausea and/or vomiting | *At least 2 of these features:*<br>Pressing, tightening, or non-pulsatile character<br>Mild-moderate intensity<br>Bilateral location<br>No aggravation with routine activity<br><br>*Both of the following features:*<br>No nausea or vomiting (may have anorexia)<br>No photophobia and/or phonophobia | 5 attacks, frequency; 1-8 attacks on any given day. NB: *Headaches often recur at the same time each day during the cluster period, which can last for weeks to months.*<br>Severe, unilateral, bilateral, supraorbital, or temporal pain lasting 15minutes to 3 hours (untreated)<br><br>*with at least one of the following features on the same side as the pain:*<br>Lacrimation<br>Nasal congestion |

| Migraine Headache | Tension Headache | Cluster Headache |
|---|---|---|
| ↓ Photophobia and phonophobia | | ↓ Rhinorrhea<br>↓ Forehead and/or facial sweating<br>↓ Ptosis<br>↓ Miosis<br>↓ Eyelid edema |
| Management:<br>*See the text* | Management<br>Pharmacological therapy<br>➢ Analgesics: Paracetamol, NSAIDs<br>➢ Antidepressants: Amitriptylline, Mirtazapine OR venlafaxine.<br>Non-pharmacological therapy<br>➢ Stress management (physical and mental)<br>➢ Regular meals<br>➢ Hydrate well<br>➢ Exercise,<br>➢ Sleep therapy<br>➢ Massage<br>➢ EMG Biofeedback<br>➢ Cognitive behavior therapy (CBT)<br>➢ Relaxation training | Management<br>**Acute Cluster Headache**<br>Triptans:<br>➢ Sumatriptan<br>➢ Zolmitriptan<br>Supplemental oxygen may be required during the attack.<br><br>**Chronic Cluster Headache**<br><br>**CCB:** Verapamil |
| **Cluster headaches:** Have a genetic component. More common in males 20-40 years of age. May start during sleep.<br>**Tension Headache:** Gradually developing, constant bilateral dull ache or squeezing band-like headache. Triggered by stress both physical and emotional, dehydration, lack of sleep. Typically starts from midday/ towards the end of the day. | | |

## 4.2 Seizure Disorders

Epilepsy is a clinical syndrome characterized by the presence of recurrent seizures. Seizures are a result of excessive electric impulse discharge of cerebral neurones.

### 4.2.1 CLASSIFICATION AND TREATMENT OF SEIZURES

Seizures are classified as partial and generalized. These seizures can be explained as follows:

#### PARTIAL SEIZURES

- Simple partial seizures; can be motor, sensory and sensory-motor (consciousness not impaired).
- Complex partial seizures; starting with anaura (later impairment of consciousness) and often accompanied by automatic behaviour.
- Partial seizures becoming progressive (Jacksonian seizures) or generalized.

#### GENERALIZED SEIZURES

**Initially generalized:**

- Absence seizures
- Tonic seizures
- Myoclonic seizures
- Tonic-clonic seizures
- Clonic seizures
- Atonic seizures

**Clinical Features**

Meticulous history from patient and reliable witness is critical in diagnosing a seizure disorder. Ask about the prodromal phase, aura and the type, duration, frequency, and the age at onset of seizures. Details about the post ictal phase are important. Ask about precipitating factors, for example alcohol use.

**Investigations**

- Thorough physical examination including fundoscopy in newly diagnosed cases
- Skullx-ray: All cases for possible radiolucent focal lesion, raised intracranial pressure
- Full haemogram
- Malaria parasites (MPs) especially in children
- Blood sugar, urea, and electrolytes in cases where metabolic conditions are considered as a cause of a seizure disorder
- Electroencephalogram
- CTscan
- MRI scan

**Management**

**First Aid**

- During an epileptic attack:
  - Patient should be placed on the left lateral position with head turned to the same side.
  - Remove or loosen tight fitting clothing around the neck.
  - Remove dentures.
  - DO NOT attempt to insert any instrument into the mouth to avoid tongue biting as this may have already happened.
  - Do not allow patient to be surrounded by too many eager observers.
  - Allow seizure to complete its course without physically attempting to hold down the patient. However, remove patient from danger, e.g.,fire.
- After an attack:
  - Investigate patient as outlined above and started on therapy.

**General Management**

- Treat underlying diagnosed condition if possible,e.g.,hypoglycaemia, meningitis.
- Establish firm diagnosis before starting therapy.
- For most patients, start on therapy as outpatients.
- Start therapy if patient has had 2 or more seizures within 1year.
- Advise patient that treatment is usually life-long. Therapy maybe discontinued after a seizure-free period of at least 2 years. Reduce dose gradually over many months. Sudden discontinuation of drugs may precipitate status epilepticus. Complex partial seizures will require lifelong drugs.

**Pharmacological Management**

- Start therapy with one drug, usually phenobarbitone. Increase at regular intervals until seizures are controlled or side effects appear. If side effects appear and seizures are still not controlled, introduce other drugs and taperoff the first drug. Refer to Table 4.1 for a summary of the administration of the drugs of choice for the different types of seizures.

**—Drugs used at maximum recommended dose should be withdrawn if seizures are not controlled.**

- Admit if underlying metabolic cause is suspected or raised intracranial pressure is present.

**Patient Education**

- Avoid becoming drunk, especially drinking sprees during weekends.
- Eat at regular intervals.
- Stress, physical or mental may precipitate a fit, thus manage stress.
- Avoid sleep deprivation.
- Never swim alone and all precautions should be taken when swimming.
- Avoid operating heavy or sharp edged machinery.
- To prevent burns, protective shield should be made around “jikos”(braziers).

## 4.2.2 STATUS EPILEPTICUS

This is a succession of seizures in which the patient does not regain consciousness between attacks. It could be due to partial, complex partial, absence, tonic-clonic, or clonic. Only the last 2 are life threatening.

**Clinical Features**

Patient is not able to talk, the tonic phase is not clear and the patient appears in continuous clonic phase, the short tonic phases being difficult to see. May bein respiratory embarrassment with cyanosis or may be hypoglycaemic.

**Table 4.2: Pharmacological management of common seizures**

**a.) Drugs of choice**

| Partial | First drug | Other drugs |
|---|---|---|
| Simple | Phenytoin | Carbamazepine, valproic acid, gabapentin |
| Complex | Carbamazepine | Phenytoin, valproate, gabapentin, zonisamide |
| Secondarily generalized | phenytoin | Lamotrigine, valproate, carbamazepine |
| Generalized | First drug | Other drugs |
| Absence | Ethosuximide | Valproic acid, clonazepam, valproate, lamotrigine, |
| Tonic-clonic, clonic | Phenobarbitone | Carbamazepine, phenytoin, lamotrigine, |
| Tonic | As above | As above |
| Atonic | As above | As above |
| Myoclonic | Clonazepam | Nitrazepam, valproic acid, phenobarbitone, carbamazepine, |

**b.) Drug dosage and frequency**

| Drug | Dose | Frequency |
|---|---|---|
| Phenobarbitone | 60–240mg | Once daily |
| Phenytoin | 50–400mg | Once daily |
| Carbamazepine | 400–1,400mg | In 2–3 divided doses |
| Sodium valproate | 600–2,400mg | In 3 divided doses |
| Ethosuximide | 20–40mg/kg/day | In 2 divided doses |
| Clonazepam | 1–12mg | Once daily |

**Management**

**Supportive**

Place patient by the side (lateral position). Do NOT attempt to put anything into the patient's mouth to stop the biting of the tongue. You are likely to cause more damage.

**Pharmacological**

- Give IV (not IM) diazepam 10mg STAT then infuse IV phenytoin 15–20mg/kg at a rate not exceeding 50mg/minute (for adults). Maintenance dose of 100mg 8 hourly. To be administered in normal saline. If no response use IV phenobarbitone. Maintenance 300–500mg/day, preferably oral.
- Phenobarbitone second line after phenytoin. Loading dose of phenobarbitone 20mg/kg IV at a rate of 50–75mg/minute. If no response repeat at 5–10mg/kg. Maintenance 1–5mg/kg/day PO.

- Rectal diazepam 10–20mg may be as effective as intravenous diazepam. Use rectal solution at 0.5mg/kg.

**Phenobarbitone should only be used where respiratory support is available.**

## 4.3 Ischaemic Stroke

Stroke is a group of diseases that are of abrupt onset and cause neurological damage. The majority of strokes result from interrupted supply of blood to the brain (ischaemic), while about 10–15% arise from haemorrhage into the brain substance or its surrounding spaces (haemorrhagic). Ischaemic stroke commonly arises from mural thrombi forming at the site of atherosclerotic lesions, blocking blood flow. Alternatively, ulceration or rupture of an atherosclerotic plaque may lead to the formation of a clot and distal embolization, or still, haemorrhage into an atherosclerotic plaque may obstruct the artery.

Commonly, emboli arise from the left side of the heart, from mural thrombi, vegetations from infected heart valves, or arrhythmias. Paradoxical emboli can arise from venous circulation and access cerebral circulation through right to left cardiac shunts.



**Clinical Features**

Rapid onset of neuronal malfunction referable to the area of the brain for which blood supply is disrupted.

**Diagnosis**

From history and physical examination.

**Investigations**

- Non-contrast CT scan brain
- Cerebral angiography

**Management**

- Thrombolytic therapy useful if initiated within 3 hours of onset of symptoms
- Drugs are intravenous tissue plasminogen activator (tPA), streptokinase, and intra-arterial recombinant prourokinase (rproUK).

## 4.4 Haemorrhagic Stroke

Hypertension and vascular malformations are the commonest causes of haemorrhagic stroke, both subarachnoid and intracerebral haemorrhages, both of which are associated with very high mortality.

**Clinical Features**

Intense headache of sudden onset, commonly associated with elevated blood pressure. In half of patients there is transient alteration of the level of consciousness, commonly going into coma. If there is subarachnoid bleed there are features of meningism including stiff neck and a positive Kernig's sign.

**Diagnosis**
History and physical examination usually suggest the diagnosis.

**Investigations**
- CT scan performed within 24 hours of onset of symptoms
- Lumbar puncture
- Cerebral angiogram

**Management**
Management depends on the cause of the haemorrhage. Control of blood pressure controls hypertensive haemorrhage. For A-V malformations treatment options include surgical resection, embolization of the feeding arteries and radiation-induced thrombosis.

# 5. Endocrine System

These are conditions associated with difference hormone abnormalities from the endocrine systems They include; diabetes, thyroid diseases, pituitary gland disease and adrenal gland diseases.

## 5.1 Diabetes Mellitus

Diabetes mellitus (DM) is recognized by chronic elevation of glucose in the blood (hyperglycaemia)

**NOTE**: The terms insulin dependent diabetes (IDDM) and non-insulin dependent diabetes mellitus (NIDDM) for Type 1(T1DM) and Type 2 DM (T2DM) respectively are absolete and no longer used as patients with T2DM may require insulin as discussed under insulin use in T2DM.

**Clinical Presentation**

Commonest symptoms are polyuria, polydipsia, polyphagia and weakness. Wasting tends to occur in type 1 diabetes while obesity may predominate in type 2. Patients with T2DM may be asymptomatic. Some patients with diabetes may present with features of target organ damage or complications involving the kidneys (proteinuria/renal failure), heart (heart attack or heart failure), nerves (peripheral neuropathy) eyes (DM retinopathy, cataracts) brain (stroke) or sexual dysfunction.

**Table 5.1: Classification of diabetes**

| Type | Pathogenesis | Description |
|---|---|---|
| Type 1 diabetes mellitus (T1DM) | Autoimmune destruction of the beta cells of the pancreas. Leads to absolute insulin deficiency | Affects children from 6 months to young adults < 30 years. Acute presentation Associated with DKA Positive autoantibodies (anti-GAD 65, islet Cell antibodies (ICA). |
| Type 2 diabetes mellitus (T2DM) | Insulin resistance | Older overweight/obese Strong genetic link |
| Gestational Diabetes Mellitus (GDM) | Insulin resistance Risk factors include overweight and obesity, advanced maternal age, a family history or any form of diabetes, large babies (=>4.5kgs), prior history of GDM. | Diabetes appearing for the first-time during pregnancy in the 2nd of 3rd trimester. Tends to resolve after delivery but predisposes a mother to T2DM in the future. Should be differentiated from diabetes in pregnancy which is pre-existing other type of diabetes during pregnancy. |
| Maturity Onset Diabetes of the Young (MODY) | Monogenic diabetes due to a single gene mutation in the enzymes for glucose metabolism. | Young non-obese patient 10-40 years Have =>2 consecutive generations of patients with diabetes in the family (T1/T2 DM) diagnosed at < 45 years of age. Often-times misclassified as T1DM. Genetic testing is important for diagnosis. Respond to sulphonyureas therapy |
| Latent onset Diabetes in Adults (LADA or Type 1.5 diabetes) | Autoimmune process leading to insulin deficiency like in type 1 diabetes but in older patients 30-50 years | Have features bordering both types 1 and 2 diabetes. Lean patient Positive autoantibody tests (Anti-GAD 65). Maybe associated with other autoimmune diseases. |
| Secondary Type of diabetes | Steroid induced diabetes Pancreatic disease (type 3c diabetes);chronic pancreatitis, pancreatectomy, pancreatic cancer) etc. | Evidence of underlying disease condition of drug use. |

**Table 5.2: Diagnosis of Diabetes**

| Measurement | Diagnostic Value |
|---|---|
| Fasting Blood Sugar (FBS) | =>7mmol/l (126mg/dl) |
| Random Blood Sugar (RBS) *with symptoms of hyperglycaemia* | =>11.1mmol/l (200mg/dl) |
| 2-Hour-Post Prandial Glucose (2-hr-PPG)2 or more consecutive generations of patients with diabetes in the family (T1/T2 DM) at < 45 years of age. | =>11.1mmol/l (200mg/dl) |
| Glycated Haemoglobin (HBA1c) | =>6.5% |
| FBS: Fasting Blood Sugar: No food eaten for at least 8 hours<br>Hour PPG: Performed by giving 75grams of glucose load.<br>HBA1c: Performed in a certified laboratory.<br>Conversion of blood sugar from mmol/l to Mg/dl: Multiply by 18.<br>Conversion of blood sugar from Mg/dl to mmol/l: Divide by 18. | |

Source**:** International Diabetes Federation Diagnostic Criteria

### PREDIABETES STATES

Transitional conditions, a step away from overt diabetes. Risk of progressing to T2DM.

- Impaired Fasting Glucose (IFG): FBS: 6.1-6.9mmo/l
- Impaired Glucose Tolerance: IGT: 2-Hr PPG: 7.8-11mmol/l.

## 5.1.1 PHYSICAL EXAMINATION

- Anthropometric measurements: blood pressure, weight, height (body mass index; BMI), waist vircumference.
- Features of insulin resistance: acanthosis nigricans.
- Features of complications: Diabetic foot, edema (kidney or heart failure).
- Other tests done in patients with diabetes:
  - Urinalysis: ketones, proteinuria
  - Renal function tests/urea, electrolytes and creatinine (UECr)
  - Lipid profile: dyslipidemia.
  - Glycated Hemoglobin (HBA1c): for monitoring sugar control: target < 7%.

## 5.1.2 MANAGEMENT OF DIABETES MELLITUS

Goals of management
- Symptomatic relief
- Symptomatic relief
- Correct hyperglycemia
- Long term glycemic control
- Prevent DM complications and diabetes related deaths.

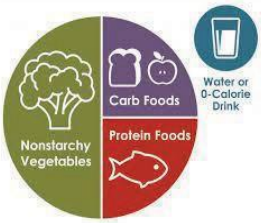


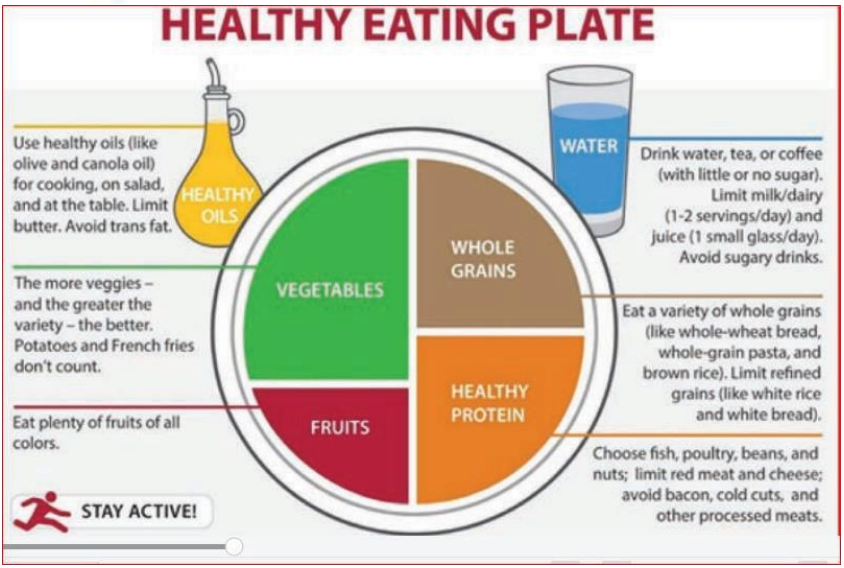
**Non-pharmacological therapy**
This is important for most types of diabetes and include; healthy diets, cessation of tobacco use, moderation or cessation of alcohol use, weight loss for the overweight/obese patients and aerobic physical activity/exercise. For the diet, consultation with a nutritionist/dietician is important and should be individualized.
One can use a plate model to advise on diet;

- Half of a plate: vegetables
- A quarter of a plate: proteins
- A quarter of a plate: starches/carbohydrates.
- Limit fruits intake.

**Model plate**

**Figure 5.1: Plate Model as adapted from the Kenya National Diabetes Management Guidelines (2018).**




**Food composition**

- Carbohydrate: 50–60% in complex form, e.g., rice, beans, peas, etc.
- Protein: 10–20%. Vegetable protein source include soyabeans, lentils and beans.
- Fat: 25–30%.
- Fiber in diet can prolong absorption of sugar. Fiber containing foods include beans, legumes and bran.
- Artificial sweeteners e.g., saccharin and aspartate, are helpful in maintaining a palatable diet.

Consider the Glycemic Index (GI) of various foods. Encourage low GI foods and avoid high GI foods. The GI of various foods is as indicated in the table below as adapted and modified from the 2018 Kenya Diabetes Study Group Guidelines.

**Table 5.3:Glycaemic index of selected types of food**

| Low Glycemic Index (GI) Foods | High Glycemic Index (GI) Foods |
|---|---|
| Whole wheat or bran bread<br>Brown rice<br>Sweet potatoes<br>Whole wheat pasta<br>Fresh peas<br>Whole wheat sugar free cereal<br>Oatmeal<br>Whole grain pasta<br>Kidney beans<br>Raw carrots<br>Dairy products<br>Dried beans<br>Brown or yellow lentils<br>Chick peas<br>Green vegetables (tomatoes, eggplant, zucchini, garlic, onions etc).<br>Green beans<br>Green Lentils<br>Split peas<br>Dark Chocolate (>70% cocoa).<br>Fruits (Green apples, cherries, berries, ). | Glucose<br>Potatoes: (mashed, baked, chips/French fries, boiled peeled).<br>Rice Flour<br>Honey<br>Cooked Carrots<br>Corn Flakes<br>Cooked Broad beans<br>Pumpkin<br>Sugar (Sucrose)<br>White bread<br>Refined sweetened cereal<br>Chocolate bars<br>Soda<br>Cookies<br>Corn<br>White rice<br>Noodles<br>Raisins<br>Fruits: Watermelon, bananas.<br>Fruit juices. |

### Meal Schedules

Strict adherence to meals schedule is important.

**Table 5.4: Pharmacological Therapy in DM.**

| Diabetes Type | Choice of Therapy | Remarks |
|---|---|---|
| Type 1 DM | Insulin | Insulin therapy is mandatory in T1DM.<br>No role for OGLAs |
| Type 2 DM | ● Oral Glucose Lowering Agents (OGLAs).<br>● Insulin<br>● GLP-1 agonists | OGLAs can be used alone or in combination with insulin/GLP-1 agonists. |
| Gestational Diabetes (GDM) | Insulin<br>Metformin | Other anti-daibetic drugs are contraindicated in pregnancy |
| Maturity Onset Diabetes of the Young (MODY) | Sulphonyureas | Depends on the type of gene mutation diagnosed by genetic testing.<br>MODY 2 (GCK) mutation does not require treatment<br>Some patients may respond to insulin |
| Latent Onset Diabetes of Adults (LADA) | Insulin | Requires insulin use within 6 months from diagnosis |
| Secondaty types of diabetes (Steroid induced DM, pancre | Insulin | Manage the underlying cause;<br>Withdrawal of steroids in steroid-induced DM. |

**Drugs used in Management of Diabetes**

- Insulin
- Oral Glucose Lowering Agents
- Glucagon-Like Peptide (GLP)1-Receptor Agonists

**Insulin**

This is the drug of choice in type 1 diabetes, GDM and LADA. Type 2 Diabetes may require insulin in the course of their disease. It is available only in injectable form. There are various types of insulins available classified according to their duration of action as summarized in the table below.

**Table 5.5: Types of insulin according to their duration of action**

| Insulin type | Examples | Onset of Action | Duration of Action | Frquency of Dosing |
|---|---|---|---|---|
| Rapid-Acting Insulin | Lispro, Aspart, Glulisine | 5-15 minutes | 3-5hours | As a bolus at meal time |
| Short-Acting | Regular or soluble insulin | 1 hour | 5-8 hours | Three times daily |
| Intermediate Acting | NPH | 1-2 hours | 14+ hours | Once or twice daily |
| Long-Acting or Basal insulin | Determir, Glargine, Degludec | 1-4 hours | 20-24 hours Degludec upto 42 hours | Once daily |
| Pre-Mix Insulin | Combination of either; intermediate (NPH) and short or rapid acting insulins | 0.5-1 hour | 7.5 - 24 hours | Twice Daily |

**Indications of Insulin in T2DM**

- OGLAs are not effective/ poor glycemic control e.g., persistent polyuria, hyperglycemia, HBA1c > 8%. This is due to the progressive nature of T2DM.
- Acute complications of DM (DKA, HHS).
- Infection
- Acute medical emergencies (acute coronary syndromes, stroke, sepsis).
- Presence of complications, e.g., renal failure, retinopathy, diabetes foot.
- Organ failure (renal, liver, heart).
- Secondary types of diabetes: Steroid induced DM, secondary DM.
- Patients undergoing surgery.

**Insulin therapy in inpatients:**

- Insulin measurement: start soluble insulin 10-16 units thrice a day.
- Injection techniques: intravenous/ intramuscularly, I.v infusion, subcutaneously. Avoid subcutaneous injection in dehydrated patients due to poor absorption.

- When blood glucose level is between 8.3 and 11.0 mmol/L, change from soluble/ regular insulin to an intermediate acting or premix insulin at a dose of two-thirds (2/3) of the total daily soluble insulin requirement.

**At Discharge: Ensure that the patient;**

- Can self inject insulin or has someone who can do it for them.
- Understand how to measure insulin dose.
- Can recognise symptoms of hypoglycemia and manage accordingly.
- Can do Self Blood Glucose monitoring (SBGM) at home before and after meals (paired blood glucose monitoring) and at bed-time.
- Can do Self-insulin adjustments when blood sugars are more or less than target levels (by 2-6 units when necessary).
- Understands proper insulin storage:
  - If unopened can be refrigerated between 2-8 degrees centigrade;
  - If open can be be stored in a clay pot.
  - Avoid extremes of temperature.
  - Avoid shaking.
  - Those insulin on insulin-pen can be stored at room temperature for upto one month (28 days).

**Oral Glucose Lowering Agents (OGLAs)**

- Biguanides: Metformin the only drug in this class. 1st line for type 2 diabetes, overweight/obese.
- Sulfonylureas (Sus):
  - 1st generation Sus: Chlorpropamide, Tolbutamide, Tolazamide
  - 2nd generation Sus: Second: Glibenclamide/ glyburide, Glipizide, Glimepiride, gliclazide, gliquidone.
- Thiazolidinediones: Pioglitazone
- Di-Peptidyl Peptidase Inhibitors: (DPP-4 Inhibitors/ Gliptins): Gliptins: Vildagliptin, Sitagliptin, Linagliptin.
- Sodium Glucose Transporter 2: (SGLT-2) Inhibitors (Gliflozins): Dapagliflozin, Empagliflozin, Canagliflozin, Ertugliflozin).
- Meglitinides: Repaglinide, Nateglinide.
- Alpha-glucosidase inhibitors: Acarbose, Miglitol, Voglibose.

**Table 5.6: Oral glucose lowering agents (OGLAs) and their mechanism of action, dosing, side effects caution/contraindications.**

| Examples | Daily dosing | Side effects | Caution or contraindications | |
|---|---|---|---|---|
| A. Biguanide: *increases insulin sensivity and reduces glucose production from the liver* | | | | |
| Metformin | 500mg to 2gms | GI side effects: flatulence, abdominal pains, vitamin 12 deficiency | Avoid in advanced renal failure eGFR<30 | |
| Sulphonyureas: *stimulates insulin release from the pancreas* | | | | |
| Gliclazide MR<br>Glimepride<br>Glibenclamide<br>Glipizide | 0-120mg<br>1-8mg<br>2.5-15mg<br>5-40mg | hypoglycemia<br>weight gain | Organ failure (liver, kidney, heart)<br>Gliclazide is safe in renal failure<br>pregnancy | |
| Thioziolindiones (Glitazones): *Improves peripheral insulin sensitivity, Reduce glucose production from liver*, | | | | |
| Pioglitazone | 5-45 mg | hepatotoxicity, fluid retention, weight gain, osteoporosis | Heart and liver failure, pregnancy, osteoporotic fractures | |
| Di-Peptidyl Peptidase Inhibitors/ DPP-4 Inhibitors/ Gliptins: *an incretin that increases glucose-mediated insulin secretion. Inhibits the DPP-4 enzyme responsible for the breakdown of GLP 1* | | | | |
| Vilagliptin<br>Sitagliptin<br>Linagliptin | 0-100mg<br>0-100mg<br>mg | urticaria, angioedema, acute pancreatitis, hypoglycaemia especially when combined with SUs. | pancreatitis<br>allergy, pregnancy, lactation<br>caution with heart failure | |
| Sodium Glucose Transporter 2: (SGLT-2) *Inhibitors (Gliflozins): inhibits the sodium-glucose co-transporter 2 in the renal proximal convulated tubules (PCT) which is responsible for glucose and sodium reabsorption resulting in in increased urinary glucose and sodium excretion.* | | | | |
| Dapagliflozin<br>Empagliflozin<br>Canagliflozin | 5-10mg<br>0-25mg<br>00-300mg | | polyuria, dehydration, hypotension, DKA or euglycemic ketoacidosis, genital candidiasis, limb amputation (canagliflozin). | Renal failure, pregnancy |
| Meglitinides/ Glinides: *Stimulate insulin release from the pancreatic beta cells by inhibiting ATP-sensitive potassium channels (insulin secretagogues).* | | | | |
| Repaglinide<br>Nateglinide | 1-16mg<br>60-120 three times a day | | liver impairment, fluid retention, weight gain, dilutional anaemia | Liver disease, pregnancy, renal impairment. (repaglinide)<br>Nateglinide is safe in renal impairment |

| Examples | Daily dosing | Side effects | Caution or contraindications | |
|---|---|---|---|---|
| Alpha-Glucosidase Inhibitors (AGI): *Slow digestion of complex sugars (sucrose and starch) therefore delays absorption, resulting in slow post-prandial increase in blood glucose.* | | | | |
| Acarbose<br>Miglitol<br>Voglibose | 25-300mg<br>25-300mg<br>0.2-0.9mg | | dyspepsia, diarrhea, flatulence | intestinal obstruction, chronic intestinal disease, DKA |

**Glucagon-Like Peptide 1 (GLP-1) Agonists**

- Include: Exenatide, Dulaglutide, Liraglutide, Semaglutide
- Mechanism of action
  - Are incretins that increase insulin secretion in a glucose-dependent mechanism as well as inhibition of glucagon relase, increase satiety and gastric emptying.
- Administration: subcutaneus injections.
- Dosage:
  - Exenatide: 5-10mcg twice a day SC.
  - Liraglutide: 0.6-1.8mg/day SC.
- When choosing therapy is T2DM, consider the following;
  - Drug availability and accessibility
  - Cost of drug
  - Side effect profile: hypoglycemia, weight gain
  - Presence of complications and comorbidities.

**Sugar Targets**

- In-patients: 8.3–13.4mmol/L in the hospital
- Home:
  - FBS: --6.9 (<7mmol/l for but not < 4mmol/L).
  - RBS: <10mmol/l.
- HBA1c: done 3-6monthly: <7%.

**Patient Education**

- Teach patients how to avoid foot injury. Hospital occupational therapist should advise patients on foot care.
- Patients with any injury, however minor, should seek medical advice.
- Patients should eat regularly.
- Patients should carry sweets or glucose and chew them if they experience any symptoms of hypoglycaemia.
- Patients should always carry “Diabetic Alert” card with them.
- Patients should join any branch of the Kenya Diabetic Association for support and continuing education.
- Patients should be followed up in the diabetic clinics or non-communicable diseases centers.
- Advise on annual eye examination.

**Psychosocial Support**

Offer psychosocial support to patients with diabetic aptientswith their caregivers as they experince a lot of stress.

## 5.2 Diseases of Pituitary Gland and Adrenals

Pituitary gland disorders can be either pituitary hyperfunction or hypofunction. These are reflected in the disorders of adrenals, thyroid and ovarian functions.

### 5.2.1 THYROID DISEASES

These are conditions affecting the thryoid gland.
The thyroid is an endocrine glands that produces thyroid hormones (T3 and T4) under the stimulation of TSH from the anterior pituitary gland.
These diseases include**:**

- ♦ Hyperthyroidism/Thyrotoxicosis: Excessive thyroid hormones.
- ♦ Hypothyroidism: Reduced thyroid hormones.
- ♦ Goiter: Enlargement of the Thyoid Gland.
- ♦ Thyroid Nodules: Discrete thyroid mass that can be single or in multiple.

Table 5.7 provides a summary of the thyroid diseases.

**Table 5.7: Summary of thyroid diseases**

| Thyroid Disease | Examples | Clinical Features | Remarks |
|---|---|---|---|
| Hyperthyroidism | Hyperthyroidism with thyoid gland hyperfunction (Graves Disease, excessive iodine). | Palpitations, excessive sweating, weight loss, increased appetite, heat intolerance, irritability/ nervousness, menstrual disturbance, hair changes (thinning), eyes involvement (proptosis), tachycardia | Elevated thyroid hormones (T3/T4). Suppressed TSH.<br><br>Subclinical hyperthyroidism (Normal T4/T3 and low TSH). |
| Hypothyroidism | Hashimoto Thyroiditis<br>Post thyroid surgery (thyroidectomy)<br>Iodine deficiency | Depression, weight gain, reduced appetite, cold intolerance, hair changes (brittle), bradycardia | Low thyroid hormones (T3/T4), elevated TSH<br><br>Subclinical Hypothyroidism (Normal T3/T4, high TSH). |
| Goiter | Endemic goiter (iodine deficiency) | Features of hyper/hypothyroidism, Obstructive symptoms (dysphagia, difficulty in breathing). | The Thyroid profile may be normal (euthyroid, hyper or hypothyroid). |
| Thyroid Nodules | Incidentalomas (picked incidentally when investigation for other unrelated conditions).<br>Single thyroid nodules<br>Multiple thyroid nodules<br>Toxic nodules<br>Non-Toxic nodules | Asymptomatic<br>Features of hyper/hypothyroidism, Obstructive symptoms (dysphagia, difficulty in breathing). | Can be benign or malignant.<br>Requires thyroid ultrasound and Fine Needle Aspiration (FNA).<br>Carry a risk of thyroid cancer. |

**Management of thyroid diseases**

Patients with thyroid diseases should be managed by a multidisciplinary team comprising by either a physician or an endocrinologist, surgeon (General or ENT), radiologist and nuclear physicist. Table 5.8 summarises the management of thyroid diseases.

**Table 5.8:Summary of management of thyroid diseases**

| Thyroid disease | Management | Remarks |
|---|---|---|
| Hyperthyroidism/ thyrotoxicosis | -Anti-thyroid drugs (carbimazole, propylthiouracil (PTU)<br>-Beta-Blockers (Propranolol, bisoprolol)<br>-Surgery (thyroidectomy)<br>Radioiodine therapy (RAI) | Carbimazole: metabolized to methimazole. Methimazole is not available in the country.<br>PTU is safe in pregnancy as carbimazole is teratogenic and contraindicated.<br>Treatment lasts from 12-18months. Monitor TBC for agranulocytosis and LFTs for hepatotoxicity, TSH/free T4 and adjust dose.<br>RAI: If no response on ATD, in non-pregnant adults. Avoid in active thyroid eye disease<br>Surgery: For those who relapse, no response to ATD, RAI is contraindicated or unavailable, presence of goiter with obstructive symptoms, when malignancy is suspected of confirmed. Levothyroxine replacement therapy should be given post total thyroidectomy. |
| Hypothyroidism | -Levothyroxine (T4) Replacement Therapy | 25-125mcg per day or 1.6mcg/kg/day<br>Dose adjusted according to the TSH/free T4.<br>Lower doses for older patient |
| Thyroid nodules | -Non-toxic nodules<br>No treatment if asymptomatic<br>-Aspiration of thyroid cysts<br>Surgery if obstructive symptoms are present<br>-Toxic Nodules<br>Anti-Thyroid drugs (ATDs)<br>Radioactive iodine<br>Surgery | Always rule out thyroid cancer by thyroid ultrasound done by a radiologist and a FNA-c and use the appropriate radiological and cytology classification in place. |

## 5.2.2 PITUITARY GLAND DISORDERS

The pituitary gland is located in the pituirary fossa in the base of the skull. It has two lobes that secrete stimulating hormones. When you suspect a patient has a pituitary gland disorder, refer to a physician or endocrinologist for further evaluation and management. These are summarized in the table 5.9.

**Table 5.9: Summary of pituitary gland disorders**

| **Anterior Pituitary Gland Hormones** | | | |
|---|---|---|---|
| **Hormone** | **Target Organ** | **Function** | **Disorders** |
| Prolactin hormone (PRL) | Mammary glands | Milk Excretion | Hyperprolactinemia Prolactinomas (No disorder for PRL deficiency). |
| Growth Hormone (GH) | Peripheral tissues | Growth and development | GH excess (Acromegaly) GH Deficiency |
| Thyroid Stimulating Hormone (TSH) | Thyroid Gland | Secretion of thyroid hormones | Hyper/Hypothyroidism |
| Gonadotropin Hormones; Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH) | Gonads (testes, ovaries) | Reproduction | Infertility, Menstrual abnormalities |
| Adrenocortical Stimulating Hormone ACTH) | Adrenal Cortex | Secretion of Cortisol | ACTH/Cortisol excess: Cushing's Disease Cortisol deficiency: Addison's Disease |
| **Posterior Pituitary Gland Hormones** | | | |
| Anti-Diuretic Hormone (ADH) | Adrenal cortex | Fluid balance | Diabetes Insipidus |
| Oxytocin | Mammary Glands | Milk let Down | |

**Investigations**

- Investigations for pituitary gland disorders
- Hormone Profile
- Imaging: MRI/ CT scan
- Management: drugs or surgery.

## 5.2.3 ADRENOCORTICAL DISORDERS

The adrenal gland is an endocrine gland located are at the suprior pole of each kidney. It is divided into two layers; The outer layer Cortex and the inner layer Medulla. The Cortex has 3 zones that secretes different hormones. The medulla produces catecholamines**.**

Table 5.10 provides a summary of the adrenocortical disorders.

**Table 5.10: Summary of adrenocortical disorders**

| Layer | Zone | Hormone | Function | Disorder |
|---|---|---|---|---|
| Cortex | Zona Glomerulosa | Aldosterone | Salt regulation | Hyperaldosteronism (Conn's Syndrome) Hypoaldosteronism |
| | Zona Fasciculata | Cortisol | Glucose regulation | Cortisol Excess/ Hypercortisolism Cortisol Deficiency (Addison's Disease) |
| | Zona Reticularis | Adrenal Androgens | Sex hormones | Androgen hormone excess (Hyperandrogenemia) hirsutism, masculinization, ambiguous genitalia, Congenital Adrenal Hyperplasia Androgen hormone deficiency (feminization |
| Medulla | | Catecholamines (adrenaline, noradrenaline) | Flight and flight hormones/neurotransmitters | Phaeochromocytomas and paragangliomas |

## 5.2.4 GLUCOCORTICOID EXCESS

This is also called hypercortisolism or Cushing's Syndrome.
It arises from a functional adrenal cortex tumor (malignant or benign) producing excess cortisol. Exogenous use of steroids can also lead to Cushing's Syndrome. Cushing's Diseases arise from adenocorticotropic hormone producing adenoma from the anterior pituitary gland.

**Clincal Features:**
Weight gain, moon-facies, hypertension, skin striae, hirsutism, acne, buffalo hump, easy bruisability, hyperpigmentation, glucose intolerance, plethora, proximal muscle weakness, menstrual dysfunction, osteopenia, hypokalaemia, and metabolic alkalosis.

**Investigations**
Rule out exogenous use of steroid use. Three sets of tests are done:

- **Tests to confirm excess cortisol production.**
  - 24-hour urinary free/unfractionated cortisol levels
  - Serum cortisol levels ( cortisol is secreted in a circadium fashion with the highest secretion being in the morning hours and the lowest/nadir at midnight. So mornining cortisol (8-9am) or midnight serum cortisol is preferred. Avoid random cortisol measurements.
  - Late-night Salivary cortisol levels (11pm to midnight).

- **Tests to confirm the loss of negative feedback mechanism**
  - Low-Dose Dexamethasone Suppression Test (1mg-DST).
  - High-Dose Dexamethasone Suppression Test (8mg-DST)

- **Tests for localisation**
  - Imaging: Pituitary imaging, Adrenal gland imaging

Other tests include: urea and electrolytes (sodium, potassium), blood sugar

**Management**
This should be guided by the endocrinologist and the surgical team.

Goals of therapy for Cushing's Syndrome
- Remission of hypercortisolism or cortisol excess
- Reversal of the clinical picture of Cushing's Syndrome
- Management and prevention of comorbidities and clinical complications
- Disease remission and prevention of recurrence

For those with exogenous use of steroids, gradual withdrawal of the steroid can be done. Avoid sudden withdrawal of steroids due to the risk of triggering an adrenal crisis. Surgical resection of the tumor is the first line of therapy in cases of Cushing's Disease with high remission (80-90%) and cure rates.

**Medical therapy**
Second line therapy for those not fit for surgery. It can also be used pre-operatively for those with severe disease. The drugs used in Cushing's Syndrome management can be classified as follows;

- ***Adrenal Directed Drugs*: inhibitors of cortisol synthesis/ steroidogenesis inhibitors**
  - Ketoconazole
  - Metyrapone
  - Mitotane (also has additional cytotoxic effects). Useful in adrenocarcinomas.
  - Etomidate
  - Osilodrostat

- ***Pituitary Directed Drugs*: inhibitors of adenocorticotropic horome secretion from corticotrophin adenomas (pituitary cells)**
  - Cabergoline (dopamine agonist
  - Pasireotide (Serotonin Receptor Ligand (SRL)/ SSAs

- ***Receptor Directed Drugs*: glucocorticoid receptor antagonist**
  - Mifepristone

## 5.2.5 ADRENAL INSUFFICIENCY (AI)

Defined as an endocrine disorder where the adrenal glands are incapable of producing glucocorticoids and minerolocorticoids. It is due to a primary destruction of the glands (Primary AI) or lack of stimulation of their secretion due to deficient adenocorticotrophic hormone (ACTH) from anterior pituitary or corticotrophin-releasing hormone from the hypothalamus (Secondary AI).

It can present acutely termed as addisonian's / adrenal crisis or chronic adrenal insufficiency. Primary adrenal insufficiency (Addison's Disease): Condition affecting the adrenal gland; infections (TB, HIV), surgery, tumors, enzyme defects (congenital adrenal hyperplasia), autoimmune conditions.

*Secondary:* Disorders of the pituitary gland leading to ACTH deficiciency (pituitary surgery, pituitary tumors, drugs, infiltrative conditions, traumatic brain injury). Exogenous use of steroids will suppress the ACTH production whereas their sudden withdrawal precipitates an adrenal crisis.

*Tertiary:* Conditions affecting the hypothalamus production of corticotropin releasing hormone. Hypothamalus tumor, infiltrative diseases, surgery, traumatic brain injury.

**Clinical Features**

Acute AI: This is a medical emergency associated with significant mortality if not identified and promptly treated. Common clinical presentations; weakness, weight loss, diarrhoea, vomiting, hypotension, darkening of skin palms, and recent scars. It is important to note that therapy for suspected AI should not be delayed pending confirmatory tests.
In acute ill patients, treatment should be commenced with immediate effect and the tests done once stable.

**Investigations**

- **Serum cortisol**
  - Random cortisol: levels done at any time of the day > 400nmmol/l makes AI unlikely. Useful as a screening test.
  - Morning cortisol: cortisol has a circadium rhythm with the highest secretion occurring in the morning. An 8am cortisol level < 100nmol/l strongly suggests AI.

- **ACTH Stimulation Test (Synacthen test)**
  - 250mcg of synthetic ACTH injected parenterally having taken a basal cortisol sample, followed by a cortisol level at 30 and 60 minutes. A value > 500nmol/l excludes AI.
  - This test should not be performed for Secondary AI of recent onset (less than 4-6 weeks) before the adrenals have atrophied.

- **Serum ACTH levels**
  - To differentiate between primary and secondary AI (ACTH dependent Vs ACTH-independent). The leves are high in primary AI and low/normal in secondary AI.

- **Testrs for investigating the specific underlying pathologies**
  - CT scan abdomen: hemorrhage, infiltrative and infective pathologies
  - 21-hydroxylase antibodies: autoimmune addison's disease
  - Pituitary MRI: infiltrative, tumors, hemorrhage
  - Anterior pituitary hormones (TSH, LH, FSH, Prolactin).

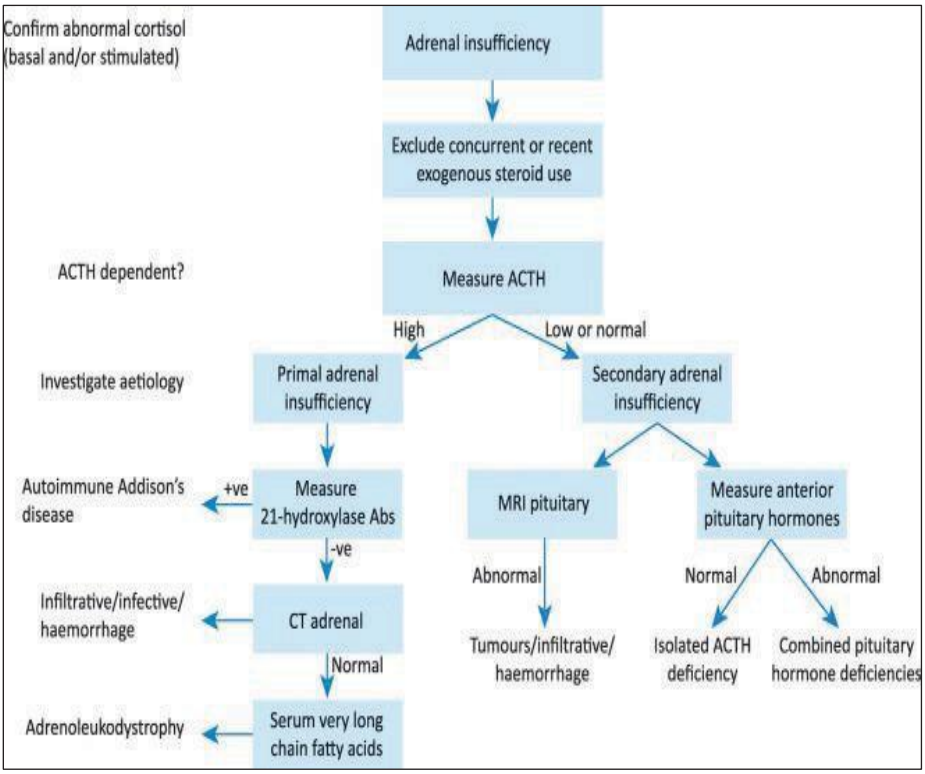



**Figure 5.2: Algorithm adapted from Pazderska (2017) Algorithm for the initial investigation of adrenal insufficiency. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6297573/bin/clinmed-17-3-258fig1.jpg.**

## 5.2.6 ADRENAL CRISIS

Defined as a medical emergency with hypotension, abdominal symptoms and laboratory abnormalities requiring emergency treatment due to acute adrenal insufficiency.

**Clinical Features**

- Symptoms: severe weakness, abdominal pains, (like acute abdomen), nausea, vomiting, syncope, back pain, confusion.
- Signs: Low blood pressure, abdominal tenderness/guarding, reduced level of consciousness, delirium
- Laboratory tests: hyponatremia, hyperkalemia, hypoglycemia (especially in children), hypercalcemia.

**Precipitating factors**

Infections, surgery, trauma, emotional stress, pregnancy, strenuous exercise, thyrotoxicosis, drugs (anti-adrenals and those that increases cortisol metabolism), gastrointestinal symptoms, hot weather, dehydration, alcohol intoxication.

**Management of adrenal crisis**

- Symptoms: severe weakness, abdominal pains, (like acute abdomen), nausea, vomiting, syncope, back pain, confusion.
- Immediately a diagnosis of adrenal crisis is suspected, patient should receive parenteral (intravenous or intramuscular) hydrocortisone (100mg stat for adults and 50mg/m2 for children).
- This is followed with appropriate fluid resuscitation. (normal saline 4-6 liters in the first 24 hours. Avoid rapid correction of hyponatremia due to the risk of central pontine mylenosis/ osmotic demyelination syndrome). ICU admission is warranted.
- 200mg (adults) and 50-100mg (children) of Hydrocortisone per day should then be given as a continuous intravenous infusion or given as a bolus injection 6 hourly till full recovery which can be 2 to 3 days.
- Prednisolone/ methylprednisolone is the next alternative if hydrocortisone is not available.
- Dexamethasone is not preferred because it has no minerolocorticoid effect and can only be given in the absence of hydrocortisone and prednisolone.
- Search for the precipitating factors should be done. Empirical antibiotics can be given if infection is suspected.
- Gradually taper the steroids.

**Long-term advice to patients on steroids**

Patient education: this is important in order to prevent the high risk patient from going into a adrenal crisis.

- Sick days rule: increase the glucocorticoid dose to 2-3 times during a minor illness.
- Stress dose is required in the event of stressful situations like trauma, surgery, major procedures, and severe illness.
- Signs and symptoms recognition of AI; including; nausea, vomiting, abdominal pain, hypoglycemia, hypotension, weight loss among others.
- Advise on importance of wearing medical alert identifications like bracelets or necklaces (''Adrenal Insufficiency- needs steroid!').
- Equip patient at risk with steroid emergency cards.
- Provide an emergency kit (100 mg hydrocortisone sodium succinate for injection or dexamethasone 4 mg, prednisolone suppositories, vials of sterile 0.9% normal saline and syringes) and teach injection technique.

## 5.2.7 PHAEOCHROMOCYTOMAS

Phaechromocytomas are catecholamine-secreting endocrine tumors arising from chromaffin cells in the adrenal medulla. The rule of 10: 10% prevalence, 10% bilateral, 10% extra-adrenal, 10% malignant, 10% in children, 10% familial and 10% recurrent. Associated with: Multiple Endocrine Neoplasia Syndrome (MEN 2A/2B), Von Hipplel Lindau (VHL) syndrome and neurofibromatosis.

**Clinical Features**

- Episodic/ labile hypertension (resistant hypertension) and the classic triad of episodic headache, diaphoresis and palpitations.
- Others are: tremors, fatigue, weight loss, anxiety attacks, hot flushes, nausea, pallor, orthostatic symptoms

**Investigations**

- Biochemical evaluation with plasma free metanephrines and urinary unfractionated metanephrines.
- Imaging studies: CT Scan is the first choice for tumor location due to its excellent spatial resolution as compared to MRI scan.
- Metastatic disease: 18F-FDG PET/CT Scan, $^{123}$I-metaiodobenzylguanidine (MIBG) scintigraphy for metastatic lesions.
- Genetic Testing: multidisciplinary team should be involved.

**Management**

- Patient should be managed by a multidiscilplicary team; physician/endocrinology, surgeon.
- Surgery offers cure.
- Medical treatment:
  - Should be started 1-2 weeks pre-operatively to minimize perioperative cardiovascular complications.
  - **Phenoxybenzamine** (*a non-competitive and non-selective alpha-receptor antagonist*) is the treatment of choice.

- **Doxazosin** is another option and it is a competitive, short-acting selective alpha-1 receptor blocker.
- Malignant Phaechromocytoma: CVD-Averbuch Regimen: Cyclophosphamide, Vincristine, Darcarbazine.

# 6. Gastrointestinal Conditions

## 6.1 Diarrhoeal Diseases

**Diarrhoea is defined as the occurrence of at least 3 loose or watery stools in a day.**

**Classification**

There are three categories of diarrhoeal diseases:

- ***Dehydration:*** This is the major cause of death from diarrhoea. Management is aimed primarily at evaluation, prevention, and treatment of dehydration.
- ***Dysentery:*** This is bloody diarrhoea.
- ***Persistent diarrhoea:*** This is diarrhoea that lasts for 14 days or more.

**Clinical Evaluation of Dehydration**

Refer to Table 6.1 for a summary of the signs of dehydration, whether severe, moderate, or mild.

### 6.1.1 REHYDRATION PROTOCOL

**In using the protocol summarized in Table 6.2, bear in mind:**

- The volumes indicated are guidelines only.
- Rehydration must be evaluated in terms of clinical signs, not in terms of volume of fluids given.
- If necessary, the volumes given below can be increased or else the initial high rate of administration can be maintained until there is clinical improvement.
- Periorbital oedema is a sign of fluid overload in infants or hypernatreamia in those on oral rehydration salts (ORS).
- Maintenance therapy should begin as soon as signs of dehydration have resolved, but not before.

### 6.1.2 FLUID MAINTENANCE THERAPY

- Fluid to be given after correction of dehydration.
- Adapt rehydration treatment to the clinical status of the patient.

**Note the following:**

- Other liquids such as plain water, rice water, uji, mala, etc., can also be given.
- ORS should constitute about two-thirds of the fluid intake until diarrhoea ceases.
- Thirst is the best guide for maintenance fluid therapy in older children and adults. Let them drink as much ORS (and other liquids) as they desire.
- Give fresh fruit or mashed bananas to provide potassium.
- Return to health worker if no improvement in 3 days or if patient develops the following: many watery stools, very poor drinking, repeated vomiting, fever, marked thirst, and/or blood in stool. Also if the caregiver is not happy with the condition.

**Table 6.1: :Clinical signs of dehydration**

| Clinical feature | Mild dehydration | Moderate dehydration (2 signs present) | Severe dehydration (≥2 signs present) |
|---|---|---|---|
| General appearance: Older children and adults | Thirsty, alert | Thirsty, alert | Generally conscious, anxious, cold extremities, clammy, cyanosis, wrinkled skin of fingers, muscle cramps, dizzy if standing |
| Pulse | Normal | Rapid | Rapid, thready, sometimes absent |
| Respiration | Normal | Deep, sometimes rapid | Deep and rapid |
| Systolic BP | Normal | Normal | Low, sometimes unmeasurable |
| Skin elasticity | Normal: fold of pinched skin disappears at once | Decreased | Fold disappears very slowly (>2 seconds) |
| Eyes | Normal | Sunken | Severely sunken |
| Tears | Present | Absent | Absent |
| Mucous membranes (test mouth with a clean finger) | Moist | Dry | Very dry |
| Urine output | Normal | Reduced, urine dark | Anuria, empty bladder |
| % of body weight loss | 1–5% | 6–9% | 10% or plus |
| Estimated fluid deficit | 10–50ml/kg | 60–90ml/kg | 100ml/kg |

**Table 6.2: Rehydration protocol**

| Degree of dehydration | Age | Type of liquid | Volume to give | Rate |
|---|---|---|---|---|
| Mild | All | ORS | 50ml/kg | In 4 hours |
| Moderate | All | ORS | 100ml/kg | In 4 hours |
| Severe | Older children and adults | Hartmann's solution, Ringer's lactate | 110ml/kg | In 4 hours: at first as rapidly as possible until a radial pulse is palpable |

NOTES: (a) Initially, adults can usually ingest up to 750ml of ORS/hour, and older children 300ml/hour. (b) If Ringer's lactate or Hartmann's solution are not available, use:

- Half-strength Darrow's solution
- Normal saline with sodium bicarbonate and potassium chloride added
- Normal saline diluted to half-strength with 5% glucose (dextrose)

**None of these solutions is as effective as Ringer's lactate or Hartmann's solution.**

## 6.1.3 MAINTAINING NUTRITION

It has been shown that there is no physiological reason for discontinuing food during bouts of diarrhoea and that continued nutrition is beneficial. Continued feeding should be encouraged.

## 6.1.4 PHARMACOLOGICAL MANAGEMENT

**Note that 50–60% of acute gastroenteritis is viral. Also note the following:**

- Always treat the fever and consider other diseases associated with diarrhoea (e.g., malaria, otitis media,pneumonia).
- Antimicrobial drugs should be used only as follows:
  - Antibiotics only for dysentery and suspected cholera with severe dehydration.
  - Antiprotozoal drugs(e.g.,metronidazole) for suspected amoebiasis only after antibiotic treatment of bloody diarrhoea has failed or faeces shows trophozoites of E.histolytica.
  - Antiparasite drugs for giardiasis when diarrhoea has lasted over 14 days and cysts or trophozoites of giardia are seen in faeces.
- Antibiotics for specific intestinal infections are listed in Table 6.3.

**Table 6.3: Antibiotics used in the treatment of diarrhoea**

| Infection | Management |
|---|---|
| **Cholera:** Very profuse watery diarrhoea (rice-water stools),frequent vomiting | **Doxycycline:**100mg BDx7days<br>***OR***<br>**Erythromycin:** 250mg QDS x 5 days |
| **Shigella dysentery:** Blood&mucus stools, cramps,tenesmus,fever | **Cotrimoxazole:**960mg BDx5days in<br>**Amoxicillin:**500mg QDSx5days<br>Tabs Ciprofloxacin 500mg OD x 5 days |
| **Intestinalamoebiasis:**<br>Acute amoebic dysentery:Aswith shigella, but usually no fever(except amoebic liver abscess) | **Metronidazole:** 800mg TDS x5–10 days<br>***OR***<br>Tinidazole 2g OD for 3days |
| **Acutegiardiasis:**Prolonged diarrhoea, often marked eructation (belching),flatulence | **Metronidazole:** 800mg TDS x 5–10days<br>***OR***<br>Tinidazole 2g OD for 3 days |

### 6.1.5 PREVENTION OF DIARRHOEAL DISEASES

- Proper sanitation: Provision of safe drinking water in sufficient quantities and disposal of faeces.
- Hygiene during food preparation: Remember the 4C's–Clean hands, Clean food, Clean utensils, Clean storage.
- Cholera vaccine.

## 6.2 Gastritis

This is an acute ulceration of the stomach, usually multiple lesions, non-recurrent and self-limiting.

**Aetiology**
Drugs (NSAIDs), alcohol, acute stress associated with massive burns, head injuries

**Clinical Features**
Epigastric pain with or without vomiting. May follow ingestion of drugs and herbal preparations. Heartburn may be a feature. Examination reveals tenderness in the epigastrium and the regions around it.

**Investigations**
Not always necessary if cause is obvious. Otherwise barium meal and endoscopy.

**Management**
- Treat the primary disease, e.g., head injury, renal failure.
- Avoid drugs known to cause ulceration.
- Magnesium trisilicate tabs 2–4 QDS or frequently ***OR*** mistantacids 30ml 1 hour and 3 hours after meals. Adjust dose according topain.
- Role of triple therapy (see Section 6.4, below, on peptic ulcer disease).

## 6.3 Gastro-Oesophageal Reflux Disease (GORD)

This is the physiological process characterized by effortless movement of gastric contents from the stomach to the oesophagus. Symptoms and pathology occur when the oesophageal mucosa has excessive contact with gastric contents as a consequence of continual failure of anti-reflux mechanism.

**Clinical Features**
- Heart burn is the characteristic symptom of GORD, with or without regurgitation of gastric contents into the mouth.
- Pain on swallowing hot drinks or alcohol.
- Oesophagitis causes bleeding, which can be massive.
- Peptic stricture causes gradually progressive dysphagia.
- Aspiration of gastric contents resulting in aspiration pneumonia.
- Oesophageal ulcers cause same type of pain as gastric or duodenal ulcer.

**Diagnosis**

- Detailed history points to the diagnosis
- Barium swallow, will show oesophagitis, ulcers or stricture.
- Oesophagoscopy: With oesophageal washing or biopsy confirms diagnosis.

**Management – Uncomplicated GORD**

- Elevate head of bed 6 inches.
- Advise to avoid strong stimulants of acid production (e.g., coffee, alcohol, fatty foods, smoking).
- Take antacids 30ml 1 hour after meals and at bed time.
- Give H2 receptors antagonists and proton pump inhibitors (see peptic ulcer).
- Give cholinergic agonists (e.g.,metoclopramide 10mg PO 30 minutes before meals and at bedtime).

## 6.4 Peptic Ulcer Disease

It is the ulceration of gastroduodenal mucosa that has tendency to be chronic and recurrent. Can be duodenal or gastric.



**Clinical Features**

- Duodenal ulcer:
  - Epigastric pain, typically at night and when hungry
  - May present for the first time with complications (see later in this section)
  - Wide individual variation in symptoms and food that give pain
  - 95% of duodenal ulcers are caused by Helicobacter pylori (H.pylori).

**Gastric ulcer:**

- Epigastric pain, worse with food
- Other features as in duodenal ulcer above.

**Investigations**

- Stool for occult blood
- Barium meal.
- Upper GIT endoscopy, where available and biopsy of the gastric mucosa for H. pylori.

**Management**

- Avoid any foods that in the patient's experience cause pain.
- Avoid obviously acidic foods, e.g., cola drinks.
- Limit alcohol intake and smoking.
- Advise bed rest in acute attacks.
- Avoid gastric irritating drugs (NSAIDs).
- Give magnesium based antacids or combined magnesium-aluminium compounds,liquid preferred. Maximum dose is 6 tablets a day. Adjust dose to limit pain. If no response; give cimetidine 800mg or ranitidine 300mg nocte for 4–6 weeks then 400mg or 150mg, respectively, as maintenance.

♦ Aim for H. pylori eradication by triple therapy:
- Regime I:
  – Omeprazole 20mg BD 14 days
  – Clarithromycin 500mg BD 14 days
  – Amoxicillin 1g BD 14 days
- Regime II:
  – Omeprazole 20mg BD 14 days
  – Amoxicillin 1g BD 14 days
  – Metronidazole 400mg TDS 14 days

Other PPI such as esomeprazole can be used instead of omeprazole.
♦ Admit for all the above management.
♦ Indications for surgery in peptic ulcer disease:
- Intractable haemorrhage of more than 5 units of blood in 24 hours
- Recurrent bleeding after non surgical management during same hospitalization
- Perforation
- Penetration to the pancreas
- Intractable ulcer pain
- Suspicion of malignancy, especially in gastric ulcers.
- Gastric outlet obstruction.

**Complications**
Haematemesis, obstruction, perforation, penetration to the pancreas, malignancy.

## 6.5 Upper GIT Bleeding

Bleeding from the GIT above the ligament of Treitz.

**Aetiology**
♦ Oesophageal varices
♦ Gastritis and gastric ulcers
♦ Duodenal ulcers
♦ A-V malformation
♦ Malignancies – Stomach and oesophagus
♦ Mallory-Weiss syndrome
♦ Polyps

**Clinical Features**
Vomiting of fresh bright blood or coffee-ground vomitus (haematemesis). Forceful vomiting followed by haematemesis suggests gastroesophageal junction tear.

Excessive alcohol intake or ingestion of anti-inflammatory drugs may suggest erosive gastritis; previous epigastric pain suggests peptic ulcer. In massive haemorrhage, blood may appear per rectum.

**Investigations**
♦ Haemoglobin, platelet count

- Investigate as per cause, if obvious, e.g., liver function test in liver disease
- Barium swallow/meal after patient is stable
- Endoscopy if available.

**Management**

- Set up large IV line, start infusion of normal saline.
- Group and cross-matchat least 3 units of blood.
- Perform nasogastric suction to assess blood loss.
- Infuse fluids to maintain normal pulse, blood pressure, and urine output and substitute with whole blood as soon as possible.
- Assess any further loss of blood as evidenced by :persistent tachycardia, postural hypotension, continuing haematemesis.
- Admit all patients with haematemesis.

## 6.6 Lower GIT Bleeding

This may be frank bleeding (haematochezia) or occult bleeding, depending on the cause.



**Common Causes**

- Haemorrhoids
- Anal fistula and fissures
- Tumours:
  - Benign: Polyps, leiomyoma,fibromas
  - Malignant
- Infections
  - Bacterial: shigella, campylobacter,salmonella
  - Protozoa:amoebiasis
  - Parasite:schistosomiasis
- Trauma
- Angiodysplasia
- Inflammatory bowel disease (Crohn's disease and ulcerative colitis)
- Diverticular disease
- Bleeding disorders

**Investigations**

- Haemogram and ESR
- Stool for microscopy,C&S
- Double contrast bariumenema
- Proctoscopy, sigmoidoscopy, colonoscopy, and biopsy
- Coagulation screen

**Management**

- Group and cross match if necessary.
- Treat the cause.
- Refer to the surgeons any suspicious rectal bleeding.

**— No physical examination is complete without a rectal examination.**

## 6.7 Pancreatitis

Pancreatitis is an inflammation of the pancreas. There are two forms of pancreatitis:

- Acute pancreatitis is a sudden and short bout of inflammation
- Chronic pancreatitis is ongoing inflammation.

### 6.7.1 ACUTE PANCREATITIS

**Clinical features**

- Moderate to severe upper abdominal pain that may spread to the back.
- Pain that comes on suddenly or builds up over a few days.
- Pain that worsens when eating and relieved when leaning forward lying in a fetal position.
- Swollen, tender abdomen.
- Nausea and vomiting.
- Fever.
- Increased heart rate.

### 6.7.2 CHRONIC PANCREATITIS

- Constant, sometimes disabling pain that spreads to the back.
- Unexplained weight loss.
- Foamy diarrhea with visible oil droplets (steatorrhea).
- High blood sugar, if insulin-producing pancreas cells are damaged.

**Diagnosis**

- Ultrasound or computed tomography
- Oral glucose tolerance test
- Endoscopy
- Magnetic resonance imaging (MRI)
- Stool tests
- Endoscopic retrograde cholangiopancreatography

**Treatment**

- Intravenous (IV) hydration
- Analgesics
- Antibiotics
- Surgical intervention

## 6.8 Irritable Bowel Syndrome

This is a common chronic disorder that affects the large intestine.

**Clinical features**

- Cramping,
- Abdominal pain,
- Bloating,
- Diarrhea or constipation, or both.

**Investigations**

- Clinical Presentation
- Stool Exam to rule out infection
- Lactose intolerance tests
- Colonoscopy
- Endoscopy
- X-ray or CT scan to R/O other abdominal conditions

**Management**

- There is no definitive treatment for the condition.
- Treatment aimed at symptomatic relief.
- Lifestyle remedies aimed at relieving the condition include;
  - Participating in regular physical exercise
  - Cutting back on caffeinated beverages that stimulate the intestines
  - Eating smaller meals
  - Minimizing stress (talk therapy may help).
  - Taking probiotics ("good" bacteria normally found in the intestines) to help relieve gas and bloating. (Live bacteria and yeasts that are good especially for the digestive system.)
  - Avoiding deep-fried or spicy foods.

## 6.9 Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is a group of disorders that cause chronic inflammation (pain and swelling) in the intestines. IBD includes Crohn's disease and ulcerative colitis.

**Causes of IBD**

- Genetics: family history of the disease
- Immune system response: food mistaken for foreign substances
- Environmental triggers: exposure to an environmental trigger like smoke, stress, medication use, depression etc.

**Investigations**

- Upper endoscopy
- Colonoscopy
- Sigmoidoscopy
- Full blood count
- Inflammatory Marker Tests (C-Reative Protein)
  - Liver function tests to rule out primary sclerosing cholangitis

- Urea and Electrolytes to rule out dehydration in acute diarrhoea
- Stool test

**Management**

- Mild disease requires only symptomatic relief and dietary manipulation.
- Mild to moderate disease can be managed with 5-aminosalicylic acid compounds, including olsalazine and mesalamine.
- Mesalamine enemas and suppositories are useful in treating proctosigmoiditis.
- Metronidazole may be required in patients with Crohn's disease.
- Corticosteroids are beneficial in patients with more severe symptoms
- Immunosuppressant therapy may be considered in patients with refractory disease that is not amenable to surgery.
- Inflammatory bowel disease in pregnant women can be managed with 5-aminosalicylic acid compounds and corticosteroids.

## 6.10 Ascities

This is an abnormal accumulation of fluid within the (peritoneal) cavity.

**Causes**

- Liver cirrhosis
- Cancer within the abdomen
- Congestive heart failure
- Tuberculosis.
- Renal failure
- Chronic (long standing) pancreatitis

**Clinical Presentation**

- Abdominal pain
- Bloating
- Shortness of breath
- An abnormally enlarged belly
- Nausea
- Less hungry feeling than usual
- Tiredness/exhaustion
- Breathlessness
- Urinary urgency and constipation

**Investigations**

- Physical examinations
- Liver function tests
- Urea, electrolytes and creatinine
- Abdominal ultrasound
- CT abdomen

**Management**

- Treatment of the underlying condition
- Reliving the symptoms; Ascetic tap, diuretics

- Supportive measures including; reduce salt intake, amount of fluids intake, alcohol intake etc.

## 6.11 Cholecystitis

Cholecystitis is inflammation of the gallbladder that occurs most commonly because of an obstruction of the cystic duct by gallstones arising from the gallbladder (cholelithiasis).

**Clinical features**

- Tenderness in the abdomen when it's touched. Pain begins in the mid to upper right abdomen and may spread to the right shoulder blade or back.
- Nausea and bloating.
- Vomiting.
- Fever above 100.4 F (38 C)
- Chills.
- Abdominal pain that gets worse when taking a deep breath.
- Abdominal pain and cramping after eating – especially fatty foods.
- Jaundice

**Investigations**

- Abdominal ultrasound
- MRI
- Abdominal CT scan

**Management**

- Fasting, to rest the gallbladder
- IV fluids to prevent dehydration.
- Analgesics.
- Antibiotics to treat infection.
- Surgery (cholecystectomy)

## 6.12 Viral Hepatitis

**This is liver inflammation caused by viruses, including hepatitis A,B,C,D (delta), and E.**

- Hepatitis A and D are transmitted through the fecal oral route; the rest are by blood and blood products. The hepatitis A virus causes an acute hepatitis, which is usually self-limiting, while the rest can go to the chronic stage.
- Chronic B and C infections can lead to cirrhosis and hepatocellular carcinoma.

**Clinical Features**

Symptoms and signs of acute hepatitis include yellowness of eyes, fever, nausea, anorexia, vomiting, right upper quadrant pain. Physical examination reveals upper abdominal tenderness.

**Investigations**
- For hepatitis A
  - 1g specific anti-HAV titres
- For hepatitis B
  - Hepatitis B surface antigen
  - 1g anti-hepatitis B core antigen
- For hepatitis C
  - Hepatitis C antigen
- Liver function tests

**Management**
- General: supportive of liver function.
- Specific:
  - Hepatitis B and Hepatitis C immunoglobulins.
  - Interferon alpha 2b pegelated; interferon alpha 2a nucleoside
  - Tenofovir, lamivudine, entecavir
    adefovir,telbivudine
    NB: Use dual therapy for Hepatitis C.

**Prevention**
- Hygiene
- Safer sex practices
- Vaccination
- Precaution in handling biological fluids and laboratory equipment
- Vaccination against hepatitis A and B



## 6.13 GIT Parasitic Infestations

### 6.13.1 AMOEBIASIS

This is an infection usually of the colon caused by Entamoeba histolytica. Most cases can be prevented if at level 1 strict attention is paid to personal hygiene, availability of clean, uncontaminated water, environmental sanitation and waste disposal.

**Clinical Features**
Amoebic dysentery. Amoebic liver abscess. Amoebiasis and "vague" abdominal complaints. Asymptomatic cyst carrier.

**Investigations**
- Stool for microscopy–trophozoites with ingested RBCs and cysts of entamoeba histolytica in amoebic dysentery
- Chest x-ray
- Full haemogram
- Liver ultra-sound scan
- Needle aspiration for microscopy in amoebic liver abscess

**Management**

- Amoebic dysentery:
  - Correct dehydration
  - Give metronidazole 400mg TDS for 5 days
- Amoebic liver abscess
  - Give metronidazole 750g OD for 3–5 days
  - Refer pointing abscesses for surgical drainage
- Amoebiasis and "vague" abdominal complaints:
  - Where amoebiasis is common, there is a tendency to blame any abdominal complaints on amoeba. Usually these patients have cysts in stool but no evidence of invasive disease, e.g., ingested RBC in trophozoite. Exclude other causes of abdominal pain.
- Asymptomatic cyst carriers:
  - Treat cyst carrier only if patient is a food handler. Use diloxanide furoate 500mg twice daily for ten days, or a combination of diloxanide furoate with metronidazole (entamizole) 1 tab 3 times a day for 10 days.

**Prevention**

- Provision of safe drinking water and sanitary disposal of faeces are important preventive measures.
- Regular examination of food handlers and appropriate treatment when necessary.

## 6.13.2 INTESTINAL WORMS

These infections comprise a large group of parasitical cestodes, schistosomes, flukes, nematodes, and filarial worms. Only nematodes are dealt with in this section. They include hookworm disease, ascariasis, enterobiasis, trichuriasis, trichostrongyliasis, anisakiasis, capillariasis, and gnathostomiasis. Still, only the common ones are highlighted. Table 6.5 (overleaf) summarizes the most common worm infections with their clinical features and the method of detection.

**Management**

Management of the more common intestinal worms is summarized in Table 6.4.

**Table 6.4: Treatment regimens for common intestinal worms**

| Worms | Adult treatment dosages |
|---|---|
| Ascaris lumbricoides(roundworms) | Albendazole 400mg STAT ***OR*** Levamisole 2.5mg/kg as a single dose |
| Hookworms | Albendazole 400mg STAT ***OR*** Levamisole 2.5mg/kg as a single dose |
| Trichuris trichiura(whipworms) | Albendazole 400mg STAT |
| Strongyloides stercoralis | Albendazole 400mg BD×3days |
| Enterobius vermicularis(pinworms) | Mebendazole 500mg STAT; Levamisole 2.5mg/kgas a single dose REPEAT AFTER 10 days Taenia |
| saginata(beeft apeworms) | Praziquatel 25mg/kg/dose TDS for 2 days; albendazole 400mg once daily for 3 days |

**Table 6.5:Common intestinal worms – Features and investigations**

| Worms | Clinical features | Investigations |
|---|---|---|
| Ascaris lumbricoides (roundworms): Large round, cream coloured worms that live in the small intestines | • Infection by swallowed embryonated eggs<br>• Loef?er'ssyndrome<br>• Mild bouts of recurrent colic<br>• The mother has seen the worm in stool or vomitus<br>• Complications such as obstruction, vomiting may occur | Stool for ova |
| Hookworms | • "Grounditch"<br>• Features of anaemia (iron deficiency) | Stool for ova<br>Haemogram |
| Trichuris trichiura (whipworms) | • Diarrhoea with blood<br>• Rectal prolapse<br>• Anaemia<br>• Wasting | Stool for ova<br>Worms may be seen adhering to rectal mucosa |
| Strongyloidess tercoralis | Most infections are asymptomatic but the following may occur:<br>• Larva currens(buttocks)<br>• Soiling of inner wear with stool<br>• Hyperinfection syndrome<br>• Diarrhoea<br>• Gram-negative septicaemia<br>• Bacterial peritonitis<br>• Encephalitis | Direct stool microscopy (motile larvae, adult worms) |
| Enterobius vermicularis oxyuriasis (pin worm) Synonyms: Threadworm, pinworm, seatworm.<br>The worm is 4mm long and is just visible to the human eye | Mode of spread<br>*Auto-infection:*<br>• Direct anal to mouth transfer via the fingernails<br>• Retro- infection; eggs may hatch into larvae at the anal-rectal area. Then larvae move retrogradely to the caecum.<br>*Cross infection:*<br>• Contamination of fingers by clothing, objects, toilet seats,etc.<br>• By inhaling and swallowing eggs in the dust<br>• Main presentation: perianal and perineal itching. Migrating larvae may cause:<br>• Vaginitis, vulvitis, salpingitis, and peritonitis<br>• Irritation, insomnia may occur | Stool for ova<br>Ova can be obtained from the perianal region by use of adhesive tape |
| Taenia saginata (beef tapeworm) | • Non-specific symptoms,irritability<br>• Segment may be passed with stools<br>• Egg instools | Stool for ova (motile proglottides) |

# 7. Selected Infections and Related Conditions

## 7.1 Parasitic Infections

The leading causes of morbidity and mortality are parasitic, bacterial, fungal and viral infections. Accurate diagnosis and appropriate, cost-effective treatment are essential in overcoming these infections. Individual infections are discussed depending on their clinical importance.

### 7.1.1 MALARIA

Malaria parasites are usually transmitted by the bite of an infected female anopheles mosquito. Plasmodium falciparum is the commonest malarial parasite in Kenya and it is associated with significant morbidity and mortality. The other species are: P. malariae, P. vivax, P. ovale.

**Clinical Features**

**Uncomplicated Malaria**

- Classically, malaria presents with paroxysms of fever, chills, rigors, and sweating.
- Other features include malaise, headache, myalgia, joint pains, refusal to feed, nausea, vomiting, abdominal discomfort, and diarrhoea.

**Severe Malaria**

Severe malaria presents with a combination of most of the above plus either one or more of the following:

- Parasitaemia>5%or>200,000 parasites per µl of blood in high transmission area or>100,000 parasites per µl of blood in low transmission area
- Anaemia Hb<5gm%
- Cerebral malaria manifesting as confusion, stupor, convulsions orcoma
- Jaundice
- Hyperpyrexia, temperature>39ºC
- Hypoglycaemia (blood sugar<2.2mmol/L)
- Pulmonary oedema
- Disseminated intravascular coagulopathy (DIC – spontaneous bleeding)
- Malaria haemoglobinuria (Coca-cola coloured urine)
- Oliguria
- Hypovolaemic shock
- Fluid electrolyte imbalance

**Investigations**

- OPD cases:
  - Thick blood smear for malaria parasites (several slides may need to be done)

- Inpatient cases:
  - Thin blood smear for parasite count, species identification, and RBC morphology:
    - Haemoglobin
    - Blood sugar
    - Urinalysis

— A negative slide does not necessarily rule out malaria. Where cerebral malaria is suspected, begin appropriate therapy promptly.
— Exclude other diseases, e.g., meningitis, which may present with similar features. ***Do not assume a positive slide explains the cause of afebrile illness.***
— 20–30% of the normal population in endemic parts of Kenya will have positive slide for malaria parasites without symptoms and signs of malaria.

**MANAGEMENT**

**Uncomplicated Malaria**
**These cases are treated as outpatients:**

- The current recommended treatment of patients with uncomplicated malaria is a combination of artemether-lumefantrine. This is available as a fixed-dose combination with a tablet containing 20mg of artemether and 120mg of lumefantrine. Treat adults with a 6-dose regimen of 4 tablets STAT, then 4 on hours 8, 24, 36, 48, and 60.
- Should the patient deteriorate clinically at any time or symptoms persist 3–14 days after initiation of treatment, this should be considered as treatment failure. Treat such patients with quinine. Tablets come in 200mg or 300mg preparations. The dose is approximately 10mg/kg of body weight 8 hourly for 7 days.

**Recommended second line drugs are dihydroartemisinin plus piperaquine.**

**Severe Malaria**
**Prompt diagnosis and management of the specific complication is vital. Quinine is the recommended treatment for severe malaria.**

- Give quinine injection as a loading dose of 20mg/kg IM then refer. Where referral is not possible, continue with a maintenance dose of 10mg/kg 8 hourly.

**OR**

- Give artemether as loading dose 3.2mg/kg IM injection, then 1.6mg/kg maintenance dose until the patient can take oral therapy, then put on a full course of AL.

**Management – General**

- Reduce temperature if hyperpyrexia if present.
- Maintain fluid and electrolyte balance especially if there has beens ignificant fluid loss.
- Monitor output. Output should be atleast 30ml per hour. If hydration is inadequate and oliguria persists give frusemide 40–80mg IV STAT.

- Convulsions: Use diazepam 0.3mg/kgIV/IM ***OR*** Rectal 0.5mg/kg OR Paraldehyde 0.2ml/kg IM.
- Hypoglycaemia: Monitor blood glucose regularly. Large doses of dextrose may be required 25% 2ml/kg or 50%1ml/kg.

- Anaemia: Monitor Hb regularly. Transfuse if Hb is less than 5g% AND patient develops cardiorespiratory distress (grunting, nasal flaring, chest indrawing, heart failure).
- Check bloodslide for malaria parasites daily to confirm if parasitaemia is falling.



**Management – Specific**

**The management of adults with severe malaria must be appropriate to each complication that develops. Quinine is not contraindicated in pregnancy. Fluid and antimalaria drugs are given as for children. IV quinine should be given as follows:**

- First dose 20mg/kg in ½ litre of fluid in 5% dextrose given over 4 hours (max 1,200mg).
- Then give 10mg/kg in ½ litre of fluid over 4 hours (max 600mg) 8hours after commencing the initial dose
- Repeat 10mg/kg 8 hourly until the patient can take orally.
- Change to oral AL full dose or oral quinine to complete 7days therapy. Assess fluid regularly, including urine output.

**Monitoring response:**

**It is similar to that for children, with special attention to the complications.**

- If patient cannot be weighed, loading dose should be 900mg, followed by 600mg 8 hourly.
- Monitor for and correct hypoglycaemia with 50% dextrose(1ml/kg).NB:Each infusion of quinine should be given over 4 hours.
- Use quinine IM if IV drip cannot be monitored or fail to get IV access **—Quinine hydrochloride may be given IM in emergencies as shown in Table 7.1.**

**Table 7.1:Dosing of intra-muscular injection of quinine hydrochloride**

| Weight range (kg) | Volume of quinine injection (ml) | No. of injection sites |
|---|---|---|
| 31 – < 36 | 3.2 | 2 |
| 36 – < 41 | 4.0 | 2 |
| 41 – <46 | 4.5 | 2 |
| 46 – < 51 | 5.0 | 2 |
| 51 –< 56 | 5.5 | 2 |
| 56 – < 60 | 6.0 | 2 |
| 60 + | 6.0 | 2 |

Use 10ml sterile syringe. Draw up 4ml of sterile water for injection. Then into the syringe, draw up 600mg (2ml) from an ampoule of quinine and shake. The syringe now contains 100mg quinine per ml. NOTE: Each injection should not be more than 3ml per injection site.

**The dose for adults above 60kg should not exceed 600mg.**

- ♦ Quinine hydrochloride may be given IM in emergencies.
- ♦ Oral quinine may be introduced intragastrically by NG tube in situations when parenteral quinine is not available.
- ♦ Look out for renal failure.

**Chemoprophylaxis**

- ♦ Anti-malaria prophylaxis should be given to the following groups when going to malaria prone areas:

- All non-immune visitors to malarious areas:
  – Long-term residence >4weeks
  – Short-term residence <4weeks
- Patient with sickle cell disease and thalassaemia
- Children with impaired immunity (e.g., HIV,leukaemia)
- Patients with hyperimmune malaria syndrome, leukaemia or splenectomy
- Pregnant women (minimum of 2 IPT doses a month apart)
  - ♦ Chemoprophylaxis regimen:
- Current recommended antimalaria prophylaxis for those at risk is mefloquine 250 mg given weekly starting 2 weeks before travel to a malaria endemic area and continued for up to 4 weeks after return to a non malarious area.

**Patient Education**

- ♦ Seek early treatment for fever.
- ♦ Cover exposed skin in the evenings.
- ♦ use long lasting insecticide treated nets (LLINs).
- ♦ As a community, participate in indoor residual spraying(IRS)in epidemic prone areas.

## 7.1.2 TRYPANOSOMIASIS (SLEEPING SICKNESS)

This is a zoonotic disease caused by Trypanosoma brucei, trypanosomiasis is transmitted by bites of the tsetsefly(glossinaspp.).There are 2 types in Africa, T. brucei rhodesiense (East Africa) and T.brucei gambiense (WestAfrica).

**Clinical Features**

Disease caused by T. brucei rhodesiense is an acute febrile illness complicated by myocarditis and meningoencephalitis that is rapidly fatal if not treated, while that caused by T. brucei gambienseis a chronic debilitating illness with mental deterioration and physical wasting. History of travel to an endemic area helps in the diagnosis.

**Investigations**

Laboratory demonstration of trypanosomes in blood, bone marrow, CSF, and scraping from chancre.

**Management**

- Suramin for early cases 20mg/kg body weight, maximum single dose of 1g. Test dose of 200mg required initially. Treatment given on days1,3,7,8,14, and 21.The total single course is 5g and should not exceed 7g.
- Pentamidineisethionate (lomidine) 4mg/kg IM on alternate days for a total of 10 injections.
- Melsoprol(MelB)for CNS disease 2–3.6mg/kg per day IM in 3 divided doses in day1, 2, and 3,then repeat on day 10,11,and 12 and again on day 21,22, and 23.



## 7.1.3 LEISHMANIASIS

Disease caused by Leishmania species.

**VISCERAL LEISHMANIASIS**

Visceral leishmaniasis (kalaazar) is caused by Leishmania donovani. It is transmitted by a sandfly, which has an animal reservoir in domestic dogs and other canines.

**Clinical Features**

Presents with a massive enlargement of spleen and liver, as well as wasting despite a good appetite. It occurs as an opportunistic infection in the immunocompromised.

**Management**

- Sodium stibogluconate (pentostam) 20mg/kg/day for 28 days ***OR***
- Liposamal amphotericin B 3mg/kg daily on days 1–5,14–18,and 21–25; aminosidine IM OD for 3 to 4 weeks.

**CUTANEOUS LEISHMANIASIS**

Not common in Kenya. This disease is caused by Leishmania tropica.

**Clinical Features**

Presents as ulcers or skin lesions that may be confused with fungal disease or even neoplasm.

**Management**

- Sodium stibogluconate (Pentostam)

## 7.1.4 TOXOPLASMOSIS

Caused by T. gondii. Common in immunocompromised persons. Transmitted by blood products, ingestion of contaminated foods, tissue and organ transplantation, and laboratory accidents.

**Clinical Features**

Presents with lymphadenopathy, CNS, and ocular manifestations.

**Investigations**

- Isolation of organisms from body fluids and tissues
- PCR of tissue/body fluids
- Detection of 1gG and 1gM antitoxoplasmosis antibodies

**Management**

- Pyrimethamine and sulphadiazine combination pyrimethamine 200mg in 2 divided doses on day 1,then 25–l00 mg daily for 6–8 weeks and sulphadiazine 75mg/kg day 1 (4g maximum), then l00mg/kg per day (up to 6g) in 2 divided doses per day for 6–8 weeks.
- Folinic acid supplementation to be given with pyrimethamine.
- Clindamycin 600–1,200mg 8 hourly can be given, in combination with pyrimethamine or sulphadiazine for 6–8 weeks then maintenance till CD count is above 200.

## 7.1.5 SCHISTOSOMIASIS

This is an infection with blood flukes of the genus Schistosoma, which may cause chronic disease of intestines, liver, or genito-urinary tract. Adult flukes are white worm-like creatures that inhabit parts of the human venous system. All need a molluscan intermediate host. Important species of schistosomiasis in Kenya are S. haematobium and S. mansoni. Adult worms live and copulate within the veins of the mesentery. The sexually mature ones are found in the intestinal veins for S. mansoni mainly, while those of S. haematobium are mainly located in the venous plexus of the genitourinary tract.

Some eggs penetrate the intestinal or bladder mucosa and are passed in faecesor urine. Eggs hatch in fresh water, liberating cercariae that multiply in snails (intermediate host) and produce thousands of cercariae. These penetrate human skin within a few minutes after exposure and transform themselves into schistosomes, which develop into sexually active adult worms in the intestinal veins or venous plexus of the genitourinary tract depending on the species. The lifespan of adult worms ranges from 3 to 37 years. S. haematobium is common along the coastline, Tana river, Kwale, and Lamu. S. mansoniis widespread, particularly in Machakos, rice schemes, parts of Nyanza, and even Nairobi.

**Clinical Features**

Acute dermatitis and fever after exposure is a rare presentation. Occasionally transverse myelitis and convulsions may occur. Chronic schistosomiasis with S. mansoni may result in portal hypertension, splenomegaly, anaemia, and oesophageal varices, while terminal haematuria, dysuria, progression to obstructive uropathy, and bladder cancer may occur in the case of S. haematobium

Metastatic eggs can be found in other organs such as the spinal cord and brain. Salmonella infection in patients with schistosomiasis is difficult to eradicate until the schistosomiasis has been treated. Salmonella infection may present as recurrent pyrexia. Treatment consists of the following:

- Pyrimethamine and sulphadiazine combination pyrimethamine 200mg in 2 divided doses on day 1, then 25–100 mg daily for 1–2 weeks and

sulphadiazine 75mg/kg on day 1 (4g maximum) ,then 100mg/kg per day (up to 6g) in 2 divided doses per day for 1–2 weeks.

- Folic acid supplementation to be given with pyrimethamine.
- Clindamycin alone can be given, orcombined with pyrimethamine or sulphadiazine.

**Investigations**

- For S.mansoni:
  - Stool for ova, use concentration or Kato technique
  - Rectal snip for histological examination
  - Barium swallow and endoscopy to demonstrate oesephageal varices
  - Abdominal ultrasound
- For S.haematobium:
  - Urine for RBC and for ova of S.haematobium
  - Hatching test
  - X-ray lower abdomen – May show calcified bladder (sandy patches)
  - Intravenous urogram when obstructive uropathy is suspected

**Management**

Praziquantel 40mg/kg BD for a day is effective against all types of shistosomiasis.

**NB: Patients should be examined for living eggs; if positive, re-treat.**

**Prevention**

- Pyrimethamine and sulphadiazine combination pyrimethamine 200mg in 2 divided doses on day 1, then 25–100mg daily for 1–2 weeks daily and sulphadiazine 75mg/kg on day 1(4g maximum),then 100mg /kg per day(upto 6g) in 2 divided doses per day for 1–2 weeks.
- Folic acid supplementation to be given with pyrimethamine.
- Clindamycin alone can be given, in combination with pyrimethamine or sulphadiazine.

## 7.1.6 FILARIASIS

This is an arthropod-borne disease caused by thread like nematodes that in their mature adult stage reside in lymphatic or connective tissue (refer to Table 7.2).

**Investigations**

**Demonstration of microfilariae in blood or tissues.**

**Management**

- Lymphatic types: Ivermectin 150–200µg/kg in a single dose repeated at 6 and 12 months.
- Onchocercavolvulus: Ivermectin 150µg/kg in a single dose repeated at 6 and 12 months.

**Prevention**

Vector control: avoid bites of mosquitoes and other vectors by wearing clothing that covers the limbs, applying repellent creams, and using insecticide treated nets.

**Table 7.2:Summary of species, vectors, and pathologies for filariasis disease**

| Species | Vector | Pathology |
|---|---|---|
| Wuchereria bancrofti | Mosquitoes | Lymphatic (elephantiasis) and pulmonary |
| Brugia malayi | Mosquitoes | Lymphatic (elephantiasis) and pulmonary |
| Brugia rimori | Mosquitoes | Lymphatic (elephantiasis) |
| Onchocerca volvulus | Black fly | Skin, eye, and lymphatics |
| Loa loa | Deer fly | Allergy |
| Mansonella perstans | Midges | Allergy |
| Mansonella streptocerca | Midges | Skin |
| Mansonella ozzardi | Midges | Vague |

## 7.2 Viral Diseases

### 7.2.1 MEASLES

Measles occurs mainly in children; although rare in adults. In the case of adults, it carries a much higher mortality rate. For a full description see Part II, Section 21.

### 7.2.2 VIRAL HAEMORRHAGIC FEVERS

These are viral infections characterized by fever and haemorrhage. Refer to Table 7.3 for a summary of clinical signs and management.

**Table 7.3:Summary of viral haemorrhagic fevers**

| Condition | Vector | Clinicalmanifestations anddiagnosis | Management |
|---|---|---|---|
| Yellow fever | Aedes mosquitoes | Severe fever Jaundice Vascular permeability, shock, and DIC Diagnosis: Blood and liver examination for viruses | Treatment is supportive |
| Dengue fever | Aedes mosquitoes | Severe fever Vascular permeability, shock, and DIC Diagnosis: RT-PCR for virus | Treatment is supportive |
| Tickbornediseases | Ticks | Severe fever Vascular permeability, shock, and DIC Diagnosis: Blood and tissue examination | Treatment is supportive |
| Congo Crimean fever | Contaminated materials | Severe fever Vascular permeability, shock, and DIC | Treatment is supportive |
| African haemorrhagic fevers:Marbug and Ebola viruses | | Fatal haemorrhage,f ever, rash, hepatic and pancreatic inflammation. Diagnosis: ELISA | Treatment is supportive |

# 7.3 Bacterial Infections

## 7.3.1 MENINGITIS

This is an acute inflammation of the pia and arachnoid coverings of the brain with spread into the cerebro-spinal fluid (CSF). Most commonly due to invasion by bacteria (Pyogenic meningitis), and less so by viruses (Aseptic meningitis), tubercle bacilli (Tuberculous meningitis), or fungi (Fungal meningitis). The commonest bacterial organisms are Streptococcus pneumoniae (Pneumococcus), Haemophilus influenzae, and Neisseria meningitidis (Meningococcus), but almost any other bacteria may be involved depending on circumstances of the invasion and the age of the person.

Predisposing factors are low immunity, prematurity and septicaemia; infections in the nose, sinuses, ears, throat, and lungs; penetrating injuries of the skull and spinal column, and congenital malformations of the brain and spine.
Meningococcal meningitis often occurs in epidemics.

**Clinical Features**

Neck stiffness, positive Kerning's sign, altered level of consciousness, headaches, fever, vomiting, convulsions, photophobia are common features.

**Investigations**

- Lumbar puncture–Mandatory
- Fundoscopy –Also mandatory
- Where possible a head CTscan where there is evidence of focal neurological deficit
- Haemogram and ESR
- CXR
- Others: Mantoux test, history of contact withTB

**Table 7.4:CSF characteristics**

| Type | Colour | Protein | Sugar | Cells |
|---|---|---|---|---|
| Normal | Crystal clear | Below 0.4g/L | Above 2.5mmol/L | 0–5(x10/L) |
| Pyogenic | Cloudy | High | Low or nil | Hundreds to thousands mainly polymorphs |
| Tuberculous | Clear OR opalescent | Moderately raised | Low | A few hundreds mainly lymphocytes |
| Viral | Clear OR opalescent | Moderately raised | Normal | A few hundreds mainly lymphocytes |

— **Admit patient if meningitis is suspected. Initiate treatment immediately.**

**Management – General**

♦ When seizures occur:

- Stop seizures bygiving IV/IM diazepam 0.3mg/kg ***OR*** 0.5mg/kg STAT rectally. Repeat as necessary.
- Prevent seizures by giving phenobarbitone 3–6mg/kg IM BD or TDS ***OR*** 3–6mg/kg/day orally.

♦ Treat coma as follows:

- Keep airway clear and suck out secretions.
- Nurse the patient on the side; turn every 2 hours.
- Give oxygen, if necessary, 0.5–1L/min by intra nasal catheter.
- Give IV fluids if necessary.
- Observe vital signs carefully every 2 hours until awake.

♦ Follow the patient's progress:

- Take the temperature and pulse.
- Assess neck stiffness/Kerning's sign.
- Maintain fluid and electrolyte balance.
- Ensure patient is passing urine well.
- Ensure patient does not go into further seizures.

♦ Treat for malaria if in endemic area for malaria.

♦ Give dexamethasone 4mg IM/PO TDS for 72 hours in adults to reduce sequel of meningitis such as deafness. Carry out physiotherapy on the patient.

**Management – Pharmacological**

If CSF is normal, discontinue antimeningitis therapy and investigate and treat patient in line with other clinical and laboratory findings.

**Antibiotics**

♦ Streptococcus pneumoniae: Benzylpenicillin 4 mega units IV 6 hourly for 14–21 days ***OR*** chloramphenicol 1g IV 6 hourly for 14 days, ***OR*** ceftriaxone 2g/day IV 12 hourly for 14–21 days, Vancomycin 2g/day IV 8–12 hourly ***OR*** meropenem 2g/day IV 8 hourly may also be given.

♦ Neisseria meningitidis: Benzyl penicillin 4 mega units IV 6 hourly for 10 days ***OR*** chloramphenicol 1g IV 6 hourly for 10 days. Ceftriaxone may also be given as above. Dexamethasone 0.15mg/kg IM/PO 8 hourly for 4 days in adults to reduce sequel of meningitis such as deafness.

**Prophylaxis**

♦ To close contacts or household members for meningococcal meningitis:

- Sulphadiazine 1g BD PO for 2 days (if the organism is susceptible)***OR***
- Rifampicin 600mg BD PO for 2 days,***OR***
- Minocycline 100mg BD PO for 2 days for adults only

♦ Purified capsulate polysaccharide vaccine is available to control outbreaks, but it must be administered within 3–7 days of case identification to prevent an epidemic. Note: The vaccine is not very useful for children <2years.

**Complications**

- These include subdural effusion, hydrocephalus, blindness or deafness, secondary epileptic seizures, mental and physical retardation.
- Note: Notify the medical officer for health if meningococcal meningtis is diagnosed

## 7.3.2 TETANUS

It is a neurological disorder characterized by muscle spasms due to endotoxin produced by Clostridia tetani. Tetanus occurs in several clinical forms, including generalized, neonatal, and localized disease.

**Clinical Features**

Trismus(lockjaw),opisthotonos(rigid arching of back muscles),dysphagia, laryngospasm. Diagnosis is mainly clinical.

**Management**

- Maintain adequate airway (intubation is necessary)
- Insert a nasogastric tube as early as possible for nutrition and drug administration
- Neutralize toxin:1,000–3,000IU of human tetanus immunoglobulin IM wound. Horse serum is an alternative.
- Eliminate toxin production:
  - Crystalline penicillin 1 mega unit IV QDS for 10 days (children 50,000IU/kg/ day; Neonates BD, older children QDS)
  - Metronidazole 2g/day for 7–10 days
  - Other agents that can be used include cephalosporins, imipenem, macrolides and tetracyclines.
  - Surgical toilet of the wound
- Control spasms –general
  - Diazepam is the drug of choice. Add phenobarbitone or chlorpromazine if additional sedation is required. All 3 drugs may be needed in severe cases. (Refer to Table7.5 for a guide to the dosage of these drugs.)
  - Diazepam 10–60mg IV/ rectally QDS
  - Phenobarbitone30–90 mg IV/M every12 hourly chlorpromazine 100mg IM QDS alternating with diazepam. Maintain fluid balance.
  - Monitor for and treat intercurrent infections.
  - Nurse in a dark, quiet isolation.

**Prevention**

People with open wounds should be given 2 doses of tetanus toxoid at least 4 weeks apart. Only 1 dose if immunized during the last 3 years and adequate surgical toilet.

**Table 7.5:Guideline for dosage administration for tetanus drugs**

| Drugbeing administered | Time inhours | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 6 | 9 | 12 | 15 | 18 | 21 | 24 |
| Diazepam | + | | + | | + | | + | | + |
| Chiorpromazine | | + | | + | | + | | + | |
| Phenobarbitone | + | | | | | | | | + |

Note: Frequency of drug administration should be titrated against clinical condition. Optimum level of sedation is achieved when patient remains sleepy but can aroused.

## 7.3.3 TUBERCULOSIS

Tuberculosis is caused by Mycobacterium tuberculosis (M-TB). This is commonly M-TB hominis, but M-TB bovis also causes human infections .Transmission is by droplet infection through coughing and sneezing. The bovine type is mainly contracted by drinking unpasteurized milk. The incidence of TB is on the increase because of its association with HIV/AIDS, poverty, malnutrition, and overcrowding.

**Clinical Features**

Most cases of tuberculosis in Kenya (80%) are pulmonary. Features of pulmonary tuberculosis are cough for 3 weeks or more, haemoptysis, chest pain, fever and night sweats, weight loss, and breathlessness.

Extrapulmonary tuberculosis symptoms depend on the organs affected. TB adenitis manifests as lymphadenopathy, TB arthritis as painful swollen joints, TB meningitis as meningitis with features of meningitis, TB peritonitis as ascites and TB pleural as pleural effusion.

**Investigations**

- Sputum for AAFB (2 sputums pot and early morning).
- GeneXpert
- Mantoux test.
- Chest x-ray.
- Lymph node biopsy.
- Fine needle aspirate of lymph nodes.
- Body fluids for biochemistry and microscopy (CSF, pleural, pericardial, and peritoneal fluids)
- Sputum for AAFB culture and sensitivity(before the start of treatment in those on treatment and suspected drug resistantTB).

**Management**

The success of tuberculosis treatment depends on strict adherence to WHO's DOTS (directly observed treatment short-course) strategy.

**General Management**

- Follow national treatment guidelines.
- Ensure adequate supply of drugs.
- Use correct regimens and dosages.
- Ensure regular patient attendance.
- Always supervise initial phase of treatment.
- Trace defaulters promptly.
- Maintain accurate patient information and clinic attendance records.

**Pharmacological Management**

Pharmacologic management depends on the classification of the patient and the presence of other conditions, such as HIV.

**Classification of TB Patients. Patients are classified into the following groups for epidemiological and treatment reasons depending on the site, microbiology, severity of disease, and history of previous treatment. These same categories are used in the TB register for reporting:**

- New (N): Patient who has never been treated for TB before.
- Relapse(R): Patient who has received treatment and was declared cured,but now has TB again.
- Transferred in(TI):Patient who was registered in another county initially and has now reported to continue treatment.
- Treatment resumed(TR):Patient who interrupted treatment, and was declared "out of control", but is now resuming treatment.
- Other(O):Other types of patients,e.g.,failure cases put on retreatment.

**Short Course Chemotherapy(SCC).SCC is given to all TB patients registered by the National Leprosy and Tuberculous Programme (NLTP). Different SCC regimens are used for the different categories of tuberculosis. The following apply:**

- In the first 2 months (initial phase of treatment) the drugs should be administered under the direct observation of either a health care provider in a health facility or another reliable member of the household or community.
- Drugs and tools for registration and reporting should be available before treatment is started. Patient should be admitted if is too ill or DOTS cannot be ensured.
- During the continuation phase the patient should collect a supply of drugs 2 weekly for daily self-administration at home.

**Treatment Regimens and Drug Dosages. The treatment regimen for new adult smear-positive patients and other seriously ill cases of TB, e.g., TB meningitis, military TB, and TB of vital organs is summarized in Table 7.6**

**Table 7.6:Dosage of individual anti-TB drugs according to body weight**

| Drug | Recommendations Average dose in mg/kg | Range in mg/kg | Maximum dose |
|---|---|---|---|
| Isoniazid | 10 | 7-15 | 300mg |
| Rifampicin | 15 | 10-20 | 600mg |
| Pyrazinamide | 35 | 30-40 | 2.0mg |
| Ethambutol | 20 | 15-25 | 1.0mg |

Treatment of TB in HIV/AIDS Patients. HIV increases a person's susceptibility to infection with M. tuberculosis. In individuals infected with M. tuberculosis, HIV is a potent cause of progression of tuberculosis infection to disease. In HIV infected children, dissemination of TB is common. TB meningitis, miliary tuberculosis, and widespread tuberculous lymphadenopathy occur. Offer all TB patients HIV testing

and counselling, and pit all HIV-positive patients on cotrimoxazole preventive therapy. For these patients, do a work upfor ART and request them to bring their regular sexual partners for counselling and HIV testing.

**Complications of TB**
These include haemoptysis (coughing up blood), spontaneous pneumothorax, bronchiectasis, lung fibrosis and lung abscess.

**Acquired Drug Resistant TB**
Acquired drug resistance results from inappropriate use of one drug or poor adherence to treatment. This suppresses the growth of organisms susceptible to the drugs but encourages the multiplication of isolated strains with spontaneous drug resistance.

Multiple Drug Resistant TB (MDR-TB). This is resistance to at least both rifampicin and isoniazid as a consequence of poor adherence to recommended treatment regimens by clinicians and patients. This resistance is further associated with increasing poverty and the HIV/AIDS epidemic. MDR-TB is confirmed on culture and sensitivity.

**Prevention of MDR-TB.** Drug resistance can be prevented by:
- Strengthening TB programmes.
- Ensuring directly observed therapy whenever rifampicin is used.
- Using fixed dose combination tablets containing rifampicin.
- Referring all drug-resistant TB patients to higher level for appropriate management.

## 7.3.4 SALMONELLA INFECTIONS

Disease caused by the following salmonella: Salmonella typhi and Salmonella paratyphi A, B, and C commonly cause enteric fever. Salmonella enteritis causes gastroenteritis.

**TYPHOID FEVER**
Systemic disease caused by S. typhi. Typhoid bacilli are shed in the faecesof a symptomatic carrier or in the stool or urine of those with active disease.

**Transmission is via contaminated food or water by:**
- Direct contamination by faeces or urine.
- Flies from faeces to food.
- Healthy carriers who are food handlers.
- Health personnel through inadequate hygiene when changing soiled linen.
- Healthy carriers, who can shed organisms for more than ayear.

**Clinical Features**
These include high fever, headaches, anorexia, weight loss, diarrhoea, consti- pation, abdominal tenderness, changes in sensorium, splenomegaly, relative bradycardia, and Rose Spots (blanching lesions). A high index of suspicion for typhoid is required when investigating any patient with unexplained fever.

**Investigations**

- ♦ Full haemogram: Relative leukopaenia in relation to the fever
- ♦ Cultures: Positive in blood in first week; stool and urine cultures become positive in the third week
- ♦ Widal test: Four fold rise in separate specimens acquired 2 weeks apart suggest

S.typhi infection. Rising titres of O antigen are significant. NB: Only titres of O antibody of 1:160 or more are significant. The gold diagnostic standard should be isolation of bacilli in cultures.

**Management**

- ♦ Chloramphenicol:(2–4g in adults ***OR*** 50mg/kg body weight per day in children) for 2 weeks
- ♦ Cotrimoxazole 4 tabs BD for 2 weeks
- ♦ Amoxicillin 4–6g or 100mg/kg/day in 3 divided doses for 2 weeks
- ♦ Ciprofloxacin 500–750mg BD for 14 days ***OR***
- ♦ Ofloxacin 400mg BD for 14 days ***OR***
- ♦ Norfloxacin 400mg BD for 14 days ***OR***
- ♦ Ceftriaxone 1g OD IV for 7–14 days

**Complications**

- ♦ Intestinal haemorrhage.
- ♦ Chronic carrier state for Salmonella typhi.
- ♦ Intestinal perforation leading to peritonitis, sepsis, and septicaemia.

- Clinical features: Abdominal pain and distension with rebound tenderness.
- Investigations: Plain x-ray abdomen–erect and decubitus may show pneumoperitoneum or multiple fluid levels.
- Management: Drugs as above and surgical laparatomy.

**Prevention**

- ♦ Drink wholesome drinking water (boil water for 10 minutes or use water that is chlorinated).
- ♦ Drink pasteurized milk or boiled milk.
- ♦ Prevent healthy carriers of Salmonella typhi from handling food.
- ♦ Treat healthy carriers.
- ♦ Ensure hygienic waste disposal.
- ♦ Vaccination:

- Live attenuated oral vaccine 4 capsules given on alternate days. Avoid antibiotics for 1 week NB: contraindicated in immuno-suppression cases.
- Typhim VI vaccine–Single dose 0.5 ml IM (70%efficacy; booster dose needed every 2–3 years).

## 7.4 Other Selected Infections and Related Conditions

Some common conditions with their recommended first and second line antibiotic treatment are presented in Table 7.7.

**Table 7.7:Selected infections with recommended antibiotic treatment**

| Infection or related condition | First line antibiotic treatment | Second line antibiotic treatment |
|---|---|---|
| Acute rheumatic fever | Benzathine penicillin | Erythromycin |
| Acute osteomyelitis | Clidamycin<br>Cloxacillin + gentamicin | Cloxacillin + chloramphenicol |
| Cellulitis | Cloxacillin | |
| Conjunctivitis (bacterial) | Tetracycline eye ointment | Chloramphenicol eye drops |
| Dysentery (shigella) | Ciprofloxacin | Ceftriaxone |
| Ludwig's angina | Benzyl penicillin | |
| Otitis media | Cotrimoxazole | Amoxicillin |
| Pneumonia (mild) | Cotrimoxazole | Amoxicillin |
| Pneumonia (severe) | Benzyl penicillin + gentamicin | Ceftriaxone<br>Amoxicillin + clavulinate |
| Septic arthritis | Cloxacillin + gentamicin | Amoxicillin + gentamicin |
| Urinary tract Infections | | |
| Lower | Cotrimoxazole | |
| Upper (outpatient) | Amoxicillin + clavulin | Cotrimoxazole |
| Upper (inpatient) | Gentamicin | Ciprofloxacin |

# 8. Musculoskeletal Conditions

## 8.1 Non-Specific Arthralgia

Joint pain without features of inflammation.

**Clinical Features**
General malaise and joint pains; joint mobility not affected, joint not red, not warm, not tender or only slightly tender. Usually it is a feature of another illness and careful systemic examination is warranted.

**Investigations**
None except for the illness of which arthralgia is a feature.

**Management**
Ibuprofen 400mg 8 hourly *OR* paracetamol 1g 8 hourly.

## 8.2 European League Against Rheumatism (EULAR) defined characteristics describing arthralgia at risk for rheumatoid arthritis.

These parameters are to be used in patients with arthralgia without clinical arthrisitis and without other diagnosis or other explanation for the arthralgia.

**History taking**
- Joint symptoms of recent onset (duration <1 year)
- Symptoms located in metacarpophalangeal joints
- Duration of morning stiffness >60 min
- Most severe symptoms present in the early morning
- Presence of a first-degree relative with rheumatoid arthristis

**Physical examination**
- Difficulty with making a fist
- Positive squeeze test of metacarpophalangeal joints

## 8.3 Rheumatoid Arthritis

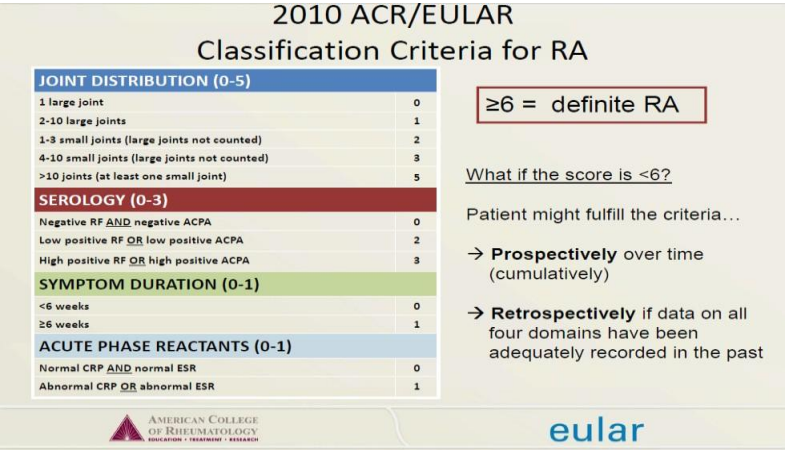
A chronic inflammatory disease that affects the joints. It is symmetrical, peripheral, polyarthritic, most commonly involving the small joints of hands, wrists,metacarpophalangeal joints, ankles, knees, and cervicals pine.

**Clinical Features**

- Articular: Symmetrical peripheral polyarthritis mostly of small joints (warm, painful, stiff, swollen). Stiffness worse in the morning. Muscle wasting. Deformities may occur, including ulnar deviation, boutonniere deformity.
- Extra-articular: Fever,weight loss,lassitude,anaemia,subcutaneous nodules, splenomegaly, lymphadenopathy, keratoconjuctivitis, pericarditis,pleuritis.

The figure 8.1 shows the criteria used for cliical classification of RA.

**Figure 8.1: Classifaction criteria for Rheumatoid Arthritis**




**Investigations**

- Haemogram–Moderate hypochromic, microcyticanaemia;or leucopaenia in Felty's syndrome
- Acute phase reactants such as ESR or C-reactive Protein—Elevated
- Rheumatoid factor
  - Anticitrullinated cyclic peptides (ACCP)-highly specific
  - Imaging such as radiographs or ultrasounds of involved joints

**Management**

- Initiate physiotherapy.
- Initiate occupational therapy.
- Provide drug treatment as indicated:

- Use of NSAIDs such as Ibuprofen 400mg tabs 8 hourly until pain is relieved. Refer early as NSAIDS do not treat RA sufficiently when used alone
- Glucocorticoids, especially prednisone at doses not exceeding10mg/day.
- Disease modifying antirheumatic drugs (DMARDs). These drugs should be started as soon as diagnosis is confirmed and should aim to achieve remission or low disease activity. These include methotrexate 7.5–15mg once weekly- supplement folic acid at 5mg weekly, **OR** Leflunomide 10-20mg daily ***OR*** sulfasalazine 500mg twice daily increased to 1g twice daily in 2 weeks up to a maximum of 3g, hydroxychloroquine 200–400mg once daily.
- Intraarticular steroids –use methylprednisolone acetate, Triamcinolone acetonide 40mg for large joints and 20mg for small joints.
- For severe disease not responding to the conventional DMARDs after 3-6months, start on biologics: etanercept, adalimumab or tocilizumab. For systemic disease, Tocilizumab may be considered first line treatment.

♦ Refer for orthopaedic review if:

- Deformities are present
- Disease not responding to non-steroidal anti-inflammatory drugs (NSAIDs).
- There is systemic organ involvement.

♦ Admit for:

- Management of acute exacerbation.
- Bed rest (may need to splint the affected joint).
- Intensive physiotherapy.
- Systemic complications.

**Complications**

All the systems are involved in this disease. It needs specialist attention, as does the use of steroids or chloroquine. Refer patients.

## 8.4 JUVENILE IDIOPATHIC ARTHRITIS (JRA)

**Clinical Features**

This type of arthritis begins at or before the age of 16 years and persists for more than 6 weeks. It tends to affect large and small joints and may interfere with growth and development. Refer to Table 8.1 for a summary of characteristics and the clinical classification.

**Management**

♦ Supportive treatment including Ibuprofen at 10mg/kg every 8 hours; physiotherapy and occupational therapy.

♦ Aspirin is used with caution because of concerns about Reyes syndrome. For dosage see under adult treatment or paediatric schedule.

♦ Intraarticular steroid injection for inflamed joints with Methylprednisone acetate, Triamcinolone acetonide. 40mg for large joints and 20mg small joints.

♦ Concomitantly, start disease modifying antirheumatic drugs (DMARDs) as soon as diagnosis is confirmed.These are methotrexate 0.5mg/kg once weekly, ***OR*** sulfasalazine 30-50mg/kg daily in 2 divided doses to maximum of

2g per day, hydroxychloroquine 5mg/kg/day maximum 200mg once daily. These may be given singly or in combination.

- For severe disease not responding to the conventional DMARDs above after 3-6months, start on biologics: etanercept, adalimumab or tocilizumab. For systemic disease, Tocilizumab may be considered first line treatment.

**Prognosis**

- Complete remission occurs in 10–60% of patients depending on subtype. However, overral, JRA is a chronic disease and majority of children will have active disease into adulthood.
- Those with oligoarticular, ANA+ disease are at high risk for vision threatening chronic anterior uveitis. Thus, theres need for routine ophthalmologic evaluation (3-6 monthly) for children with JRA.
- Those with polyarticular and RhF positive have a less favourable prognosis. NB: For osteomyelitis and septic arthritis see Chapter 53 (Orthopaedics).

**Table 8.1: Summary of juvenile rheumatoid arthritis (JRA)**

| Characteristic | The clinical classification of JRAnoted or observed | | |
|---|---|---|---|
| Systemic disease | Pauciarticular | Polyarticular | |
| Percentage | 20% | 40% | 40% |
| Rheumatoidfactor | -ve | -ve | +/-+ve/-ve |
| Antinuclearfactor | -ve | 75% | |
| HLAB27 | | +/-+ve/-ve | -ve |
| Clinicalpresentation | Highfever,rash, splenomegaly, generalized arthritis lymphadenopathy, serositis, striking leucocytosis, and thrombocytosis | Younger age | As for adult rheumatoid |

## 8.5 Gout

Gout is a metabolic disorder due to hyperuricaemia. The causes may be primary or secondary (e.g., myeloproliferative, lymphoproliferative disorders, haemolytic anaemia, polycythaemia; tumour lysis syndrome following cytotoxic therapy and thiazide diuretics).

## 8.6 ACUTE GOUT

**Clinical Features**

Excruciating joint pain, usually single joint commonly the big toe. Pain becomes more severe as attack progresses but subsides spontaneously in about 4 days. Tophi are found primarily in the pinna, and overlying olecranon bursa. There is erythema and warmth over the affected joint.

**Management**

- If severe, Diclofenac 75mg IM STAT/PRN then 50mg PO 8 hourly ***OR*** **Ibuprofen 400–800mg 8 hourly.**

- ♦ Several other NSAIDs may be used as long as side effects, especially renal and gastrointestinal, are taken into consideration.
- ♦ Colchicine 0.5mg hourly till patient improves or GIT side effects appear,or maximum of 6mg has been taken.

### 8.6.1 INTERCRITICAL GOUT

This is defined as the period between attacks. Initially inter-critical periods are long, but later acute attacks occur more frequently. If arthritic attacks are frequent, renal damage is present, or serum uric acid levels are significantly elevated, then serum uric acid should be lowered.

- ♦ Colchicine at maintenance level should be started before manipulation of uric acid at 0.5–0.6mg given BD a few days prior to initiation of uric acid lowering drugs.
- ♦ Allopurinol 300mg OD is drug of choice for lowering uric acid levels. (Allopurinol should not be given when the patient is in pain.)

### 8.6.2 ASYMPTOMATIC HYPERURICAEMIA

This is the situation that arises when there is hyperuricaemia without any symptoms.

**Management**

- ♦ No drug treatment is needed.
- ♦ Advise patient to reduce weight.
- ♦ Avoid alcohol consumption.
- ♦ Avoid heavy consumption of foods containing high concentrations of purines, e.g., roasted meat.

### 8.6.3 TOPHACEOUS AND GOUTY ARTHRITIS

This describes the situation in which there is deposition of uric acid crystal in cartilage, tendons,and soft tissue. In 90% of cases there is renal involvement.

**Management**

- ♦ Treat with allopurinol 300mg per day or probenecid 250mg BD.
- ♦ Initiate colchicine prophylaxis before starting allopurinol.
- ♦ Watch out for:
  - Renal impairment.
  - Uric acid nephrolithiasis.
  - Failure to respond to the therapy.

## 8.7 Osteoarthritis

This is a degenerative joint disease characterized by cartilage degeneration and bone hypertrophy at the articular margins. It is chronic but does commonly present with acute-on-chronic flares.

**Clinical Features**

Pain, stiffness, immobility, and “cracking”of the joints. Pain worse towards end of day. Joint tenderness, bony swelling, loss of full range of movement, and crepitus on movement. Heberden’s nodes. Joints commonly involved are cervical and lumbar

spines, the knees and hips, as well as the hands and feet. It may also occur secondarily in response to severe or chronic joint injury (e.g., after fractures).

**Investigations**

- Haemogram, ESR
- X-ray, joints – Loss of joint space, osteophytes, marginal bone lipping, bone cysts
- Arthroscopy
- MRI scan

**Management**

- Resting of joints, including use of crutches; involve physiotherapist.
- Ibuprofen 400mg tabs 8 hourly until pain is relieved.
- Others are:
  - Non-selective NSAIDs combined with gastric mucosal protectant.
  - Pure analgesics such as tramadol.
  - Intra-articular glucocorticoids,e.g.,methylprednisolone acetate and betamethasone. Dipropionate/sodiumphosphate (2mg/5mg) depot preparation.

## 8.8 Systemic Lupus Erthematosus (SLE)

A multisystemic autoimmune disease that commonly affects females. It has a broad range of clinical and immunologic manifestations. The various clinical symptoms do not always occur simultaneously and may develop at any stage of the disease. Diagnosis of SLE can be made if a client satisfies 4 of the clinical and immunologic criteria used in the SLICC classification criteria, including at least one clinical criterion and one immunologic criterion, OR if he or she has biopsy-proven nephritis compatible with SLE in the presence of ANAs or anti-dsDNA antibodies.

### 8.8.1 CLASSIFICATION OF SLE: THE SYSTEMIC LUPUS INTERNATIONAL COLLABORATING CLINICS (SLICC)

**Clinical Criteria**

- Acute cutaneous lupus erythematosus (including “butterfly rash”)
- Chronic cutaneous lupus erythematosus (eg, localized or generalized discoid lupus erythematosus
- Oral ulcers (on palate and/or nose)
- Non-scarring alopecia
- Synovitis (≥ 2 joints) or tenderness on palpation (≥2 joints) and morning stiffness (≥30 min)
- Serositis (pleurisy or pericardial pain fore more than 1 day)
- Renal involvement (single urine: protein/creatinine ration or 24-hour urine protein, >0.5g)
- Neurological involvement (e.g., seizures, psychosis, myelitis)
- Hemolytic anaemia
- Leukopenia (<4000/uL) or lymphopenia (<1000/uL)
- Thrombocytopenia (<100000/uL)

**Immunological Criteria**

- ANA level above laboratory reference age
- Anti-dsDNA antibodies
- Anti-Sm antibodies
- Antiphospholipid antibodies (anticardiolipin and anti- β 2-glycoprotein I [IgA, IgC, or IgM ] antibodies; false positive VDRL[Veneral Disease Research Laboratory test].
- Low complement (C3, C4, or CH50)
- Direct Coombs test (in the absence of hemolytic anaemia)

For classification as SLE, four criteria (at least one of them clinical and at least one immunological) have to be fulfilled or lupus nephritis has to be diagnosed histologically in the presence of ANA or anti-dsDNA antibodies. The SLICC criteria are not diagnostic criteria.

**Management**

- Hydroxychloroquine- recommended for all patients with SLE, at doses not exceeding 5mg/kg.
- Monitoring of retinopathy should be done.
- Glucocorticoids- may provide rapid symptom relief but should not be a mainstay of treatment due to the side effects and risk of organ damage. medium to long-term aim should be to minimise daily dose to ≤7.5 mg/day prednisone equivalent or to discontinue them.
- Pulsing with high dose steroids may be necessary; High-dose intravenous Methylprednisone (usually 250–1000 mg/day for 3 days) is often used in acute, organ-threatening disease (eg, renal, neuropsychiatric) after excluding infections.
- Immunosuppressive therapy: the choice of agent depends on prevailing disease manifestation(s), patient age and childbearing potential, safety concerns and cost. Methotrexate (MTX) and azathioprine (AZA) should be considered in patients with poor symptom control after a trial with GC and HCQ or when HCQ alone is unlikely to be sufficient, due to the large experience gained with their use and their relatively safe profile
- Biologics- add-on treatment with *belimumab* should be considered when disease does not respond to the immunosuppressive agents.
- In organ-threatening disease refractory or with intolerance/contraindications to standard immunosuppressive agents, *rituximab* can be considered .
- Skin disease- Effective protection from ultraviolet exposure with broad-spectrum sunscreens and smoking cessation are strongly recommended.
- Treatment of skin disease includes topical agents.

Comorbidities commonly occur in SLE;

- Antiphospholipid Syndrome-The presence of aPL is associated with thrombotic and obstetric complications and increased risk of damage.
- Infections - commonly result in patients with SLE , as a result of the disease or the treatment with immunosuppressive therapy.
- Cardiovascular disease.

## 8.9 Systemic Sclerosis

Systemic sclerosis (SSC) is a connective tissue disease, characterised by excessive production and accumulation of collagen, called fibrosis, in the skin and internal organs and by injuries to small arteries.

**Diagnosis**

- Mostly clinical; As per the American College of Rheumatology 2013 criteria for classification of systemic sclerosis, Patients with a score of at least 9 points are classified as having systemic sclerosis.

**Table 8.2: Classification criteria for systemic sclerosis issued in 2013 by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR)**

| Criteria | Points |
|---|---|
| Skin thickening of the fingers of both hands extending proximal to the metacarpophalangeal joints | 9 |
| Puffy fingers or<br>Sclerodactyly (skin thickening of the fingers) | 2<br>4 |
| Digital tip ulcers<br>or<br>fingertip pitting scars | 2<br><br>3 |
| Telangiectasia | 2 |
| Abnormal nailfold capillaries | 2 |
| Pulmonary arterial hypertension or<br>Interstitial lung disease | 2<br><br>2 |
| Raynaud's phenomenon | 3 |
| Systemic sclerosis-related autoantibodies (anti-centromere, anti-topoisomerase I, anti-RNA, polymerase | 3 |

**Investigations**

- Kidney function tests
- ANA profile
- SS autoantibodies- anti-centromere, anti-topioisomerase, anti-RNA polynmerase are specific for the condition
- Echocardiogram
- Chest Radiographs and CT scans

**Management**

- Specific management of systemic sclerosis may depend on the specific symptom or system affected and complications of systemic sclerosis such as renal crises, pulmonary arterial hypertension may require multidisciplinary and specialist review.
- Raynaud's Phenomenon: nifedipine may reduce the frequency of Raynaud's phenomenon; Prostanoids such as intravenous iloprost may be used in severe illness.
- Monitor blood pressure closely
- Digital ulcers: PDE-5 inhibitors may heal and prevent digital ulcers, ERA such as bosentan- may reduce number of new digital ulcers.

- Skin and lung disease: immunosuppressive agents such as methotrexate and cyclophosphamide will be useful .
- Gastrointestinal disease: Proton-pump inhibitor and prokinetic gents may help in GERD and motility disturbances and to prevent esophageal ulcers and strictures.

## 8.10 Large Vessel Vasculitides

Large-vessel vasculitis includes giant cell arteritis (GCA) and Takayasu's arteritis (TAK). Early diagnosis of these two diseases is quite challenging in clinical practice and may be accomplished by combining the patient symptoms, physical examination findings, blood test results, imaging findings, and biopsy results, if available.

Rapid diagnosis and effective treatment are required in large vessel vasculitis (LVV) to reduce the risk of complications such as blindness in giant cell arteritis (GCA) and aortic aneurysm or vascular stenosis in GCA and Takayasu arteritis (TAK).

### 8.10.1 GIANT CELL ARTERITIS

GCA affects patients aged over 50, females being affected two to three times more often than males. GCA mainly involves large- and medium-sized arteries, particularly the branches of the proximal aorta including the temporal arteries.

Vasculitic involvement results in the typical manifestations of GCA including temporal headache, jaw claudication, and visual loss. A systemic inflammatory response and a marked response to glucocorticoids is characteristic of GCA. GCA usually remits within 6 months to 2 years from disease onset. However, some patients have a chronic-relapsing course and may require longstanding treatment.

Mortality is not increased, but there is significant morbidity mainly related to chronic glucocorticoid use and cranial ischaemic events, especially visual loss.
All patients presenting with signs and symptoms suggestive of GCA should be urgently referred to a specialist team for further multidisciplinary diagnostic work-up and management

Untreated active GCA is an emergency and carries a substantial risk of permanent visual loss and other ischaemic complications. It is recommended that all patients ≥50 years of age presenting with acute or subacute onset of signs and symptoms suggestive of GCA especially and raised inflammatory markers without explanation (eg, infection) should be referred urgently to a specialist team/experienced centre for further diagnostic work-up.

**Diagnosis**
- Raised inflammatory markers-ESR, CRP
- Temporal artery biopsy- the gold standard for diagnosis
- Imaging

**Management**
Glucocorticoids are the mainstay of treatment for GCA - 40–60 mg/day prednisone-equivalent.

### 8.10.2 TAKAYASU ARTERITIS

**Takayasu arteritis (**TAK) mainly involves the aorta and its main branches. Women are particularly affected with a female:male ratio of 9:1. In most patients, age of onset is between 20 and 30 years. Early manifestations of TAK are non-specific and include constitutional and musculoskeletal symptoms. Later on, vascular complications become manifest. Most patients develop vessel stenoses, particularly in the branches of the aortic artery, leading to manifestations of vascular hypoperfusion. Aneurysms occur in a minority of cases.

**Diagnosis**
- Inflammatory markers- ESR, CRP
- Imaging
- Histology

**Management**
- Glucocorticoid treatment- 40–60 mg/day prednisone-equivalent.
- However, many patients have an insufficient response to glucocorticoids alone, or relapse when they are tapered or discontinued.

- Immunosuppressive agents and, in refractory cases, biological drugs can often attain disease control and prevent vascular complications.
- Surgical review for Revascularization procedures may be required in patients with severe established stenoses or occlusions.

## 8.11 Chronic inflammatory Muscle Disease

Polymyositis, dermatomyositis and inclusion body myosistis are chronic autoimmune, inflammatory muscle diseases, characterized by muscle weakness. The muscles affected are typically those closest to the trunk or torso, affecting both sides of the body. The onset can be gradual or rapid. Patients with inflammatory myopathies have increasing difficulty with tasks requiring the use of proximal muscles, such as getting up from a chair, climbing steps, or lifting objects. Tasks requiring distal muscles, such as buttoning or holding objects, are affected early in inclusion-body myositis but only in advanced cases of polymyositis, dermatomyositis, and necrotizing autoimmune myositis.

Polymyositis is common in females and may have flares or relapses, and periods with minimal or no symptoms, known as remissions.

Dermatomyositis is characterized by distinct skin manifestations accompanying or preceding muscle weakness; the skin manifestations include periorbital heliotrope (blue purple) rash with edema; erythematous rash on the face, knees, elbows, malleoli, neck, anterior chest (in a V-sign), and back and shoulders (in a shawl sign); and a violaceous eruption (Gottron's rash) on the knuckles, which may evolve into a scaling discoloration. The lesions are photosensitive and may be aggravated by ultraviolet radiation. Dilated capillary loops at the base of the fingernails, irregular and thickened cuticles, and cracked palmar fingertips ("mechanic's hands") are characteristic of dermatomyositis. Subcutaneous calcifications, sometimes extruding to the surface of the skin and causing ulcerations and infections, may occur and are especially common among children.

**Diagnosis**

- Physical examination of muscle strength- weakness
- Muscle enzymes - Creatine phosphokinase (CPK), aldolase, SGOT, SGPT, and LDH are usually elevated
- Autoantibodies
- Electrical tests of muscle and nerves
- Muscle biopsy-gold standard for diagnosis
- MRI

**Treatment**

- High doses of corticosteroid treatment- Prednisone at 1mg/kg upto 100mg daily
- In rapidy deteriorating patients; intravenous methylprednisolone at a dose of 1000 mg per day for 3 to 5 days may be given before starting treatment with oral glucocorticoids. These can then be tapered off after 3-4 weeks.
- In patients with dermatomyositis, topical glucocorticoids or calcineurin inhibitors and sunlight avoidance are recommended.
- When glucocorticoids fail to induce remission or in severe and rapidly progressive cases, intravenous immune globulin therapy (2 g per kilogram in divided doses over a period of 2 to 5 consecutive days) is appropriate.
- Immunosuppressive medication- azathioprine, mycophenolate mofetil, methotrexate, or cyclosporine
- Physical therapy

Referrals and follow up care: may be made to rheumatologists and other specialists as needed. Investigation for malignancies may be required.
Vaccinations

# 9. Neoplasms

Neoplasms can be benign or malignant. Malignant neoplasms are also referred to as cancers. Neoplams most commonly present as swellings, and at times pain and malfunction of the affected organs or tissues. Patients with suspected malignancies should be urgently referred to appropriate consultants for diagnostic examinations and treatment. Neoplasms can occur in any age group. In general, most will require treatment in referral hospitals (level 6). Refer to Table 9.1 for site-specific investigations and management and to Table 9.2 for a summary of common malignancies.

Psychosocial support for the patient and caregivers is essential as these are chronic diseases with lots of psychological and social impact.

**Table 9.1:Site-specific investigations and management of malignancies**

| Tumoursite | Clinicalfeatures | Investigations | Management |
|---|---|---|---|
| Nose and paranasal sinuses | Nasal blockage, rhinorrhoea, epistaxis, nasal mass, facial swelling, paraesthesia, headaches, proptosis, and necknode(s) | CT scan EUA and biopsy Nasal endoscopy and biopsy | Surgery, radiotherapy, chemotherapy, targeted therapy (levels 5 andabove) |
| Nasopharynx | Nasal obstruction, epistaxis | CT scan, EUA, and biopsy | Same |
| Oropharynx | Sore throat, mass, pain radiating to the ear, trismus, bleeding | CT scan, EUA, and biopsy | Same |
| Hypopharynx | Pain on swallowing radiating to the ear, increasing dysphagia, nodes,and hoarseness | Barium swallow, CT scan, endoscopy, and biopsy | Same |
| Larynx | Persistent hoarseness, stridor, cough, neck nodes appear late | Endoscopy, CT scan, and biopsy | Same |

**Table 9.2 continued :Common malignancies, clinical manifestations, investigations, and management options**

| Tumour | Clinical features | Investigations | Management |
|---|---|---|---|
| Leukaemias occur in children and adults. Can be acute or chronic, lympho- blastic, lymphocytic or myelogenous | Anaemia Bone pains Haemorrhagic tendencies, epistaxis and gum bleeding Repeated infection, malaise | Haemogram Bone marrow Cytochemistry Flow cytometry | Refer to oncologist/ haematologist for specialized care (level 5 and above) for chemotherapy. For chronic leukaemias, drugs used depend on type and disease phase. For acute leukaemias, drugs used depend ontype and treatmentphase. |
| Burkitt's lymphoma (predominant in children, also seen in young adults, rarely in older adults, but more in those with AIDS) | Usually a jaw tumour May also present as an abdominal mass OR central nervous system tumour | Biopsy of the mass for histology, fine needle aspiration cytology, immunohistochemistry. Haemogram, bone marrow, x-ray, Ultrasound scan, CT scan, PET scan Lumbarpuncture | Refer for specialized care (level 5 and above) Chemotherapeutic protocols commonly combine cyclopho- sphamide, doxorubicin, methotrexate, vincris- tine, andprednisone |
| Hodgkin's disease (seen in children and adults) | Lymph node enlargement, usually cervical Splenomegaly abdominal masses | Haemogram Chest x-ray Lymph node biopsy for histology and immunohistochemistry Bone marrow | Refer for specialized care (level 5 and above) for chemo- therapy with or without radiotherapy Chemotherapy commonly used combines doxorubicin, bleomycic, vinblastine, dacarbazine (ABVD) |
| Nephroblastoma (Wilms' tumour) A paediatric tumour | Average age 2 years: Embryonal tumour Early childhood Painless loin mass (abdominal mass) Fast growing | Full haemogram U/E in normal IVU (Intravenous urography) shows displaced calicyes FNAC shows malignant embryonal tumour cells CXR formetastasis | Refer to specialized care: Chemotherapy and surgery – nephrectomy with post surgical chemotherapy has good prognosis (level 5 and above) Drugs used include actinomycn D, doxorubicin,vincristine andcyclophosphomide |
| Neuroblastoma | Embryonal tumour Abdominal mass in loin region Markedly elevated blood pressure Fast growing often crossingmidline Child is sick looking | Full haemogram IVU shows caudally displaced normal kidney FNAC – malignant embryonal cells Ultra sound shows supra renal tumour with normal kidney CXR – look for metastasis, 24-hour urine – VMA grossly elevated | Refer to specialist centre (level 5 and above) Treatment depends on stage and involves surgery, radiotherapy and chemotherapy. The chemotherapy pro- tocols commonly used combine vincristine, actinomycin D, doxoru- bicin, and ifosfamide. Carboplatin and etopo- side are also used. |

*Continued*

**Table 9.2 *,continued***

| Tumour | Clinical features | Investigations | Management |
|---|---|---|---|
| Dysgerminoma | Commonest mid- line tumour in neo- natal period Commonest in ovary, testis, thymus, sacrococcygeal (most dramatic – teratoma) Presents with pressure symp- toms; may ulcerate especially when malignant | Plain x-ray may show calcification U/S – defines extent/site of tumourFoetoproteintum our marker | Good prognosis. Surgical excision Radiotherapy and chemotherapy are highly effective. Etoposide, bleomycin, and cisplatin combination commonly used. |
| Rhabdomyo-sarcoma | Tumour of muscle Juvenile and adult variants occur. Can occur anywhere; commonest in pel- vis; bladder, vagina may present with a fungating mass (sarcoma botryoid) May ulcerate and bleed | Good physical examination: Full haemogram U/S, CXR CT scanwhen available Biopsy FNAC | Juvenile type favourableprognosis, adult variant poor prognosis Surgery and chemotherapy used. Doxorubicin,ifosfamide or doxorubicin, cyclophosphamide, vincristine combinations used |
| Retinoblastoma | Age usually below 3 years Inherited through chro- mosome 13 May be unilateral or bilateral Yellowish whitish reflex | Skull x-ray Urine catecholamines Fundoscopy CT scan head | Refer to ophthal- mologist and oncologist for specialized treatment (level 5 and above) Surgery and chemo- therapy are offered. Combination of cyclophosphamide, doxorubicin, vincristine, or etoposide and carboplatin |
| CNS tumours | Headache, convul- sions, vomiting, papilloedema disturbance of gait & vision | X-ray skull CT scan MRI scan | Refer to neurosurgeon (level 5 and above) Surgery, radiotherapy, chemotherapy |

# 10. Haematological Conditions

## 10.1 Anaemia

Patients with anaemia have a reduction in total red blood cell mass, decreased concentration of red blood cells (RBC) and haemoglobin (Hb) in the peripheral blood and a corresponding decrease in the oxygen carrying capacity of the blood. Normal Hb levels are:

- Males:13.5–17.5g/dl
- Females:12.0–16.0g/dl.

**Common causes of anaemia in Kenya are:**

- Haemolysis due to infections especially malaria and haemoglobinopathies, especially sickle cell disease.
- Iron deficiency due to chronic blood loss, nutritional deficiency and intestinal parasites, e.g., hookworm.
- Bone marrow depression (aplastic anaemia).

**Clinical Features**

Meticulous history is essential, e.g., history of previous hospitalization fo rsickle cell, blood loss due to menorrhagia. Clinical features include irritability, listlessness, anorexia, easy fatigability, and pallor of the mucous membranes (conjunctivae, lips and tongue), nail beds, and palms. There may be splenomegaly and a short, soft, apical "haemic" systolic murmur. Severe cases may present in heart failure and shock.

**Investigations**

- Full haemogram/Hb estimation
- Thin blood film examination for cell morphology and blood parasites
- Stool for ova of helminthes, occult blood
- Urinalysis
- Bone marrow
- Sickling test/HB electrophoresis

**Management**

**Identify the cause and treat:**

- Malaria:
  - Give a full course of an appropriate antimalaria drug. Thereafter give antimalaria prophylaxis [see section on malaria] for 3months. If the spleen is palpable, continue prophylaxis until it is not palpable. - -
- Iron:
  - Give iron orally if the anaemia is mild or moderate. Adults: ferrous sulphate 200mg 8 hourly with folate 5mg once daily, continue for a minimum of 3 months after normal HB levels arer eached.
  - Give parenteral iron in patients who cannot receive transfusion, with chronic renal failure, or in inavailability of blood. For those who are unable to tolerate oral iron or if compliance is poor, consider iron sucrose or other similar. This also replenishes body stores of iron.
- Folic acid: Give to all patients who have malaria and anaemia. Dose is 5mg once daily.
- Hookworm treatment:
  - Give albendazole 400mg STAT for adults

- ♦ Sickle cell anaemia:
- Folic acid, malaria prophylaxis (see Section 7.1.1,malaria)

**Blood transfusion**

- ♦ Use blood only when required to save life.
- ♦ Do not give blood transfusion routinely unless the haemoglobin level is<6g/dl, or patient has early features of haemodynamic instability. The rate of loss of blood should guide the decision on bloodtransfusion.

- Transfuse any patient if the haemoglobin is less than 8g/dl and there is also:
- More than 20% blood loss (more than 1 litre in an adult).
- Active bleeding with shock, hypotension, cold extremities, slow capillary refill.

**Admit patients with:**

- ♦ Severe anaemia.
- ♦ Active and severe bleeding.
- ♦ Anaemia (any degree of severity) that is accompanied by pneumonia, heart failure, dizziness, confusion, oedema.



## 10.2 SICKLE CELL DISEASE (ANAEMIA)

This is a chronic haemolytic anaemia found mainly in Nyanza, Western and Coast regions. Sickle cell anaemia is characterized by sickle-shaped RBCs as a result of homozygous inheritance of HBS. In HBS, amino-acid valine is substituted for glutamic acid in the position 6 of the b-chaun. This Hb polymerizes at sites of low partial pressures of oxygen (PO2) and the RBCs assume the “sickle shape”. Such cells adhere to vascular endothelium and plug small capillaries and arterioles leading to occlusion and infarction. Because sickled RBCs are fragile and cannot withstand the trauma of circulation, haemolysis occurs in the small blood vessels. These abnormal RBCs are also destroyed within the spleen.

**Clinical Features**

- ♦ Impaired growth and development
- ♦ Susceptibility to infections (malaria, H. influenza,pneumococcal)
- ♦ Anaemia and mild jaundice
- ♦ Hepatosplenomegaly in young children
- ♦ Bone pain (especially long bones in children)
- ♦ Pain and swelling of the hands and feet (hand and foot syndrome)
- ♦ Arthralgia with fever may occur
- ♦ Avascular necrosis of the femoral head is common
- ♦ Severe abdominal pain with vomiting
- ♦ Occlusion of major intracranial vessels may lead to hemiplegia, cranial nerve palsies and other neurological deficits
- ♦ Acute chest syndromes (sudden onset of fever, chest pain, leukocytosis, and pulmonary infiltrates on x-ray) may be fatal.
- ♦ Tower shaped (“bossing”) skull.

**Investigations**

- ♦ Full haemogram to include peripheral smear,Hb
- ♦ Sickling test
- ♦ Hb electrophoresis
- ♦ X-ray:
  - Long bones: Cortical thinning noted with irregular bone densities and new bone formation.

**Management**

Transfuse for very severe anaemia (aplastic crisis, infections)

**Sickle Cell Crisis**

There are 3 types of crises: thrombotic (vaso-occlusive, painful or infarctive), aplastic (sequestration), and haemolytic.

♦ Management of the crisis

- Give IV or oral fluids until they produce dilute urine.
- Give analgesics regularly. In the acute phase if pain is severe, give narcotic analgesics (e.g., morphine injection 10mg PRN)

♦ Treat infections vigorously and promptly if present by use of ceftriaxone 1g IV once daily for 7 days or coamoxiclav 1.2g 8 hourly for 7 days.

- Treat malaria if present endemic areas.
- Give supplementary folic acid but AVOID iron.

♦ Blood transfusion

- Blood must be given immediately at the time that it is needed. Re-evaluate the patient immediately prior to transfusion to ensure that blood is still required to save life.
- Use only blood that is free of HIV, has been properly grouped and cross matched, and is in the correct bag labelled for the patient.
- Remove the bag of blood from the Blood Bank refrigerator just before transfusion.
- Never transfuse blood that has been out of the refrigerator for more than one hour or out of the donor for more than 21days.
- Give frusemide (1mg/kg STAT) IV at the beginning of the transfusion (but only if the patient is NOT actively bleeding). If patient has heart failure, give frusemide immediately; do not wait until blood is available.
- Give antimalaria drugs (full course) to all patients having blood transfusion only in malaria endemic areas.
- Transfusion of adults requires a minimum of 2 units of blood. Transfusion of only 1 unit in an adult is probably not needed.

♦ Management of transfusion reactions:

- If the patient develops fever, skin rash or becomes ill, then:
  - Stop blood transfusion immediately.
  - Give chlorpheniramine 5mg IV STAT ***OR*** 5mg IM STAT.
  - Return blood to the bank with a fresh sample of patient's blood.
  - Monitor urine output.

♦ Monitor cardiovascular and renal function

- If hypotension develops start IV fluids.

- Hydroxyurea should be given to patients with more than 3 crises per year. This can be started at a dose of 10mg/kg orally and escalated by 5mg/kg to a maximum dose of 25mg/kg/day.

- -

# 11. Conditions in Pregnancy

## 11.1 Anaemia in Pregnancy

This is a major obstetric problem in Kenya. In Kenya, anaemia is generally accepted as Hb <10gm%. Degrees of anaemia are categorized as "mild anaemia" at haemoglobin levels of Hb 8–10gm, "moderate anaemia" at Hb 6–7gm, "severe anaemia" at Hb 4–5gm, and "very severe anaemia" at below Hb 4 gm.

In severe anaemia the pregnancy is in danger of abortion, premature labour, or IUFD, while in very severe anaemia the mother's life is also in danger. Most cases are due to iron deficiency resulting from dietary deficiency, or blood loss from hookworm infestations, haemolysis due to malaria and sickle cell disease. Anaemia can also be due to folate deficiency resulting from inadequate intake. Iron deficiency and folic acid deficiency often occur together causing "Dimorphic Anaemia".

**Clinical Features**

General weakness, dizziness, pallor, and oedema may occur, while haemolytic anaemia may be associated with jaundice and hepatosplenomegaly.

**Investigations**

- Full haemogram (Hb, PCV,PBF)
- Stool for hookworm ova and schistosomal ova, where applicable
- Urine urobilinogen and schistosomal ova, where applicable
- Blood slide for malaria parasites
- Sickling test

**Management**

- Raise Hb (oral or parenteral haematinics,transfusion).
- Eradicate cause–dietary deficiency, treat malaria, treat hookworms, give haematinics if dietary deficiency exists.
- Prevent recurrence.

**Table 11.1: Management of anaemia in pregnancy**

| Severity of anaemia | Corresponding level of haemoglobin | Management options recommended |
|---|---|---|
| Mild | 8–10 | Treat cause.Oralhaematinics, as for prophylaxis |
| Moderate | 6–7 | As above.Iron dextran (Imferon) |
| Severe | 4–5 | As above.Transfuse and iron depot. |
| Very Severe | Below 4 | Resuscitation and treat as for severe anaeemia – |

**Prevention**

- Prophylaxis iron throughout pregnancy
- Antimalaria prophylaxis (see Section 11.5, Malaria in Pregnancy)
- Balanced diet
- Routine antenatal screening at first visit and visits neart e

## 11.2 Cardiac Disease in Pregnancy

In Kenya, the origin of cardiac disease in pregnancy is often rheumatic involving the valves.

**Clinical Features**

History of rheumatic fever in childhood, known rheumatic heart disease. Features of dyspnoea, palpitations, body oedema, cough, easy fatigability, evidence of heart enlargement, murmurs, thrills, left parasternal heave, raised jugular venous pressure, tachycardia. Hepatomegaly, ascites and basal crepitations may be present.

**Investigations**

- Routine antenatal profile (Hb, VDRL, blood group,urinalysis)
- Urine for microscopy and culture and sensitivity
- Shielded chest x-ray in early pregnancy
- ECG, echocardiogram

**Approach to Management**

**This depends on the following functional classification devised by the New York Heart Association:**

- Class I –Asymptomatic
- Class II – Symptomatic with heavy work
- Class III – Symptomatic with light work or exercise
- Class IV – Symptomatic at rest

**Approach to management of cardiac disease in pregnancy according to classification:**

- ClassI and II are managed as outpatients until 34–36 weeks of gestation, when they are admitted for bed rest and observations in hospital.
- Class III and IV are admitted on first visit at any gestation for the entire duration of pregnancy.

**Management – Supportive**

- Order bed rest.
- Give haematinics supplementation.
- Treat intercurrent infections.
- Avoid undue physical and emotional stress.
- Do regular urine analysis and culture.
- Ensure dental hygiene.
- Carry out regular U/E estimations.

**Management – Pharmacological**

- Note that digitalization is indicated in imminent and overt cardiac failure, if not previously on digoxin. Rapid digitalization by mouth,1–1.5 mg in divided doses over 24hours, less urgent digitalization 250–500µg daily (higher dose maybe divided).
- Continue maintenance therapy with digoxin 250µg, frusemide 20mg as needed.
- Continue prophylactic benzathine penicillin 2.4 MU monthly.

## 11.3 Diabetes in Pregnancy

Diabetes mellitus is a metabolic disorder characterized by hyperglycaemia**.**

**Clinical Features**

- Overt diabetes: If not already diagnosed, the symptoms include polydipsia, polyuria, weightloss, blurred vision, lethargy. Glycosuria is common but not diagnostic.
- Gestational diabetes: This will occur in 1–5% of pregnancies. Historical risk factors include previous gestational diabetes, family history of diabetes, previous macrosomic infant, previous unexplained stillbirth, polyhydramnios, obesity, and advanced maternalage. Glycosuria maybe present but is not diagnostic.
- Complications of diabetes include Chronic hypertension and nephropathy, pregnancy-induced hypertension, foetal macrosomia, intrauterine growth retardation, polyhydramnios, foetal distress, and foetal hypoglycaemia.

**Investigations**

- Postprandial blood glucose level
- Glucose tolerance test (GTT) to confirm diabetes

**Management**

- Stabilize uncontrolled diabetes in pregnancy in hospital.
- Maintain regular daily physical activity to the extent possible.
- Ensure appropriate diet: 30–35 calories/kg/day, i.e.,1,800–2,400 calories per day, carbohydrate 200g/day and protein 90g/day.

Manage non-insulin requiring gestational diabetes by diet alone and monitor with serial blood sugar. If not controlled by diet, start the patient on insulin soluble under the supervision of the diabetic team during admission. Start with 10 units of soluble insulin TDS subcutaneous to maintain the sugar under 7–10mmol/L. Change the dosage as required. Once controlled, convert to insulin 70/30; give 2/3 of the daily dose of soluble in the morning and 1/3 in the evening.To prevent PET, start aspirin 60–75mg OD to start at 16th week and stop at 36th week to avoid excess bleeding.

**Delivery**

- Non-insulin requiring gestational diabetic should be delivered at term.
- Well controlled insulin-requiring diabetic should go to 38 weeks before delivery.
- Insulin dependent diabetic with hypertension, renal, retinal or cardiac disease, PET, intrauterine growth retardation must be delivered at 37th week. When in labour give 1/2 of the daily dose as souble insulin STAT subcutaneously and then put the other half of the daily dose as soluble insulin in an infusion of 1 litre 5% dextrose to be given given over 8 hours.
- Intrapartum blood glucose is monitored hourly and insulin doses adjusted accordingly in small doses (discontinue usual insulin regime) as soluble insulin subcutaneously to maintain the sugar at 7–10mmol/L.

**Postpartum Care**

- Insulin requirement usually reduces after delivery, so serial glucose monitoring should be done hourly for the first 4 hours and then monitor 2 hours after a meal. Give soluble insulin subcutaneously and this needs to be done while allowing adjustment of insulin dose to achieve stable control.

**Patient Education**

- Pre-pregnancy counselling: Achieve optimum glucose control before pregnancy to minimize foetal complications in diabetic pregnancy.
- Family planning: Advise on a small family.
- Recommended family planning methods: These include voluntary surgical contraception, barrier methods, norplant, intrauterine device, and progesterone-only pill.

## 11.4 Drugs in Pregnancy

Drugs taken by the mother during pregnancy can be harmful to the developing foetusin a variety of ways. Drugs taken just before delivery can also affect the baby. Table 8.6 provides guidelines on drugs that are considered safe or relatively safe in pregnancy, drugs that should be used with caution and only when necessary, and drugs that are contraindicated.

## 11.5 Malaria in Pregnancy

Falciparum malaria is particularly dangerous in pregnant women. The clinical features of malaria in pregnancy depend, to a large extent, on the immune status of the woman, which in turn is determined by her previous exposure to malaria. (Refer also to Section 7.1.1, on malaria.)

**Clinical Features**

- Non-immune (women from endemic area): These have a high risk of maternal perinatal mortality. Clinical features include acute febrile illness, severe haemolytic anaemia, hypoglycaemia, coma/convulsions, and pulmonary oedema. Abortion, intrauterine death, premature labour, and intrauterine growth retardation are other possible outcomes.
- Semi-immune (women from endemic area): These may be asymptomatic, despite placental infection. They may develop severe anaemia and deliver low birthweight babies. More common in primigravidae than multigravidae. One of the dangers of malaria in these settings is that it is not detected or suspected. Antimalarials should form part of the case management of all women with severe anaemia who are from endemic area irrespective of whether they have a fever or a positive blood slide (see Section 8.10.1, Anaemia in Pregnancy).

**Investigations**

- Hb,PCV
- Blood slide: peripheral blood film for identification of parasites. This may be negative in a woman from endemic areas, despite the presence of malaria parasites in the placenta.

**Table 11.2: Guidelines for drug use in pregnancy**

| Types of medication | Degree of safety for use in pregnancy | | |
|---|---|---|---|
| | **Safe or relatively safe** | **Some risk – Use with caution** | **Contraindicated in pregnancy** |
| Analgesics | Codeine, morphine, pacacetamol, pethidine | Indomethacin, salicylates | |
| Anti-convulsants | Ethosuximide, phenobarbitone, primidone | Clonazepam, phenytoin | |
| Anti-microbials | Ampicillin, amoxycillin, cephalosporins, clidamycin, dicloxacilin, erythromycin, gentamicin, isonizid, miconazole, oxacillin, penicillin | Chloramphenicol, metronidazole, nitrofuratoin, streptomycin, sulfonamides, trimethoprim, rifampicin, kanamycin | Tetracycline |
| Anticoagulants | Dipyridamole, heparin | Dicumarol, warfarin | |
| Antiemetics | Hydroxyzine, meclizine, prochlorperazine | Phenothiazines | |
| Antihypertensive | Hydralazine, methyldopa, propranolol | Diazoxide | Nitroprusside |
| Bronchodilators | Aminophylline, beclomethasone | Cromolyn sodium | |
| Cardiac drugs | Atropine, digoxin, lidocaine, procainamide, quinidine | Dispyramide, nifedipine | |
| Decongestants | Pseudoephedrine | | |
| Diuretics | Frusemide, Hydrochlorothiazide | | Acetazolamide |
| Gastrointestinal drugs | Antacids, cimetidine, ranitidine | | |
| Hypoglycemics | Insulin | | Chlorpropamide, tolbutamide |
| Sedative & psychiatrics | Barbiturates, flurazepam | Diazepam, chlordiazepoxide, haloperidol, lithium, phenothiazines, tricyclic antidepressants | |
| Thyroid preparations | L-thyroxine, propylthiouracil | | Iodide |
| Vaccines | Polio, tetanus, rabies | | Rubella, measles, smallpox |
| Other drugs | Ferrous sulphate, probenecid | | Antineoplastic drugs, oestrogens, DES |

**Management – Supportive**

- Check blood sugar regularly as hypoglycaemia is a common problemin women with severe disease.
- Correct dehydration.
- Evacuate if incomplete/inevitable abortion.
- Deliver if foetal death or established labour.

**Management – Pharmacological**
**See Section 7.1.1, on malaria.**

## 11.6 Pueperal Psychosis

The following aspects in the patient's history may help to identify high-risk patients and to facilitate early identification of patients with puerperal psychosis:

- Family history of major psychological illness of close relative, e.g.,mother.
- Major emotional complications during and after a previous pregnancy.
- "Reaction" of current pregnancy.
- "Fear" of labour from a previous experience.
- Traumatic childhood.
- Deprivation of emotional support during adult life, e.g., single mother.
- Severe prolonged or multiple somatic symptoms with no apparent organic cause during current or previous pregnancy.
- Major sustained mood changes or repeated rapid mood swings or abnormal sleep patterns.
- Refer to Chapter 16, Mental Illness, for clinical features and management.

# 12. Lower Respiratory Tract Conditions

## 12.1 Pneumonia – Adults

This is consolidation of the lung parenchyma due to infection.

**Clinical Features**
Breathlessness, cough with or without sputum (which may be rust coloured), fever, pleuritic chest pain, bronchial breathing, reduced chest movements, reduced breath sounds, tachypnoea, crackles and percussion dullness. Features less pronounced in elderly patients.

**Classification**
- Primary: Occurring in a previously healthy person living in the community. This is usually lobar due to pneumococci. Usually a very short history.
- Secondary: Develops in association with prior respiratory disease, immunocompromised patients, debilitated patients, alcoholics, or post operative patients.

**Investigations**
- Haemogram - PBF,WBC
- Sputum microscopy
- Chest x-ray PA

**Management – Community Acquired Pneumonia**

**Outpatients**
- IM benzyl penicillin 2 MU STAT, then amoxicillin 500mg TDS for 7days.
- If penicillin allergy is present: Erythromycin 500mg QDS for 7days. Alternative antibiotics include cotrimoxazole.
- Analgesics: Paracetamol ***OR*** acetyl salicylic acid (aspirin).

**Inpatient care**
- Admit for inpatient care in the presence of the following:
  - Cyanosis
  - Respiratory distress (RR >25 perminute)
  - Heart failure or pleural effusion
  - More than one lobe is involved.
  - Poor response as outpatient.
  - Patient is dehydrated.
  - Secondary pneumonia is suspected.
- After admission, give the following treatment:
  - IV/IM Crystalline penicillin 2 mega-units QDS till response, then discharge on amoxicillin 500mg TDS. If allergic, give erythromycin 500mg QDS or cotrimoxazole for 5 days.
  - If no response, consider investigation for TB.

**Management – Secondary Pneumonia**
- Admit patient.

- Treat with benzyl penicillin 2 mega units IM IV 6 hourly + gentamicin 240mg IM IV once a day 5 days ***OR*** IV ceftriaxone 2g every 24 hours ***OR*** erythromycin 500mg 6 hourly for 5days.
- In case of aspiration add metronidazole 500mg IV 8 hourly or use coamoxiclav 1.2g 8 hourly in place of benzylpenicillin.
- If staphylococcus is suspected add flucloxacillin 500mg IV 6 hourly.
- As a special precaution, consider pseudomonas and staphylococcus.

**Prevention**

**Give Pneumovaccine to those who have sickle cell disease and with impaired spleen function.**

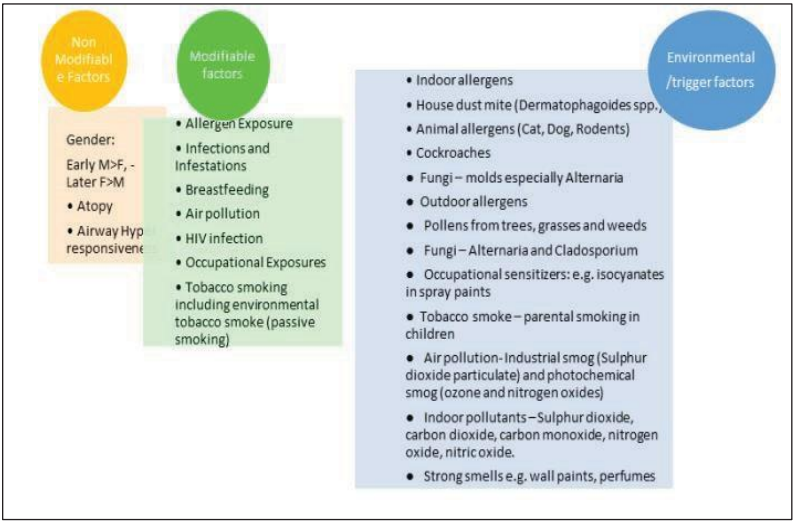
## 12.2 Asthma

According to the global initiative for asthma (GINA), asthma is defined as a chronic inflammatory disease process of the airways.

The essential elements of asthma from this definition include:

- The presence of airway inflammation.
- Airway hyper responsiveness.
- Recurrent episodes of symptoms of wheezing, breathlessness, chest tightness and coughing.
- Reversible airways obstruction that is demonstrable by changes in lung function tests.

**Figure 12.1: Risk factors for Asthma**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Clinical Features**

Asthma has variable clinical presentation, but the classical symptoms are wheezing, chest tightness, difficulty in breathing and cough. Lung function tests (spirometry) should be done where available. Below is a table summarizing both typical and atypical presentation of asthma.

**Table 12.1: Typical and atypical presentation of asthma**

| Typical presentation | Atypical presentation |
|---|---|
| • Frequent episodes of cough, chest tightness, breathlessness and wheezing that vary in duration and severity<br>• Symptoms occur mainly at night and wake up patient, usually in the early hours of the morning<br>• Symptoms disappear spontaneously or after bronchodilator use<br>• Persistent breathlessness can occur in the most severe form of asthma, due to progression from reversible to irreversible airflow limitation<br>• Severe progression is rare and linked to irreversible airway remodeling<br>• Several risk and trigger factors usually present | • Mainly in children<br>• Recurrent attacks of cough, particularly in the evening and/or at night, which do not respond to symptomatic treatment<br>• Chest tightness with wheezing that occurs only after exercise<br>• Clinical pattern similar to an acute respiratory infection but frequently recurs during a short period |

Source: Integrated Guideline for Tuberculosis,Leprosy and Lung Disease, 2021

**Diagnosis**

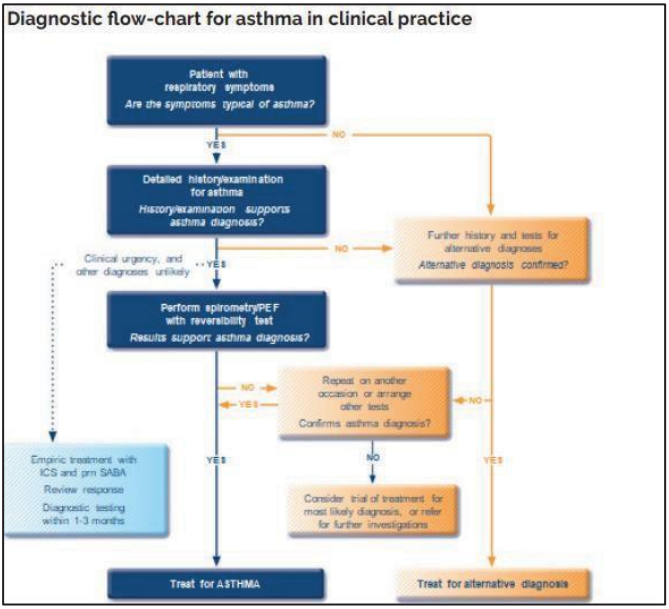
♦ **History**

- Is there recurrent or episodic wheeze, cough, chest tightness or shortness of breath
- Are the symptoms particularly troublesome at night or early morning?
- Are the symptoms triggered by factors such as dust, cold exposure, strong smells
  or exercise.

  Is there a consistent response to asthma-specific treatment?
- Is there a family history of allergy/atopy i.e allergic conjunctivitis, allergic rhinitis,
- asthma, eczema and food (protein) allergy?

♦ **Obtain a Lung Function Test** to assess airway hyper-responsiveness (measure forced vital capacity i.e. FVC, forced expiratory volume i.e. FEV1 and peak expiratory flow i.e. PEF) by Spirometry, where available. Peak expiratory flow meter (PEFM) is cheaper and should be used where there's no spirometery.

**Figure 12.2: Diagnostic flow-chart for asthma in clinical practice**



Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.2: Classification of Asthma**

| Classification | Symptoms | Nocturnal symptoms | PEF or FEV |
|---|---|---|---|
| **Intermittent** | < 1 time week<br>Asymptomatic and normal PEF between attacks | ≤ 2 times a month | ≥ 80% predicted<br>Variability < 20% |
| **Mild persistent** | > 1 time a week, but < 1 time a day | > 2 times a month | ≥ 80% predicted<br>Variability < 20%-30% |
| **Moderate persistent asthma** | Daily Attacks affect activity | > 1 time week | 60-80% predicted<br>Variability > 30% |
| **Severe persistent asthma** | Continuous Limited physical activity | Frequent | ≥ 60% predicted<br>Variability > 30% |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Management of asthma**

The aim of management is to achieve asthma control and to return patients to productive lives.

*Goals of asthma care/management are to:*

- Achieve and maintain control of symptom
- Prevent asthma exacerbation
- Maintain lung function as close to normal as possible
- Maintain normal level of activity including exercise
- Avoid adverse effects of asthma medications
- Prevent development of irreversible airflow limitation
- Maintain normal growth velocity in children
- Prevent asthma mortality

***Management of acute asthma***

Acute asthma is classified as:

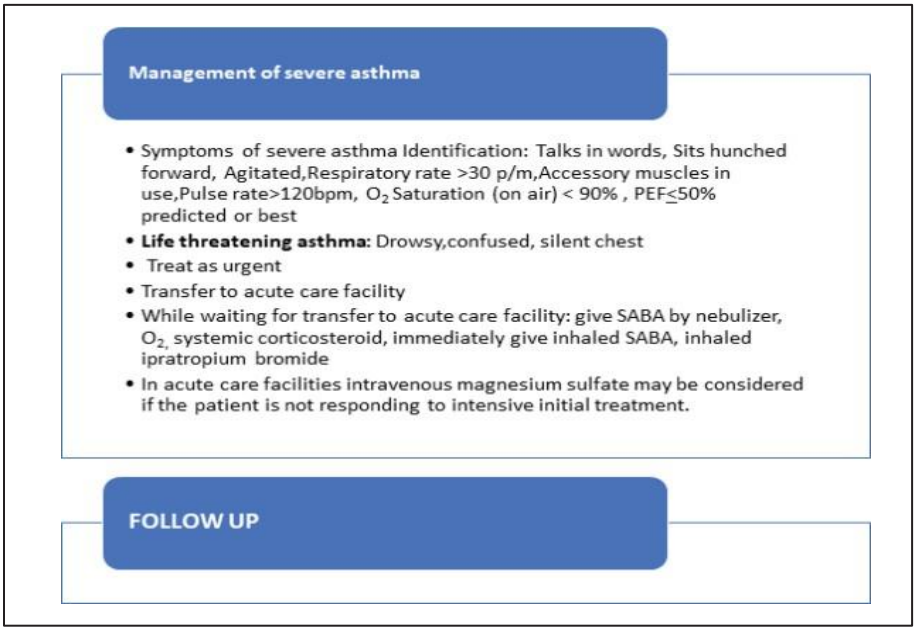
- Mild asthma attack
- Moderate asthma attack and
- Severe asthma attack.

**Table 12.3: Classification of Asthma Medications**

| | |
|---|---|
| **Relievers** | They reverse broncho-constriction and relieve its symptoms. They include rapid and short acting, rapid and long acting bronchodilators.<br>Short acting bronchodilators: Salbutamol |
| **Controllers** | They are taken daily to keep asthma under control through their anti – inflammatory effects |
| | Long Acting $_2$ Agonists (LABA) have anti-inflammatory effects, are used in combination with inhaled corticosteroids for the long term control of asthma, they also inhibit mast cell mediator release, plasma exudation and reduce sensory nerve activation.<br>Beclomethasone Dipropionate<br>Budesonide<br>Ciclesonide<br>Fluticasone Propionate |

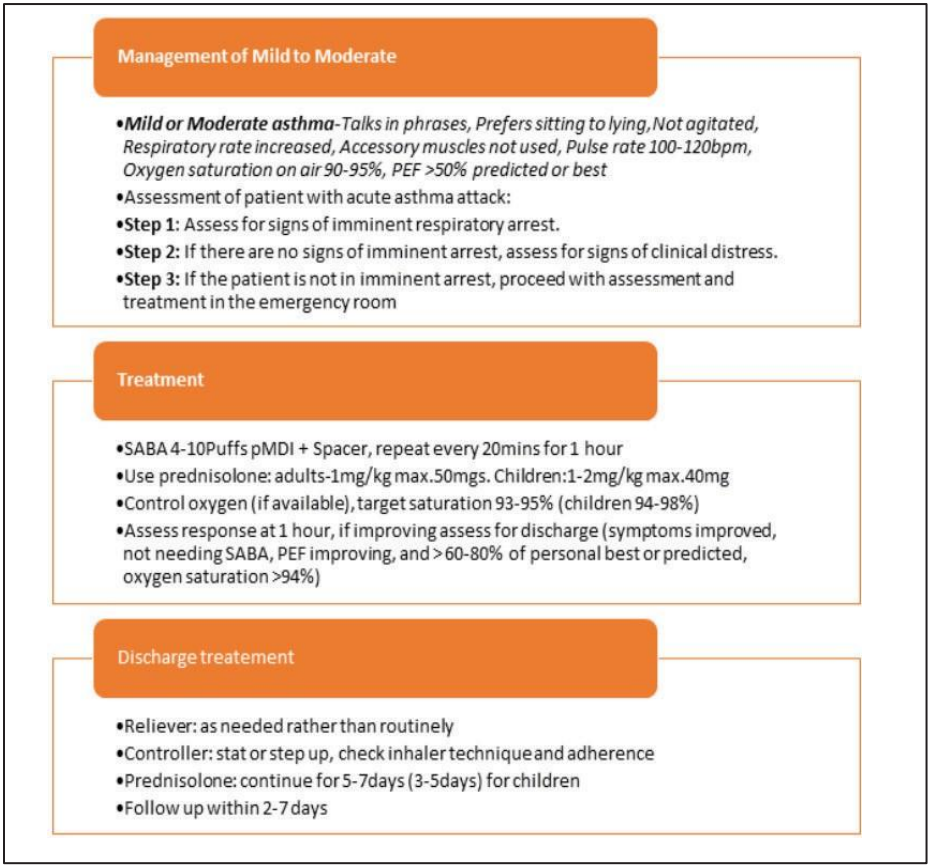
Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Figure 12.3: Management of Severe Asthma**



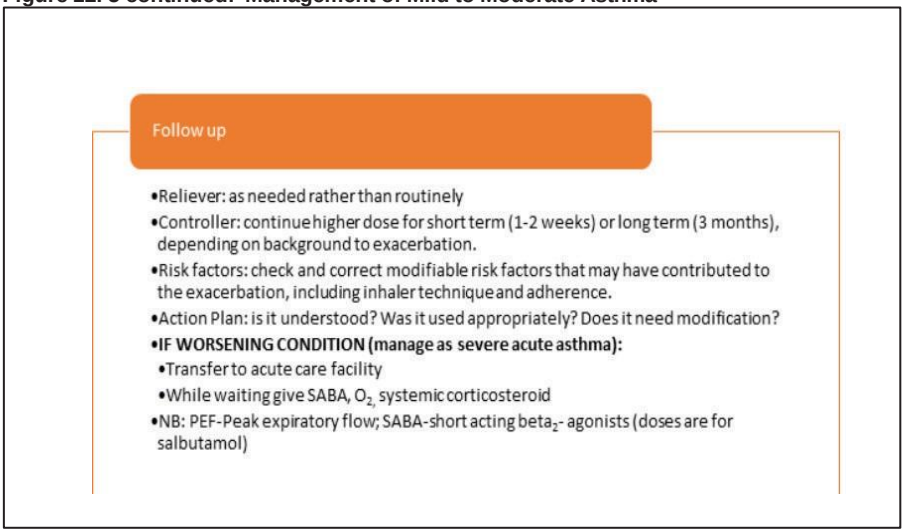

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Figure 12.4: Management of Mild to Moderate Asthma**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Figure 12. 5 continued: Management of Mild to Moderate Asthma**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Management of chronic asthma**

***Routine care for asthma patients***

Asthma is a chronic illness and therefore the clinical team and patients need to develop a long-term plan for the patient management. The patient – health provider partnership includes:

- Personalized education: ensure the following is included in the patient education. Basic information about the disease.
  - Medication including relievers and preventers.
  - Potential side effects of medicines.
  - Training on the medicine inhaler technique.
  - Recognition of worsening asthma and actions to be taken.
- Self-monitoring of asthma control.
  - If no response, consider investigation for tuberculosis.
  - Regular review to assess control and adjust treatment as may be necessary.
  - Identification and avoidance of symptom trigger factors (indoor and outdoor pollutants).
- A written asthma management plan.
- Regular assessment of patients for their symptom control.

**Table 12.4: Assessment of Asthma Symptom Control**

| In the past 4 weeks, has the patient had; | Yes | No |
|---|---|---|
| Daytime symptoms of asthma (cough, wheeze, shortness of breath more than twice/week etc)? | | |
| Any night waking due to asthma? | | |
| Reliever needed more than twice/week? | | |
| Any activity limitation due to asthma? | | |
| **Score: Well controlled - None of these, Partially controlled - 1-2 of these, Uncontrolled - 3-4 of these** | | |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021



**Medication for chronic care**

- Long Acting β2 Agonists (LABA) have anti-inflammatory effects and are used in combinationwith inhaled corticosteroids (ICS) for the long-term control of asthma. Anti-inflammatory therapy (ICS) forms the backbone of asthma control.
- All asthma patients should be on inhaled corticosteroids. Bronchodilators should not be used without combining with ICS, since asthma is an inflammatory disease.
- Frequent use of SABA can lead to excess deaths due to side effects and failure to address the inflammatory aspect.

**Table 12.5: Other medications used in the treatment of asthma**

| | |
|---|---|
| Anticholinergics | They are used for the treatment, especially in the acute care setting. Ipratropium bromide is usually combined with a short acting $B_2$ agonist |
| Leukotriene Modifiers | They are used as add on therapy in patients who fail to achieve control.<br>Used with low dose inhaled corticosteroids or as alternatives to low dose inhaled corticosteroids and in aspirin induced asthma (AIA).<br>It is useful in the presence of allergic rhinitis and asthma to relieve both nasal and chest symptoms |
| Systemic Corticosteroids | They are recommended for patients with moderate to severe acute exacerbations of asthma. In some patients with steroid dependent asthma the lowest possible dose of should be used |
| The cromones, Anti IGE | Refer to the National asthma Guidelines |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.6: Approaches to the differential diagnosis of asthma**

| *Diagnosis* | *Evaluation* |
|---|---|
| Upper Airway Disease<br>·Adeno-tonsillar hypertrophy<br>·Rhino-sinusitis<br>·Post Nasal Drip | • Clinical ENT examination<br>• Sinus X-ray<br>• CT Paranasal Sinuses<br>• ENT Specialist referral |
| | |
| Congenital Structural Bronchial Disease<br>·Tracheo-bronchomalacia<br>·Cartilage Rings<br>·Cysts<br>•Webs | • Bronchoscopy<br>• CT Scan Chest |
| | |
| Bronchial/ Tracheal Obstruction<br>·Vascular Rings/ Slings<br>·Enlarged Cardiac Chamber<br>·Lymph Node Enlargement from TB or lymphoma | • CXR<br>• CT Scan Chest<br>• Echocardiogram<br>• Mediastinoscopy |
| | |
| Endobronchial Disease<br>·Foreign body/tumour | • Chest X-ray<br>• Bronchoscopy |
| Esophageal / Swallowing Problems<br>·Reflux<br>·Uncoordinated Swallowing<br>·Laryngeal Cleft<br>·Tracheo-esophageal Fistula | • Upper GI Studies/Barium Swallow<br>• Upper Endoscopy<br>• PH Probe<br>• Milk Scan |
| Pulmonary Suppuration<br>·Cystic Fibrosis<br>·Primary Ciliary Dyskinesia<br>·Severe Immunodeficiency Syndromes<br>·Agammaglobulinemia | • Sweat/Genetic testing<br>• Lung/Sinus Biopsy/Molecular Genetic Testing<br>• Complete Blood Count<br>• Immunoglobulin Levels<br>• Complement Levels |
| Miscellaneous<br>·Post Viral Wheeze<br>· Acute Bronchiolitis.<br>•Laryngo-Tracheobronchitis | • Characteristic Viral Syndrome<br>• Antigen Tests for RSV<br>• Viral Cultures<br>• PCR<br>• CXR |
| Chronic obstructive pulmonary disease | • Spirometry |
| Congestive heart failure | • Echocardiogram<br>• Serum BNP |
| Pulmonary embolism | • Chest X-ray<br>• Lower limb doppler ultrasound<br>• CT pulmonary angiogram<br>• Echocardiogram<br>• Perfusion/ventilation scans |
| Tumors | • Contrast enhanced CT Scan Chest |
| Pulmonary eosinophilia | • Sputum eosinophilia<br>• Elevated eosinophil counts in full haemogram. |
| ACE Inhibitor induced cough | Medication review and discontinuation of ACE inhibitors |
| Vocal cord dysfunction | Bronchoscopy |
| Laryngeal dysfunction | Laryngoscopy |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

## 12.3 Chronic Obstructive Pulmonary Disease (COPD)

This is a progressive lung disease that makes breathing difficult. It encompasses chronic bronchitis and empysema. It is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases.

**Table 12.7: Risk factors for COPD**

**RISK FACTORS**

- Age and Sex: Elderly >40 years and Female > Male
- Environmental factors:
  - Active tobacco smoking
  - Secondary tobacco smoking
  - Indoor air pollution - bio fuels and coal

  Outdoor air pollution-also contributors to the lungs' total burden of inhaled particles
- Occupation exposure:
  - Organic and inorganic dust
  - Chemical agents and fumes
- Genetic factors-
  - Severe hereditary deficiency of alpha-1 antitrypsin (AATD), the gene encoding matrix metalloproteinase-12 (MMP-12) and glutathione S-transferase have also been related to decline in lung function or risk of COPD
- Lung growth and development – any factor that affects lung growth during gestation and childhood (low birth weight, respiratory infections, etc.) has the potential to increase an individual's risk of developing COPD
- Socio-economic status -poverty is consistently associated with airflow obstruction and lower socio-economic status is associated with an increased risk of developing COPD.
- Asthma and airway hyper-reactivity - asthma may be a risk factor for the development of airflow limitation and COPD
- Infections-
  - History of severe childhood respiratory infection has been associated with reduced lung function and increased respiratory symptoms in adulthood
  - Post Tuberculosis lung damage
  - Chronic bronchitis - may increase the frequency of total and severe exacerbations

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.8: Dignosis of COPD**

Consider COPD in a patient with:

- Dyspnea that is progressive, persistent and worsens with exercise
- Chronic cough which may be intermittent and productive or not productive
- Chronic sputum production
- Wheezing
- History of exposure to risk factors which may include tobacco smoking, smoke from home cooking and heating bio fuels, occupational dusts and chemicals
- Patient with above symptoms and previously treated for Tuberculosis
- Age of 40 years and above
- Family history of COPD

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.9: Lung function testing: Spirometry**

- Spirometry is the Gold standard for clinical diagnosis and monitoring COPD
- Post bronchodilator FEV1/FVC less than 70% confirms the presence of persistent airflowlimitation
- To diagnose, manage and follow up COPD patients one should have access to Spirometryfacilities

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Distinguishing Asthma and COPD**

When a patient presents with a chronic cough, other causes of chronic cough should be excluded. These include chronic bronchitis, tumors, post-infectious cough, tuberculosis and heart failure. COPD and asthma both present with cough, wheezing, chest tightness and difficulty in breathing. The table below summarises the differences in the two.

**Table 12.10: Difference between asthma and COPD**

| ASTHMA | COPD |
|---|---|
| • Onset before 20 years of age<br>• Associated hay fever, eczema, allergies<br>• Intermittent symptoms, with normal breathing in between<br>• Symptoms worse at night, early morning, with cold or stress<br>• Personal or family history of asthma<br><br>**Asthma likely**<br>Confirm diagnosis - Give routine asthma care | • Onset after 40 years of age<br>• Symptoms are persistent and worsen slowly over time<br>• Cough with sputum starts long before difficult breathing<br>• Client is or was a heavy smoker and/or had TB<br>• Previous diagnosis of COPD<br>**COPD likely**<br>• Confirm diagnosis - Give routine COPD care |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.11:Management of COPD**

| Goals of COPD management |
|---|
| • To relieve symptoms<br>• Prevent disease progression<br>• Prevent and treat complications and exacerbations<br>• Reduce risk of death |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

Assess COPD severity and the extent of exacerbation. The assessment is aimed at determining disease severity, its impact on patient's general health status, risk of exacerbations and death.

**Table 12.12:Classification of the severity of COPD based on post bronchodilator FEV1 (in patients with FEV1/FVC <0.70)**

| | | |
|---|---|---|
| GOLD 1 | Mild COPD | $FEV_1 \geq 80\%$ predicted |
| GOLD 2 | Moderate COPD | $50\% \leq FEV_1 < 79\%$ predicted |
| GOLD 3 | Severe COPD | $30\% \leq FEV_1 < 49\%$ predicted |
| GOLD 4 | Very Severe COPD | $FEV_1 < 30\%$ predicted |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021



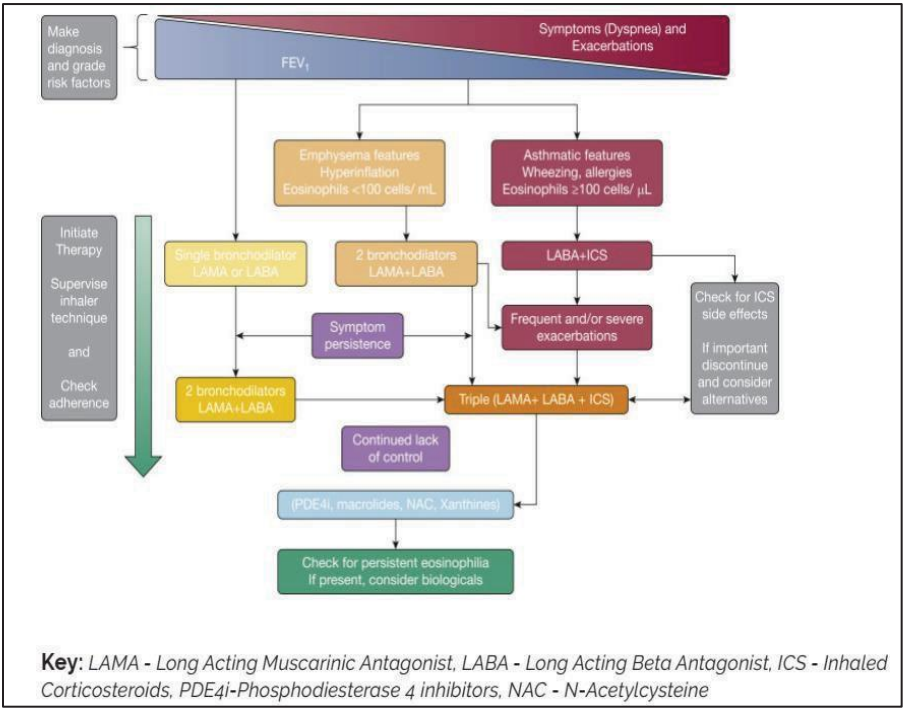
**Assess risk for exacerbations**

Exacerbation of COPD is an acute event characterized by a worsening of the patient's respiratory symptoms that is beyond normal day-to-day variations and leads to a change in medication. The best predictor of frequent exacerbations (2 or more per year) is ahistory of previous treated events.

**Management of COPD includes:**

- Smoking cessation: This has the greatest impact in reducing the disease progression. This can be done through:
  - Patient counseling
  - Nicotine replacement therapy e.g. nicotine patches, nicotine gums, sublingual tablets etc.
  - Institution of smoking prevention and tobacco control strategies (Refer to National Tobacco Control policies)
- Prevention of occupational exposure.
- Reduction of exposure to indoor pollutants e.g. bio fuels in poorly ventilated houses.
- Physical exercise.
- Pharmacotherapy – the drugs used in the management of COPD are aimed at reducing symptoms, frequency and severity of exacerbations and improving health status. They include:
  - Inhaled bronchodilators
  - Inhaled corticosteroids
  - Combined inhaled corticosteroid/ bronchodilator therapy is more effective than individual components. Antibiotics are not recommended except for treatment of suspected bacterial infections.
  - Mucolytic agents for patients with viscous sputum
  - Oxygen therapy - long term administration of oxygen for > 15 hours per day has been shown to increase survival in patients with severe COPD
  - Palliative care/ hospice care is important for patients with advanced COPD which iis marked with deteriorating health status, increasing symptoms, frequent acute exacerbations with frequent hospitalizations and associated comorbidities e.g. cardiovascular diseases, malignancies and progressive respiratory failure.

**Figure 12.6: Guide for the management of COPD based on severity of the disease**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Treatment of acute exacerbations**

Lower respiratory tract infections (LRTI) occur commonly in patients with COPD. In patients previously treated for tuberculosis, repeat tuberculosis tests only if other TB symptoms develop. If patient's sputum increases or changes in color to yellow/green, treat for LRTI:

- Give doxycycline 100mg 12 hourly for 10 days or amoxicillin 500mg 8 hourly for 10 days.
- Give short course oral prednisone 40mg daily for 7 days if patient has severe COPD
- High dose (800pg) inhaled corticosteroids are effective in patients with severe COPD with more than 2 infective exacerbations per year.
- Give influenza vaccine yearly and pneumococcal vaccine 5 yearly.
- Identify and manage complications. Treat fluid retention with a low dose diuretic.
- Encourage patient to exercise daily e.g. walking, gardening, household chores, using stairs instead of lifts etc.
- Refer to a chest specialist for follow-up of the disease once patient is stable.

## 12.4 Post Tuberculosis Lung Disease (PTLD)

Post TB Lung Disease (PTLD) is defined as a chronic respiratory abnormality with or without symptoms attributable, at least in part, to previous pulmonary TB. Patients with PTLD may present with persistence of symptoms or decline in lung function despite successful completion of treatment or cure.

**Table 12.13: Presentation of Post TB Lung Disease**

| Post TB lung disease may present as the following: |
|---|
| 1. Lung scarring (fibrosis) |
| 2. Bronchiectasis |
| 3. Chronic Obstructive Pulmonary Disease (COPD) |
| 4. Lung abscess |
| 5. Aspergillus-related lung disease |
| 6. Spontaneous Pneumothorax |

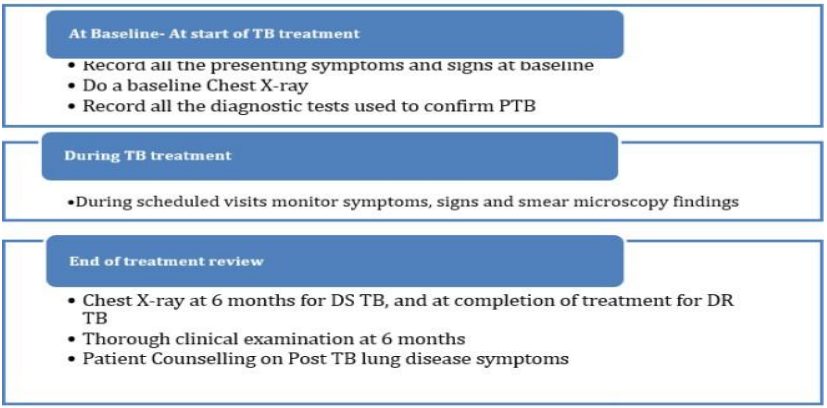
Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

### Clinical Presentation of PTLD

This is dependent on the type of lung impairment and presentation is usually variable. The key presentation of PTLD is persistence of clinical symptoms or occurrence of new symptoms e.g. cough, chest pains, breathlessness or decline in lung function despite successful completion of PTB treatment.

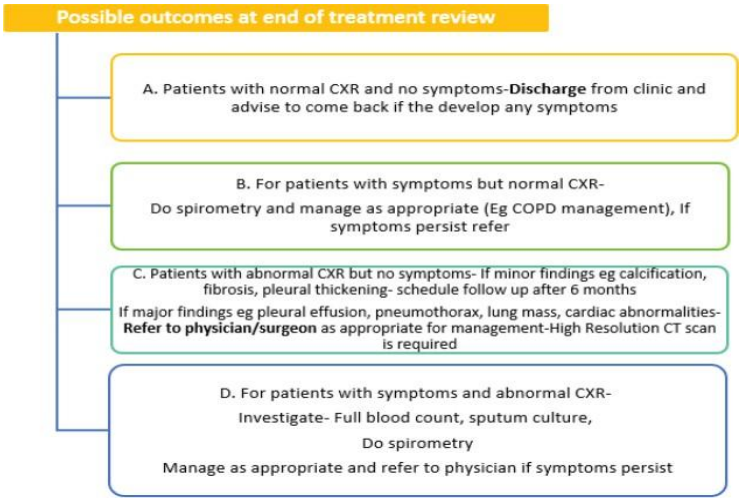
### Assesment of Post TB Lung Disease

**Figure 12.7: Assessment for Post TB Lung Disease**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Figure 12.8: Possible outcomes at end of treatment review**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.14: Post treatment review assessment**

| Review at Month 12, 18 and 24 after the end of treatment |
|---|
| •All B,C,D patient categories above should be reviewed at 12, 18 and 24 months. |
| •History: Symptom enquiry form<br>•Cough<br>•Hemoptysis<br>•Sputum<br>•Chest pain<br>•Breathlessness<br>•Clinical examination<br>•Vital signs- Respiratory rate, Heart rate<br>•Nutritional status- BMI<br>•Full physical examination |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Table 12.15: Other radiological investigations**

| Other radiological investigations as indicated: |
|---|
| Lung parenchyma diseases (Interstitial diseases) - High Resolution CT Scan |
| Cardiac involvement - Cardiac CT scan |
| Mediastinum, chest wall involvement - Chest CT Scan |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

**Management of Post TB Lung Disease conditions:**
The management is dependent on the diagnosis:

## 12.4.1 LUNG SCARRING (FIBROSIS)

This is the thickening, scarring or stiffness of lung tissue making it less efficient in the ability to get oxygen into the bloodstream. Normally, there is associated volume loss. Develops as a consequence of lung healing, e.g from TB. The stiffness causes difficulty in lung expansion leading to shortness of breath. This could be a sequelae of extensive TB disease.

- Clinical Features
  - Symptoms depend on the extend of fibrosis. Patients may be or experience dry cough. In severe cases, shortness of breath on exertion, decreased exercise tolerance, finger clubbing.
- Investigations
  - Commonly occurs at the apices and upper lobes, with fibronodular opacities and associated loss of lung volume.
  - Elevation of the adjoining fissure or hilum may be associated.
  - Scaring appearing beyond 6 months can help distinguish active TB from healed TB.
- Management
  - There is no cure and management is to relieve symptoms and reduce further lung scarring.
  - In severe terminal cases, long-term oxygen therapy may be required.
  - These patients should be referred for review and specialised care by a physician.

## 12.4.2 BRONCHIECTASIS

This is a chronic lung disease often secondary to an infectious process that results in the abnormal and permanent distortion and widening of airways leading to build-up of excess mucus. Mechanisms for pathogenesis of bronchiectasis infection, airway obstruction and peribronchial fibrosis.

- Clinical Features

- Cough and daily mucopurulent sputum production, often lasting months to years (classic).
- Blood-streaked sputum or hemoptysis from airway damage associated with acute infection.
- Dyspnea, pleuritic chest pain, wheezing, fever, weakness, fatigue, and weight loss.
- Rarely, episodic hemoptysis with little to no sputum production (i.e., dry bronchiectasis).

♦ Investigations
- The diagnosis of bronchiectasis involves the following (i) a compatible history of chronic respiratory symptoms (e.g., daily cough and (ii) purulent sputum production. Tests recommended for aetiological testing in adults:
  1) Differential blood count
  2) Serum immunoglobulins (total IgG, IgA and IgM)
  3) Testing for allergic bronchopulmonary aspergillosis (ABPA)
  4) Sputum culture for bacterial infection
  5) Mycobacterial culture for NTM

  Additional tests may be appropriate in response to specific clinical features, or in patients with severe or rapidly progressive disease.

- Radiological diagnosis

  Imaging plays a pivotal role in the diagnosis of bronchiectasis.
  - **High-resolution computed tomography** (HRCT) is the cornerstone in the radiological diagnosis of clinically suspicious cases and the most sensitive and specific non-invasive method for diagnosing bronchiectasis

  .

  **The CXR:** This has a role largely in surveillance for intercurrent infection, progressive lobar collapse, or suspected development of cavitary disease in patients with known bronchiectasis.

The affected individuals are often normal or show nonspecific findings. The key changes to look for include parallel line opacities (tramtrack appearance), tubular opacities (mucus plugging) and ring opacities (dilated end on bronchi). Others are lobar atelectasis and compensatory hyperinflation.

♦ Management
- Treatment is mainly aimed at reducing exacerbations. These are associated with increased airways, systemic inflammation and progressive lung damage In addition, more severe and more frequent exacerbations are associated with worse quality of life, daily symptoms, lung function decline, and mortality. The cycle below shows the different cycles that a patient goes through.

**Table 12.16: Treatments for bronchiectasis according to the vicious cycle concept**

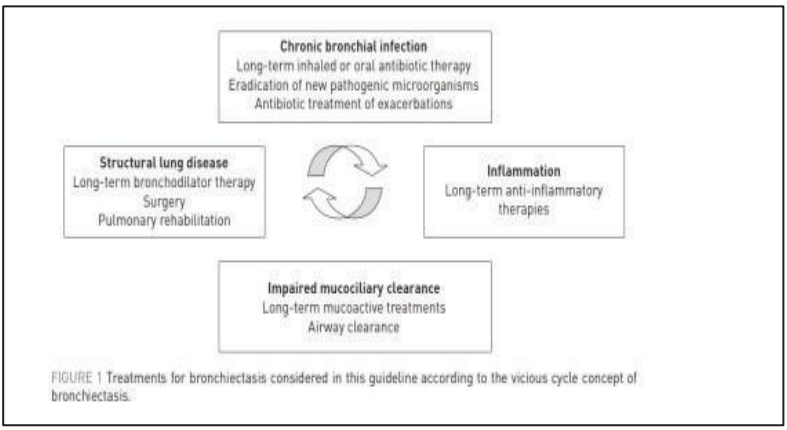


FIGURE 1 Treatments for bronchiectasis considered in this guideline according to the vicious cycle concept of bronchiectasis.

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

- Chest physiotherapist: This includes postural drainage and other maneuvers aimed at improving drainage of respiratory secretions.
- Antibiotics: Infective exacerbations will require antibiotics. Broad-spectrum antibiotics like amoxicillin-clavulanate, metronidazole or clindamycin for anaerobic infection. Antipseudomonal antibiotic like ciprofloxacin, 3rd generation cephalosporin (e.g., ceftazidime) should be used when colonization with Pseudomonas is suspected.
- If haemoptysis is severe and life threatening, patients should be admitted to hospital for more specialized treatment
- ***NB:*** *Once a diagnosis is made, REFER to a chest physician for further specialized care.*
-

## 12.4.3 CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Fore detailed information refer to COPD section 12. 3 above

## 12.4.4 LUNG ABSCESS

A lung abscess is a bacterial infection that occurs in the lung tissue. The infection causes tissue death, and pus collects in that space. A lung abscess can be challenging to treat, and it can be life threatening. Often seen in a patient with extensive damage to the lungs after TB.

- Clinical Features
  - The most noticeable symptom of a lung abscess is a productive cough. The contents that are coughed up may be bloody or pus-like, with a foul odour, fever of 38 degrees celsius or higher, chest pain, shortness of breath, sweating or night sweats, weight loss, fatigue.
  - Dullness on percussion and decreased or absent breath sounds with an intermittent pleural friction rub (grating or rubbing sound) on auscultation, crackles may present.

- Investigations
  - Rule out TB and other infections by conducting sputum or pus analysis, CXR and/or CT.
  - Full blood count and/or a blood culture will support establishing the causative agent/

- Management
  - Antibiotic treatment is given. The choice of antibiotic is adided by the results of a pus culture-sensitivity test.
  - Surgical intervention may also be necessary.

**NB:** Once a diagnosis is made, REFER to a chest physician for further specialized care

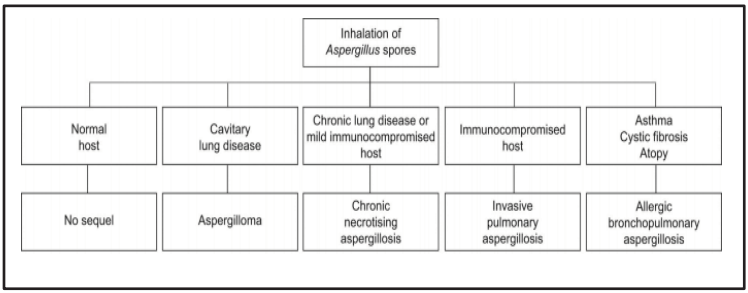
## 12.4.5 ASPERGILLUS-RELATED LUNG DISEASE

This results from colonization of tuberculous cavities or bronchiectatic lesions with the fungus Aspergillus. Three distinctive patterns of aspergillus-related lung disease are recognized:

- Saprophytic infestation of airways, cavities and necrotic tissue
- Allergic disease including extrinsic allergic alveolitis, asthma, allergic bronchopulmonary aspergillosis, bronchocentric granulomatosis and

- Chronic eosinophilic pneumonia.
  They manifest depending on tunderlying lung pathology and host immune status into the following 5 types:

**Figure 12.9: Classification of Aspergillosis**




Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

- **Clinical Features**
  These vary depending on the severity and type of illness one develops.

  Three distinct types are known with specific signs and symptoms.

**Table 12.17: Signs and Symptoms of Aspergillosis**

| Type of illness | Signs / symptoms | Predisposing condition |
|---|---|---|
| ***Allergic reaction*** *(allergic bronchopulmonary aspergillosis)* | • *Fever*<br>• *A cough that may bring up blood or plugs of mucus*<br>• *Worsening asthma* | *Develops in asthma or cystic fibrosis* |
| ***Aspergilloma*** | • *Normal initially*<br>• *A cough that often brings up blood (haemoptysis)*<br>• *Wheezing*<br>• *Shortness of breath*<br>• *Unintentional weight loss*<br>• *Fatigue* | *Emphysema, tuberculosis or advanced sarcoidosis,* |
| ***Invasive aspergillosis*** *(Most severe form and fatal)* | *Signs and symptoms depend on which organs are affected, Generally:*<br>• *Fever and chills*<br>• *A cough that brings up blood (haemoptysis)*<br>• *Shortness of breath*<br>• *Chest or joint pain*<br>• *Headaches or eye symptoms*<br>• *Skin lesions* | *In people whose immune systems are weakened as a result of cancer chemotherapy, bone marrow transplantation or a disease of the immune system to the brain, heart, kidneys or skin* |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 202

**Table 12.18: Diagnosis of Aspergillosis**

| | | |
|---|---|---|
| ***-Minor criteria -*** *positive Aspergillus radioallergosorbent assay test results*<br>*Sputum culture for Aspergillus in sputum and*<br>*Culture / sensitivity* | | |
| ***Imaging*** | | |
| ***Chest radiography***<br>*-Fleeting pulmonary infiltrates*<br>*- Mucoid impaction*<br>*-central bronchiectasis*<br>*- Lobulated infiltrate, which has been likened to a cluster of grapes or a hand in a mitten*<br>***HRCT***<br>*Mucus filled bronchi*<br>*Areas of atelectasis* | ***Chest radiography***<br>*-Variable features with variable, solitary or multiple nodules*<br>*- Cavitary lesions*<br>*- Alveolar infiltrates that are localized or bilateral*<br>*- Diffuse infiltrates as disease progresses*<br>***HRCT***<br>*-Characteristic halo sign (i.e., an area of ground-glass infiltrate surrounding nodular densities)*<br>*-Later disease may show a crescent of air surrounding nodules, indicative of cavitation.*<br>*-Because Aspergillus is angioinvasive, infiltrates may be wedge-shaped,*<br>*-Pleural-based, and cavitary, which is consistent with pulmonary infarction* | ***Chest radiography***<br>*-A mass in a preexisting cavity,*<br>*-Usually in an upper lobe*<br>*- manifested by a crescent of air partially outlining a solid mass.*<br>*- Movement of mass with position*<br>***HRCT***<br>*- Better definition of the mass within a cavity*<br>*- May demonstrate multiple aspergillomas in areas of extensive cavitary disease (supine and prone CTs to be considered)* |

- High levels of specific immunoglobulin G against Aspergillus in blood (Confirmatory test)

**Table 12.18 continued: Diagnosis of Aspergillosis**

| *Diagnostic spectrum* | | |
|---|---|---|
| Allergic bronchopulmonary aspergillosis (**ABPA**) | **Invasive aspergillosis (Chronic necrotizing Aspergillus pneumonia)** | **Aspergilloma** |
| ***Laboratory testing*** | | |
| ***Major:***<br>Blood: *CBC for Eosinophilia.*<br>*-Skin test - positive result for A. fumigatus*<br>*-Marked elevation of the serum immunoglobulin E (IgE) level to greater than 1000 IU/dL*<br>*- Aspergillus Precipitin test: positive results for Aspergillus precipitins (primarily immunoglobulin G [IgG], but also immunoglobulin A [IgA] and immunoglobulin M [IgM])* | Demonstration of the organism in sputum | |

Source: Integrated Guideline for Tuberculosis, Leprosy and Lung Disease, 2021

- Management
  - The only effective treatment is surgical removal of the aspergilloma.
  - In addition to surgical removal: Oral itraconazole may provide partial or complete resolution of aspergillomas in 60% of patients.
  - Antifungal medicine can be used for invasive pulmonary aspergillosis e.g., Amphotericin B and voriconazole.

## 12.4.6 SPONTANEOUS PNEUMOTHORAX

It is the presence of air in the pleural cavity the results in the impairment of oxygenation and ventilation. It is a medical emergency and results from rupture of a TB cavity adjacent to the pleura. It may be associated with formation of pus in the pleural space (empyema) leading to a pyopneumothorax.

- Clinical Features
  - Acute onset shortness of breath
  - Chest pain

- Investigations
- Pneumothorax is generally diagnosed using a chest X-ray. In some cases, a computerized tomography (CT) scan may be needed to provide more-detailed images

- Management
  - The patient should be admitted to hospital for appropriate management.
  - Underwater seal drainage.

***NB:*** *Once a diagnosis is made, REFER to a chest physician for further specialized care*

# 13. Mixed Selection of Common Conditions

## 13.1 Coma

Coma is a state in which the patient is unarousable and unresponsive to external stimulation. In profound coma, brain stem and myotatic reflexes may be absent. Coma noticed for the first time is always an emergency. It is only after the cause is known and its implications are understood that it may be treated otherwise.

**Aetiology**

Infections (malaria, meningitis, encephalitis), trauma, tumours, cerebro-vascular accidents, diseases (diabetes, epilepsy, liver failure), drugs (alcohol, methyl alcohol, barbiturates, morphine, heroin), chemicals, and poisons (see Section 1.5, on poisoning).

**History**

Detailed history from relative or observer to establish the cause if known or witnessed. The circumstances and temporal profile of the onset of symptoms of critical importance in ascertaining the cause of the coma. Documentation of use of drugs and pre-existing diseases is important.

**Examination**

- ♦ Secure a patent airway.
- ♦ Determine if cardiac output is adequate (BP, pulse rate).
- ♦ Evaluate and monitor according to Glasgow Coma Scale (see Section 51.1 on head injury).
- ♦ Monitor temperature, pulse, respiratory rate, and their pattern
- ♦ Consider leads to possible causes:

- Hypothermia: Occurs in alcohol, barbiturate, and sedative poisoning, hypoglycaemia, and hypothyroidism.
- Hypotension: Occurs in internal haemorrhage, myocardial infarction, septicaemia, alcohol, or barbiturate poisoning.
- Hyperventilation with a change in pulse rate may signify increased intracranial pressure.
- Hypertension may signify hypertensive encephalopathy or a cerebrovascular accident.
- Fever occurs in systemic infection with meningitis or encephalitis.
- Neck stiffness could signify meningitis, subarachnoid haemorrhage, or cerebral malaria.

- ♦ Determine the muscle tone and deep tendon reflexes. Note any asymmetry.

**Investigations**

**These vary according to findings but generally include:**

- ♦ Blood slide for malaria parasites
- ♦ Blood sugar
- ♦ U&E
- ♦ Liver functiontests
- ♦ Lumbar puncture (after fundoscopy)
- ♦ Skull x-ray (if there is evidence of trauma)
- ♦ CT scan, whereavailable

**Management to Be Initiated at Any Level where It Occurs**

- Maintain adequate airway – nasal, oral or endotracheal intubation.
- Ensure adequate circulation – always fix a large IV canula immediately in anticipation of drug administration.
- Monitor vital signs.
- Turn patient 2 hourly to avoid pressure sores
- Condom catheters in males(uricondom)
- Urethral catheters in females. Change regularly and repeat urine and catheter tip cultures at least fortnightly.
- Prevent contractures by regular daily passive exercises (physiotherapy)

**Management – Specific**

- Identify and treat cause appropriately.
- Rapidly and assiduously correct hypertension, hypoxia, hypercapnia, hypoglycaemia, hypothermia.
- Give 50ml of 50% dextrose IV diluted in an equal volume of normal saline or 5% dextrose or water for injection if blood glucose is low(<3.5mmol/L).
- Begin therapy for meningitis immediately if suspected.
- Treat malaria if confirmed or suspected.
- Treat the underlying cause when identified.

## 13.2 Fever

This is where there is an elevation of core body temperature above the normal circadian (daily) range. Normal body temperature in adults 18–40 years is 36.8$^{o}$C ±0.4$^{o}$C. Substances that cause fever are called pyrogens. Fever accompanies a wide variety of illnesses and need not always be treated on its own. In general, the cause should be ascertained before therapy as far as possible.

**Management – General**

**Conditions that merit lowering the temperature on their own:**

- Precipitation of heart failure
- Delirium/confusion,
- Convulsions,
- Coma,
- Malignant hyperpyrexia or
- Heat stroke, and
- When the patient is extremely uncomfortable.

**Treat by:**

- Immersing in cold water at 20–25$^{o}$C or tepidsponging.
- Treating cause of thefever.
- Treatingthefeverwithacetylsalicylicacidinjectionortablets***OR***paracetamol tablets.

**— Fever alone is not a reason to give antibiotic.**

## 13.2.1 FEVER OF UNKNOWN ORIGIN

This describes fever of more than 3 weeks duration, the cause of which is not apparent after at least 1 week of intensive investigations. Assessment should include observation of the fever pattern, detailed history and physical examination, laboratory tests, and non-invasive and invasive procedures. This definition excludes common short self-limiting infections and those that have been investigated and diagnosed within 3 weeks.

**For common diseases to be considered it is worth noting that:**

- ♦ Most cases of prolonged obscure fever are instances of well-known diseases presenting atypically.
- ♦ Actual pattern of graphic record, despite emphasis in traditional books, is so variable as not to be practically helpful.
- ♦ Aggressive diagnostic effort is justified as cure is possible in some cases.
- ♦ Infections (accounts for 50% being due to viral infection):

• Tuberculosis: This is the commonest cause of pyrexia of unknown origin in Kenya. The lesions of military TB may not be visible easily on x-rays until disease is well advanced. Sites like kidneys and tubo-ovarian region raise diagnostic difficulties.
• Specific bacterial infections without distinctive localizing signs. The commonest here are salmonellosis and brucellosis.
• Deepseated bacterial abscesses, e.g., subphrenic or periphrenic abscess, purulent infections of large bowel or female pelvic organs. Reactivated old osteomyelitis should be considered as well.
• Infective endocarditis especially due to atypical organisms, e.g., Q-fever, aspergillus.

- ♦ Viral infections:

• Anicteric hepatitis virus infection
• Slow viruses: commonest is HIV.
• Neoplasms (10–20% inchildren).

- ♦ Lymphomas: These are the commonest among the neoplastic causes of PUO. Diagnosis may be difficult iflesions are deep seated retroperitoneal nodes.
- ♦ Leukaemia: Contrary to common belief, it is extremely rare for leukaemia to present with fever only.
- ♦ Solid tumours: The commonest among solid tumours is hypernephroma with pancreatic carcinoma, and sarcomas coming next although presentation with fever alone is rare.
- ♦ Immunogenic diseases: These diseases may present with fever only for several months. The common ones are rheumatoid arthritis, systemic lupus erythematosus, polyarthritis nodosa, rheumatic fever, and cranial arteritis.
- ♦ Other causes:

• Chronic granulomatous hepatitis – steroids would be useful.
• Recurrent small pulmonary thromboembolism.
• Drug fever.
• Liver cirrhosis.

- ♦ Habitual hyperthermia: Usually young adult female with imperfect thermo-regulation.
- ♦ Cause may remain unknown in 10–20% of children.

- Temperature: Rarely exceeds 37.6°C. It is mentioned because no action need be taken.

**Investigations**

The routine investigations listed below should be done before a diagnosis of PUO is made:

- Blood count
- Blood C&S
- Urinalysis
- CXR
- Urea and electrolytes
- LFTS

**Additional investigations that need to be done include the following:**

- Repeated history taking and examination may detect:
  - New clinical features that give a clue.
  - Old clinical signs previously missed or overlooked.
- New tests:
  - Immunological: rheumatoid factor (Rh factor), antinuclear antibody (ANA), anti-streptolysin O titre (ASOT).
  - Most PUOs have abdominal involvement hence, do: barium studies of GIT; intravenous urography; scan liver, spleen, kidneys either computerized axial tomography or ultrasound.
  - Withhold drugs for a few days. Fever disappears in drug fevers.
- ECG may detect right heart strain in embolism
- Invasive procedures
- Liver biopsy
- Finally diagnostic laparotomy maybe justified.NB: Very experienced surgeon required.

**Refer to levels 5 and 6 if:**

- Patient deteriorates rapidly.
- New tests described above are not available in your centre.
- Invasive procedure is required.

**— Prognosis: 10–20% causes remain unknown; 5–10% mortality rate.**

## 13.3 Hepatosplenomegaly

This is the enlargement of the liver to more than 3cm below the costal margin and the spleen to more than "just palpable". The liver size should be described as centimetres below costal margin and below xiphisternum. Since splenomegaly is an extremely common sign and commonly related to malaria, probably splenomegaly smaller than grade 3 Hacket will not cause major concern

**Causes of Hepatosplenomegaly**
Condition responsible: Hepatomegaly splenomegaly
Infections: Malaria, kalaazar, schistosomiasis, infectious hepatitis, amoebic hepatitis/abscess

**Management**

- ♦ No other symptoms, see as outpatient:
- Exclude schistosomiasis (stoolx3),rectal snip,blood diseases (Hb,WBC, sickle cell test), brucellosis (brucella test blood), malaria (malaria slide).
- If tests normal, treat as idiopathic splenomegaly syndrome with proguanil 50mg daily below 3 years, 100mg in older children for 6 months or until spleen is definitely smaller.
- ♦ Admit
- If patient is anaemic
- If patient is febrile
- For invasive diagnostic tests, e.g., bone marrow, liver biopsy.

## 13.4 Jaundice

It presents as a yellow colouration of skin and mucous membranes due to excess bilirubin. Serum bilirubin >2mg% (34.2µmol/L). In general terms, hyperbilirubinaemia may be pre-hepatic, hepatic, or post-hepatic.

- ♦ Pre-hepatic: Due to excess intravascular release of bilirubin by haemolysis.
- ♦ Hepatic: Due to hepatocyte dysfunction (faulty uptake, metabolism or excretion of bilirubin)
- ♦ Post-hepatic: Due to impaired removal of bilirubin from biliary system(e.g., common bile duct obstruction, intrahepatic cholestasis)

Common causes, as summarized in Table 13.1, include viral hepatitis, haemolytic anaemia (e.g., sickle cell, malaria), cirrhosis, biliary obstruction, hepatoma, drug induced (e.g., alcohol, isoniazid).

**Clinical Features**
Meticulous history and physical examination are important before ordering investigations. History should include exposure to hepatotoxic drugs pre-existing known haematological disorder. History of anorexia, nausea, and aversion to smoking is suggestive of viral hepatitis, while history of dark urine, pale stool, and pruritus is suggestive of obstructive jaundice. Physical examination should include observation

for presence of spider naevi, gynaecomastia, loss of axillary hair, parotid gland enlargement, and ascites, which is suggestive of cirrhosis.
Splenomegaly is suggestive of parenchymal liver disease or haemolytic jaundice.

**Investigations**

- ♦ Blood slide for malaria parasites. ***Jaundice in a patient with malaria is a medical emergency.***
- ♦ Urine –Bilirubin:
  - Absence of bilirubin in a patient suggests haemolytic anaemia.
  - Presence of bilirubin suggests hepatobiliary jaundice.

**Table 13.1:Common causes of jaundice**

| Condition responsible | Hepatomegaly | Splenomegaly |
|---|---|---|
| Infections | Malaria | Malaria/tropical splenomegaly |
| | Kala azar | HIV |
| | Schistosomiasis | Kala azar |
| | Infectious hepatitis | Schistosomiasis, Infectious hepatitis |
| | Amoebic hepatitis/abscess | Brucellosis |
| | Brucellosis | Other infections like SBE, typhoid fever, infectious mononucleosis |
| Blood conditions | Haemolytic anaemia | Haemolytic anaemia, e.g., sickle cell |
| | Leukaemia | anaemia in child <3 years autoimmune haemolytic anaemia Leukaemia |
| Nutrition | Kwashiorkor | Iron deficiency |
| Congestion | Cardiac failure | Portal vein thrombosis |
| Other | Liver tumour | Liver cirrhosis |
| | Displaced rather than enlarged liver | Rheumatoid arthritis (Felty's syndrome) SLE |

  - Urine –Urobilinogen:
  - Excessive urobilinogen suggests haemolysis. Urobilinogen is absent in obstructive jaundice.
- ♦ Liver function tests:
  - Gamma globulin transaminase – Elevated levels suggest alcohol abuse.
  - Alkaline phosphatase – Elevated levels suggest obstruction.
  - SGOT (AST) – Elevated levels suggest hepatocellular damage.
  - SGPT (ALT) – Elevated levels suggest hepatocellular damage.
- ♦ Serumproteins:
  - Albumin – Low levels in chronic liver disease such as cirrhosis.
  - Globulins–Hyperglobulinaemia is found in chronic active hepatitis, cirrhosis.
- ♦ Fullhaemoglobin–Polymorphonuclear leukocytosis is found in leptospirosis. Sickle cells may be seen in the peripheral blood smear.
  - Reticulocyte count–Increased reticulocyte count indicates a haemolytic anaemia.
- ♦ If above investigations are not diagnostic, consider:

- HBsAg, HAV–Ab. TORCHES (toxoplasmosis, rubella,cytomegalovirus, herpes, syphilis) in young infants.
- Ultrasound: useful in obstructive jaundice, gallstones, differentiating between abscess and tumour.
- Alpha-foeto proteins: Substantial elevations of alpha-foeto proteins are found in hepatocellular carcinoma.
- Paracentesis of fascistic fluid: Protein content<3g%is found in cirrhosis, tuberculosis, peritoneal tumours, peritoneal infection, or hepatic venous obstruction. Blood-stained ascites usually indicates a malignant disease–cytology is mandatory.
- Liver biopsy is important in diagnosis of chronic hepatitis and cirrhosis and hepatocellular carcinoma.



**Management**

- Patients with history and physical findings suggestive of viral hepatitis can be managed as outpatients requiring advice on bed rest, avoidance of alcohol. Prescribe multivitamin tablets.
- Admit for diagnostic evaluation if cause not apparent.

**Consider hepatic encephalopathy in any patient who has jaundice and mental complaint. Early treatment of hepatic encephalopathy may reduce mortality.**

## 13.4.1 OBSTRUCTIVE JAUNDICE

This refers to jaundice resulting from obstruction of bile in the biliary tree (post-hepatic jaundice). Causes include:

- Intraluminal (within the lumen) include gallstones, which dislodge from the gall bladder and are impacted in common bile duct (CBD), and helminthiasis (ascaris and liver flukes).
- Mural (within the wall of ducts) due to inflammation, benign and malignant tumours of bile duct wall, e.g., cholangiocarcinoma, cholangitis, etc.
- Extramural (outside the walls) include choledochal cysts, enlarged lymph nodes of any cause, and carcinoma of thepancreas.
- Other causes are congenital biliary atresia, iatrogenic trauma to the ducts during surgery (especially cholecystectomy), and strictures after cholangitis and cholecystitis.

**Clinical Features**

**It presents as painless jaundice, with pruritus that can be severe; jaundice progresses steadily**

- Distended gallbladder, which is present in 60% of carcinoma of the head of the pancreas.
- Anorexia, which is usually present.
- Diarrhoea that is troublesome with foul smelling pale stool.
- Dark urine, history of flatulence, and dyspepsia in fat females are suggestive of gallstones.

**Investigations**

- Hb, WBC, ESR
- Liver function tests
- Prothrombin time index
- Plain abdominal x-rays
- Abdominal ultrasound and CT scan

**Management**

- Carry out adequate investigations and surgical management.
- Give cholestyramine 4–8g once or twice daily, maximum dose 24g a day.

## 13.5 Lymphadenopathy

It is where there is an abnormal increase in size or altered consistency of lymph nodes. It is manifestation of regional or systemic disease.

**The following common diseases are associated with lymph node enlargement:**

- Infectious diseases
  - Viral diseases: HIV
  - Bacterial infections: Pyogenic, tuberculosis
- Malignant diseases
  - Haematological: Hodgkin's and non-Hodgkin's lymphoma
  - Metastatic tumours to lymph nodes: Head and neck, breast, prostate
- Immunological disease
  - Connective tissue disorders.

**Clinical Features**

Clinical features depend on underlying cause.

**Investigations**

Careful clinical examination is vital before ordering investigations, e.g., axillary lymph nodes in the presence of a breast mass points to cancer of the breast.

- Full haemogram
- Chest x-ray
- Blood for HIV test
- Bone marrow
- Lymph node biopsy

**About 25% of patients will have non-diagnostic results from the biopsy. A repeat biopsy should be performed if enlarged lymph nodes and symptoms persist.**

**Management**

Further diagnostic evaluation depends on the initial results, e.g., a thorough ENT work-up if biopsy indicates a secondary tumour deposit from the post-nasal space. Specific management depends on the specific cause of lymphadenopathy.

# 14. Skin Diseases

## 14.1 Inflammatory Skin Conditions

### 14.1.1 ATOPIC DERMATITIS

**Clinical Features**

It is an acute, sub-acute but usually chronic pruritic inflammation of the epidermis and dermis often occurring in association with a personal or family history of hay fever, asthma, allergic rhinitis, or atopic dermatitis. Sixty per cent of patients with this condition begin to suffer from it sometime during the first year of their life, but onset is most frequent in the first 2–3 months.

It commonly presents with the following skin lesions: erythema, papules, scaling, excoriations and crusting. Pruritus is the cardinal feature of eczema and the constant scratching leads to a vicious cycle of itch-scratch-rash-itch. Subsequentlythe skin becomes thickened (lichenified) presenting mainly on cheeks and extensor surfaces of limbs of an infant; it later localizes on the flexural areas of the limbs in both older children and adults. The natural history is that the disease clears with age in the majority of children. (See appendix 1 for detailed list of common skin conditions.

**Management**

- ♦ Parents should be educated on the disease and its natural history and be advised to avoid any precipitating factors if identified.
- Encourage wearing of clothing made of cotton
- Avoid any food substance that seriously aggravates the eczema
- Avoid agents that will cause the skin to dry excessively, e.g., detergents and medicated soaps, etc. NB: One should use normal toilet soaps.
- Avoid any of the petroleum jelly products on those who react.
  - ♦ Chlorpheniramine maleate 4mg 8 hourly can be used to alleviate itch.
  - ♦ Steroids: Topical and oral steroids are the mainstay treatment. Use of the mildest steroid that controls the problem is advocated.
- 0.1% betamethasone or 1% hydrocortisone ointments/creams.

NB: If a large body surface area is involved (e.g., 50% and over) or the disease is very severe, one is advised to consult a dermatologist who may choose to use systemic steroid prednisone 20–40mg daily. The main complications of infection need prompt treatment, e.g., bacterial, fungal, and viral. As with other atopic conditions, stress may aggravate eczema and thus older children should be assisted and encouraged to minimize stress.

### 14.1.2 CONTACT DERMATITIS

It is the acute or chronic inflammation produced by substances contacting the skin and causing toxic (irritant) or allergic reactions. Primary irritants include acids, alkalis, soaps, detergents, acetone, etc. Among the causes of allergic

contact dermatitis are topical drugs, plants, shoes, clothing, metal compounds, dyes, and cosmetics. Sensitivity to latex in gloves is a particular problem for many health workers, and sensitivity to latex may preclude the use of condoms by some men.

**Clinical Features**
Lesions may be acute vesicles or weeping subacute erythema, or dry and scaly with papules, or chronic lichenified (thickened) excoriated and hyper pigmented rash. The lesions may take the shape of the contact with the offending item, for example shoes, watch, gloves, etc., but may be asymmetric and not have any particular shape.

**Management**
- Identify and remove causative agent.
- Drain large blisters but do not remove tops (roofs).
- Apply gauze or thin cloths dipped in water or normal saline.
- Apply topical 1% hydrocortisone ointment for dry lesions and cream for wet lesions.

## 14.1.3 NUMMULAR ECZEMA

This is also known as nummular dermatitis or discoid eczema. It is a chronic but treatable condition that causes coin-shaped spots to develop on the skin. These spots are often very itchy and well-defined. The lesions may ooze clear fluid or become dry and crusty.

## 14.1.4 DYSHIDROTIC ECZEMA

This type of skin disorder presents with blisters that may last for three to four weeks before clearing. Some people never develop blisters again. Dyshidrotic eczema can also be a lifelong, debilitating disease if not well treated.

Common triggers include skin care products like shampoo or soap. Coming into contact with nickel or cobalt can also lead to blisters. These metals are found in a variety of things ranging from jewelry and cell phones to the foods taken.

Other common triggers include stress, sweat, and hot climates. Having wet hands for long periods can also trigger dyshidrotic eczema. For some people, dyshidrotic eczema is also seasonal as it develops every spring or summer and then clears when the temperature starts to fall. Exposure to cutting oil and coming in contact with cement at work is occasionally responsible.
Recent research has led to new treatments for difficult-to-treat dyshidrotic eczema. One newer option is use of biologics like omalizumab that helps lesions improve when other drugs do not work.

## 14.1.5 SEBORRHEIC DERMATITIS

This is an inflammatory scaling disease of the scalp, face, and occasionally other areas with high density of oil glands (axilla, upper chest, anogenital areas).

**Clinical Features**

**Symptoms develop gradually as:**

- Dry or greasy diffuse scaling of scalp (dandruff) with pruritus.
- Yellow-red scaling papules in severe cases found along the hairline, external auditory canal, eyebrows, conjunctivae, and naso-labial folds. Does not cause hair loss.
- Cradle cap (thick yellow crusted scalp) in newborns.

**NOTE: Severe seborrhoeic dermatitis is found in neurological disorders (Parkinson's disease) and HIV infection.**

**Management**

- Control scaling by 2% salicylic acid in oil.
- Use shampoos containing selenium sulphide, sulphur, and salicylic acid, or tar shampoos daily till dandruff is controlled as advised by dermatologists. (More recently ketaconazole shampoo is excellent.)
- To treat superimposed bacterial, fungal, or viral infections, which are prevalent in HIV patients.

## 14.1.6 PSORIASIS

This is a common papulosquamous skin disease that occurs in 2–3% of the general population.

**Clinical Features**

Clinical presentations are erythematous macules, papules, or plaques that are usually covered with silvery scales.

**Diagnosis**

Is made by observation of the characteristic lesions and demonstration of Auspitzspots and Koebner's phenomenon. A skin biopsy may be needed in atypical cases.

**Management**

- Try topical therapy with corticosteroids, dithranol, calcipotriol, tazarotene, and tar; UV light-based treatment using narrow bands, broad bands, and PUVA. More recently, biological therapy like etanercept, infliximab, and afalizumab are being used.
- Refer to a dermatologist.

## 14.1.7 PITYRIASIS ROSEA

Pityriasis rosea is a self-limiting skin condition that presents as discrete scaly papules and plaques along the Langer lines (cleavage lines) over the trunk and limbs. This generalized rash is usually preceded by a Herald patch on the trunk. The incidence is 150 cases per 100,000 persons per year. It typically affects persons 10 to 35 years of age. Some studies report that males and females are equally affected, whereas others report that females are affected more often. It has seasonal variation, as studies show a higher prevalence during winter.

**Clinical Features**
The classic feature is a Herald (mother) patch on the trunk in up to 90% of cases. The patch is erythematous with slightly elevated scaling borders and a lighter depressed center. It can measure 3 cm or more in diameter and may be the only skin manifestation for approximately two weeks but is associated with generalized smaller lesions. Prodromal symptoms (e.g., general malaise, fatigue, nausea, headaches, joint pain, enlarged lymph nodes, fever, sore throat) do present before or during the course of the rash in 70% of patients. The generalized rash, also known as secondary eruption, presents on the trunk along the Langer lines of the skin usually on the torso, upper arms, thighs or neck.

Relapses are common during which there is no Herald patch, and the lesions may be smaller or fewer than in the initial episode. The low relapse rate, between 1.8% and 3.7%, suggests the development of immunity. Pityriasis rosea in children presents similarly to that in adults. Pruritus has been reported more often in this population. Children have more facial (30%) and scalp involvement, and post-inflammatory pigmentary changes.

**Investigations**
Skin biopsy for histology usually show focal parakeratosis, spongiosis, and acanthosis in the epidermis, and extravasated red blood cells with perivascular infiltrates of lymphocytes, monocytes, and eosinophils in the dermis.

**Differential Diagnosis**
The differential diagnosis of pityriasis rosea includes several conditions like If the diagnosis is uncertain, skin biopsy will help exclude other conditiions.

**Treatment**
- Topical medications, such as calamine lotion or zinc oxide gel are used.
- Mild topical corticosteroids e.g., hydrocortisone and betamethasone are applied twice daily for several weeks.
- Antihistamines are useful for allergy and reduce itching.
- Taking baths using lukewarm water or soaking in oatmeal baths.
- In some cases, drugs, such as Azithromycin and Acyclovir (Valtrex, Zovirax) can be used.
- Phototherapy or artificial UV light is used for stubborn cases. The ultraviolet rays are believed to reduce the duration of the rash.

## 14.2 Bacterial Infections

### 14.2.1 BOILS AND CARBUNCLES

Boils and carbuncles are superficial skin infections usually caused by Staphylococcus aureus or Streptococcus epidermidis and occasionally by Gram negative bacteria. These staph infections form pockets in the skin that are filled with pus, a fluid that includes bacteria, dead skin cells and infection-fighting white blood cells. Whether the pocket of pus is called a boil or a carbuncle depends on its location and size.

A boil, also called a furuncle, begins as a painful infection of a single hair follicle. Boils can grow to be larger than a golf ball, and they commonly occur on the buttocks, face, neck, armpits and groin while, a carbuncle is a deeper skin infection that involves a group of infected hair follicles in one skin location. Carbuncles often are found on the back of the neck, shoulders, hips and thighs, and they are especially common in middle-aged or elderly men. People with diabetes or HIV infection are more likely to develop carbuncles due to compromised immune system.

**Clinical Features**

- A boil presents as a red, swollen, painful and fluctuant mass under the skin.
- As the infection gets worse, a whitish tip, also called a point or head, can appear at the center of the boil. This tip is usually the area from which the boil's pus will drain. A carbuncle looks like a cluster of interconnected boils.

**Treatment**

- Incision and drainage: is done for large boil or carbuncle and the wound is cleaned and dressed.
- Analgesics: for pain depending on severity. Paracetamol, Ibuprofen or Meloxicam.
- Antibiotics: are prescribed to help heal severe or recurrent infections.
- The commonly used are Cloxacillin, Ampiclox, Cephalexin and Erythromycin or other macrolides.
- NB: If there is any underlying condition then treat it as well.

### 14.2.2 FOLLICULITIS

Superficial folliculitis is more common in children, whilst folliculitis of the beard area is more common in adult males.

**Aetiology**

It can be caused by microorganisms such as

- Staphylococcus aureus is the most common.
- Pseudomonas spp. - occurring in outbreaks associated with hot tubs, paddling pools, etc. cause intense pruritus, particularly in areas under a bathing suit.

- Other gram-negative folliculitis affects patients with a history of long-term antibiotic therapy for acne: including Klebsiella spp., Enterobacter spp. and Proteus ssp.
- Malassezia folliculitis (previously called pityrosporum folliculitis) causes itchy acneiform eruption on the upper back, upper arms, chest, neck, chin and face, affecting younger patients.
- Other fungal folliculitis are due to Candida spp. and Trichophyton spp are commonly in men in form of tinea barbae in the beard area. It may also be caused by contact with domestic animals like dogs, cattle and other animals.

The lesions from bacterial infection may have a clearly demarcated flaking edge of confluent erythema.

- Herpetic folliculitis is due to herpes simplex viruses (HSV); often in men who shave near oral cold sore lesions.
- Compromised immune system commonly leads to Eosinophilic pustular folliculitis; sterile and intensely itchy eruption associated with HIV infection. This is a rare autoimmune disease, more common in Asian races.
- Another type that results from physical irritation - eg, traction folliculitis as a result of a hairstyle and from oily overalls.

The condition is due to obstruction or flow disruption in pilosebaceous glands ± infection. Furuncles are a more deep-seated infection of the base of the hair follicle, characterised by inflammatory nodules and pus formation, which may result from folliculitis. They may develop into carbuncles.

**Risk factors**

- Overgrown beard, shaving 'against the grain' and particularly thick hair.
- Excessive friction from clothing or overly tight-fitting clothing is another common cause.
- Excessive sweating and hyperhidrosis and high external humidity.
- Pre-existing dermatitis.
- Reduced host immunity - eg, poorly controlled diabetes, immunosuppression.
- Occluded skin - particularly for dermatological treatment with topical corticosteroids.

**Clinical features**

- Folliculitis may occur as a relatively trivial irritation - superficial folliculitis, or as a more deep-seated process involving the lower hair follicle. Often the cause of superficial folliculitis is unclear. The most common infecting organism is S. aureus
- The lesions often start as a rash or a set of slowly evolving red lumps on the skin, usually on hairy areas
- The rash may be pain-free or cause irritation and pruritus
- If mild and left alone, the rash usually resolves without scarring
- Patients may notice small pustules with hairs at the centre of the lesions

- Commonly affected areas are the axilla, beard, face, scalp, thighs and inguinal regions.

- Check temperature and exclude signs of systemic toxicity.
- The central hair shaft may not be easy to see if hair is fine and/or fair; a magnifying glass can help demonstrate its presence. Erythematous papules form in a relatively regular, sometimes 'grid-like', pattern. Small pustules may be seen.
- Deep folliculitis tends to cause more erythema, becoming more confluent between the lesions, with no noticeable surface pustules and intense irritation of the skin. It can cause scarring, keloid formation and hair loss.
- Regional lymph nodes are involde (adenitis), even in simple or mild folliculitis. Folliculitis of the eyelash is known as **a stye or hordeolum**.



**Treatment options**

- **Supportive measures**
  - Use moisturizers and clean shaving implements with surgical spirit.
  - Reduce frequency of shaving and ensure shaving 'with the grain' (or grow a beard).
  - Advise patients to use hair removing creams.
  - Maintain good skin hygiene with non-allergenic cleaning agents.

- **Specific treatment**
- Treat nasal carriage of S. aureus with topical Fucidin® in those with recurrent folliculitis.
- Mild, superficial folliculitis may resolve without treatment but reassure.
- Topical antiseptics such as triclosan, clorhexidine or povidone-iodine may be used to treat and prevent superficial folliculitis.
- For deeper folliculitis, topical or oral antibiotics are usually required; preferred agents are flucloxacillin, erythromycin or cephalosporins and topical mupirocin ointment BD x 7 days.
- In severe or recurrent cases, antibiotic therapy may be required for 4 to 6 weeks.
- Other antibiotics may be used depending on culture and sensitivity results

## 14.2.3 CELLULITIS

Cellulitis is a common bacterial infection of the dermal and subcutaneous tissue. Erysipelas is best regarded as a more superficial form of cellulitis. Cellulitis / erysipelas usually follow a breach in the skin, although a portal of entry may not be obvious. If treated promptly the infection is usually confined to the affected area, however, more severe episodes can lead to septicaemia. Necrotising fasciitis (NF) is an uncommon but rapidly progressive and life-threatening infection of the deep dermis, adipose tissue and subcutaneous fascia.

**CELLULITIS AND ERYSIPELAS**

Streptococcal infection, especially group A (Streptococcus pyogenes), is the most common cause of cellulitis and erysipelas. Staphylococcus aureus can occasionally cause cellulitis.

Other infections are rarely associated, except in certain groups eg immunocompromised patients (a range of organisms), unilateral childhood facial cellulitis can be due to Haemophilus influenza type b (although less so now since the introduction of the Hib vaccination), and orbital cellulitis (as opposed to the more common periorbital cellulitis) is often caused by Streptococcus pneumonia or one of the other sinus pathogens

**Risk factors for cellulitis and erysipelas**

- Skin conditions of the lower legs such as gravitational eczema, leg ulcers, lymphoedema, tinea, trauma.
- Previous episodes of cellulitis
- Obesity
- Immunocompromised patients
- Chronic disease such as diabetes mellitus, chronic liver, or renal disease
- Pregnancy
- Necrotising fasciitis

**Clinical features**

- For both cellulitis and erysipelas, the commonly involved areas are the legs followed by the face, but any other site can be affected.
- Facial involvement tends to be more superficial in erysipelas.
- In cellulitis the rash is normally unilateral, except on the face where it may occasionally be bilateral.
- Morphologically, there occur erythema, the edge is more well-demarcated in erysipelas than in cellulitis.
- Oedema accompanied with increased local temperature and tenderness.
- Sometimes haemorrhage, bullae, or lymphangitis are also present, and these suggest streptococcal infection as opposed to staphylococcal infection. Severe cellulitis can lead to ulceration and more deep-seated tissue damage.

**Complications**

Several complications may follow cellulitis or erysipelas and include:

- Lymphangitis presents with a red line originating from the cellulitis and spreading proximally to lymph nodes along the lymphatic vessels.
- Deep-seated fasciitis and myositis.
- Septicaemia, which can occasionally be fatal
- Nephritis
- Necrotising fasciitis

NOTE: Early diagnosis of necrotizing fascitis is vital in terms of reducing morbidity and mortality, and should be suspected in the following scenarios:

- If the level of pain and tenderness, or systemic upset, is out of proportion to the physical signs
- Dusky-violaceous (pinkish) areas along with erythema
- Crepitus
- Over a short period of time blisters develop, the affected area becomes necrotic, and the patient very toxic

**Differential diagnosis**

- Deep vein thrombosis
- Gravitational eczema: many patients with gravitational eczema are incorrectly diagnosed of having bilateral cellulitis, which is extremely rare, and are inappropriately given systemic antibiotics over several months. Patients with gravitational eczema will have itch, non-tender erythema and sometimes areas of brown discoloration. Gravitational eczema can become infected, which occasionally evolves into a secondary cellulitis
- Contact allergic dermatitis: can present acutely as erythematous, sore and tender areas of skin, sometimes with blisters.
- Panniculitis including sclerosing panniculitis (Syn. Acute lipodermatosclerosis). This causes multiple tender nodules and plaques on affected parts. The acute stage of lipodermatosclerosis causes tender erythema of the lower legs. However, compared to cellulitis, lipodermatosclerosis is usually bilateral and patients are afebrile
- Eosinophilic cellulitis (Syn. Wells syndrome): is rare that clinically the condition presents with large, indurated erythematous plaques, and less commonly nodules that evolve over several weeks.
- Eosinophilic fasciitis: is also rare, and the cutaneous manifestations evolve as the condition progresses. The acute inflammatory stage consists of pain, swelling and tenderness of the distal limbs. These findings are later replaced by induration, and eventually fibrosis with limitation of the movement of the hands and feet. The affected skin is taut and firmly adherent to underlying tissue with dimpling and a peau d'orange appearance. The condition has a symmetrical distribution of the swelling with reddening and tenderness.

**Investigations**

- Skin swabs should be taken, and if present from open areas / areas of exudate, although in many cases results are negative. If bullae or abscesses form, culturing the fluid from inside these lesions yields an organism in more than 90% of cases.
- Blood cultures will be needed for some patients particularly those admitted.

**Management**

- **Antibiotics therapy**
  - First line treatment - Flucloxacillin 500 mg QID for 10 - 14 days (adjust doses accordingly in children), which is bactericidal on streptococci and staphylococci.
  - For penicillin allergy use clarithromycin 500 mg BD (adjust doses accordingly in children).
  - For patients on statin therapy who are penicillin allergic, clarithromycin can be used if the statin is withheld during treatment. Alternatively use doxycycline 200 mg taken for the first day and then 100 mg daily for 7-10 days.
  - Facial cellulitis - consider Amoxiclav if sinus pathogens are a possible cause and refer to local antibiotics guidelines.

- **Adequate analgesia**
  - For the lower leg - rest and elevate the affected area where possible to reduce pain, swelling and damage to the venous system.
  - Give advice the patient to report immediately if the condition deteriorates (spreading infection / systemic symptoms), or if the antibiotics are not tolerated or effective.
- **Post-treatment of cellulitis**
  - Identify and manage the cause or risk factors, which may include: poor hygiene, Injury, weakened immunity, obesity or other skin diseases.
  - Treatment of lower leg skin conditions such as gravitational eczema, leg ulcers, and oedema. If tinea is present, treat accordingly, and then consider prophylactic treatment with terbinafine cream once or twice a week to the plantar surface of the feet and in-between the toes.
  - Weight control and adequate glycaemic control if diabetic.
- **Treatment of recurrent cellulitis**
  - Antibiotic prophylaxis should be offered to patients who have two or more attacks of cellulitis per year: cloxacillin 500 mg QID (1g if weight >75kg) should be the first choice. The dose may be reduced to 250 mg QID after 2 weeks of successful response.
  - For those allergic to penicillin, clarithromycin 500 mg daily (OR erythromycin 250 mg BD) is recommended. For those both allergic to penicillin and on statins use doxycycline 100 mg OD x 10 days.
  - Prophylaxis may need to be prolonged if relapse occurs when antibiotics are discontinued after one year period of successful prophylaxis.

## 14.2.4 IMPETIGO CONTAGIOSUM

It is a contagious intradermal infection caused by streptococcus or staphylococcus. Commonly associated with poor hygiene, crowded living conditions and neglected minor trauma. Frequently complicates scabies, purpura urticaria, and insect bites. Presents as bullous lesions that rupture and crust, occurring on the face, arms, legs, and buttocks.

**Management**

- Local treatment by cleaning with saline water.
- Systemic antibiotics: only for extensive lesions (flucloxacillin 500mg 6 hourly for 5 days ***OR*** erythromycin 250mg 6 hourly for 5 days).

## 14.2.5 BULLOUS IMPETIGO

This is common in neonates (pemphigus neonatorum) although any age can be affected. Caused by staphylococcal infection. Affects mainly axilla and groin.

Causes large bullae containing pus and clear serum. These rupture easily, leaving raw areas. They do not form crusts as in impetigo contagiosum.

**Treatment**

- Treat as above.
- Admit for inpatient care if patient is toxic or septicaemia is suspected.

**Patient Education**

- Spreads easily in schools.
- Isolate and treat infected individuals.
- Separate towels and bath facilities.

## STAPHYLOCOCCAL SCALDED SKIN SYNDROME (SSSS) – RITTER'S DISEASE

Toxin-mediated epidermolytic disease leading to detachment of superficial epidermal layers to resemble scalding. Mainly occurs in children under 2 years of age. Severity varies from localized form (bullous impetigo) to generalized form of epidermolysis. Also found in immuno-compromised adults and in renal failure.



**Clinical Features**

- Vesicles that are flaccid; gentle lateral pressure causes shearing off, leaving raw areas.
- Focus of infection may be found in the nose, umbilical stump, purulent conjunctivitis, otitis media, or nasopharyngeal infection.

**Investigations**

Pus swab for culture and sensitivity is essential.

**Management**

- Admit and treat with the following:
- Parenteral cloxacillin or flucloxacillin preferred. Change antibiotics according to culture and sensitivity results.
- Skin care:
  - Topical care baths with normal saline.
  - If widespread and weeping lesions are present, treat like burns (refert o Chapter 45, Burns)

**Do not give corticosteroids**

**Detect carriers to prevent nursery epidemics.**

## 14.3 Fungal Infections

### 14.3.1 SUPERFICIAL FUNGAL INFECTIONS

The dermatophyte infections are caused by fungi (genus microsporum, trichophyton, and epidermophyton) and thrive on non-viable keratinized tissue of the skin (stratum, comeum, hair, nails). Sources of infection include other persons, animals such as puppies or kittens, and more rarely the soil. The nomenclature is "tinea" followed by the Latin name of the appropriate part.

**Clinical Features**

- ***Tinea pedis (athlete'sfoot): Scaling*** or maceration between toes, particularly the fourth interspace. Causative organismis T. rubrumor T. interdigitalae. Hot humid weather and occlusive footwear are predisposingfactors.
- ***Tinea cruris:*** An erythematous and scaly rash with distinct margin extending from groin to upper thighs or scrotum. Itching may be severe. Common in males.
- ***Tinea corporis (body ringworm)***: Characteristically annular plaque with raised edge and central clearing with variable levels of scaling and itching.
- ***Tinea capitis (scalp ringworm)***: Mainly disease of children and has spontaneous recovery at puberty in normal circumstances. Scaling, itching and loss of hair are common also "Mashillingi". Scarring, alopecia may result.
- ***Tinea anguum:*** Involves the nails and presents with nail discolouration and subungual hyperkeratosis (friable debris).

**Investigations**

**Direct microscopy of skin scale in 20% potassium hydroxide mounted on a slide to demonstrate hyphae.**

**Management**

- For dry lesions, apply 1% clotrimazole ointment 12 hourly until 1 week after lesions have healed.
- 2% miconazole ointment applied once or twice a day.
- Terbinafin 250mg daily ***OR*** weekly fluconazole where indicated and prescribed by dermatologist.

### 14.3.2 DEEP FUNGAL INFECTIONS

Deep fungal infections comprise two distinct groups of conditions: subcutaneous mycoses and systemic mycoses. Neither are common, and the subcutaneous mycoses, with some exceptions, are largely confined to the tropics and subtropics. In recent years, the systemic mycoses have become important opportunistic infectious complications

Examples of deep fungal infections include;

- Aspergillosis
- Blastomycosis
- Cryptococcosis
- Chromoblastomycosis
- Histoplasmosis
- Mycetoma, and
- Sporotrichosis

Subcutaneous mycoses are due to a large and diverse group of fungus that produce infection when traumatically introduced into the skin and subcutaneous tissue or one has depressed immunity. A variety of skin changes may be seen in association with systemic mycoses. The skin lesions depend partly on which fungus is the cause.

Some examples and their presentations are summarized on table 14.1.

**Diagnosis**

This is made by history, careful physical examination and investigations including skin biopsy and skin scrapings for fungal cultures.

**Treatment**

A number of agents are now available for treating deep fungal infections, including Amphotericin B in conventional and liposomal formulations, and the triazoles eg Itraconazole and fluconazole given for 4-6 weeks. It is important to note that there is lack of uniformity concerning the ideal therapy but depends on the facility and level of training.

**Table 14.1:Presentation of Selected Deep Funga Skin Infections**

| Infection | Clinical features |
|---|---|
| Candidiasis | • Single or widespread lesions<br>• Small red papules or larger nodules<br>• Purpuric lesions can resemble ecthyma gangrenosum or purpura fulminans. |
| Aspergillosis | • Few or many lesions<br>• May result in rapidly spreading red patch with a necrotic centre (blackened dead tissue)<br>• May resemble pyoderma gangrenosum. |
| Cryptococcosis | • Most often a skin rash is the first sign of infection<br>• Extremely varied appearance<br>• Papules, abscesses, plaques, blisters, sinuses, ulcers, cellulitis or purpura (bleeding into the skin). |
| Blastomycosis | • Papules, nodules, warty lesions<br>• Pustules, abscesses, ulcers and scars<br>• May cause oral lesions. |
| Histoplasmosis | • Scaly (psoriasis-like) or soft papules, nodules, abscesses, sinuses or ulcers<br>• May cause mouth ulcers<br>• May cause erythema multiforme, erythema nodosum or toxic erythema. |
| Coccidioidomycosis | • Papular lesions, plaques, abscesses, sinuses, ulceration or toxic erythema (a generalized red rash)<br>• Hypersensitivity reactions may result in erythema multiforme. |
| Mycetoma<br>(Eumycetoma) | • Causes masses that are painless but may extend to the bone and cause destruction to tissues. |
| Zycomycosis | • Mucorales infection causes plaques, pustules, abscesses and ulcers<br>• Entomophthorales infection causes slowly progressive subcutaneous firm or hard nodules, often affecting the nose or sinuses. |

## 14.4 Parasitic Skin Infestations

### 14.4.1 SCABIES

Scabies is a cutaneous parasitosis caused by a mite, Sarcoptes scabiei hominis within the epidermis. It exists in two forms: ordinary scabies, relatively benign and moderately contagious; and crusted scabies, favoured by immune deficiency, extremely contagious and refractory to conventional treatment.

Person to person transmission takes place chiefly through direct skin contact, and sometimes by indirect contact (sharing clothing, bedding). The challenge in management is that it must include simultaneous treatment of both the patient and close contacts, and at the same time, decontamination of clothing and bedding of all persons undergoing treatment, to break the transmission cycle.

**Clinical Features**

**Scabies presents in two stages:**

- Primary skin lesions
  - The typical primary lesions are multiple papules called burrows accompanied with itching, worse at night, very suggestive of scabies if close contacts have the same symptom.
  - Scabies burrows (common): fine wavy lines of 5 to 15 mm, corresponding to the tunnels made by the parasite within the skin. Burrows are most often seen in the interdigital spaces of the hand and flexor aspect of the wrist, but may be present on the areolae, buttocks, elbows, axillae. The back and the face are spared. Burrows may be associated with vesicles, corresponding to the entry point of the parasite in the skin.
  - Scabies nodules (less common): reddish-brown nodules, measuring 2 to 20 mm, on the genitals in men, persisting after effective treatment (they are not necessarily indicative of active infection).
- Secondary skin lesions
  - Resulting from scratching (excoriations, crusts) or super-infection (impetigo).
  - The typical lesions and secondary lesions may co-exist, or specific lesions may be entirely masked by secondary lesions.
  - In infants and young children, vesicular eruption; often involving palms and soles, back, face, and limbs occur.
  - Secondary infection or eczematization is frequent. Isolated scabies nodules in the axillae may be the only manifestation.
  - Examination of the mother's hands may support the diagnosis.

**Treatment**

- In all cases of scabies, the close contacts of the patient are treated simultaneously, even in the absence of symptoms.
- Clothing and bedding (including that of contacts) are changed after each treatment. They are washed with hot water at ≥ 60 °C then dried in the sun, or exposed to sunlight for 72 hours, or sealed in a plastic bag for 72 hours.

- Topical treatment
  - Topical scabicides namely, benzyl benzoate emulsion apply OD for 3 days, and 1% Gammabenzene hexachloride lotion is applied over the entire body (including the scalp, post-auricular areas, umbilicus, palms and soles) only once (stat dose), avoiding mucous membranes and face, and the breasts in breastfeeding women.
  - Particular attention should be paid to common infestation sites. The patient must not wash his hands while using the , benzyl benzoate emulsion (or the product should be reapplied if the hands are washed).
  - In children under 2 years, the hands must be wrapped to prevent accidental ingestion of the product and contact with eyes.
  - Topical scabicides should not be applied to broken or inflamed skin.
  - Treatment of secondary bacterial infection, if present, should be initiated 24 to 48 hours before using topical scabicides
- The preferred alternative treatment is 5% permethrin cream:
  - Children 2 months and over and adults: one application, with a contact time of 8 hours, then rinse thoroughly. Repeat the application after 7 days.

## 14.4.2 LICE (PEDICULOSIS OR PHYTHIRIASIS)

Pediculosis is a benign contagious parasitic infection due to three species of lice specific to humans: head lice, body lice and pubic lice. Transmission from person to person occurs through direct or indirect contact. Body lice are potential vectors of relapsing fever, typhus, eruptive rickettsioses, and trench fever.

**Clinical Features**

- **Head lice** mainly affect children: itching and scratch marks (nape of neck and around the ears), which may become secondarily infected (impetigo) in prolonged infestation; presence of live lice and/or live (shiny, grey) nits attached to the hair shaft within 5 mm of the scalp.
- **Body lice** mainly affect populations living under poor conditions (refugees, prisoners, the homeless): itching and scratch marks (back, belt line and armpits), often inflamed and infected; presence of lice and nits in the clothing (parasites are not found on the body).
- **Pubic lice** (also called, crab lice) are considered to be a STI: itching and scratch marks (pubic and perianal area), but other hairy areas may also be affected (armpits, thighs, eyelashes); lice and nits at the base of the hair shaft, rarely visible.
- Examine contacts; check for associated systemic infection (body lice) or STI (pubic lice).

**Treatment**

- **Head lice:**
  - Apply 4% dimeticone to scalp and dry hair, paying particular attention to the areas behind the ears and around the nape of the neck. Do not reduce or exceed the recommended duration of application.
  - Children 6 months and over and adults: leave on hair for 8 hours, then rinse thoroughly.

- Keep away from flames and/or intense heat sources (including cigarettes) during application and until rinsing (risk of ignition).
- Or, if dimeticone is not available or in children 2 to 6 months you can use 1% permethrin lotion
- Children 2 months and over and adults: leave on hair for 10 minutes, then rinse thoroughly.
- Repeat application of either treatment after 7 days.
- Decontaminate combs, headwear and bedding (wash using hot water ≥ 60 °C 30 minutes, iron or dry in the sun or, if not feasible, seal in a plastic bag for 2 weeks.
- Treat as all contacts with live lice and/or live nits. Do not treat those with dead nits alone (dull, white, > 1 cm from scalp).
- Sometimes, mass treatment is done if there is outbreak with the Norwegian type.
- Apply 30 to 60 g (2 to 4 heaped soup spoons) of **0.5% permethrin powder** to the inside of the clothes and underclothes in contact with the skin (front and back, neck and waistline, sleeves and socks) in a fully clothed patient, then rub in the powder by hand. Leave for 12 to 24 hours.
- Treat other clothing (including headwear) and bedding in a plastic bag with 0.5% permethrin powder. Repeat in 8 to 10 days if the infestation persists.
- It is also recommended to do disinfection of clothing and bedding as above or as for head lice.

♦ **Pubic lice**
- Shave and/or apply 1% permethrin lotion to hairy areas (as for head lice).
- Treat the partner at the same time. Decontaminate clothing and bedding (as for head lice).
- Repeat the application after 7 days.

♦ **Oral treatment**
- Ivermectin PO 200 micrograms/kg single dose is an alternative: it is more practical than topical treatment (e.g. in the case of an epidemic or for treating contacts) and can be started right away in the case of secondary infection. A single dose may be sufficient; a second dose 7 days later reduces the risk of treatment failure.
- Ivermectin is not recommended for children < 15 kg or pregnant women (safety not established).
- Administration of ivermectin to patients with loiasis carries a risk of severe neurological complications when significant Loa loa microfilaraemia is present.
- Treatment of secondary bacterial infection, if present, should begin 24 to 48 hours before local antiparasitic treatment; local treatment is applied later when tolerated.

### 14.4.3 JIGGERS (TUNGA PENETRANS)

Diagnosis is not a problem, but education to the community on treatment is mandatory.

- Use 5% chlorohexidine to suffocate the jiggers.
- Extract the jiggers with clean pin.
- Suffocate jiggers by soaking feet in liquid paraffin or kerosene.
- The topical application of a two-component dimeticone with a defined viscosity, as found in treatments for headlice, is highly effective
- Give tetanus toxoid.
- Dust earthen floors with insecticide powders – this is highly recommended.
- Keep the patient comfortable and give adequate analgesia.
- Offer supportive feeding.
- Restore normal health and independence.

## 14.5 Vitamin Deficiencies Skin Conditions

### 14.5.1 PELLAGRA (NIACIN DEFICIENCY)

Pellagra is a disease caused by deficiency of niacin, also known as vitamin B-3. It is marked by dementia, diarrhea, and dermatitis, also known as “the three Ds”. If left untreated, pellagra can be fatal.

While it is less common than it used to be, it is still a significant problem in many developing countries. It can also affect people whose bodies don’t properly absorb niacin.

There are two types of pellagra, known as primary pellagra and secondary pellagra.

- Primary pellagra is caused by diets low in niacin as some people depend on corn as a staple food and can’t digest and absorb it unless prepared properly or tryptophan. Tryptophan can be converted to niacin in the body, so not getting enough can cause niacin deficiency.
- Secondary pellagra occurs when your body can’t absorb niacin. Things that can prevent your body from absorbing niacin including: alcoholism, eating disorders, medications, including anti-convulsants and immunosuppressive drugs.

The other causes are gastrointestinal diseases, such as Crohn’s disease and ulcerative colitis (IBD), cirrhosis of the liver and carcinoid tumors.
Pellagra can be difficult to diagnose because it causes a range of symptoms. There’s also no specific test for diagnosing niacin deficiency. You need to check for any gastrointestinal problems, rashes, or changes in your mental state. In many cases, diagnosing pellagra involves observing for the response to niacin supplements.

**Clinical Features**

- ♦ The main symptoms of pellagra are dermatitis, dementia, and diarrhea. This is because niacin deficiency is most noticeable in body parts with high rates of cell turnover, such as your skin or gastrointestinal tract.
- ♦ Dermatitis related to pellagra usually causes a rash on the face, lips, feet, or hands. In some people, dermatitis forms around the neck, a symptom known as Casal necklace.
- ♦ Additional dermatitis symptoms include:
  - Red, flaky skin
  - Areas of discoloration, ranging from red to brown
  - Thick, crusty, scaly, or cracked skin
  - Itchy, burning patches of skin
- ♦ In some cases, the neurological signs of pellagra appear early on, but they're often hard to identify. As the disease progresses, possible dementia symptoms include:
  - Apathy, depression, confusion, irritability, or mood changes. Others are; headaches, restlessness or anxiety, disorientation or delusions, sores on the lips, tongue, or gums, anorexia, difficult eating and drinking, nausea and vomiting.

**Management**

- ♦ Treatment is achieved through making dietary changes and a niacin or nicotinamide supplement.
- ♦ It may also need to be given intravenously. Nicotinamide is another form of vitamin B-3. With early treatment, many people make a full recovery and start feeling better within a few days of starting treatment.
- ♦ Skin improvement may take several months. However, if left untreated, primary pellagra usually causes death after four or five years.
- ♦ While recovering from either primary or secondary pellagra, it's important to keep any rashes moisturized and protected with sunscreen.

**Patients with pellagra may die if they are not treated.**

### 14.5.2 FLACKY PAINT DERMATOSIS

This is common in kwashiorkor resulting from inadequate protein or fat intake. The condition is characterized by generalized edema with a "flaky paint" dermatosis.

Lesions are darkly pigmented patches form, and these may peel or desquamate, rather like old, sun-baked blistered paint. This has led to the terms "peeling paint" or "flaky paint" dermatosis. Underneath these flakes are atrophic depigmented areas that may resemble a healing burn.

## 14.6 Connective Tissue Disorders

The exact cause of some forms of connective tissue disease is not known but researchers believe the disorder may be triggered by something in the environment of people who may be genetically susceptible. In these diseases,

the body's normally protective immune system produces antibodies that target the body's own tissues for attack.
The diseases include:
- Polymyositis
- Dermatomyositis
- Scleroderma
- Systemic Lupus Erythematosus
- Vasculitis
- Mixed Connective Tissue Disorder (MCTD)

## 14.6.1 POLYMYOSITIS AND DERMATOMYOSITIS

These are two related diseases in which there is inflammation of the muscles (polymyositis) and skin (dermatomyositis).
Symptoms of both diseases can include:
- Muscle weakness
- Fatigue
- Difficulty swallowing
- Shortness of breath
- Fever
- Weight loss

People with dermatomyositis may also have a skin involvement around the eyes and the hands.

**SCLERODERMA (OR SCLEROSIS)**

Scleroderma is a term for a group of disorders that causes thick, tight skin, buildup of scar tissue, and organ damage. These disorders fall into two general categories: localized scleroderma and systemic sclerosis.
Localized scleroderma is confined to the skin and, sometimes, the muscle beneath it. Systemic sclerosis also involves the blood vessels and major organs.

**SYSTEMIC LUPUS ERYTHEMATOSUS (SLE OR SIMPLY LUPUS)**

This is a disease characterized by inflammation of the joints, skin, and internal organs. There is a localized form called Discoid Lupus Erythematosus (DLE).

Clinical features of SLE include: a butterfly-shaped rash on the face, cheeks and bridge of the nose, sensitivity to sunlight, mouth ulcers and hair loss
Other signs include fluid around the heart and/or lungs, kidney problems, anemia or other blood cell problems, problems with memory and concentration or other nervous system disorders.

**VASCULITIS**

Vasculitis is a general term for more than 20 different conditions characterized by inflammation of the blood vessels. These can affect blood flow to the organs and other body tissues. Vasculitis can involve any of the blood vessels and the skin.

**MIXED CONNECTIVE TISSUE DISORDER (MCTD)**

People with MCTD have some features characteristic of other diseases, including lupus, scleroderma, polymyositis or dermatomyositis, and rheumatoid arthritis. While many people with mixed connective tissue disease have mild symptoms, others may experience life-threatening complications.

**Diagnosis**

To diagnose these conditions, various tests depending on what type of connective tissue disorder suspected. First take a detailed medical history, a family history followed by a physical examination. Further investigations or tests include:

- **Blood tests**
  - Markers of inflammation, such as C-reactive protein and Erythrocyte sedimentation rate (ESR)
  - Tests for antibodies, especially for autoimmune conditions
  - Tests for dry eyes or dry mouth
  - Blood and urine tests
  - Tissue biopsy

- **Others are imaging techniques**
  - X-rays
  - CT Scans and
  - Magnetic resonance imaging (MRI) scans

**Treatment**

Due to the many different types of connective tissue disorders, the treatments will vary depending on the person and the disease.

- Drug treatments include
  - Vitamin supplements
  - Corticosteroids – prednisone at 1-2 mg/Kg OD or BD for several weeks
  - Immunomodulators - monoclonal antibodies, azathioprine and cytokines
  - Antimalarial drugs – like hydrochloroquine
  - Calcium channel blockers – nifedipine
  - Methotrexate – at a dose of 7.5 to 25mg weekly
  - Cyclophosphamide and Corticosteroids combination
  - Pulmonary hypertension medications – Lasix, nitroglycerine, morphine and inotropes.

- **Physical therapy**

Patients also need regular schedule of appointments for follow up. Other specialists, such as ophthalmologist, physicians, rheumatologists can be consulted depending on what type of connective tissue disorder the patient has.

## 14.7 Skin Pigmentation Disorders

Normal skin color depends on melanin, hemoglobin (oxidized and reduced), and carotenoids. Melanin is the major color determinant and is responsible for variations in skin color. Pigmentary disorders typically indicate an increased amount of melanin, leading to darker color of the skin, called hypermelanosis or hyperpigmentation. Decreased or absent pigment makes the skin appear lighter or white, known as hypomelanosis or hypopigmentation.

Some skin diseases and conditions cause generalised or localised hyperpigmentation (increased skin colour, hypermelanosis), hypopigmentation (reduced skin colour, hypomelanosis), or depigmentation (absent skin colour, leukoderma).

A wood's lamp may be used to assess pigmentation during the examination of the skin, as pigmentary changes are often easier to identify while exposing the affected skin to long-wavelength ultraviolet ray.
These are many but the common examples include; piebaldism and post-inflammatory hypopigmentation.

### 14.7.1 PIEBALDISM

This results from congenital absence of melanocytes in affected areas of the skin and hair (depigmentation) that may have spontaneous expansion and contraction. This is a disorder of hypopigmentation, which involves skin and hair.

**Aetiology**
It is autosomal dominant mutation of the c-kit or SNA12 gene and is rare. The changes that occur include;

- Hypomelanosis or loss of colour on affected parts.
- Histologically, absence or markedly decreased melanocytes in affected areas

**Clinical Features**

- The characteristic pattern of the patient hairing;
  - White forelock
  - White macules/patches on forehead and the chin
  - Areas with lack of pigmentation on the trunk, anterior thorax, abdomen, mid-arm to wrist, mid-thigh to mid-calf (anterior and posterior)and normal pigmentation of hands, upper part of arm, shoulders, upper thighs, and feet to mid-calf.

- The features that distinguish piebaldism from vitiligo are
  - Presence of islands of normal pigmentation in areas of hypomelanosis.
  - Characteristic distribution of the patches.
  - May be congenital in origin.

- Frequently hereditary autosomal dominant pattern can be observed in family members.

Diagnosis is made through **history taking** during which you enquire about presence of deafness and screen for congenital megacolon particularly in infants.

**Manangement**

- Dipigmented areas are generally benign
- Treatment is mainly cosmetic
- Refer to a dermatologist for further management



### 14.7.2 POST-INFLAMMATORY HYPOPIGMENTATION

This occurs when there is a loss of skin color (pigmentation) after the skin heals from an injury. The pigment-producing cells (melanocytes)

**Management**

The following agents can be used to treat epidermal melanosis or hypomelanosis singly or, more effectively, in combination:

- Hydroquinone cream 1-3%
- Topical retinoids e.g., Isotretinoin cream 0.1 %
- Topical corticosteroids
- Glycolic acid and other fruit acids
- Azelaic acid

Other treatment modalities are;

- Skin grafting using skin obtained from large and flat parts of the body with normal skin.
  - Minigrafts or pinch grafts: most successful, and
  - Culture grafts: not always cosmetically acceptable
  - Photo-chemical method using - PUVA: not always easily available

## 14.8 Dermatological Emergencies

Although fairly uncommon, dermatological emergencies occur and are important to recognize as they can be life-threatening and easily missed in clinical practice.

The common examples of dermatological emergencies include:

- Eczema Herpeticum
- Erythroderma (exfoliative dermatitis)
- Stevens-Johnson syndrome (SJS) and Toxic Epidermal Necrolysis (TEN)
- Drug reactions eg Anaphylaxis, Fixed Drug Eruption and Photosensitive Drug Eruptions
- Erythema Multiforme

## 14.8.1 ECZEMA HERPETICUM

Eczema herpeticum is a complication of atopic eczema that occurs with infection of the herpes simplex virus (HSV). EH is thought to be due to a reduced level of immunity to HSV in patients with atopic dermatitis and the lesions become secondarily infected with Herpes simplex hominis virus.

**Risk factors**

EH can affect any age but most commonly affects infants and children. It is also more common in those with severe atopic dermatitis/eczema. The other conditions related to EH include burns, pemphigus vulgaris and cutaneous T-cell lymphoma.

**Clinical Features**

Typical symptoms of eczema herpeticum include:

- General malaise, fever, and itchy, painful lesions
- Gritty or sore eyes (if eye involvement present)
- The symptoms tend to develop 5-12 days following contact with HSV.[1]\
- On physical examination, typical clinical findings of eczema herpeticum include:
  - Groups of itchy painful blisters, erosions and crusted papules
  - Local lymphadenopathy near the site of the lesions
  - Evidence of secondary bacterial infection (e.g. cellulitis/impetigo).

Eczema herpeticum can be mistaken for impetigo as the typical golden crusting of impetigo can mask the primary herpetic lesions.

**Investigations**

Diagnosis is mainly based on clinical findings. However, viral and bacterial swabs can be taken from the base of a new blister.

Swabs for viral studies (including Tzank smears): shows the presence of HSV type 1 or 2 would confirm the diagnosis.

Bacterial swabs for staining and culture: may reveal secondary infection with Staphylococci or Streptococci.

**Management**

All patients with eczema herpeticum should be referred to a dermatologist for urgent assessment. If there is any ocular involvement an ophthalmology review should also be requested.

The mainstay of treatment is antivirals (e.g. Aciclovir or Vancyclovir). In serious cases, intravenous Acyclovir can be considered.

If a secondary bacterial infection is present, antibiotic treatment is promptly given.

**Complications**

- Herpes hepatitis
- Encephalitis
- Disseminated intravascular coagulation
- Death (very rare)

## 14.8.2 ERYTHRODERMA (EXFOLIATIVE DERMATITIS)

Erythroderma is the intense redness of the skin covering at least 90% of the skin surface area, usually secondary to pre-existing inflammatory skin disease like eczema, psoriasis (pustular), scabies and drugs.

The pathology of erythroderma is still not fully understood. It is thought to be a complex process leading to rapid epidermal cell turnover.

Erythroderma usually occurs in those with inflammatory skin disease. The most common precipitants are eczema and psoriasis. The other causes include Norwegian scabies, drug eruptions (e.g. allopurinol, gold, sulfonamide, sulfonylureas, isoniazid), and cutaneous lymphoma (Sezzary syndrome).

**Clinical Features**

- Red, painful and itchy skin over a large area
- Malaise, anorexia, fever and restlessness
- Hot, erythematous skin covering at least 90% of the skin surface
- Signs of desquamation or peeling of the skin
- Generalised lymphadenopathy
- Erythroderma
- Important areas to cover in the history include past medical history (e.g. history of inflammatory skin diseases such as psoriasis or eczema) and drug history (e.g. the recent commencement of a new drug).

**Investigations**

The diagnosis of erythroderma is usually clinical; however, baseline observations and blood tests should be completed. If the diagnosis is unclear, a skin biopsy should be considered.

**Management**

- All patients with suspected erythroderma should be admitted and be reviewed by a dermatologist.
- Patients with erythroderma who are systemically unwell may be admitted to a burn's unit or intensive care unit.
- Drug management
  - Emollients are used in abundance and cool, wet dressings are the mainstays of treatment.
  - Treat any underlying causes (e.g. discontinuing suspected trigger medication).
  - Topical corticosteroids like Betamethasone or other stronger preparations are used in many cases to reduce inflammation and itching.
  - Patients should be managed in a warm room (~30°C) and have their fluid balance, electrolytes and body temperature monitored closely.

**Complications**

- Dehydration
- Electrolyte imbalance
- Secondary bacterial infection

- Hypothermia secondary to impaired thermoregulation
- Cardiac failure
- Death

### 14.8.3 STEVENS-JOHNSON SYNDROME AND TOXIC EPIDERMAL NECROLYSIS

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are variants of the same condition. They are severe mucocutaneous reactions, almost always secondary to medications.

The mechanisms of SJS and TEN are not fully understood. SJS and TEN most commonly occur due to medications including allopurinol, anti-epileptic drugs, sulfonamides, antivirals, nonsteroidal anti-inflammatories, salicylates, sertraline and imidazoles

SJS and TEN are statistically more common in females and in those with HIV. Infections such as mycoplasma and cytomegalovirus can also trigger the condition.

The classification of SJS SJS-TEN and TEN is that.
SJS has <10% of body surface, SJS/TEN is between 10-30% and TEN is >30%

**Clinical Features**
- Prodromal flu-like/non-specific upper respiratory tract illness.
- A painful rash starting on the trunk which then spreads over several hours to days onto the face and limbs.
- Mouth ulcers or soreness.
- Painful or irritated eyes.
- A rash which initially starts as macules which then progresses to blisters and eventually sheets of desquamation.
- Positive Nikolsky's sign: gentle rubbing of the skin causing desquamation or scaling.
- Ulceration, erythema and blistering in the oral cavity.
- Conjunctivitis or corneal ulceration.
- Important things to enquire about in the history include drug history (e.g. newly commenced medication – symptoms can occur 5-28 days after starting the causative medication), past medical history (e.g. HIV)

In young children, staphylococcal scalded skin syndrome can present with similar clinical features. A skin biopsy with histology is required for a definitive diagnosis.

**Investigations**

- The diagnosis is usually made clinically although a skin biopsy is required to confirm the diagnosis
- The condition is classified based on percentage body surface area of the detached epidermis.

**Management**

- These patients require hospital admission, urgent dermatology referral and admission to a specialist burns unit or intensive care unit.
- Any suspected causative drugs should be stopped.
- Fluid balance and electrolytes should be monitored closely.
- Patients will require adequate analgesia as the condition is extremely painful.
- Ophthalmology opinion will be required if there is evidence of ocular involvement (e.g. corneal ulceration).



**Complications**

Complications of SJS and TEN include:

- Dehydration/hypovolaemic shock
- Secondary infection of the skin or mucous membranes
- Other infections
- Disseminated intravascular coagulation
- Thromboembolism
- Death (mortality rates are approximately 10% for SJS and ~30% for TEN).

## 14.8.4 ACUTE DRUG REACTIONS

Cutaneous drug reactions usually develop 1–2 weeks following initiation of a medication but, some severe adverse reactions may present later (e.g., 4–6 weeks) after treatment initiation. The specific conditions include:

**ANAPHYLAXIS**

Most cases due to an IgE-mediated allergic reaction; however, certain drugs (e.g., penicillins, opiates, and radiocontrast media) can act directly on mast cells to liberate histamine or lead to leukotrienes (e.g., NSAIDs). Urticarial eruptions due to serum sickness may persist and have associated systemic symptoms.

**FIXED DRUG ERUPTION**

**I**solated, well-demarcated erythematous lesions, often on the extremities, face, or genitalia that can be painful/blister. Rechallenge may cause recurrent lesions at the same site. Common drugs include sulfonamides, tetracyclines, barbiturates, and NSAIDs.

**PHOTOSENSITIVE DRUG ERUPTIONS**
Cutaneous reaction limited to sun-exposed sites. May be due to either a phototoxic reaction (non-immune, e.g., tetracyclines, NSAIDs, and fluoroquinolones) or a photoallergic reaction (immune-mediated, e.g., thiazide diuretics and sulfonamides). Some drugs cause photosensitive porphyria cutanea tarda or photo-onycholysis.

**URTICARIA AND ANGIOEDEMA**
The two (urticaria & angioedema) account for 25% of drug reactions. Urticaria occurs in 2 forms: acute and chronic based on the duration of onset of symptoms. Sudden onset of intensely pruritic erythematous, oedematous skin lesions that resolve within 24 hours is in the acute type. Chronic urticarial is often idiopathic but can be due to physical causes, e.g., dermographism, cold/heat contact, delayed pressure, vibration, and sunlight exposure. Other cases are cholinergic in origin from heat orexercise, contact, aquatic animals, or water. Urticaria may be associated with angio-oedema where there is deeper tissue oedema that can involve mucous membranes. May also be associated with life-threatening anaphylaxis in which oropharyngeal irritation, bronchospasm, hypotension, and tachycardia may occur.

**ERYTHEMA MULTIFORME**
This is the rapid onset of erythematous lesions with a typical 'target' appearance (see Fig. 12.2), often affecting the extremities or the face. Severe 'EM major' variant involves mucous membranes and becomes SJS. EM more commonly has an infectious aetiology but may be triggered by certain medications.

**ACUTE GENERALIZED EXANTHEMATOSUS PUSTULOSIS**
Rapid onset of widespread sterile pustules, often starting in skin creases. Associated with fever and leucocytosis. More rapid onset than generalized pustular psoriasis.

**DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS**
Presents as an exanthematous drug eruption, but associated with fever and systemic symptoms, which commonly include facial oedema, lymphadenopathy, and drug-induced hepatitis. Common drugs include anticonvulsants, sulfonamides, and allopurinol. It is associated with 10% mortality.

**Investigations**
The range of investigations is wide, but the common ones include:

- Blood tests e.g., Serum immunoglobulin levels (IgE, etc), complete blood count and swabs for culture.
- Skin prick test for IgE-mediated hypersensitivity or if no clear trigger for acute urticarial or angio-oedema can be identified.
- Skin patch tests are not usually indicated in urticarial or angio-oedema.
- Skin biopsy for histopathology and immunoflorescence

**Treatment**

- Anaphylactic reactions require immediate treatment, but the holistic care is provided by a multidisciplinary team.
  - First lay the patient flat on the couch or bed.
  - Secure the airway and give Oxygen by mask or nasal prongs.
  - Ensure good cardiac function and do pulse oximetry monitoring.
- Drugs used include:
  - Giving S/C or IM adrenaline 0.5mg (0.5mL of 1:1000 adrenaline injection), and repeat every 5minutes according to BP, pulse, and respiratory function. IV adrenaline may be required if the patient is severely ill with poor circulation.
  - Establish IV access (large bore) and start IV fluids if hypotensive.
  - Giving IV hydrocortisone 100–300mg and IV chlorpheniramine 10–20mg. Continue H1-antagonist (e.g., oral chlorpheniramine 4mg every 4–6h) for at least 24–48h and continue if urticaria and pruritus persist.
  - If the patient deteriorates, start IV aminophylline infusion. Patients on β-blockers require IV salbutamol infusion.
  - Acute urticaria ± angio-oedema is usually not life-threatening, unless associated with anaphylaxis or upper airway obstruction. If airway obstruction occurs, treat as anaphylaxis and get urgent other specialists' advice for airway management.
  - Give oral antihistamines such as hydroxyzine 25mg or chlorphenamine 4mg.
  - A single dose of oral prednisolone (0.5–1mg/kg) may be given but should not be continued without specialist advice.
  - When the patient's condition has stabilized, discharge on regular maintenance treatment with oral non-sedating antihistamine (nsAH) (e.g., cetirizine 10mg/24h, levocetirizine 5mg/24h, desloratadine 5mg/24h, or fexofenadine 180mg/24h).
  - For resistant chronic urticarial, the treatment should include high-dose combination nsAH, leukotriene antagonists, or systemic immunosuppressants, e.g., omalizumab (anti-IgE monoclonal antibody).

## 14.8.5 EXFOLIATIVE DERMATITIS

Synonyms: Exfoliative erythroderma syndrome, erythroderma.

**Clinical Features**

Serious, life-threatening reaction pattern of the skin characterized by generalized and confluent redness with scaling and associated systemic toxicity, generalized lymphadenopathy, and fever. The disease presents as an acute and also as a chronic one. More than 50% of the patients have a history of pre-existing dermatosis, commonly eczematous dermatitis (atopic, contact), psoriasis, drug reaction. They may also have pre-existing leukaemia, lymphoma, or other malignancy. In up to 10–20% no possible cause is identified.

Constitutional symptoms include fatigue, weakness, anorexia, weight loss, malaise, feeling cold with (shivering), clinically red appearing skin that is thickened and with scaly lesions and no recognizable borders. Oedema of lower legs and ankles may occur. When palms and soles are involved there is thickening and fissuring. There tends to be alopecia (hair loss, but not uniform) and nails tend to be shed.

Prognosis is guarded and therefore this is a medical problem that should be dealt with using modern inpatient dermatology facilities and personnel. The disease has many multi-systemic complications.

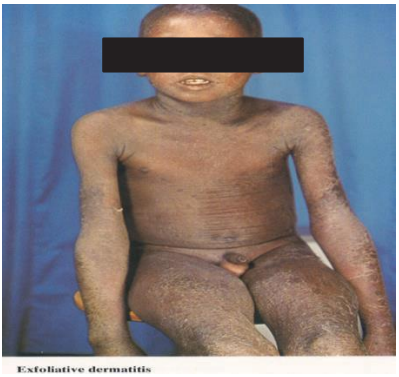
**Management**

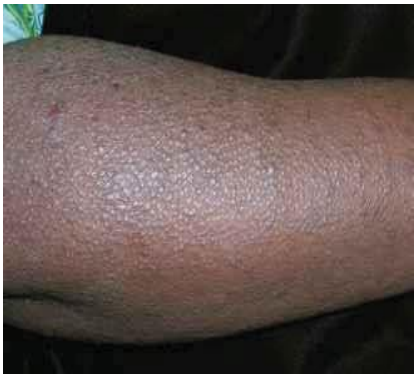
- ♦ Bath soaking
- ♦ Bland emollients: Liquid paraffin, emulsifying ointment.
- ♦ Nursing care: Single room, keep warm,etc.
- ♦ Systemic management:
- Supportive – Fluid, electrolyte, protein replacement.
- Systemic steroids used under specialist care are prednisone or prednisolone 0.5mg/kg/day in 2 divided doses.
- ♦ Confirm primary skin disorder by skin biopsy.

**Note: Erythroderma may be purely secondary to HIV infection.**

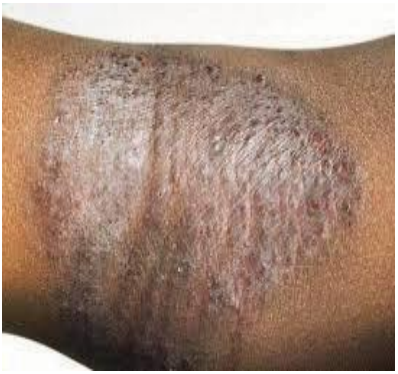
**Figure 14.1: Pictures of Common Skin Conditions**

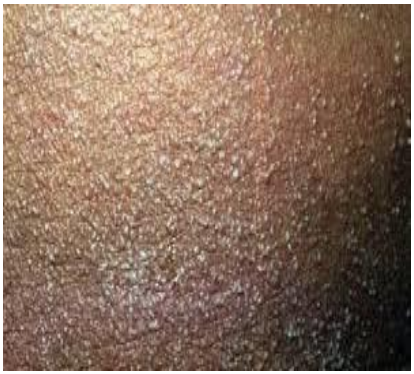
**A. Common Inflammatory Conditions**



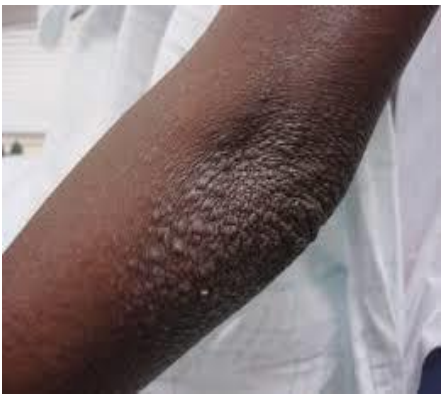



Pictures of exfoliative dermatitis and lichen simplex chronicus on the lower limb

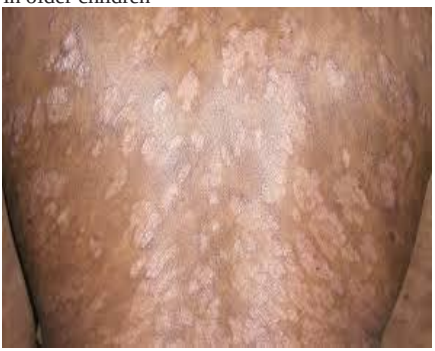




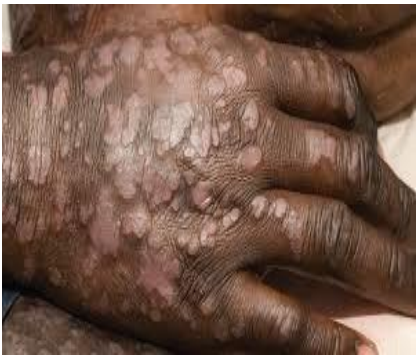
Lichen simplex chronicus and lichenoid eruption

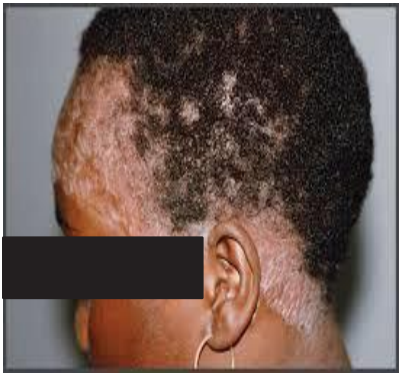



Sites of Atopic eczema (flexure) and extensor surface in older children

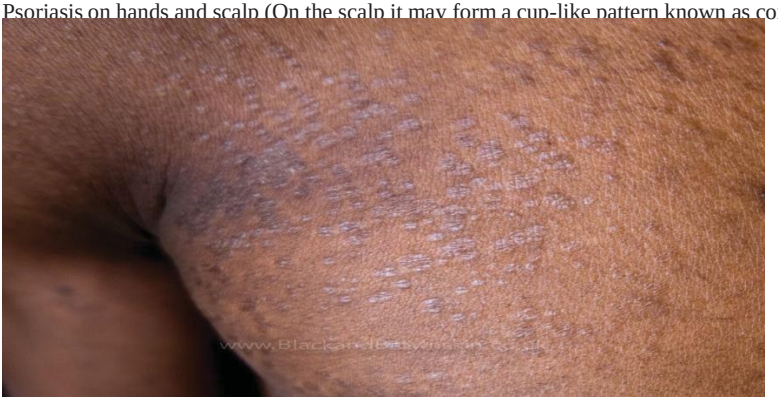


Pictures of plaque psoriasis on lower limbs and on the trunk

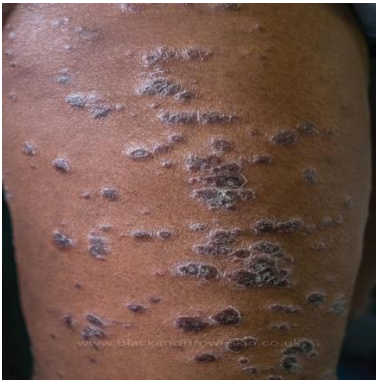


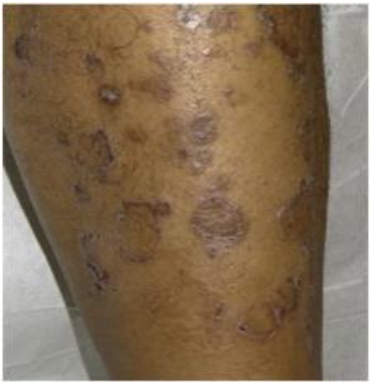


Psoriasis on hands and scalp (On the scalp it may form a cup-like pattern known as corona psoriatica)

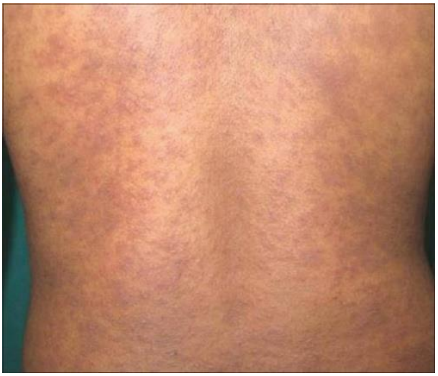


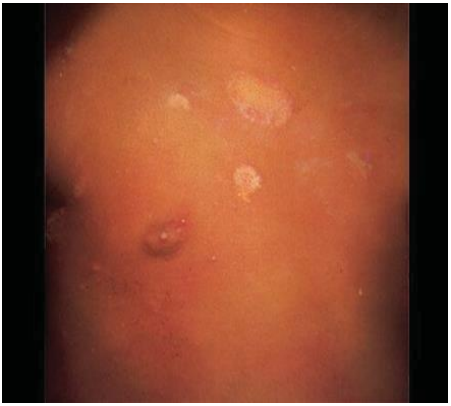
Shiny papules or plaques of Lichen planus



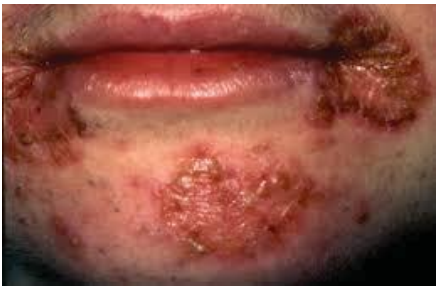


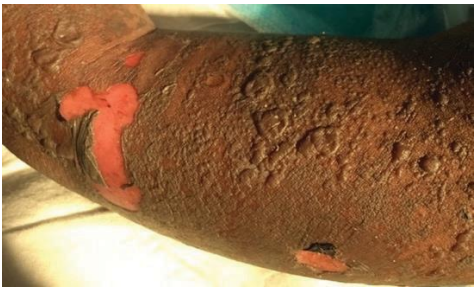
Lichen planus lesions on the lower limbs





Picture of pityriasis rosea, usually it has a larger area of lesions termed a herald (mother) patch

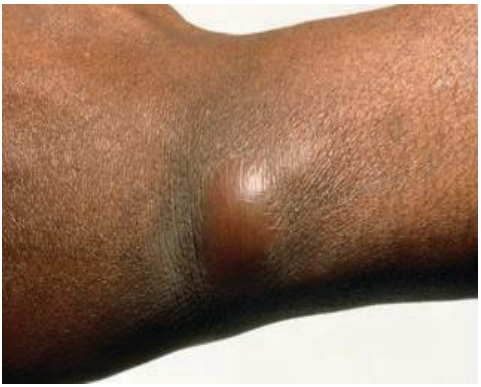




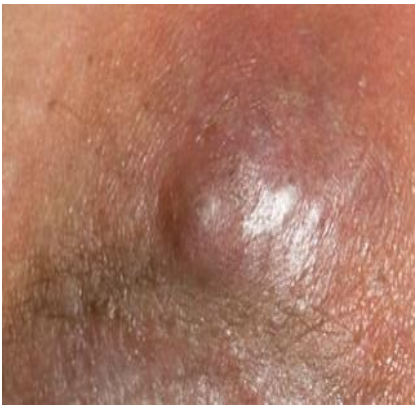
Pictures of Impetigo and Staphylococcal Scalded Skin Syndrome in children

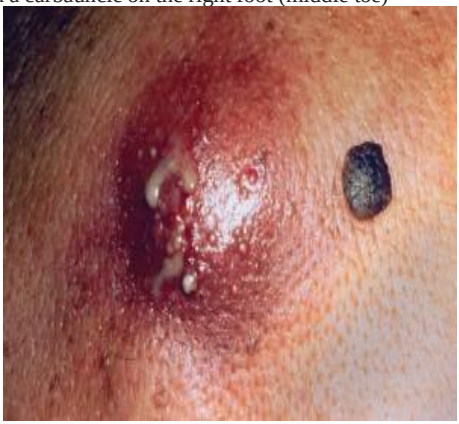
**Figure 14.1 continued: Pictures of Common Skin Conditions**

**B. Bacterial skin infections**

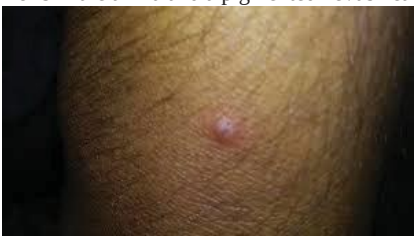


Boil above the wrist (may be confused for ganglion) and a carbauncle on the right foot (middle toe)





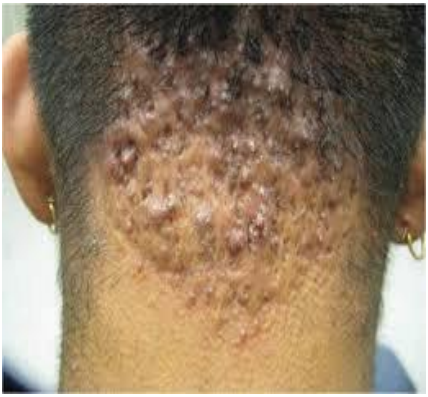
Boils in the axilla and a pigmented nevus near the lesion in the picture on the right side

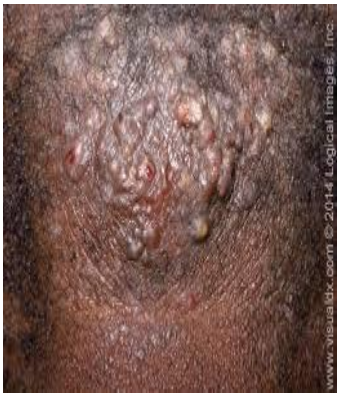


Folliculitis on upper and lower limbs of adult patients

**Figure 14.1 continued: Pictures of Common Skin Conditions**

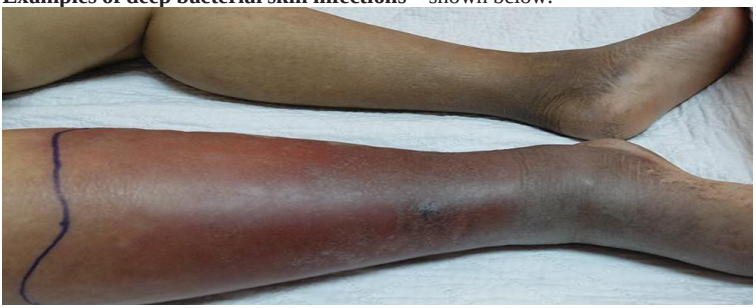
**Bacterial skin infections**



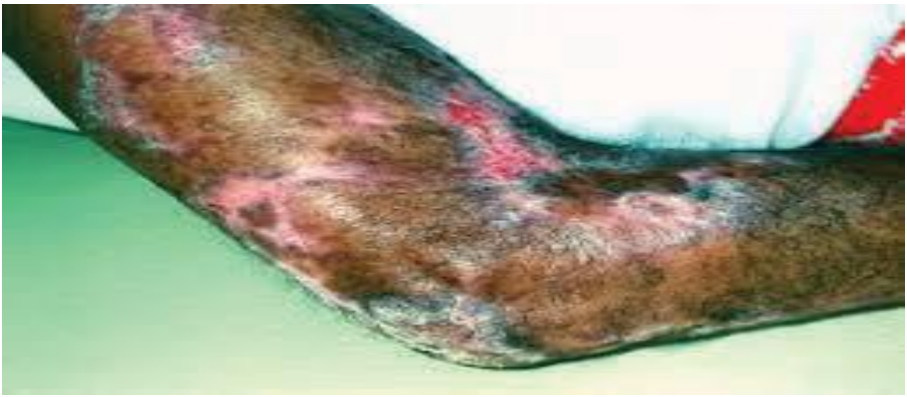



Sycosis barbae and Acne keloidalis nuchae (on nuchal areas) both of which are differentials of folliculitis

**Examples of deep bacterial skin infections –** shown below:

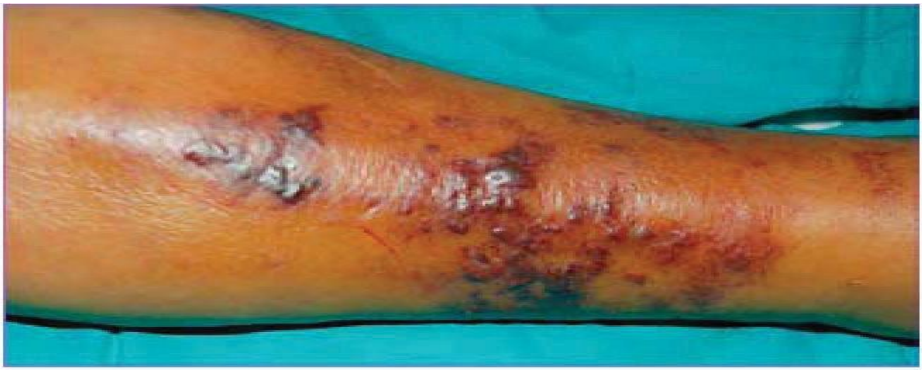


Cellulitis on right lower limb



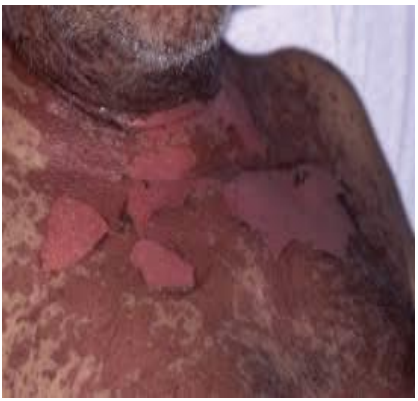
Necrotizing fasciitis on the arm with evidence of half-healing, hypopigmentaion and scarring

**Figure 14.1 continued: Pictures of Common Skin Conditions**



Necrotizing fasciitis in early stages, may be mistaken with burns blisters

**C. Steven -Johnsons Syndrome**

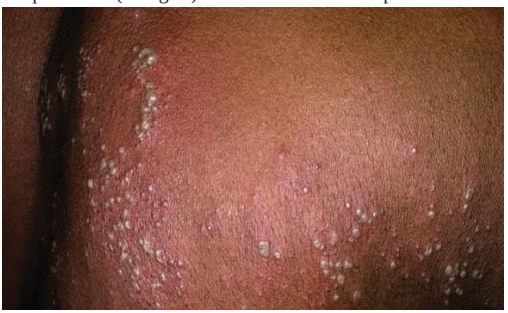


Patient with Steven Johnsons Syndrome (SJS) lesions on the back and front parts of the trunk

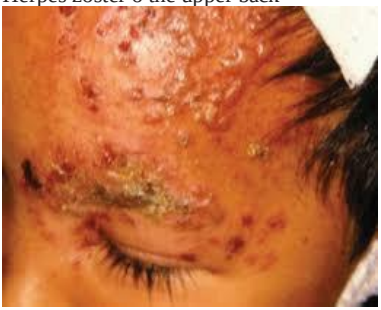
**Figure 14.1 continued: Pictures of Common Skin Conditions**

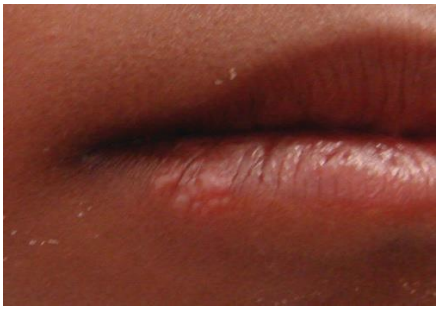
**D. Viral Skin Infections**

Herpes zoster (Shingles) on the back and H. simplex around the mouth.

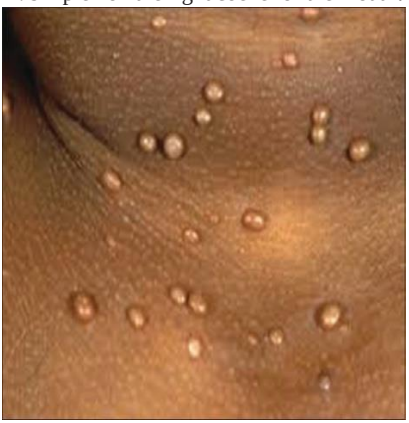


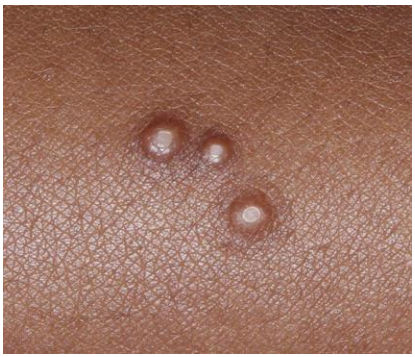
Herpes zoster o the upper back





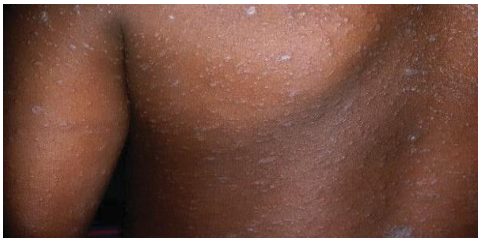
Herpes zoster ophthalmicus (involving the facial nerve), it may be serious if it involves the tip of the nose and H. simplex on the right coener of the mouth**.**



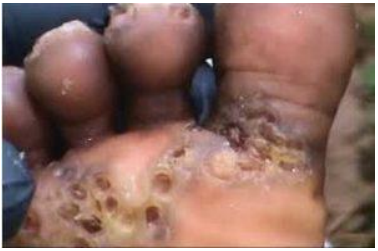
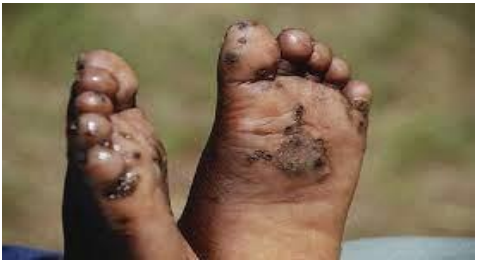


Molluscum contagiosum, the later picture shows lesions in semi-healing stage

**Figure 14.1 continued: Pictures of Common Skin Conditions**
**F: Parasitic Skin Conditions**

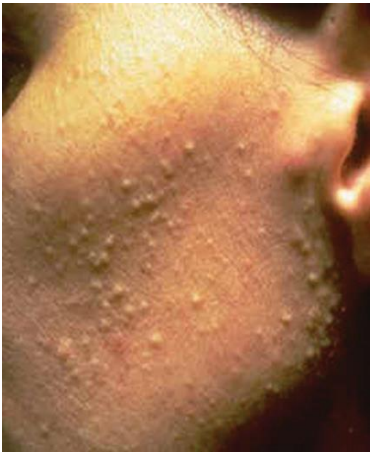
Localized and generalized forms of scabies. There is also genital that is classified as an STI

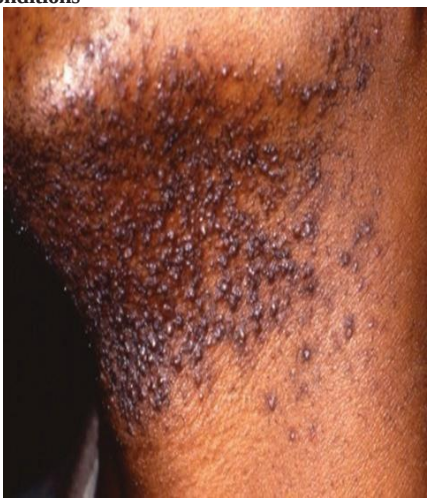



Jiggers (Tungiasis) of the feet Can be secondarily infected by bacteria


Head lice (Pediculosis) and the causative parasite that forms knits on hairs. The one involving the pubic area is called crab louse

**Figure 14.1 continued: Pictures of Comon Skin Conditions**





Sycosis barbae (due to bacterial infection), and pseudo-sycosis barbae (a transient condition due to pieces of hairs piercing the skin causing a local allergic reaction or papules

# 15. Genito-urinary Disease Urinary Tract and Renal Conditions

## 15.1 Haematuria

Presence of blood in urine confirmed by presence of at least 5 RBCs/HPF in 3 of 3 consecutive centrifuged specimens obtained at least 7 days apart.

**Classification**

- Microscopic (seen only by microscopy or urinalysis) vs. Gross hematuria (visible to the eye).
- Intermittent Vs Persistent.
- Symptomatic Vs. Symptomatic
- Initial hematuria Vs. all-stream hematuria and terminal hematuria.

**Table 15.1: Causes of Haematuria**

| Glomerular Causes | Non-Glomerular Causes |
|---|---|
| • Glomerulonephritis<br>• Lupus nephritis<br>• Polycystic Kidney Disease<br>• IgA nephropathy<br>• Good Pastures Disease | • Trauma (*external or urethral instrumentation*)<br>• Renal stones/Nephrolithiasis<br>• Exercise<br>• Menstruation<br>• Malignancy (renal cell carcinoma, bladder cancer, prostate cancer)<br>• Bleeding disorders (*thrombocytopenia, coagulopathy, use of blood thinners/ hematological disorders like sickle cell anemia*) |

**Clinical Features**

- Flank pain/mass, lower abdominal pain, dysuria, urinary urgency or frequency, fever, active menstruation, passing stone, recent throat or skin infection, joint pains, oral ulcers, rash, hemoptysis, leg swelling, hearing loss, constitutional symptoms (weight loss, anorexia, cachexia), back-pain
- In the history taking, enquire about: previous history of hematuria, family history of hematuria, any procedures in the recent past, drug history**.**
- Perform a complete medical examination: Blood pressure, edema, suprapubic/ flank tenderness or mass, palpable kidneys.

**Investigations**

- **Lab tests**: Urinalysis, urine microscopy, renal function tests, urine cytology where malignancy is suspected
- **Imaging**: ultrasound (kidney, ureter, bladder); Abdominal-pelvic CT scan, cystoscopy.
- Kidney biopsy

**Management**

This depends on the underlying cause.

- For asymptomatic, intermittent hematuria with negative imaging, stable renal functions and no presence of proteinuria: reasonable observations and follow up.
- Overt hematuria needs prompt management.
- Severe hematuria: Ensure hemodynamic stability.
- Correct any hematological abnormality with blood products, transfusions, or medications.
- If it is drug-induced hematuria (blood thinners), stop the medication and give

the appropriate antidote.

NB: Haematuria is **a serious sign of disease** and should be aggressively investigated. Refer urgently for appropriate management where necessary.

## 15.2 Pyuria

Defined as presence of >10 WBC per HPF on a urine specimen and is suggestive of urinary tract inflammation. It is most commonly associated with bacterial UTI. When pyuria is associated with haematuria or proteinuria, it is suggestive of parenchymal renal diseases interstitial nephritis.

Sterile pyuria refers to the presence of white blood cells within the urine, in the absence ofinfection as confirmed by negative urine cultures. Persistent sterile pyuria is often due to tuberculosis therefore TB cultures are recommended.

**Investigations**

- 24-hour urine collection for Mycobacteria and brucellosis
- Polymerase chain reaction (PCR) test

**Treatmen**t

- Depends on the urine culture results for sensitivity. Empirically quinolonones, cephalosporins.

## 15.3 Urinary Tract Infection (UTI)

This is an infection affecting the urinary system.

**Classification**

- Uncomplicated UTI: a bacterial infection of the bladder and associated structures with no structural abnormality, no comorbidities (diabetes,

immunocompromised state, or pregnancy). It is also known as cystitis or lower UTI.
- Complicated UTI: when it occurs in the presence of structural abnormalities and comorbidities. In males, UTI should be considered complicated.

**Risk factors**
- Being female (short urethra, proximity to anus and hormonal changes),
- Catherization
- Sexual intercourse
- Spermicides
- Frequent pelvic examination
- Immunosuppression
- Kidney transplant
- Use of antibiotics
- Diabetes mellitus.

Pathogenic bacteria ascend from the perineum, causing the UTI.

**Escherichia coli** is the most common organism in uncomplicated UTI. Other organisms include proteus mirabilis, klebsiella, and enterococcus (gram negatives).

**Clinical Features**
- Urinary frequency
- Urgency
- Suprapubic discomfort
- Dysuria.

**Diagnosis**
- Mostly clinical diagnosis from history
- Urinalysis (WBCs, pus cells).
- Urine culture to confirm the diagnosis and evaluate drug sensitivity to identify the best choice of therapy. Proper urine sample collection is recommended to avoid contamination.

**Management**
- Many cases of uncomplicated UTIs will resolve spontaneously, without treatment. Most patients seek therapy for symptoms relief.
- Encourage increased fluid intake unless there is renal/heart failure.
- Treatment is aimed at preventing spread to the kidneys or developing into upper tract disease or pyelonephritis.
- Oral antibiotics: Fluoroquinolones: *nitrofurantoin 100mg bd, ciprofloxcin 5000ng bd, ofloxacin*, 2nd/3rd generation cephalosporins; *cefixime 400mg od, cefpodoxine 100ng bd.* Trimethoprim/sulphamethoxazole (TMP/SMX) or amoxicillin/clavulanate can be used if sensitive. A lot of resistance has been reported with TMP/SMX for 3-5 days.

## 15.4 Acute Pyelonephritis

Acute pyelonephritis is a bacterial infection causing inflammation of the kidneys. Also referred to as upper tract UTI.
It usually occurs as a complication of an ascending UTI from the bladder. Less commonly, it can result from hematogenous spread especially in those in those with ureteral obstructions, immunocompromised and debilitated patients.

Classified as complicated when it occurs in pregnancy, kidney transplant, urinary tract anatomical abnormalities, renal failure, immnunocompromised patients and those with hospital-acquired bacterial infections.

**Aetiology**

- Mostly gram-negative bacteria (*Escherichia coli, Proteus mirabilis, Klebsiella pneumonia, Enterococci fecalis and Enterobacter)* from fecal flora.
- In ICUs, antibiotic-resistant enterococci, *Klebsiella. pneumoniae*, *Pseudomonas aeruginosa,* and *Candida albicans.*

**Complications:** untreated acute pyelonephritis can lead *to* abscess formation, septic shock, and kidney damage.

**Clincial Features**

- Fever
- Flank pain
- Nausea
- Vomiting
- Burning on urination (dysuria)
- Increased frequency
- Urgency.

**Investigations**

- Good history taking and physical examination.
- Tests: urinalysis (pyuria), presence of nitrite suggests E. coli infection; proteinuria, microscopic hematuria; urine cultures and sensitivity; TBC, renal function tests.
- Imaging: May not be required routinely. Indicated in thoes with complicated acute pyelonephritis. Include renal/ureter/bladder ltrasound (negative test does not exclude the pathology).

**Management**

- Can be managed as either outpatient or inpatient for 7-14 days.
- Healthy, young, non-pregnant women can be managed as outpatients while the very young, elderly, immunocompromised, poorly controlled diabetes, post-renal transplant, pregnant, structural abnormalities, those who cannot tolerate oral intake should be managed as inpatients.

**Drugs:** Antibiotics, analgesics, and antipyretics.

- **Antibiotics**: Uncomplicated infection:
  Empirical treatment based on the local antibiotic resistance patterns.
  Adjust treatment with urine culture and sensitivity results.

I) Outpatient oral management

- Fluoroquinolones: Ciprofloxacin 500mg bd, Ciprofloxacin sustained release 1000mg od, Levofloxacin 500mg or 750mg od 7 days.
- Cotrimoxazole (Trimethoprim/sulphamethoxazole) 960mg bd 14 days
- Cephalosporins: 2rd and 3rd generation: cefixime 400mg od; cefpodoxine 100mg bd.

II) Hospitalised patients with complicated acute pyelonephritis

- Intravenous (IV) antibiotic treatment until there are clinical improvements
- Fluoroquinolones
- Aminoglycoside ± ampicillin
- Second-generation cephalosporin broad-spectrum cephalosporin
- Beta-lactam ± beta-lactamase inhibitor ± aminoglycoside,
- Aminoglycoside ± beta-lactam
- Carbapenems

III) For those in ICU due to severe sepsis or septic shock (consider the antibiotic resistance pattern of the causative bacteria in your locality).

- Piperacillin/tazobactam
- Carbapenem are administered after urine cultures results

IV) For those with complicated acute pyelonephritis due to urinary tract obstruction.

- Identify the cause of obstruction using specialized, individualized diagnostic methods in cosultation with urologists and radiologists.

**Imaging methods for upper UT obstruction**

- Abdominal radiography (Kidney, Ureter, and Bladder X-ray study ± Intravenous pyelogram),
- Ultrasonography
- CT scan renal: the most useful for diagnosing emphysematous pyelonephritis and checking the scope of a lesion.

**Lower Urinary Tract Obstruction**

Commonly presents as urinary retention, residual urine volume >100ml is significant.

**Diagnosis**

- Keep a voiding diary
- Cytology, ultrasound examination, and CT scan.
- For male patients: blood prostate-specific antigen (PSA) test and a transrectal ultrasound-guided prostate biopsy.

NOTE: Consider a neurogenic bladder or obstructive UTI if patient has *overflow incontinence, Parkinson's disease, diabetic neuropathy, cerebral infarction, spine injury, a neurological disorder, or recurrent UTIs.*

**NB: All patients with acute pyelonephritis should be subjected to a urinary culture test before empirical antibiotic administration.**

**Table 15.2:Antiobotic Treatments for Acute Pyelonephritis**

| Antibiotics | Examples and dosages | Side effects |
|---|---|---|
| Fluoroquinolones | Ciprofloxacin 500mg bd<br>Ciprofloxacin sustained release 1000mg od<br>Levofloxacin 750mg od | |
| Aminoglycosides | Gentamicin<br>Amikacin 15mg bd | Nephrotoxic and ototoxic. Monitor renal functions, hydrate patient, avoid of stop in renal failure of hearing loss. |
| Beta-lactam+ beta-lactamase inhibitor | Amoxicillin-clavulinate 1gm bd<br>Piperacillin-tazobactam 3.375 g iv every 6 hours | |
| Cephalosporins | Cefpodoxime proxetil 200mg bd | |
| Carbapenems | Meropenem 500-1000mg bd<br>Imipenem+Cilastatin 500mg tid/qid | |

NOTE: Upon fever alleviation, oral antibiotics may be started basing on the antibiotic sensitivity and resistance patterns.

## 15.5 Acute Prostatitis

Prostatitis refers to inflammation of the prostate gland due to bacterial and non-bacterial causes.

**Table 15.3: Causes of Prostatitis**

| Category | Description |
|---|---|
| I | Acute bacterial prostatitis |
| II | Chronic bacterial prostatitis |
| III | Chronic prostatitis/chronic pelvic pain syndrome |
| | a. Inflammatory |
| | b. Non- inflammatory |
| IV | Asymptomatic prostatitis |

## 15.5.1 ACUTE BACTERIAL PROSTATITIS

**Aetiology***: E. coli*, *Pseudomonas aeruginosa* and *Enterococci*

**Risk factors:** Urinary tract catheterization, benign prostatic hypertrophy.

**Clinical Features**

- Symptoms of UTIs (dysuria, frequency, urgency).
- Symptoms of prostatitis (perineal pain, genital pain, painful urination, and rectal pain).
- Symptoms of bacteremia (fever, chill, joint pain, and muscle pain).

**Digital Rectal Examination (DRE):** a soft, swollen, and tense state of the prostate and severe pressure pain. NB: Avoid prostate massage due to severe tenderness and risk of inducing bacteremia/sepsis.

**Complications:** acute UT obstruction, epididymitis, prostatic abscess, sepsis, and chronic bacterial prostatitis, septicaemia, chronic bacterial prostatitis, prostatic abscesses.

**Diagnosis:** midstream urine culture, blood culture test, elevated serum PSA levels, Trans-rectal ultrasonography, or CT scan to screen for prostatic abscesses.

**Treatment:** Requires immediate inpatient management and empirical intravenous antibiotics after collection od urine and blood samples for culture and sensitivity then adjsut treatment accoding to the test results. Ensure proper hydration, bed rest, analgesics (NSAIDs).

**Antibiotics:** 3rd generation cephalosporins, a broad-spectrum β-lactam/β-lactamase inhibitor, or carbapenem.
For severe infections suspected as sepsis or recurrent infections, a combination antibiotic therapy may be considered as early empirical antibiotic treatment using β-lactams and aminoglycosides.
For prostate abscess: transrectal ultrasound-guided needle aspiration, transrectal ultrasound-guided catheter drainage, perineal ultrasound-guided drainage, or transurethral abscess resection (consult the surgical/radiology team).

**Other drugs:** Alpha-blockers (tamsulosin, terazosin, doxazosin) if urinary symptoms and significant residual urine. NSAIDs for pain and inflammation.

## 15.6 Nephrotic Syndrome

Nephrotic syndrome is defined as a clinical syndrome comprising of features of heavy proteinuria and hypoalbuminemia due to an increased permeability of the glomerular basement membrane. The consequence of this protein loss of low serum albumin is edema, dyslipidemia, coagulation/fibrinolysis abnormalities, reduced renal function, and immunological disorders.

The components of nephrotic syndrome are summarized in the table below

**Table 15.4: Clinical Definition of Adult Nephrotic Syndrome**

| **Clinical Definition of Adult Nephrotic Syndrome** | |
|---|---|
| Heavy proteinuria | =>3.5grams per day |
| Hypoalbuminemia or hypoproteinemia | Serum albumin: =<30mg/dl<br>Total protein:=<60mg/dl |
| Edema | Peripheral, ascites |
| Dyslipidemia | Elevated LDL cholesterol |

NB: The heavy and hypoalbuminemia are indispensable prerequisites for the clinical diagnosis of nephrotic syndrome.

**Classification**

- Primary nephrotic syndrome has no background diseases or idiopathic.
- Secondary nephrotic syndrome has background cause such as

**Clinical Features**

- Symptoms of bacteremia (fever, chill, joint pain, and muscle pain).
- Oedema is the predominant symptom of nephrotic syndrome (NS). In the early phase of NS, it appears on the eyelids. Progresses gradually to involve the lower limbs, sacrum. When severe, it is generalized with ascites, pleural/peeicardial effusion (Anarsaca).

**Complications**: venous thromboembolism due to loss of the coagulation proteins in urine (anti-thrombin III, protein C, protein S), renal failure. Complications related to treatment of nephrotic sundrome (steroids, cytotoxic/immunosuppressnat agents).

**Diagnosis:** urinalysis (proteinuria =>3+, hyaline/graular/fatty/waxy casts,RBCs) liver function tests (hypoalbuminemia/hypoproteinemia) lipid profile (elevated Total, LDL, VLDL cholesterol), renal function tests abnormalities). **Other tests;** HIV, HBSAg, HCV, VDRL, RBS.
Immununological tests: Anti-nuclear antibody (ANA), anti-ds-DNA antibdy, anti-Sm antibody, anti-phospholipid antibody.

**Imaging:** Renal ultrasound

**Renal biopsy:** for defitnitive dignosis of secondary causes.
6 histological entities: Minimal Change disease (MCD), Focal Segmental Glomuroslerosis (FSGS), Membranous nephropathy (MN), Membranoproliferative glomerulonephritis (MPGN), Mesangiocapillary glomerulonephritis (MCGN), Diabetes nephropathy (DN).

**Management:** Involve the physician/nephrologist in the management of the patient.

- **Supportive Treatment**
  - Low salt diet
  - Protein intake: 1.0–1.1 g/kg body weight (BW)/day in minimal change nephrotic syndrome and 0.8 g/kg BW/day in other nephrotic syndromes.
  - Calorie intake of 35 kcal/kg BW/day to maintainthe nitrogen balance
  - Fat restricted diet
  - Ascites tapping
  - Extra-Corpoeal Ultrafiltration Method (ECUM) for refractory edema and ascites.
  - Vaccination (pneumococcal and flu vaccine).

- **Adjuvant Therapy**
  - Renin-Angiotensin System (RAS)-Inhibitors (ACE-I/ ARBs) for proteinuria and hypertension.
  - Loop diuretics (Furosemide, torasemide) intravenously when edema is significant. Can take orally once edema subsides. Can be combined with thiazide diuretics in severe edema.
  - Aldosterone antagonists (Spironolactone/Eplerenone).
  - Statin therapy (atorvastatin, Rosuvastatin, Simvastatin, pravastatin).
  - Clotrimozole prophylaxis again pneumocystis pneumonia for those on immunosuppressive agents.
  - Immunoglobulin therapy in those with hypogammaglobulinemia for prevention of infectious diseases.

**No role for the following therapies in Nephrotic Syndrome**
- Immunoglobulin therapy in those with hypogammaglobulinemia for prevention of infectious diseases.
- Albmun infusion unless in severe shocl or pulmonary edema for temporary relief.
- Routine antiplatelet therapy.
- Routine anticoagulation (unless venous thromboembolism is present).

**Definitive Therapy**
- Steroid Therapy
  - Give pulse steroid therapy (high dose)with Methylprednisolone for 3 days as oraltherapy is affeected by reduced absorption due to interstitital edema.
  - Oral prednisolone 0.5-1mg/kg (upto 60mg/day, divide the dose 2/3 in the morning, 1/3 in the morning for doses =>45mg/day.Once daily morning dose for smaller doses. This is to maintain the normal physiological cortisol production. Maintain the initial dose for 2-4 weeks. Taper the steroid dose by 5-10mg every 2-4 weeks.

- Monitor urinalysis for proteinuria until remission is achieved (nil proteins in urine).
- Maintain the minimum dose to prevent relapse for about 2 years, then gradually taper and then discontinue therapy.

- Do follow up urinalysis after treatement is stopped to assess for relapse. For those whose proteinuria reverts after stopping steroids are labelled as steroid-dependent NS.
- If Proteinuria persists even on steroids (steroid-resistant nephrotic syndrome). Those who go into remission on steroids (steroid-sensitive nephrotic syndrome).
- Regular urinalysis should be done to assess the proteinuria ans dose of steroid is gradually taperred. Avoid abrupt stoppage of steroid therapy due to the risk of adrenal crisis.

♦ **Monitor for the adverse effects of long-term steroids** (steroid induced diabetes, hypertension, infection, osteoporosis, immunosuppression with infection risk, Cushing 's Syndrome).
♦ **Immunosuppressive drugs:** Can be given in combination with steroids or when thers is steroid-resistant nephrotic sundrome or steroid-dependent nephrotic syndrome. They include (cyclosporine, cyclophosphamide, azathioprine, mycophenolate mofetil, mizoribine, rituxumab). **This should be given in consultation with a nephrologist.**

## 15.7 Glomerulonepritis (GN)

Defined as a group of renal diseases characterized by immune-mediated damage to the glomerular basement membrane (GBM), mesangium, or the capillary endothelium, leading to hematuria, proteinuria, and renal failure. The blood pressure become elevated, and patient develop edema.

**Classification**
♦ Acute vs. Chronic Glomerulonephrits
♦ Primary vs. Secondary GN
♦ Nephrotic vs. Nephritic GN

**Examples of nephritic GN:** IgA nephropathy, Henoch Schonlein purpura (HSP); Post streptococcal glomerulonephritis (PSGN), Polyarteritis nodosa, Goodpasture syndrome, Lupus nephritis, Hepatitis C infection.

Some patients may also manifest symptoms of nephrotic syndrome in addition to nephritic syndrome as term referred to as **nephritic nephrotic syndrome.**

## 15.7.1 ACUTE GLOMERULONEPHRITIS

**RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS (RPGN)**

The clinical features are presented in the table below.

**Table 15.5: Clinical Features of Rapidly Progressive Glomerulonephritis**

| Type of glomerulonephritis | Age at risk | Extrarenal features | Investigations |
|---|---|---|---|
| **Post-Streptococcal GN** | Children 2-12 years | 7-10 days after a throat<br>2-3 weeks after skin infection(impetigo) | Low C3, ASOT, throat or skin swab. |
| **IgA Nephropathy** | 20-30 years | | IgA may be elevated in half of the cases<br>Normal complement |
| **Henoch-Schonlein purpura** | < 20 years | Purpuric rash on legs, arthritis, abdominal pain | Complememet normal |
| **Wegener's granulomatosis** | 50-60 years | Weight loss, malaise, upper/lower RTI, arthritis | ANCA, complement normal |
| **Lupus nephritis** | 20-30years (more females) | Arrthtis, photosensitivity (malar rash), pleurisy, pericarditis, | Low C3, ANA, Anti-DS DNA,anti-cardiolipin antibodies. |
| NB: Rapidly progressive glomerulonephritis (RPGN) Associated with the clinical picture of sudden and severe acute renal failure.It occurs in the background of any of the above types of acute GN. | | | |

**Investigations**

- Urinalysis: dysmorphic red blood cells and RBC casts are pathognomonic of glomerular inflammation.
- Total Blood count
- Urea, Creatinine electrolytes
- Hepatitis B surface antigen and Hepatitic C virus antibodies
- Renal ultra-sound.
- Renal biopsy

**Treatment** (should be managed in consultation with physician/nephrologist)**.**

- Salt (sodium/potassium) restriction
- Fluid restriction
- Loop duiretics:
  - Furosemide: iv if edema is severe/ then oral.
  - Torasemide:

- **Antihypertensives**
  - Renin Angiotensin inhibitors (ACE-I or ARBs).
  - Calcium channel blockers
- **Corticosteroids**
- **Immunomodulators**
- **Antibiotics:** indicated for Post streptococcal GN with evidence of streptococcal infection. Early treatment of streptococcal infection with antibiotics reduces the severity and incidence of glomerulonephritis.
  - Penicillins
  - Erythromycin (alternative in patients with penillin allergy.
- **Dialysis:** In some cases, the disease has a fulminating course leading to renal failure. In such cases, renal replacement therapy with dialysis is performed.

**NB:** Treatment of the underlying disease should be managed accordingly.



## 15.8 Renal Failure

Renal failure refers to the inability of the kidneys to perform excretory function leading to retention of nitrogenous waste products from the blood.

**Functions of the kidney include**
- Regulation of electrolytes and volume
- Excretion of nitrogenous waste products
- Elimination of exogenous molecules, e.g., many drugs
- Endocrine function (synthesis ofof hormones, e.g.,erythropoietin and vitamin D.

**Classification**
- Acute Kidney Injury (AKI)
- Chronic Kidney Disease (CKD)
- Acute on Chronic Kidney Disease

### 15.8.1 ACUTE KIDNEY INJURY (AKI)

The term acute kidney injury (AKI) has replaced acute renal failure because AKI indicates the entire clinical spectrum from a mild increase in serum creatinine to overt renal failure. It is an acute condition associted with sudden and often reversible reduction in kidney function where GFR declines abruptly within hours to days.
It was previously referred to as acute renal failure. It is very common especially in hospitalised patients.

According to Kidney Disease: Improving Global Outcomes (KDIGO), AKI is defined by the presence of any of the following.
- Increase in serum creatinine by =>26.5mmol/l (0.3mg/dL) within 48 hours.
- Increase in serum creatinine to =>1.5 times from baseline within the prior seven days.
- Urine volume <0.5 mL/kg/hr for at least 6 hours.

**Table 15.6: Aetiologies of Acute Kidney Injury**

| Type of AKI | Pathophysiology | Aetiologies |
|---|---|---|
| **Prenal**<br>Any cause of reduced blood flow to the kidney<br>Comprises of 60% of AKI | Hypovolumia | Vomiting,diarrhoea,severe burns, hemorrhage, renal fluid loss (overdiuresis), |
| | Hypotension from decreased cardiac output: | cardiogenic shock, massive pulmonary embolism, acute coronary syndrome, congestive cardiac failure |
| | Hypotension from systemic vasodilation: | Hypotension from systemic vasodilation: septic shock, anaphylaxis, anesthesia administration, hepatorenal syndrome |
| | Renal vasoconstriction: | NSAIDs, iodinated contrast, amphotericin B, calcineurin inhibitors, hepatorenal syndrome |
| | Glomerular efferent arteriolar vasodilation: | ACE inhibitors,<br>Angiotensin receptor blockers (ARBs) |
| **Renal Causes**<br>Intrinsic renal diseases that affect the glomerulus or tubules.<br>Comprises 35% of AKI | Acute tubular necrosis: | Ischemia from prolonged prerenal injury,<br>Drugs: aminoglycosides, vancomycin, amphotericin B,<br>Rhabdomyolysis,<br>Intravascular hemolysis |
| | Acute interstitial nephritis: | Drugs beta-lactam antibiotics, penicillins, NSAIDs, proton pump inhibitors (PPIs), 5-ASA.<br>Infections,<br>Autoimmune conditions (SLE, IgG related disease) |
| | Glomerulonephritis | Anti-GBM,<br>Immune complex-mediated diseases (SLE, post-infectious GN, cryoglobulinemia, IgA nephropathy, Henoch-Schonlein purpura. |
| | Intratubular obstruction | Monoclonal gammopathy in multiple myeloma,<br>Tumor lysis syndrome,<br>Toxins (ethylene glycol). |
| **Post-Renal causes**<br>Obstrucutive conditions<br>Comprises of 5% of AKI<br>NB: a unilateral obstruction may not always present as AKI | *Extrarenal obstruction*<br>✓ Benign prostate hypertrophy (BPH)<br>✓ Improperly placed catheter<br>✓ Bladder, prostate or cervical cancer<br>✓ Retroperitoneal fibrosis<br>*Intrarenal obstruction*<br>✓ Nephrolithiasis<br>✓ Blood clots<br>✓ Papillary necrosis | |

**Clinical Features**

- The history and physical exam should focus on determining the etiology of AKI and to differentiate AKI from CKD.
- **Examine for**: dehydration, cardiovascular system examination;(pulse rate, blood pressure, and jugulovenous pulse in establishing volume status).
- eatures suggestive of vasculitis (livedo reticularis, digital ischemia, butterfly rash, and purpura).
- Features of liver disease, band keratopathy in multiple myeloma.
- Signs of diabetes mellitus, atheroemboli in retinopathy.
- Signs of hypertension.
- Keratitis, iritis, and uveitis in autoimmune vasculitis.
- Hearing loss in Alport disease.

**Investigations**

Search for all possible etiologies of AKI, including prerenal, renal, and post renal disease. Pay special attention to those reversible ones.

- Full blood counts (infection and anaemia).
- Urinalysis (proteinuria, osmolality).
- Urine microscopy: urine sediments; muddy brown casts (acute tubular necrosis), sterile pyuria (acute interstitial nephritis).
- Urine sodium, urine/plasma creatinine ratio, fractional excretion of sodium/urea.
- Urine culture and sensitivity
- Urea and electrolytes
- Serum creatinine
- Liver function tests (hepatorenal syndrome)
- Imaging: Renal or kindey ureter bladder-prostate ultrasound if obstructive causes are suspected.
- Kidney biopsy: unexpalined rapidly declining renal function.

**Table 15.7: Guidelines for urinary indices whereby established ARF can be distinguished from renal vasoconstriction with intact tubular function (prerenal azotemia)**

Guidelines for urinary indices whereby established ARF can be distinguished from renal vasoconstriction with intact tubular function (prerenal azotemia)

| Laboratory test | Prerenal azotemia | ARF |
|---|---|---|
| Urine osmolality (mOsm/kg) | >500 | <400 |
| Urine sodium level (mEq/l) | <20 | >40 |
| Urine/plasma creatinine ratio | >40 | <20 |
| Fractional excretion of sodium (%) | <1 | >2 |
| Fractional excretion of urea (%) | <35 | >35 |
| Urinary sediment | Normal; occasional hyaline or fine granular casts | Renal tubular epithelial cells; granular and muddy brown casts |

Osm, osmole; Eq, equivalent.

Source: Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 2004 Jul;114(1):5-14. [Medline]

**Staging of AKI**

- AKIN (Acute Kidney Injury Network) Staging
- RIFLE (Risk, Injury, Failure, Loss and End-Stage Renal Disease) Criteria

**Table 15.8: AKIN Staging of Acute Kidney Injury**

| AKIN stage | Urine output | Serum Creatinine |
|---|---|---|
| Stage 1 | Less than 0.5 ml/kg/h for more than 6 hours | Increase of => 26.5 mmol/l (0.3mg/dl)<br>*OR*<br>Increase to => 1.5 to 2-fold from baseline |
| Stage 2 | Less than 0.5 ml/kg per hour for more than 12 hours | Increased to > (2 to 3-fold) from baseline |
| Stage 3 | Less than 0.3 ml/kg/h for 24 hours<br>*OR*<br>Anuria for 12 hours | Increased to >3-fold) from baseline<br>*OR*<br>Increased to => 354mmol/l (4.0 mg/dl) with an acute increase of at least 44mmol/l (0.5 mg/dl)<br>*OR*<br>On RRT |

Source: Mehta RL, Kellum JA, Shah SV, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11(2):R31. doi:10.1186/cc5713

**Table 15.9: Rifle Staging of Acute Kidney Injury**

| Criteria | Urine Output | Serum Creatinine or GFR |
|---|---|---|
| **R:** Risk | Less than 0.5 ml/kg/h for more than 6 hours | 1.5 fold increase in serum creatinine<br>*OR*<br>GFR decrease of >25% |
| **I:** Injury | Less than 0.5 ml/kg per hour for more than 12 hours | 2-fold increase in serum creatinine<br>*OR*<br>GFR decreased >50% |
| **F:** Failure | Less than 0.3 ml/kg/h for 24 hours<br>*OR*<br>Anuria for 12 hours | Serum creatinine 3-fold,<br>*OR*<br>Serum creatinine >354 mmol/l (>44mg/dl) with an acute rise of 44 mmol/l (0.5mg/dl)<br>*OR*<br>GFR decrease by >75% |
| **L:** Loss | | Persistent acute renal failure=complete loss of kidney function for > 4 weeks |
| **E:** End Stage Kidney Disease | | ESRD >3 months |

Source: Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P; Acute Dialysis Quality Initiative workgroup. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8(4):R204-R212. doi:10.1186/cc2872.

**Management**

- Fluid challenge with close monitoring of urine output and renal function.
- Avoid nephrotoxic drugs (aminoglycosides, NSAIDs, contrast media).
- Dose adjustment may be required in some drugs that are renally excreted.
- Loop diuretics (e.g., furosemide 1-5mg/kg) if fluid overload present and in the oliguric phase of acute tubular necrosis (ATN).
- Restrict dietary potassium and phosphorous intake.
- Manage hyperkalemia if present:
  - Give 10 IU of soluble insulin with 50mls of 50% dextrose. Combination of insulin and dextrose facilitate the uptake of glucose into the muscle cells accompanied by potassium via the Na-K-ATPase pump.
  - Administrate 10–30ml of 10% calcium gluconate over 10–20 minutes for cardiac cell membrane stabilisation. (This requires constant ECG monitoring for widening of QRS interval, loss of P wave, or cardiac arrhythmias).
  - Nebulise with salbutamol an adrenergic agonist via ($Na^+$ -$K^+$ -ATPase) pump stimulation, thereby shifting potassium into the intracellular compartment.
  - Loop diuretics (e.g., furosemide) results in potassium excretion.
  - Potassium binders: Use of cation exchange resins in the sodium cycle may promote GIT potassium loss. These are sodium polystyrene sulphonate(kayexelate) 20g in 70% sorbitol solution 3 to 4 times a day.

- Refer the patient to a dialysis center as soon as possible if:

- Anuria is present for more than 24 hours OR oliguria of more than 48 hours.
- Hyperkalemia not responding to above medical treatment.

**Dialysis in Acute Kidney Injury (AKI)**

**Indications**

- Volume overload unresponsive to diuretic therapy
- Hyperkalemia and metabolic acidosis unresponsive to medical management.
- Uremic pericarditis
- Uremic encephalopathy

NOTE: It is not clear the optimal timing for initiation renal replacement therapy in AKI. Weigh the benefits of early versus late initiation. Do not use a single urea or creatinine threshhold.

**Dialysis Modalities**

- Intermittent hemodialysis (IHD)
- Continous renal replacement therapy (CRRT): For hemodynamically unstable patients.
- Sustained Low Efficiency Dialysis (SLED)
- Slow Continous Ultrafiltration (SCUF)
- Peritoneal dialysis (PD)

**Table 15.10: Theoretical advantages and disadvantages of CRRT, IHD, SLED and PD**

| Modality | Potential setting in AKI | Advantages | Disadvantages |
|---|---|---|---|
| IHD | Hemodynamically stable | Rapid removal of toxins and low-molecular-weight substances<br>Allows for "down time" for diagnostic and therapeutic procedures<br>Reduced exposure to anticoagulation<br>Lower costs than CRRT | Hypotension with rapid fluid removal<br>Dialysis disequilibrium with risk of cerebral edema<br>Technically more complex and demanding |
| CRRT | Hemodynamically unstable<br>Patients at risk of increased intracranial pressure | Continuous removal of toxins<br>Hemodynamic stability<br>Easy control of fluid balance<br>No treatment-induced increase of intracranial pressure<br>User-friendly machines | Slower clearance of toxins<br>Need for prolonged anticoagulation<br>Patient immobilization<br>Hypothermia<br>Increased costs |
| SLED | Hemodynamically unstable | Slower volume and solute removal<br>Hemodynamic stability<br>Allows for "down time" for diagnostic and therapeutic procedures<br>Reduced exposure to anticoagulation | Slower clearance of toxins<br>Technically more complex and demanding |
| PD | Hemodynamically unstable<br>Coagulopathy<br>Difficult access<br>Patients at risk of increased intracranial pressure<br>Under-resourced region | Technically simple<br>Hemodynamic stability<br>No anticoagulation<br>No need for vascular access<br>Lower cost<br>Gradual removal of toxins | Poor clearance in hypercatabolic patients<br>Protein loss<br>No control of rate of fluid removal<br>Risk of peritonitis<br>Hyperglycemia<br>Requires intact peritoneal cavity<br>Impairs diaphragmatic movement, potential for respiratory problems |

CRRT, continuous renal replacement therapy; IHD, intermittent hemodialysis; PD, peritoneal dialysis; SLED, sustained low-efficiency dialysis.

**Hemodialysis catheter**

- Insert a temporary dialysis catheter/ Uncuffed non-tunneled dialysis catheter, under (ideally under ultrasound guidance).

**Site of catheter**

- Right jugular vein > femoral vein >left jugular vein. Avoid the subcalvian vein. Obtain a CXR after the jugular or subclavian vein catheter insertion.

**Choice of dialysate**

- Bicarbonate

**Choice of dialyzer**

- Use biocompatible semipermeable hollow-fiber dialyzer

**Anticoagulation during hemodialysis in AKI**

- Be cautious when anticoagualting due to the increased bleeding risk.
- Use either unfractionated heparin or low molecular weight heparins
- Avoid warfarin and direct oral anticoagulants.

**Dose of RRT in AKI**

- Deliver a Kt/V of 3.9 per week when using intermittent or extended RRT.
- Deliver an effluent volume of 20–25 ml/kg/h for CRRT in AKI (1A).
- Prescribe the dose before starting each session of RRT.
- Assess the actual delivered dose frequently and adjust prescription accordingly.
- Consider the patient's treatment goals of electrolyte, acid-base, solute, and fluid balance.

**Discontinuation of RRT**

- Discontinue RRT when it is no longer required either when;
  - the intrinsic kidney function has recovered to the point that it is adequate to meet patient.
  - RRT is no longer consistent with the goals of care.

**Peritoneal Dialysis (PD) in AKI**

- Mainly confined to pediatrics.
- Useful in regions with limited resources.
- Advantages: ease of use, low cost, and minimal requirements on infrastructure, No need for vascular access and anticoagulation, relatively good hemodynamic tolerance.
- Disadvantages: Less effective than HD (in patients with splanchnic hypoperfusion or who are on vasopressors), the risk of protein loss, the unpredictability of solute and fluid removal, the need for an intact peritoneal cavity, risk of peritonitis, diaphragmatic splinting leading to ventilatory compromise and fluctuating blood glucose levels.

**Follow up after AKI**

Evaluate patients 3 months after AKI for resolution, new onset, or worsening of pre-existing CKD.

**Possible outcomes after AKI include;**

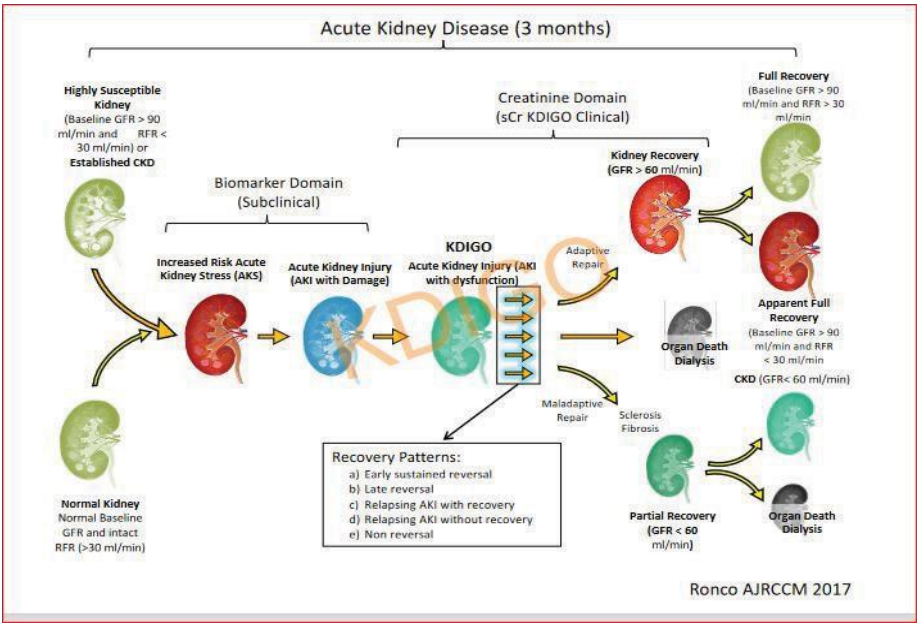
- Complete and sustained recovery (early or late)
- Partial recovery
- Relapsing with recovery

- Relapsing with no recovery
- No reversal or non-recovery
- No reversal or non-recovery

## 15.8.2 CHRONIC RENAL FAILURE

CRF or chronic kidney disease (CKD) is defined as a persistent impairment of kidney function. The serum creatinine remains abnormally elevated for 3 months or more with an estimated of GFR of <60 ml per minute / 1.73m$^2$.

**Figure 15.1: Spectrum of Acute Kidney Disease**




Source: Ronco C, Ferrari F, Ricci Z. Recovery after Acute Kidney Injury: A New Prognostic Dimension of the Syndrome. Am J Respir Crit Care Med. 2017 Mar 15;195(6):711-714. doi: 10.1164/rccm.201610-1971ED. PMID: 28294655.

**Table 15.11: Criteria for Chronic Kidney Disease**

| Criteria for CKD (either of the following present for >3 months) | |
|---|---|
| Markers of kidney damage (one or more) | Albuminuria (AER ≥30 mg/24 hours; ACR ≥30 mg/g [≥3 mg/mmol])<br>Urine sediment abnormalities<br>Electrolyte and other abnormalities due to tubular disorders<br>Abnormalities detected by histology<br>Structural abnormalities detected by imaging<br>History of kidney transplantation |
| Decreased GFR | GFR <60 ml/min/1.73 $m^2$ (GFR categories G3a-G5) |

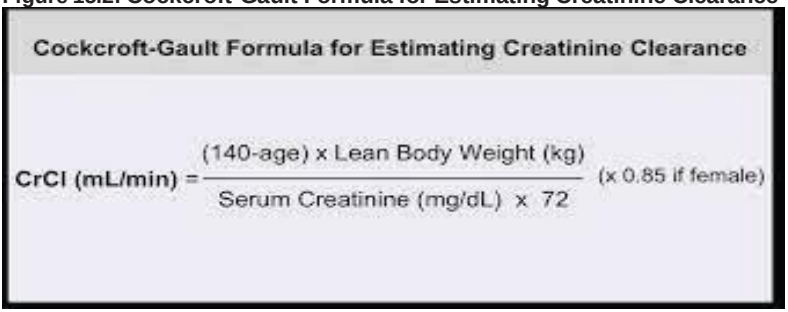
Abbreviations: CKD, chronic kidney disease; GFR, glomerular filtration rate.

Source: Levin A, Stevens PE. Summary of KDIGO 2012 CKD Guideline: behind the scenes, need for guidance, and a framework for moving forward. Kidney Int. 2014 Jan;85(1):49-61. doi: 10.1038/ki.2013.444. Epub 2013 Nov 27. PMID: 24284513.

**2012 KDIGO CKD classification**

- According to glomerular filtration rate (GFR)
- Glomerular Filtration Rate is calculated using;
  - Cockroft Gault Formula
  - Modification of Diet in Renal Disease (MDRD) formula.
  - Online eGFR calculators are available (https://www.mdcalc.com/mdrd-gfr-equation).

**Figure 15.2: Cockcroft-Gault Formula for Estimating Creatinine Clearance**




Source: Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976;16:31-41.

**GFR Grading**

- GFR Units: ml/min per 1.73 m2
- 6 categories:
  - Grade 1: GFR 90
  - Grade 2: GFR 60-89
  - Grade 3a: GFR 45-59
  - Grade 3b: GFR 30-44
  - Grade 4: GFR 15-29
  - Grade 5: GFR <15 or on dialysis

Albuminuria status should be establised and assigned to 3 categories; A1, A2 and A2.

- **A1:** Albumin Creatine Ratio of <30mg/g (<3mg/mmol
- **A2:** Albumin Creatinine Ratio of 30-300mg/g (3-30mg/mmol)
- **A3:** Albumin Cratinine Ratio of >300 mg/g (30mg/mmol)



**Risk factors for CKD:**

Hypertension, diabetes mellitus, proteinuria, obesity, smoking, dyslipidemia, genetic factors,older age, male gender, previuos history of AKI.

**Aetiology of CKD**

- *Glomerular Diseases*: diabetes mellitus, autoimmune diseases, drugs
- *Vascular Diseases*: Hypertension, atherosclerosis, vasculitis, ischemia.
- *Tubulo-interstitial disease*: urinary tract infections, nephrolithiasis, obstruction, drug toxicity.
- *Structural kidney abnormalities*: polycystic kidneys, hydronephrosis due to obstruction, vesicoureteral reflux, renal masses, infiltrative renal diseases, renal artery stenosis.
- *Renal tubular disorders*: Renaltubular acidosis, cystinuria.

**Clinical Features of Chronic Renal Failure**

- *Biochemical*: Acidosis, hyperkalaemia. Elevated blood urea, elevated serum creatinine.
- *Cardiovascular*: Pulmonary edema, hypertension, pericarditis and cardiac tamponade, heart failure.
- *Skeletal:* Bone pain and fractures(rare).
- *Nervous system*: Encephalopathy (confusion, convulsions), peripheral neuropathy.
- *Haematological system*: Anaemia, excessive bleeding, e.g., from gums, skin, nose.
- *Skin*: Scratching (pruritus), darkening of skin

**Evaluation of CKD**

- Evaluate for chronicity
  - To differentiate between AKI and CKD.
  - If the GFR is persistly reduced to <60ml/min/1.73m2 for > 3 months.
  - A repeat GFR estimation is recommended within and beyond 3 months if unclear.
  - Review past GFR, urinalysis results for albuminuria and proteinuria.
  - Presence of small sized kidneys on imaging.

- Pathological features of fibrosis or atrophy.
- Positive medical history of disorders known to cause CKD e.g., diabetes, PCKD.

**Evaluation of the cause of CKD**

- Evaluate the clinical context (personal/family history, social/ environmental factors, drug history).
- Physical examination.
- Laboratory measurements (urinalysis-hematuria, pyuria, microscopy) .
- Imaging (renal ultrasound: size, shape, symmetry, obstruction).
- Pathologic diagnosis.
- Not all evaluations are required in all patients, and will be directed by clinical context, and resource availability

**Evaluation of GFR**

- Use serum creatinine (sCr) for initial assessment, staging, and tracking the progression of CKD.
- Cystatin C can be used as an additional/confirmatory test where available.

**Management of CKD**

- Delay or prevention CKD progression
  - Adress the underlying risk factors
  - Blood pressure control (target <140/90mmhg with no or mild proteteinuria <30mg/g), or to <130/80mmhg if significant proteinuria >30mg/g).
  - Renin Angiotensin System inhibitors (ACE-I or ARB in those with CKD and proteinuria). Avoid combining ACE-I and ARBs.
  - Dietary protein intake: 0.8g/kg/day (avoid high protein diet >1.3gm/kg/day.
  - Glycemic control: HBA1c < 7% (53mmol/mol.Higher HBA1c> 7% suggested for those at risk of hypoglycemia, comorbidities, and the elderly. NB: the HBA1c will be falsely reduced in the presence of anemia.
  - Avoid nephrotoxic drugs, contrast media.
  - Stop smoking.
  - Maintain healthy BMI 20-25kg/m2.
  - Physical activity: 30 minutes 5 times a week.
  - Restrict salt intake <2gm/day.
  - Additional dietary advise: posassium, phosphates.
- Evaluate and manage for complications associated with CKD
  - Anemia (Hemoglobin <12gm/dl, females, <14gm/dl in males. Assess for secondary causes of anemia including dietary deficiencies or blood loss;
  - Iron supplentation (oral or parenteral);
  - Erythropoietin.
  - CKD-Bone mineral disorder (BMD): Evaluation recommended in those with eGFR <45ml/min/1.73m$^2$
  - Serum Calcium
  - Serum phosphate
  - Parathyroid hormone (PTH)
  - Alkaline phosphatase (ALP)
  - 25-hydroxyvitamin D(25(OH)D) (should not be done routinely, expensive).

**Table 15.12: Management of Chronic Kidney Disease Complications**

| CKD complication | Management | Remarks |
|---|---|---|
| Anemia | Erythropietin<br>Iron sucrose (parenteral)<br>Transfusion | 2000-4000units/week |
| Hyperphosphatemia | Phosphate Lowering Agents (PLA)/Phosphate binders | Examples |
| | Calciu-based PLA | Calcium citrate<br>Calcium carbonate<br>Calcium acetate<br>Calcium acetate/magnesium carbonate |
| | Non-Calcium-based PLA | Magnesium carbonate<br>Aluminium hydroxide<br>Lanthanum carbonate<br>Sevalamer HCL<br>Sevalamer carbonate |
| Hyperparathyroidism | Calcimimetics, calcitriol, and vitamin D analogues | |

NOTE: Routine Bone Mineral Density (BMD) asssessment with Dual Energy X-ray Absorptiometry (DXA/DEXA) is not routinely indicated.

**Infection prevention in CKD**

- Annual flu vaccination in all adults with CKD
- If eGFR <30ml/min/1.73m2 in those at high risk of pneumococcal infection (diabetics, nephrotic syndrome), to receive pneumococcal vaccine 5-yearly.
- Hepatitis B vaccination

**Conservative Kidney Management in CKD**

- An option for those who choose not to purse RRT.
- Patients with ESRD who are =>80years or elderly (>65 years with comorbdities) where survival benefit of dialysis is lacking.
- Dialysis prolongs life compared to CKM but associated with poor quality of life due to travels, complications.
- Patient recieve all the above therapy except dialysis.
- Dietetic-nutritional therapy can be a cornerstone in the conservative management of CKD (reducing glomerular hyperfiltration, uremic toxin generation, metabolic acidosis, and phosphorus burden).
- Provide end-of life care (pallivative care; pain management, psychological and spiritual care).

**Renal Replacememt Therapies (RRT)**
Start exploring the options of dialysis, transplantation, or conservative management with patients at least a year before they are likely to need it.

**Options**
- Hemodialysis (HD)/ Hemodiafiltration (HDF)
- Peritoneal dialysis (PD: continuous ambulatory peritoneal dialysis (CAPD) or automated peritoneal dialysis (APD),
- Kidney Transplanatation

**Dialysis Access**
- Arteriovenous (av) fistula. Fashioned around 6 months before the anticipated start of dialysis to allow for maturation.
- Peritoneal dialysis catheter; placed by an open surgical technique, around 2 weeks before the anticipated start of dialysis.
- Dialysis should be started in the presence of any of the following;
  - The estimated glomerular filtration rate (eGFR) is 5-7 mL/min/1.73 $m^2$ in asymptomatic patients.
  - Refractory fluid overload or hypertension.
  - Symptoms or signs attributable to kidney failure (serositis, acid-base or electrolyte abnormalities, pruritus) affecting daily life.
  - Progressive deterioration in nutritional status refractory to dietary intervention
  - Cognitive impairment.

**Kidney Transplantation**
- Living or deceased donors
- Living donor preemptive renal transplantation can be considered when the GFR is <20 ml/min/1.73 m2 in the background of progressive and irreversible CKD over the preceding 6-12 months.

### 15.8.3 ACUTE ON CHRONIC RENAL FAILURE
Rise in serum creatinine of ≥ 50% from baseline or a rise of serum creatinine by ≥ 26.5 µmol/L (≥ 0.3 mg/dL) in <48 h in a patient whose glomerular filtration rate is <60 ml/min for >3 months.

## 15.9 Nephrolithiasis
Also referred to as kidney stones or renal calculi.
Associated with an increased risk of CKD, ESRD, CVD, hypertension and diabetes.

**Types**
- **Calcium stones** (80% of cases): Calcium oxalate (CaOx) and Calcium phosphate (CaP, apatite).
- **Struvite (magnesium-ammonium phosphate):** 10-15% nfection stones and triple phosphate stones (infections: commonly *Proteus mirabilis,* less

commonly *Klebsiella pneumonia*, *Pseudomonas aeruginosa,* and *Enterobacter).*

- **Uric acid (Urate):** 3-10%. Risk factors; animal protein diet, hyperuricosuria, low urine volume, and low urinary pH (pH < 5.05)
- **Cystine stones:** <2% of all stone types. An autosomal recessive disorder due to defect in the rBAT gene on chromosome 2 associated with the transport of an amino acid and cystine. It results in an excess of cystinuria in urinary excretio due impaired cystine tubular absorption.

**Risk Factors**

- Personal history of prior kidney stones.
- Family history of kidney stones.
- Increased intestinal oxalate absorption due to malabsorption.
- Hypercalciuria, hyperoxaluria, hypomagnesuria, hypercystinuria,hypocitraturia.
- UTIs with uease-producing bacteria accompanied by altered urinary pH (struvite crystals).
- Low fluid intake.
- Acidic urine (pH< 5.5) promotes uric acid formation.
- Diabetes, obesity， gout, and hypertension, IBD.

**Clinical Features**

- Pain (typical reno-ureteral colic or loin pain)
- Hematuria
- Renal failure
- Vomiting
- Fever
- Passing of stone in urine.

**Dignostic work-up**

- Renal function test
- Urinalysis
- Urine elecrolytes
- Urine PH
- Serum calciul levels
- Imaging:
  - KUB (kidney-ureter-bladder) X-ray (caanot visualise uric acid stones).
  - A non-contrast CT abdomen/pelvis.
- Recurrent stone formers: Renal acidification test, parathyroid hormone levels (PTH), bone turnover markers, bone densitometry (DXA).

**Management**

- Pain management with NSAIDs (MoA: decreases smooth muscle stimulation and ureteral spasm).
- Increase fluid intake.
- Tamsulosin (aid stone passage and reduces smooth muscle stimulation).

- Stones >6mm are may require some intervention (percutaneous nephrolithotomy, rigid and flexible ureteroscopy, and shock wave lithotripsy).

**Prevention**

- Increased water intake (>2liters to >3 liters per day).
- Ensure adequate diuresis (>2liters of urine/day).
- Daily calcium intake not less than 0.8–1gm.
- Restrict animal protein to ≤0.8 g per kg of body weight.
- Reduce salt intake (<2gm/day)
- Avoid excess food intake/ vegetable consumption
- Avoid soda beverages

# 16. Mental Disorders

## 16.1 Acute Confusion (Acute Psychosis)

**Sudden onset of mental symptoms in an otherwise previously normal person.**

**Aetiology**

- Neurological causes: Cerebrovascular accidents (CVA), brain tumours, subdural haematomas, brain abscess.
- Infections: Acute meningitis, encephalitis, malaria, HIV.
- Metabolic toxic causes:
  - Metabolic derangements, e.g., DKA,hypoglycaemia.
  - Drug intoxication.
- Psychiatric causes: Schizophrenia, depression, and manic episode.

**Clinical Features**

A good history and physical examination are essential. The patient may be ill-looking, not appreciating surroundings, not alert, not aware of time, place, or who they are. They may also be unable to remember and may forget easily with poor attention and concentration. They may have visual/auditory hallucinations or delusions (grandiose or paranoid) or may be aggressive and excited. They may also have illusions (e.g., a stick is mistaken for a snake). In general, symptoms get worse at night.

**Investigations**

- HB, blood slide for MPS, culture and sensitivity, blood sugar,and serum urea and electrolytes
- CSF examination (after fundoscopy)
- X-rays –Skull
- Head CT and MRI scans

**Management — General**

Identify and manage physical (underlying) causes.

**Management – Pharmacological**

- Make appropriate psychiatric diagnosis(acute manic episode,schizophreni- form disorder).
- Give chlorpromazine 100–200mg IM STAT then 12 hourly IM/oral ***OR*** **haloperidol 5–10mg IM/oral 12 hourly.**
- Continue in patient treatment until patient develops insight,then outpatient treatment and follow up for at least 6 months.
- If after 6 months the patient relapses refer to a psychiatrist.

## 16.2 Alcohol Withdrawl (Delirium Tremens)

**Clinical Features**

Suspect if a patient with acute psychosis also has history of excessive drinking, tremors, weakness, restlessness, insomnia, hallucinations (visual), profuse perspiration. May develop features of withdrawal when admitted to hospital for another disease.

**Investigations**

- ♦ Blood sugar to exclude hypoglycaemia
- ♦ Full haemogram for evidence of macrocytosis
- ♦ Liver function test (especially liver enzymes)

**Management**

- ♦ Admit patient.
- ♦ Give thiamine 100mg parenterally IM once daily for 5 days then orally for at least 1 month to prevent brain damage ***OR***
- ♦ Inject high potency vitamin (ascorbic acid 500mg, nicotinamide 160mg, pyridoxine HCl 50mg, riboflavin 4mg, thiamine HCl 250mg)1x daily for 5 days.
- ♦ Sedate with: IVdiazepam10–40mg STAT then 10–20 mg orally 8 hourly for the first 24 hours and then gradually taper off. Aim of therapy is sedate patients until they are calm.
- ♦ Maintain fluid and electrolyte balance 10% dextrose 1,000 ml to alternate with Hartmann's solution 1,000ml 8 hourly until well hydrated.
- ♦ If hallucinations occur, give oral/IM chlorpromazine 100–200mg 12 hourly ***OR*** haloperidol PO 1.5–15 mg until symptoms are controlled (in the elderly give a half the dose), then adjust dose according to symptoms. Do not treat with chlorpromazine alone since it reduces seizure threshold.
- ♦ Provide supportive care:

- Give multivitamins containing folic acid.
- Manage head trauma; treat pneumonia and any other infections,which are common in alcohol abusers.
- Treat specific disorders symptomatically, e.g., cirrhosis,neuropathy.
- Treat seizures with diazepam IV.
- Give 50ml of 50% dextrose to correct hypoglycaemia; this should be given after thiamine injection.

- Avoid long-term use(not more than14 days) of sedatives as they may lead to addiction.
- In motivated patients, try disulfiram 05g PO once daily for 1–3 weeks, tapered to 0.25g once daily for another 3 weeks.

**Delirium tremens has a high mortality if not diagnosed and treated early.**

**Patient Education**

- ♦ Counsel the patient; abstinence may be essential.
- ♦ Encourage healthy diet.
- ♦ Involve the family in the long-term management.

## 16.3 Substance Use Disorders

These are syndromes arising out of repeated maladaptive use of substances, with substance defined as any chemical with brain altering properties. They are characterized by significant impairment in psychological, social and occupational functioning as observed over a 12-month period. Commonly abused substances in Kenya include tobacco, Cannabis sativa, khat (miraa), opioids (heroin), cocaine, and solvents (glue, petrol, wood varnish). Substance-related syndromes include intoxication, dependence, withdrawal, psychosis, mood disorders, anxiety, sleep disorders, sexual disorders. High risk groups are:

- 12–20-year-olds.
- Patients with primary mental disorders.

### 16.3.1 SUBSTANCE ABUSE BY THE ADOLESCENT

Usually present with self-neglect, slovenliness, deteriorating school/job performance, excessive sleeping, rough appearance, increasing and unexplained demand for money from caregivers, involvement in petty crime (pilfering), running away from home – in addition to aforementioned substance-related disorders.

**Investigation**

- Liver function tests
- HIV screening – especially for opioid abusers
- Urinalysis
- Blood for toxicology
- HBSAg
- HCV
- Urine for drug screen

### 16.3.2 MANAGEMENT OF SELECTED SUBSTANCES OF ABUSE

#### OPIOID DETOXIFICATION

Opioids abused include heroin, morphine, dihydrocodeine, and pethidine. Tolerance develops rapidly and withdrawal features include agitation, lethargy, sweating, goose-

flesh pimples, running nose, shivering, musculo-skeletal pains, diarrhoea, and abdominal cramps. These effects peak at 48 hours and subside over a period of 10

days. Because of the highly addictive nature of the opioids, admission to hospital is necessary for effective management.

**Management – Pharmacological**

- For agitation, use diazepam 20–80mg PO daily to be tapered off in10 days.
- For the sympathetic upsurge, use clonidine 0.15–3mg PO daily for 10 days.
- For any assaultive behaviour, use haloperidol 5–10mg 12 hourly PO/IM***OR*** **chlorpromazine100–200mg 12 hourly as necessary.**

- For pain, use paracetamol 1g PO every 8 hours asnecessary.
- Provide nutritional supportvitamins.
- Manage anycomobidities.

**CANNABIS DEPENDENCE**
Chronic users may develop psychosis, anxiety, mood disorders, and a withdrawal state. Admission is usually necessary for initiating abstinence. Treatment of the psychiatric complication is the same as for the primary syndromes.

**KHAT (MIRAA) DEPENDENCE**
Chronic users may develop anxiety, mood disorders, and schizophrenia-like psychosis. Abstinence is to be encouraged. Treatment of the related psychiatric disorders is the same as for the primary syndromes.

**SOLVENT ABUSE**
Solvents have powerful euphoriant properties. They are mainly abused by street children and the homeless. Chronic users may develop organ damage (liver, heart, kidney), apart from neurological damage. Patient education is vital. Involve family and relevant authorities in rehabilitation.

## 16.4 Substance Use Disorders

An unpleasant, vague, and diffuse feeling of apprehension. It is an alerting signal. Usually, the threat is unknown and patient functioning becomes impaired.
Pathological anxiety includes panic disorder, which may be dramatic in presentation; phobias which are fears that are out of proportion; obsessive compulsive disorder, which is characterized by an irresistible urge to act; and generalized anxiety disorder.

**Clinical Features**
The patient presents with an empty feeling in the stomach, lightness in chest, pounding heart, perspiration, urge to void, non-exertion dyspnoea, blurred vision, hyper reflexia, dizziness, and light headedness. Hypertension (transient) may be noted with some restlessness (e.g., pacing). A good history and physical

examination are of crucial importance. It is important to exclude physical causes like thyrotoxicosis, pheochromocytoma, hypoglycaemia, and temporal lobe epilepsy.

**Investigations**
Exclude organic causes like thyrotoxicosis and temporal lobe epilepsy.

**Management**
- Correct hypoglycaemia, if present.
- For uncomplicated anxiety:
  - Reassure patient.
  - Give amitriptylline 25–50mg nocte, as it may be helpful.
  - Do not use benzodiazepines.

- For complicated anxiety with presence of phobias, panic attacks, etc., refer to a psychiatrist for:
  - Psychotherapy.
  - Behaviour therapy.
  - Counselling.
  - Other pharmacological interventions, which include SSRIs, tricyclic antidepressants.

## 16.5 Post Traumatic Stress Disorder

This is a common anxiety disorder that develops after exposure to a terrifying event or ordeal in which grave physical harm occurred or was threatened. This gives rise to both psychological and social effects. Psychological effects are those that affect different levels of functioning, including cognitive (perception and memory as a basis for thoughts and learning), affective (emotions), and behavioural. "Social effects" pertain to altered relationships with the family and community networks, and effects on the economic status.

**Clinical Features**

In the acute phase, the clinical features may include intrusive flash backs, grief reaction, denial, disbelief, numbness, restlessness, anxiety, social withdrawal, and uncontrollable crying.

**Management**

- Provide psychological first aid for those showing acute distress. This is an informal, non-clinical intervention that entails:
  - Providing basic, non-intrusive care with a focus on listening but not asking to talk.
  - Showing empathy by validating the person's feelings.
  - Reminding the distressed person that their feelings are a normal reaction to an abnormal situation, and that it is expected that the uncomfortable or bothersome feelings or painful symptoms will disappear overtime.
  - Assessing needs and ensuring that these needs are met.
  - Encouraging but not forcing friendships, companionship, and otherwise positive interactions with others. For example, if the person is ready, help to join a social activity group.
  - Providing as much factual information as possible about access to services and any plans for the affected communities that may have been made.
- Refer for psychiatric management.

## 16.6 Psychosexual Disorders

These are disorders that range from deviant sexual behaviour such as homosexuality, sex change, and transvestism (tendency to appear to be of different sex) to overtly criminal activities such as rape and paedophilia.

**Management**

Refer to higher level for appropriate management.

## 16.7 Conversion Syndromes

These are mental disorders in which there is a psychogenic disturbance-of either motor or sensory function in some parts of the body.

**Clinical Features**
They may present as paralysis of a part of the body, tremors, blindness, deafness, seizures, aphonia. The severity of disability fluctuates, and the patient fails to exhibit the seriousness the disability accords. Good psychiatric history may reveal the source of conflict.

- Thorough physical examination: even though the patient often appears normal, this should be done.
- Refer to psychiatrist for appropriate management.

## 16.8 Depression

The primary and dominant characteristic of depression is a change in mood. Typically it consist of depressive mood with characteristic changes in behaviour, attitude, thinking efficiency, and physiological functioning.

**Clinical Features**
Dysphoric mood which is characterized by sadness, crying spells, irritability, or lowered ability to function socially. It further escalates to negative views of self, the environment and the future that is indicated by guilt, loss of interest, difficulties in concentrating or suicidal thoughts. There maybe insomnia with loss of or increase in appetite.There may be weight loss or gain with multiple somatic complaints, e.g., fatigue, weakness, headaches, backache, etc. A meticulous history is important as under-diagnosis is common and many patients suffering from depression are often missed and receive inadequate treatment. Many depressed patients have a precipitating factor, e.g., loss of income, death of a spouse, onset of disability, or are on drugs that produce depression as a side effect, e.g., methyldopa.

**Management – General**
Most patients are managed as outpatients. It is important for the care provider to maintain a positive and hopeful attitude towards the patient and to the extent of possible involvement of the relatives in the management of the patient, especially to improve compliance.

**Management – Pharmacological**
**Antidepressants:**

- Amitriptyline 50mg nocte: for patients who require sedation ***OR***
- Imipramine 50mg nocte for patients who do not require sedation ***OR***
- Fluoxetine 20mg once daily preferably given in the mornin for patients with BPH and cardiac disease and elderly

**OR**

- The dosage timing may improve patient compliance. Antidepressants take at least 2 weeks to take effect. If no improvement at 4 weeks review the diagnosis and medication.
- If medications are effective, they should be continued for 3 months and then reduced at 25mg/week.

**Failure to respond to therapy may be due to:**
- Poor compliance
- Inadequate dosage
- Misdiagnosis
- Inadequate therapeutic trial (usually 6 weeks) Refer to psychiatrist for:
  - Re-evaluating the diagnosis
  - Instituting chronic treatment (prophylaxis) in those with recurrent serious depression
  - Changing to second generation antidepressants,e.g.,maprotiline, monoamine oxidases inhibitors.
  - Considering electroconvulsive therapy (ECT).

**Patient Education**
- Inform the patient that there will be a delay of 2 weeks before beneficial effects of treatment are experienced.
- Explain about the side effects,e.g.,drymouth,constipation,hypotension, daytime sedation(drowsiness).
- Warn patient about dangers of alcohol consumption.
- Review the patient at least once every 2 weeks until maintenance dose is reached and then once a month until total drug withdrawal or as necessary.
- Involve the relatives in long-term management.

**— Do not give large prescriptions to patients. There is risk of suicidal overdose. Drug II administration should be monitored while at home.**

## 16.9 Bipolar Mood Disorder (Manic Episode)

The primary characteristic is a change in mood consisting of an exaggerated sense of wellbeing and enhanced esteem.

**Clinical Features**
The clinical features include hyperactivity that is usually goal oriented, over generosity, extravagance, disinhibition (promiscuity and drug abuse), irritability, accelerated speech, infectious elated congruent mood, grandiose delusions, enhanced self-esteem, insomnia, and weight loss (no time for food). In severe forms, patients appear disorganized and may be violent; legal involvement may be necessary in their management. History and physical examination are essential; it is necessary to establish if ever depressed in past.

**Management – General**

- ♦ Rule out intoxication.
- ♦ Involve family members in management.

**Management – Pharmacological**

- ♦ Immediate if disturbed:
- Haloperidol 10mg IM ***OR***
- Chlorpromazine 150-200mgIM
- Lithium carbonate 300mg 3–4 times daily. Monitor serum levels closely.
- Sodium valproate 750mg/day in 3–4 divided doses initially, increase every 3–4 days to a dose of 1,000–2,500mg/kg/day in divided doses
- Carbamazepine and other anticonvulsants such as lamotrigine, gabapentine, and topiramate can also be used.
- Olanzapine 2.5–20mg PO/IM per day
- Zuclopenthixole acuphase 100mgIM

**Long-term:**

- ♦ Start on haloperidol PO 5–10mg nocte.
- ♦ The agents used in the acute state can also be used.
- ♦ For non-compliant patients, haloperidol decanoate 100–200mg IM monthly or fluphenazine decanoate 25–50mg IM monthly.
- ♦ Clopenthixol decanoate 200mg IM monthly.
- ♦ Mood stabilizer,e.g.,carbamazepine 200mg twice a day or lithium or sodium valproate.

**Refer to psychiatrist if there is no response in 4–6 weeks and you have excluded:**

- ♦ Poor compliance
- ♦ Inadequate dose

**Electroconvulsive therapy (ECT) can be administered for acute mania or severe depression.**

**Admit if:**

- ♦ Patient is a risk to others or self.
- ♦ Patient is exhausted.

## 16.10 Schizophrenia

A form of mental illness characterized by loss of contact with reality, hallucinations, delusions, abnormal thinking, flattened affect, and disturbed work and social function, occurring in a setting of clear consciousness, memory and orientation.

**Clinical Features**

The clinical features include withdrawal and generalized loss of interest in the environment with thought disorder. The normal association of ideas is lost and there is characteristic incongruence of affect. There are also delusions, hallucinations in any

sensory modality, and disturbances in behaviour and motor function, e.g., grimacing, odd postures.

History obtained from the patient and relatives is most important. Continuous signs of illness should be present for 6 months at some point in the patient's life, with some clinical features at the time of diagnosis.



**Management – General**

Psychological and social support entails use of psychiatric community nurses and social workers in involving the family to understand the illness and help in rehabilitation of the patient into community activities. Importance of drug compliance should be explained to relatives and patients.

**Management – Pharmacological**

- Severely disturbed patient –admit:
  - Give chlorpromazine100–200mg IM and then start on oral chlorpromazine 100–200 12–24 hourly
  - Fluphenazine 2.5–40mg orally daily
  - Trifluoperazine 1–5mg orally daily
  - Haloperidol 2–25mg orally daily
- Mildly disturbed patient:
  - Manage as outpatient.
  - Give chlorpromazine 100mg TDS ***OR*** haloperidol 5mg TDS. If patient was diagnosed as a schizophrenic and missed the drugs, restart the drug as before.
- Maintenance therapy, chlorpromazine100–200mgTDS ***OR*** haloperidol 5–10mgTDS
- Onset of extrapyramidal side effects: reduce dose and start on benzhexol 2.5–5mg TDS
- For patients who are not dependable about taking oral drugs, depot preparations are available:
  - Fluphenazine decanoate 25mg IM monthly
  - Haloperidol decanoate 50mg IM monthly
  - Clopenthixol decanoate 200mg IM monthly
  - Flupenthixol decanoate 40mg IM monthly.
  - Resperidone 2–6mg PO once to twice daily.
- Electroconvulsive therapy (ECT) can be administered for refractory cases.

**Caution: Aim to use lowest dose that is therapeutic in cases of long- term use to minimize risk of side effects.**

**Admit if patient is severely disturbed, violent, or catatonic.**

**Patient Education**

Compliance to therapy is important to prevent relapses.
Relatives should bring the patient to the hospital at early signs of relapse Drugs may have to be taken for a long time depending on response.

# 16.11 Sleep Disorders

## 16.11.1 INSOMNIA

Insomnia is difficulty in initiating or maintaining sleep, leaving the patient feeling unrested. Insomnia can be a symptom of most other psychiatric and physical disorders, which should be excluded. Rule out use of addictive drugs (caffeine, etc).

**Management**

Hypnotics, e.g., diazepam 5–10mg nocte for 1–2 weeks and then taper off.

**Caution: Avoid chronic use (over 14 days) of hypnotics.**

## 16.11.2 OTHER SLEEP DISORDERS

Hypersomnia (narcolepsyicataplexy): Patient complains of excessive sleep without any demonstrable cause.

**Management**

Forced naps at regular times of the day.

Methyl phenidate 30mg morning and 20mg midday until symptoms disappear, maximum dose 60mg daily.

# 16.12 Suicide Attempts

This is the unsuccessful attempt to end one's own life.

**Clinical Features**

Suicide threats. May occur in the following conditions: depression, schizophrenia, under influence of alcohol/drugs, under severe social problems or stress, or personality disorder. Often the attempted suicide itself is the first symptom.

**Management**

- ♦ Admit patient
- ♦ Urgently restore physical fitness
- ♦ Once patient's life is out of danger, take a full history without accusing the patient. Treat the patient with understanding and respect. An empathetic approach is very important if you are to win the confidence of the patient so that he/she will be able to tell you the truestory.

♦ Assess the seriousness of the attempt: Every suicide attempt should be regarded as serious: A successful attempt may follow. Do not regard an attempt as just attention seeking. Factors indicating seriousness include:

- Patient living alone, divorced, or single
- History of a relative committing suicide
- Chronic incurable physical illness
- Suspicion of or diagnosis of cancer or AIDS
- Presence of a suicide note
- Attempt done in a place unlikely to be discovered

- Impotence in males and infertility in females
- Continuous difficulty in sleeping (insomnia)
- Alcohol and drug abuse – continuous social problems
- Whether patient regrets having failed to die
- If tablets taken, did the patient believe the dose was lethal
- Previous attempted suicide
- Failure to succeed, particularly at examinations

### 16.12.1 VALUE OF ELECTRO-CONVULSIVE THERAPY (ECT)

- Shortens hospital average length of stay, where necessary,e.g.,elderly patients, puerperal patients.
- Alternative treatment where side effect of psychotropic drugs are to be avoided, e.g., elderly patients, pregnant mothers.
- Suicidal patients.
- Refractory mental illnesses where there is an indication.

**Classical Indications**

- Major depressive disorder
- Psychosis
- Suicide attempts
- Stupor
- Schizophrenic stupor
- Bipolar mood disorder (manic episode) refractory to pharmacotherapy

Management of Side Effects of Anti-Psychotic Drugs
For extra pyramidal side effects (EPSE) and anticholinergic side effects, administer tabs benzhexhol hydrochloride 2–5mg TDS PO.

OR
Biperiden tabs 2–4mg TDS PO.

# PART II: Paediatrics and Related Disciplines

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# 17. Paediatric Emergencies

## 17.1 Recognition of a Seriously Ill Child (Triage)

It is important that all health care workers learn to recognize a child needing emergency care as soon as the child is brought to a health facility. Fortunately, this depends on a few clinical features that are easy to learn with practice. Parents/ care givers may have tried to treat the child at home or the child may have fallen sick quickly. They are advised to come to the health care facility as soon as possible if the child is weak, not able to drink, has severe diarrhoea, cold hands and feet, high fever, difficulty in breathing or convulsion.

## 17.2 Causes of Cardio-Respiratory Arrest after Neonatal Period

**These include:**

- Fluid loss: Diarrhoea, blood loss, burns.
- Fluid mal-distribution: Anaphylaxis, septic shock, cardiac disease.
- Respiratory distress: Pneumonia, asthma.
- Foreign body (obstructed airway).
- Respiratory depression: CNS infections, convulsions, poisoning.
- In addition to the above, severe malnutrition is a common cause of death in young children.
- Trauma can also be a cause.

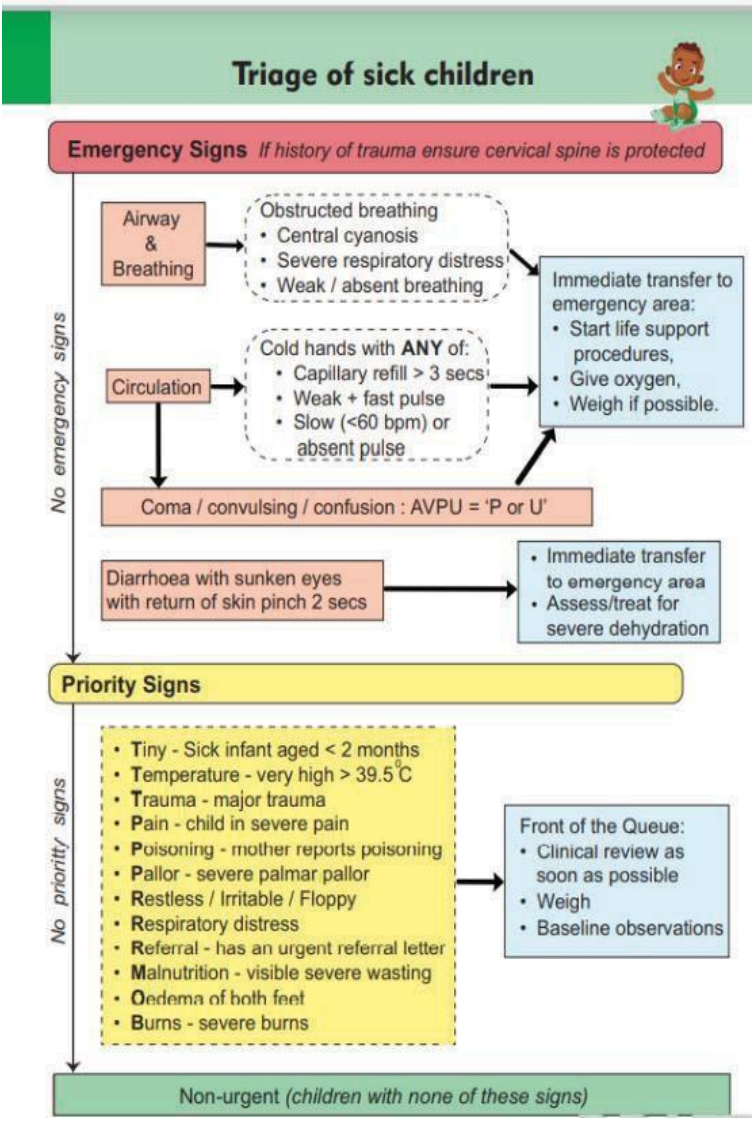
**The figures shown below assist you to triage and manage these children as they arrive at the health facility. Management in all these states includes the ABC's: Airway, Breathing, Circulation.**

## 17.3 Summary of Steps Taken: ABCD of Resuscitation

- Always have a resuscitation tray ready. Suction equipment (ambubag and mask, oxygen delivery equipment, IV fluids including dextrose. Drugs – adrenaline and diazepam
- Airway/breathing: Remove any airway obstruction. Start immediate treatment to restore or support breathing.
- Circulation: Restore circulating blood volume by giving 20ml/kg of Ringer's lactate or normal saline over 15 minutes. Repeat until return of pulse; this may be repeated up to 3 times. For shock without diarrhoea, to be given slowly over 2 hours.
- If severe anaemia start urgent blood transfusion not Ringer's.
- Convulsions: Give anticonvulsants if child is convulsing. Diazepam (IV or rectally) and then phenobarbital (IM) if no response to two doses of diazepam.
- Carry out emergency investigation if you are able: Blood glucose, malaria test, haemoglobin.
- Reasses every 2-3 minutes until stabilized following the same format– airway, breathing, circulation and intervene as needed.

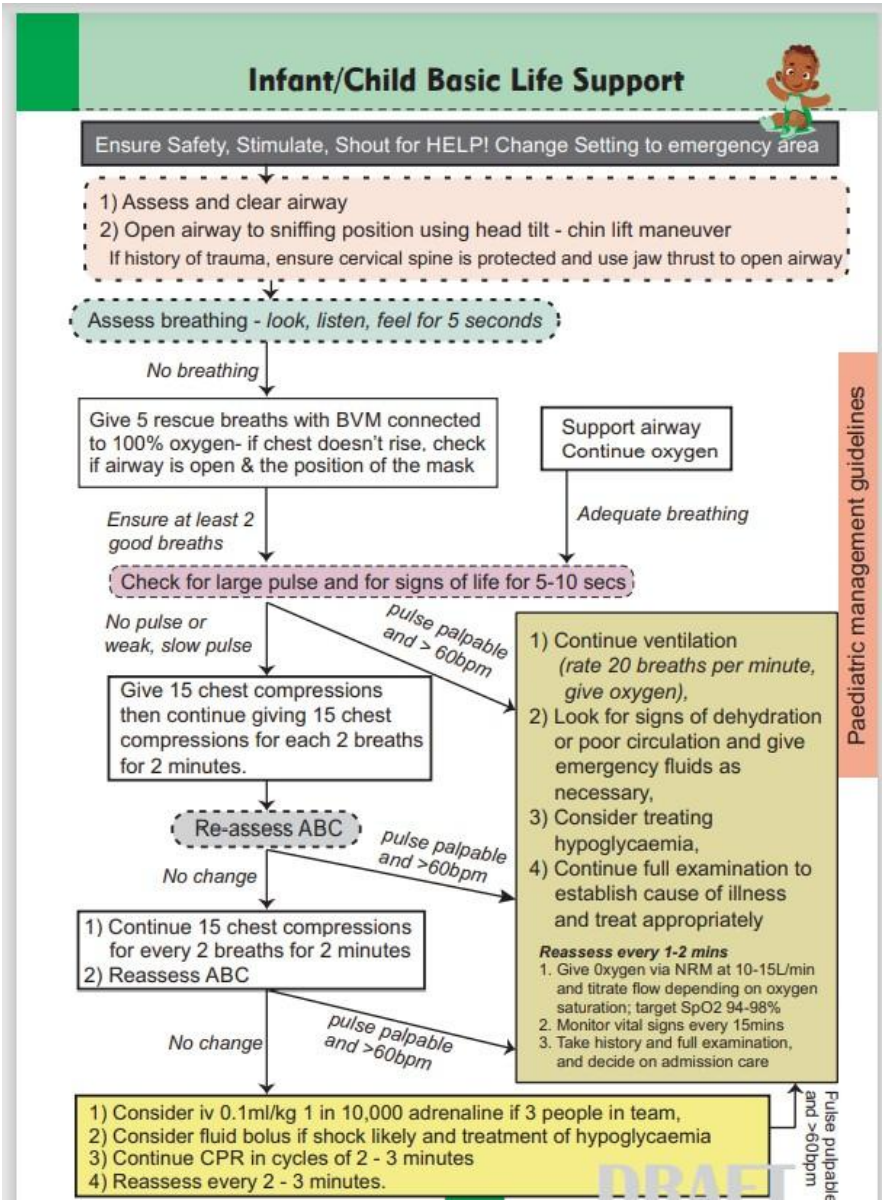
- When ventilation and chest compression are effective, carotid and femoral pulses become palpable, pupils constrict, and the colour ofucous membranes improves.
  - ♦ NOTE: Chest compressions: apply appropriate pressure over the sternum:
- For newborn or small infants, effective cardiac output can be produced by applying maximum pressure with the tip of 2 fingers placed on the sternum just below the intermammary line or hands round the infant'schest.
- For larger infants and small children, use the heel of one hand over the sternum one finger breadth above the xiphisternum.
- For big children, the heel of the right hand is placed over the heel of the left hand to provide the strength of both arms and shoulders. Hands are placed 2 finger breadths above the xiphisternum.
- Refer urgently after stabilizing the child, if need be. During transport ensure adequate airway, breathing, and circulation. Make sure you write a comprehensive report of what you have done to the receiving hospital clinician.

**Figure 17.1: Triage of sick children**




Source: Kenya Basic Paediatric Protocols, 2022

**Figure 17.2: Infant/Child Basic Life Support**




Source: Kenya Basic Paediatric Protocols, 2022

## 17.4 Shock

**Causes of shock include**

- Bleeding
- Severe infection (septic shock)
- Severe dehydration
- Cardiac disease
- Trauma

**Clinical Features**

- Cold hands
- Capillary refill >3seconds
- Weak fast pulse
- Altered consciousness AVPU<A

**- Children with these signs are in shock and need emergency treatment.**

**Treatment**

**For a child without severe malnutrition:**

- Infuse 20ml/kg of normal saline or Ringer's lactate over 15 minutes for hypovolemic shock and over 2 hours for distributive (septic and anaphylactic) shock.
- Reasses and give a second dose if there is no improvement. You may need 2 or 3 repeats to restore circulating blood volume

**For a child with severe malnutrition:**

- Give 20ml/kg of Ringer's lactate with 5% dextrose or half normal saline with 5% dextrose and infuse over 2 hours.

- ***After resuscitation: Admit and look for the cause if not already obvious and treat.***

**Investigations**

- Full blood count,
- Blood sugar
- Renal function tests
- Culture and sensitivity (blood and body fluids).
- Coagulation screen
- Radiological and other investigations as may be required

**Management – General**

- Resuscitate with normal saline or Ringer's lactate. Repeat if necessary–up to 60ml/kg may be required but watch for heart failure. A central venous catheter (CVC) line is useful.
- Hourly pulse and BP monitoring.
- Catheterize and monitor urine output hourly: If less than 1–2ml/kg/hr after adequate fluid replacement give frusemide 1–2mg/kg IV STAT. If

urine output does not increase assume renal failure and manage accordingly.
- ♦ Oxygen via nasal prongs or catheter.
- ♦ Definitive treatment of cause.

**Management – Pharmacological**
- ♦ Start empirically on:
  - Crystalline penicillin + gentamicin + metronidazole IV. Oral metronidazole can be started as soon as patient is able to swallow.
  - Specific antibiotics depend on source of infection and culture and sensitivity results.

## 17.5 Anaphylaxis

This is severe potentially life-threatening allergic reaction to drugs, food, stings, etc.,

**Clinical Features**

These include
- extensive skin rash
- pruritus
- urticaria
- respiratory distress that may be accompanied by a wheeze or a stridor (due to laryngeal oedema or bronchospasm), and hypotension.

**Management**

Parents and care givers are advised to take to a health care facility as soon as possible for a child with extensive skin rash or difficulty in breathing.

**Follow the ABC of resuscitation. In addition, do the following:**
- ♦ Adrenaline: give IM 0.01ml/kg of 1:1,000 solution; or 0.1ml/kg of 1:10,000 solution. Can be repeated every 5 minutes 2 doses; if no response after 2 doses secure IV access and infuse adrenaline IV 0.5-1ml/kg/hour and titrate according to response.
- ♦ Antihistamine: Chlorpheniramine 0.1mg/kg IV slowly or diphenhydramine. Then continue IM/SC 8 hourly for 24–48 hours.
- ♦ Hydrocortisone 4mg/kg IV is of secondary value but useful to prevent delayed recurrences.

**Subsequent management:**
- ♦ Patients with mild to moderate reaction, e.g., urticaria or mild bronchospasm, should be observed for at least 6 hours because attacks may recur after full recovery.
- ♦ Admit those with severe reactions e.g. poor circulation, severe bronchospasm.
- ♦ Continue intravenous fluid replacement, and closely monitor pulse, BP and urinary output.
- ♦ Avoid offending agents. Inform parent/child the cause of reaction so as to know and to avoid the offending agent in future.

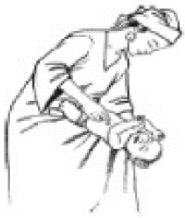
## 17.6 Choking

Choking is a blockage of the upper airway by food or other object which prevents a person from breathing effectively. Infants and young children can easily choke on a number of things. Often, they play with seeds, buttons or any small object which if put in the mouth easily goes the wrong way. Referral to facility with relevant expertise is indicated incases of failure to dislodge the object or if severe complication arise from choking

Because the procedure may be required urgently:by the time the child arrives at the health facility they may have already choked to death. Figures 17.3 and17.4 shows how this is done for infants and children, respectively.

**Figure 17.3: How to manage the choking infant**

a) Back slaps



b) Chest thrusts

1. Laytheinfantonyourarmsorthighina head downposition.
2. Give5blowstotheinfant'sbackwith heel of hand (Figure17.3a).
3. If obstruction persists, turn infantover and give 5 chest thrusts with 2fingers one on top of the other, one finger breadth below nipple level in midline (Figure17.3b).
4. If obstruction persists, check infant's mouthforanyobstructionthatcanbe removed.
5. Ifnecessaryrepeatthesequencewith back slapsagain.

PAEDIATRICS

**Figure 17.4: How to manage the choking child**

1. Give 5 blows to the child's backwith heel or hand with the child sitting, kneeling or lying (Figure17.4a).
2. If obstruction persists, go behind the child and pass your arms aroundthe child's body. Form a fist with one hand. Immediately below the child's sternum place the other hand over the fist and quickly press upwards into the abdomen (Figure 17.4b). Repeat this Heimlich manoeuvre 5 times.
3. If obstruction persist check child's mouthforanyobstructionthatcanbe removed.
4. Ifnecessaryrepeatthesequence with back slapsagain.

a) Slapping the back to clear airway obstruction in a choking child

b) Heimlich manoeuvre in a choking older child

# 18. Diarrhoeal Diseases

Diarrhoea is defined as the occurrence of at least 3 loose or watery stools in a day. Diarrhoeal illness is classified for dehydration, presence of blood in the stool and duration.

**Definitions:**

- Acute watery diarrhoea: Watery stools lasting less than 14days
- Dysentery: The presence of fresh blood in the diarrhoeal stool.
- Persistent diarrhoea: Diarrhoea that has lasted for 14 days ormore.
- Dehydration: Loss of water and electrolytes.

**Common Causes of diarrhoea in children:**

- Young children <5 years: rotavirus, Ecoli
- All ages except neonates: Shigella, cholera, salmonella spp., amoeba, giardia, candida.
- Others: Lactose intolerance, food poisoning

**The major cause of death from diarrhea is dehydration, especially in infants and young childre. Management of diarrhea is aimed primarily at evaluation, prevention, and treatment of dehydration.**

## 18.1 Assessment and Management of Acute Watery Diarrhoea

**Signs of dehydration in children:**

- Sunken eyes
- Return of skin pinch ≥ 2 seconds
- Irritability
- Restlessnes

**Severity of dehydration is classified as follows**

- No dehydration: able to drink plus less than 2 signs of dehydration.
- Some dehydration: able to drink plus 2 signs signs of dehydration.
- Severe dehydration: Inability to drink/breastfeed or altered consciousness, plus either delayed skin pinch plus sunken eyes.

**Assess for signs of shock; if present manage as outlined in paediatric emergencies then classify according to age of child and severity of dehydration (Table 18.1) and manage as given in Tables 18.2–18.4 and Figure 18.1.**

**ASSESSMENT AND MANAGEMENT OF DEHYDRATION**

- The volumes indicated are guidelines only.
- If signs of severe dehydration persists, repeat the rehydration in Plan C (Table 18.2).
- If improving and child can drink, start ORS (about 5ml/kg/hr). Show the mother how to give ORS.
- Evaluate preferably every hour until signs of dehydration disappear (usually within 4 hours).

**For other children continue ORS ( Plan A – Table 18.2):**

- Fluid to be given after correction of dehydration:
  - Up to 2 years: 50–100ml for every stool passed.
  - 2–5 years: 100–200ml for each loose stool.
  - 5 years and above: 300ml and more as desired. Thirst is the best guide for maintenance fluid therapy in older children.
- If child vomits, wait 10 minutes and give same volume slowly.
- Periorbital oedema is a sign of fluid overload: If this occurs stop the ORS and give plain water or breast milk in breastfeeding children.

**Table 18.1: Assessment and classification of diarrhoea in children below 5 years**

| Age of child | No dehydration (2 signs or less) | Some dehydration | Severe |
|---|---|---|---|
| Young infants<br>1 month to 11 months | Normal | Sunken eyes<br>Restless/irritable<br>Skin pinch goes back slowly | Lethargic/unconscious<br>Sunken eyes<br>Skin pinch goes back very slowly (>2 sec) |
| 12 months to 59 months | Normal | Able to drink<br>Thirsty<br>Restless/irritable<br>Skin pinch goes back slowly<br>Eyes sunken | Lethargic/unconscious<br>Sunken eyes<br>Not able to drink or drinking poorly<br>Skin pinch goes back |

**Table 18.2: Rehydration protocol for young children**

| Degree of dehydration | Age | Where | Type of liquid | Volume | Rate |
|---|---|---|---|---|---|
| No dehydration | 1 week– 2 months | togive Home | ORS | 10ml/ kg | After every bout of diarrhoea |
| **Plan A** | ≥2 months – 5 years | Home | ORS | 10 mg/kg | After every bout of diarrhoea |
| Some dehydration **Plan B** | 1 week – 5 years | Health unit | ORS | 75ml/kg | 4 hours, then reassess |
| Severe dehydration | less than 12 months week– | Health unit | Ringer's lactate or Hartmann's solution | 100ml/kg | 30ml/kg in 1 hr 70ml/kg in 5 hrs |
| **Plan C** | 12 months – 5 years | Health unit | Ringer's lactate or Hartmann's solution | 100ml/kg | 30ml/kg in ½hr 70ml/kg in 5hrs |

**Table 18.3: Clinical evaluation of dehydration in older children (More than 5 years)**

| Clinical features | Mild dehydration | Moderate dehydration (2 signs present) | Severe dehydration ( 2 signs present) |
|---|---|---|---|
| General: | Thirsty, alert | Thirsty, alert anxious, cold extremities, | Generally conscious, clammy, cyanosis, wrinkled skin of fingers, muscle cramps, dizzy if standing |
| Pulse | Normal | Rapid | Rapid, thready, sometimes absent |
| Respiration | Normal | Deep, some- | Deep andrapid |
| times rapid SystolicBP | Normal | Normal | Low, sometimes unmeasurable |
| Skinelasticity/ skinpinch | Immediate recoil | **Decreased** | **Fold disappears very slowly** (>2seconds) |
| Eyes | Normal | **Sunken** | **Severely sunken** |
| Tears | Present | Absent | Absent |
| Mucous membranes (test mouth witha clean finger) | Moist | Dry | Verydry |
| Urineo utput | Normal | **Reduced, urine dark** | **Anuria, empty bladder** |
| % of body weight loss | 1–5% | 6–9% | 10% orplus |
| Estimated fluid deficit | 10–50ml/kg | 60–90ml/kg | 100ml/kg |

**Table 18.4: Rehydration protocol for older children**

| Degree of dehydration | Age | Type of liquid | Volume to give | Rate |
|---|---|---|---|---|
| Mild | All | **ORS** | 50ml/kg | In 4 hrs |
| Moderate | All | **ORS** | 100ml/kg | In 4 hrs |
| Severe | Older children | Hartmann's solution, Ringer's lactate | 110 ml/kg | In 4 hrs: at first as rapidly as possible until a radial pulse is palpable |

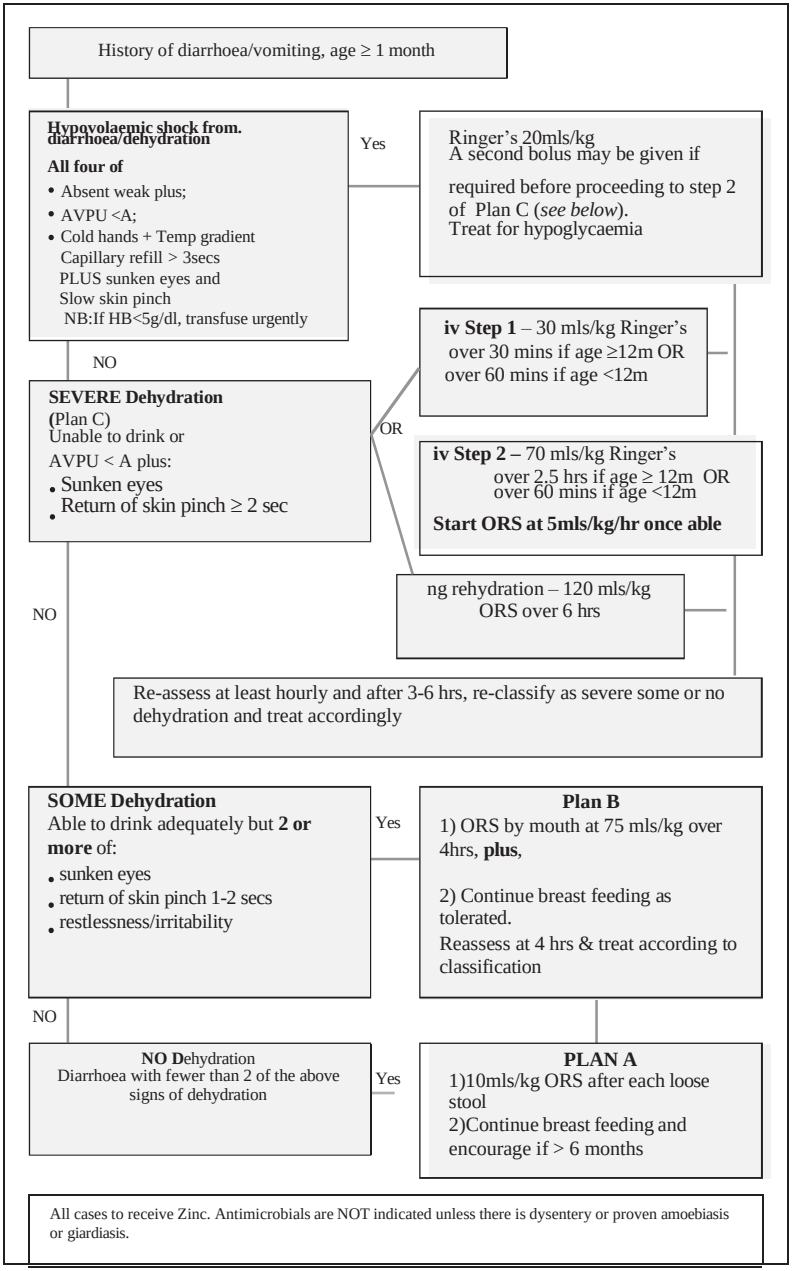
NOTES: (a) Initially, older children can drink 300 ml/hour. (b) If Ringer's lactate or Hartmann's solution is not available, use normal saline.



**Diarrhoea/GE Protocol (Excluding Severe Malnutrition)**

- Antibiotics are NOT indicated unless there is dysentery or persistent diarrhoea and proven amoebiasis or giardiasis. Refer to table 18.6.
- Diarrhoea of>14 days may be complicated by intolerance of ORS, worsening the diarrhoea. If present change to IV regimens.
- All cases to receive zinc.

**Figure 18.1: Management- Rehydration protocol for young children**



**All children under 5 years give zinc for 10–14 days:**

- Up to 6 months 10mg/day
- 6 months and above 20mg/day

**Ask caregiver to return to health facility if no improvement in 2 days or if the patient develops the following:**

Worsening of diarrhoe, not able to drink or breast, vomiting everything, fever marked thirst and blood in stool.

- Also if the child become sicker.

**At any stage if child is not improving or deteriorating consult or refer.**

**Management – Nutrition**

**It has been shown that there is no physiological reason for discontinuing food during bouts of diarrhoea and that continued nutrition is beneficial to all children. Continued feeding should be encouraged, for example:**

- Under 6 months: Breastfeed on demand as soon as baby is able to feed.
- 6–24 months: Breastfeed on demand and offer complementary food.

- 2 years and above: Provide family foods while continuing ORS:
  - Give cereal or starchy food mixed with some vegetable or proteinfoods.
  - Give fresh fruit or mashed bananas to provide potassium.
  - Food based fluids (soups, enriched uji, madafu, mala) can be used during the oral rehydration phase.
  - Add a teaspoon of vegetable oil in each serving to complementary food
  - Give an extra meal per day for 2 weeks after recovery.
  - Give vitamin A if child has not received a dose in the last 3 months.

**Management – Pharmacological**

- Majority of acute gastroenteritis in young children isviral.
- Anti-diarrhoea drugs(e.g.,absorbents) and antiemetics are not recommend in children with acute diarrhoea
- If child has fever,consider other diseases associated with diarrhoea(e.g., malaria, otitis media,pneumonia).
- Antimicrobial drugs should be used for children ***only*** asfollows:
  - Antibiotics only for dysentery and suspected cholera.
  - Antiprotozoal drugs: Metronidazole for:
    - Amoebiasis only after antibiotic treatment of bloody diarrhoea has failed or faeces shows trophozoites of E.histolytica.
    - Giardiasis when diarrhea has lasted over 14 days and cysts or trophozoites of giardia are seen in faeces.
- Antibiotics for specific intestinal infections are listed in Table18.5.

**Most acute diarrhea in children is viral AND does NOT require antibiotics**

**Table 18.5: Antimicrobials used in the treatment of diarrhoea**

| Aetiology/Clinical Features | Management |
|---|---|
| Cholera: Very profuse watery diarrhea (rice-water stools),often vomiting | Doxycycline 2 to 4 mg/ kg Max 300mg as a single dose Or Azithromycin 20mg/kg Max 1g as a single dose or Ciprofloxacin 20mg/kg max single dose |
| Shigella dysentery: Blood &mucus in stools, cramps, tenesmus, fever | Ciprofloxacin 15mg/kg bd x 3 days |
| Amoebiasis | Metronidazole 7.5mg/kg 8 hourly |

## 18.2 Persistent Diarrhoea

This is diarrhoea that starts acutely but lasts 14 days or more. Can be watery or with blood. Degree of dehydration assessed as in acute diarrhoea. Causes include:

- Malnutrition
- Occult infections
- HIV
- Candidiasis
- Amoebiasis
- Giardiasis

**Note: Persistent and prolonged diarrhoea predisposes to malnutrition especially if the nutritional status was borderline.**

**Management**

**Management for dehydration the same as that for acute diarrhoea. Then:**

- Treat underlying condition ifpresent
- Do not give antibiotics unless there is specificindication
- Rehydration as for acutediarrhoea

**Feeding Recommendation for a Child with Persistent Diarrhoea**

- Successful diet is characterizedby:
  - Weight gain
  - Adequate food intake according toage
  - Disappearance of diarrhoea.
- If still breastfeeding, give more frequent, longer breastfeeds, day andnight.
- If not breastfeeding: use fermented milk products such as“Mala”or yoghurt or any other high protein but low lactose food or drinks as these are tolerated better.The aim is to give 110 calories/kg/day,of which 10% is protein.
- Use locally available food.
- Add a teaspoonful of vegetable oil to each serving of feeds.
- For other foods, follow feeding recommendations for the child’s age. Ensure adequate intake.
- Encourage the child to feed.
- Give an extra meal per day and continue until 1 month after diarrhea has Stopped.

Give micronutrients:

- Multivitamin supplements,
- Vitamin A,
- Folate,
- Iron, and
- Zinc

**Prevention of Gastrointestinal Tract (GIT) Infections**

- Adequate nutrition
  - Breastfeed exclusively up to age 6 months and continue together with adequate complementary foods at least up to age 2 years.
- Food hygiene
  - Ensure that all food consumed by the whole household is prepared and stored hygienically. This also depends on availability of safe and adequate water supply.
  - Boil water for drinking or treat with sodium hypochlorite.
- Environmental sanitation
  - Ensure proper disposal of waste (human and household) in the homes and communal areas. This is essential.
  - Wash hands with soap after using the toilet.
- Managing food handlers
  - Require regular examination of food handlers, especially in schools.
  - Ensure appropriate treatment of food handlers when necessary.

# 19. Fever

Fever is a common but non-specific presenting sign in children. Any child with a temperature of 37.5 C or above is said to be febrile. Fever accompanies a wide variety of illnesses and does not always need to be treated on its own. In general, the cause should be ascertained as far as possible before therapy is started. History should take into account the duration, place of residence or travel to areas of high malaria transmission, pain on passing urine, pain in the ears, and whether there is a rash or not. A thorough physical examination to find localizing signs should also be done.

- Fever without localizing signs can be due to:
  - Malaria
  - Septicaemia
  - Urinary tractinfection
  - HIV
- Fever with localizing signs can be due to:
  - Ear or throat infection
  - Pneumonia
  - Septic arthritis or osteomyelitis
  - Meningitis
  - Skin and soft tissue infection

- Fever with a skin rash is commonly due to:
  - Viral infections
  - Could also be due to meningococcal infection
- Fever lasting longer than 7 days can be due to:
  - Abscesses
  - Infective endocarditis
  - Tuberculosis
  - HIV
  - Salmonella infections
  - Any chronic infection, autoimmune or inflammatory condition
  - Malignancies

**Investigations**

- Full blood count
- Blood smear for malaria parasites
- Urinalysis and microscopy
- Blood culture and sensitivity
- If fever lasts > 7 days, in addition to the above do:
  - Mantoux test
  - Chest x-ray
  - HIVtest
  - Any specific test according to suspected cause

**Management – General**

- Ask parent to reduce child's clothing to a minimum in all cases.
- Ensure adequate fluid intake.
- Ensure adequate nutrition.
- If fever is high (>39°C) or child in pain, give paracetamol
- Treat the cause if identified.
- Treat malaria if test is positive ( see section 20 on Malaria) .

Ask parent to return any time if child is not improving or is getting worse.

**Treatment**

Paracetamol 10mg -15 mg/kg 4 - 6 hourly per day

Fever alone is not a reason to give antibiotics except in a young infant (age less than 2months).

**Management – Specific**

Identification of the cause is the key to management and helps to prevent over use of specific drugs,e.g.,antibiotics or antimalarials.

**Management of Fever at the Community Level**

Since most of the cases of fever occur at the community level, it is essential to train health care providers and caregivers where applicable on early recognition and prompt initiation of treatment at the community level.

The patients should be taken immediately to a health facility if there are any features of severity as described in the section on severe malaria below.

# 20. Malaria

Malaria parasites are usually transmitted by the bite of an infected female anopheles mosquito. Plasmodium falciparum is the commonest type of malarial parasite in Kenya. It is associated with significant morbidity and mortality. The other species are: P. malariae, P. vivax and P. ovale.



## 20.1 Clinical Features of Malaria

### 20.1.1 UNCOMPLICATED MALARIA

**Classically, malaria presents with paroxysms of fever, chills, rigors, and sweating. Other features include:**

- Malaise
- Headache
- Myalgia
- Joint pains
- Nausea
- Vomiting
- Abdominal discomfort
- Diarrhoea

### 20.1.2 SEVERE AND COMPLICATED MALARIA

**This presents with a combination of most of the above plus one or more of the following**

- Severe anaemia (Hb<5g/dl)
- Inability to drink and breastfeed
- Lethargy or altered unconsciousness or coma
- Generalized convulsions 2 or more within 24 hrs
- Clinical jaundice plus presence of other vital organ disfunction
- Hypoglycaemia (blood sugar<2.2mmol/L)
- Respiratory distress, pulmonary oedema
- Acidosis
- Disseminated intravascular coagulopathy – DIC (spontaneousbleeding)
- Malaria haemoglobinuria (coca-cola coloured urine)
- Oliguria
- Shock
- Fluid electrolyte imbalance

## 20.2 Diagnosis of Malaria

### 20.2.1 CHILDREN OF ALL AGES

- In high malaria endemic areas, any child with fever or history of fever should have a parasitological test (microscopy or rapid diagnostic test (RDT) ) to confirm diagnosis.
- In low malaria endemic areas, any child with fever or history of fever in the absence of measles, running nose, or any other identifiable cause of fever shouldbe have a parasitological test (microscopy or RDT) to confirm diagnosis

### 20.2.2 ADDITIONAL INVESTIGATIONS IN PATIENTS WITH SEVERE AND COMPLICATED MALARIA

- Thick blood smear for malaria parasites (several slides may need to bedone)
- Thin blood smear for parasite count (parasitaemia>5%) species identification and red blood cell morphology
- Full blood count
- Blood sugar
- Serum bilirubin
- Urea, electrolytes, creatinine
- Urinalysis and microscopy
- Lumbar puncture in patients with altered consciousness
- Blood culture

— ***A negative slide does not necessarily rule out malaria.*** Where cerebral malaria is suspected appropriate therapy must be instituted promptly.
— **If there is no evidence of positive parasitological result in a patient who has received presumptive treatment for malaria repeat malaria test every 6 to 12 hrs**

### 20.2.3 FIRST LINE TREATMENT FOR UNCOMPLICATED MALARIA FOR ALL AGE GROUPS

The recommended first line treatment for uncomplicated malaria in Kenya is artemether-lumefantrine currently available as a co-formulated tablet containing 20mg of Artemether and 120 mg of lumefantrine. This is administered as a 6- dose regimen given over 3 days (see Table 20.1).

**Table 20.1: Dosing schedule for artemether-lumefantrine**

| Bodyweight | No. of tablets recommended at approximate timing (hours) ofdosing (each tablet contains 20mg A and 120mg L) | | | | | |
|---|---|---|---|---|---|---|
| **0h** | **8h** | **24h** | **36h** | **48h** | **60h** | |
| 5–14kg (<3 yr) | 1 | 1 | 1 | 1 | 1 | 1 |
| 15–24kg (4–8 yr) | 2 | 2 | 2 | 2 | 2 | 2 |
| 25–34kg (9–14 yr) | 3 | 3 | 3 | 3 | 3 | 3 |
| >34kg (>14 yr) | 4 | 4 | 4 | 4 | 4 | 4 |



The regimen can be expressed more simply for ease of use at the programme level as follows: the second dose on the first day should be given anytime between 8-12 hours after the first dose. Dosage on the second and third days is twice a day (morning andevening).
Treat infants weighing < 5 kg with uncomplicated P. falciparum malaria with artemisinin-based combination therapy (ACT) at the same mg/kg btw target dose as for children weighing 5 kg
(Target dose range: A total dose of 5-24mg/kg bw of artemether and 29-144mg/kg bw of lumefantrine)

**Malaria patients with HIV/AIDS should be managed according tothe same regimen above.**

**In children below 5kg (under 2 months of age), malaria is not a common cause of fever. Evaluation of other causes should be undertaken. Where malaria is diagnosed the recommended treatment is artemether-lumefantrine**

## 20.2.4 COUNSELLING AND FOLLOW UP

**For all caregivers the following counselling messages should be provided:**

- Explain dosing schedule: Use probing questions to confirm patient's understanding.
- Emphasize that all 6 doses must be taken over 3 days even if patient feels better after few doses.
- Directly observe the first treatment dose.
- Repeat the dose if vomiting occurs within 1 hr after drug administration.
- Advise that artemether-lumefantrine should preferably be taken with a meal.
- Advise caregiver to return the patient immediately to the nearest health facility if their condition deteriorates at anytime, or if symptoms have not resolved after 3 days.

## 20.2.5 SUPPORTIVE TREATMENT

- Fever management: In cases where temperature is above (temp >38.5$^{o}$C) administer an antipyretic.The recommended option is paracetamol .
- Encourage adequate fluids and nutrition: Caregivers should be encouraged to give extra fluids and where applicable to continue breastfeeding. Feeds and

fluid should be administered as small quantities in frequent intervals as tolerated.

## 20.2.6 TREATMENT FAILURE

Treatment failure is defined as a failure to achieve the desired therapeutic response after initiation of therapy. Treatment failure is not synonymous with drug resistance. Treatment failure may result from underdosing, poor adherence to treatment, unusual pharmacokinetic properties in that individual, or drug resistance.
Treatment failure could also arise because of a wrong diagnosis and thus initiating the wrong treatment. it is important when evaluating s patient with treatment failure to determine whether they vomited previous treatment or did not complete a full treatment course from their history.

Treatment failures should be suspected if patient deteriorates clinically at any time or if symptoms persists 3–14 days after initiating drug therapy in accordance with the recommended treatment regimen.

Development of symptoms 14 days after initiation of therapy where there has been prior clearance of symptoms should be considered as a new infection and be treated with the first line drug.

**Remember that not all fevers are due to malaria. A fever that does not respond to adequate antimalarials may be due to other causes.**

## 20.2.7 SECOND LINE TREATMENT FOR ALL AGE GROUPS

The recommended second line treatment for uncomplicated malaria in Kenya is oral quinine. This is administered as 10mg/kg bw per dose ever 8 hours for 7 days (refer to Table 20.2).

**Table 20.2: Dosing schedule for quinine tablets**

| Quinine sulphate 200mg | | Quinine 300mg salt (sulphate, dihydrochloride,hydrochloride) | |
|---|---|---|---|
| **Weight in kg** | **No of tabs** | **Weight in kg** | **No of tabs** |
| 4–7kg | 1/4 | 6–11kg | 1/4 |
| 8–11kg | 1/2 | 12–17kg | 1/2 |
| 12–15kg | 3/4 | 18–23kg | 3/4 |
| 16–23kg | 1 | 24–35kg | 1 |
| 24–31kg | 1 1/2 | 36–47kg | 1 1/2 |
| 32–39kg | 2 | 48kgandabove | 2 |

For children below the lowest weight category, the dosage of quinine is 10mg/kg and the tablets should be reconstituted into syrup based on the weight of the patient.

## 20.3 Management of Complicated Malaria

### 20.3.1 EMERGENCY CARE (SEE PAEDIATRIC EMERGENCIES)

— Airway, breathing, circulation
— Correct hypoglycaemia if present
— Treat convulsions if present
— Measures for unconscious patient

**Management –Specific**

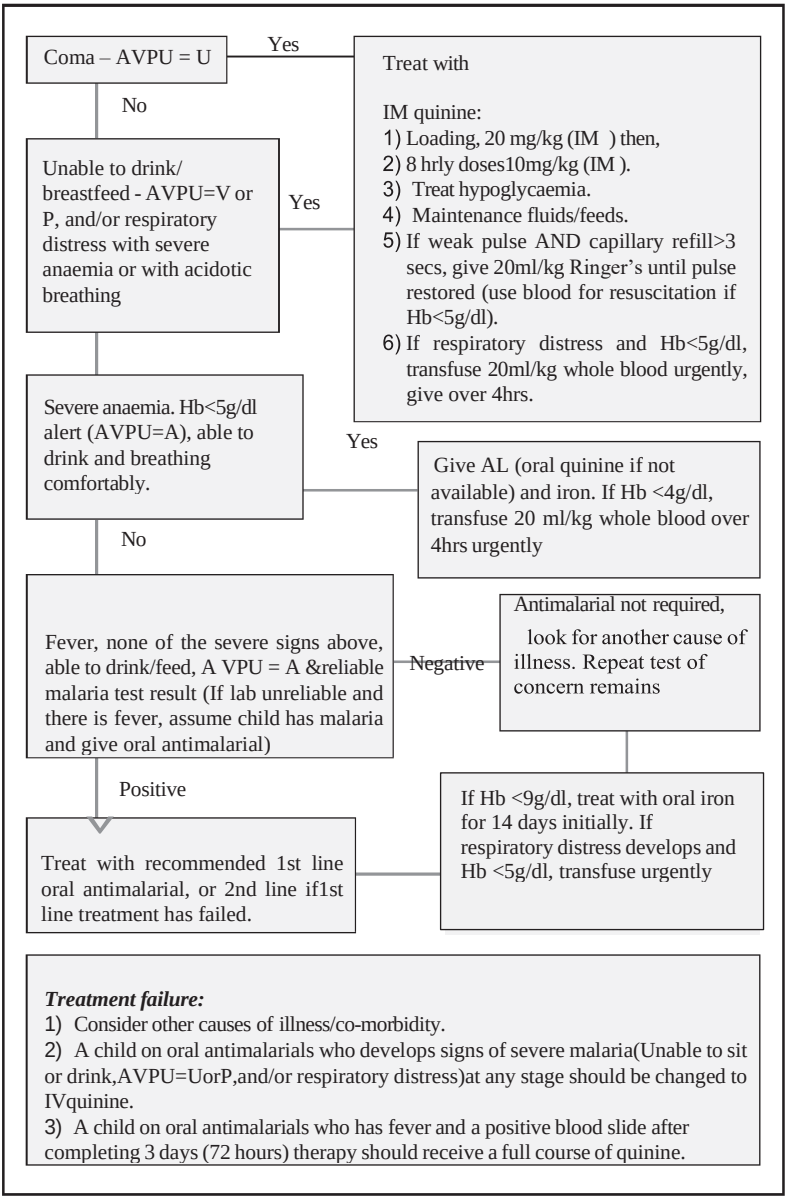
1st line drug- artesunate

- Weight ≤ 20Kg at 3mg/kg/dose and >20Kg at 2.4mg/kg/dose of Artesunate at 0,12 and 24h then daily for max 7 days.
- After the third injection of artesunate and the child can eat/drink then change to a full course of artesinin combination therapy (ACT) 8-12 hours after the last dose of artesunate (typically the 1st line oral anti-malarial, Artemether Lumefantrine).

2nd line drug- Quinine IM

- IM quinine (see Figure 20.1 and Tables 20.3 and 20.4)
- 20 mg/kg–loading dose then 10mg/kg 8 hourly until the patient is able to take orally. Thereafter, give a full dose of arthemether-lumefantrine for 3 days.

**Figure 20.1: :Management of complicated malaria**



### 20.3.2 MALARIA TREATMENT IN MALARIA ENDEMIC AREAS

If a high quality blood slide is negative, then only children in coma or those with severe anaemia should be treated presumptively for malaria.

### 20.4.1 MANAGEMENT OF COMPLICATIONS

**— Admit all patients with complications.**

- Coma: Exclude other causes especially meningitis. Do an LP and start empiric treatment for bacterial meningitis in addition to the treatment of complicated malaria.
- Shock: Besides disturbed fluid electrolytes balance, this can be due to septicaemia. Investigate for septicaemia and start empiric treatment with antibiotics.
- Severe anaemia: When Hb<4g/dl transfuse. Do not use diuretics during transfusion as often these children are hypovolaemic.
- Renal failure (oliguria and rising blood urea and creatinine). If persistent oliguria after correction of fluid electrolytes balance, refer to renal specialist.
- Refer or consult if:
  - Facility not able to manage complication
  - Patient deteriorating despite presumed adequate car
- Follow up:
  - Children with cerebral malaria may have residual neurological complications that may need rehabilitation depending on the disability.

## 20.4 Prevention of Malaria

### 20.4.1 CHEMOPROPHYLAXIS

- Anti-malaria prophylaxis should be given to the following groups:
  - All non-immune visitors to malarious areas use mefloquin, atovaquone, doxycycline or proguanil
  - Long-term residence >4weeks
  - Short-term residence <4weeks
- Use proguanil for:
  - Patient with sickle cell disease and thalassaemia
  - Patients with tropical splenomegaly syndrome or splenectomy
- Chemoprophylaxis regimes
  - Proguanil (daily dosing) non-immune visitors: Start daily 1 week before arrival and continue for 4 weeks after leaving malarious area. Refer toTable **20.4 for dosage.**
  - Others use indefinitely.

**Table 20.3: Proguanil dosage schedule**

| Proguanil | Daily PO |
|---|---|
| 1–4yrs | 50mg (½tablet) OD |
| 5–8yrs | 75mg (¾tablet) OD |
| 9–12yrs | 100mg (1tablet) OD |
| Adult | 200mg daily (2 tablets) OD |

## 20.4.2 PREVENTION OF MOSQUITO BITES

- Use insecticide treated nets (ITNs): In high malaria areas it is recommended that all sleep under ITNs but especially children under age 5 years and pregnant women.
- Screening of house inlets use wire mesh to reduce entry of mosquitoes into the house.
- Use insect repellants especially forvisitors.
- Cover exposed skin in the evenings.

## 20.4.3 VECTOR CONTROL

- Encourage all households to clear bushes around the house, drain any stagnant water, and cover or avoid throwing away containers that may collect water.
- Participate in indoor residual spraying campaigns in both endemic and epidemic prone areas.

## 20.4.4 PATIENT EDUCATION

- Seek early treatment for fever and to remember that not all fevers are due to malaria.
- Always seek medical care if the fever does not respond toantimalarials.
- Complete the antimalarial dose as prescribed for effectiveness of treatment and to prevent development of resistance.

# 21. Measles

It is also called rubella. It is one of the commonest childhood infectious exanthems. Measles is never subclinical, but the severity of the disease is related to the infective dose of the virus and the nutritional status of the child. Crowding tends to increase spread of the disease.

**Clinical Features**

Incubation 7–10 days. Fever. Catarrhal phase 2–3 days with cough, red eyes, and runny nose followed by maculopapular rash. Assess for danger signs, clouding of the cornea, or extensive mouth ulcers.

**Complications**

**These must be looked for in all patients:**

- Serious signs: Persistent fever with darkening of the rash("black measles") and subsequent desquamation.
- Stomatitis and mouth ulcers: Compromises sucking and feeding.
- Laryngitis: Distinguish a benign prodromal laryngitis from that due to a secondary infection, which may be severe.
- Bronchopneumonia: Usually severe; Gram-negative organisms or staphylococcus.
- Diarrhoea: Either due to virus or from a secondary infection.
- Vitamin A deficiency: Keratoconjunctivitis. Measles increases the consumption of vitamin A and often precipitates xerophthalmia and subsequent blindness.
- Encephalitis: Caused by the measles virus itself; it occurs on about the 5th day of the rash, sub acute sclerosing panencephalitis (SSPE) is an important late complication.
- Malnutrition: Precipitated by anorexia, stomatitis, fever, vomiting, diarrhoea, and other complications.

**Management**

- Uncomplicated cases can be treated at home.
- Treatment with antibiotics is not recommended.
- Give an antibiotic only if pneumonia (Section 24.2) or otitis media (Section 27.1)are present.Consider staphylococcal pneumonia if the child has had prior antibiotic treatment for pneumonia
- Give two doses of oral vitamin A for treatment as follows: First dose in the clinic then give the mother 1 dose to give at home the nextday:
  - 50,000 IU for young infants aged less than 6 months
  - 100,000 IU for infants aged 6–1 2months
  - 200,000 IU for children 12 months to 5years
- Treat fever (temperature 38.5°C) if present with paracetamol.
- Provide skin care as appropriate.
- Eyecare should be provided.Give antibiotic eye ointment for conjunctivitis only if there is purulent eyedischarge.
- Nutrition: Severe stomatitis/mouth ulcers may prevent feeding. Maintaining oral hygiene and,where there is candidiasis (thrush) in the mouth,application of nystatin 1ml 4 times a day is necessary. If breastfeeding advise the mother to wash the breast

after feeds and apply the same medicine after cleaning it with salt water, is necessary.

- Supervise feeding: For a patient not able to feed, feed by nasogastric tube. For breastfeeding babies who are not able to feed give expressed breast milk feeds by cup or nasogastric tube.
- Increasing the frequency of feeding (an extra meal per day over the usual feeding) after measles illlnesss is very important to help the child regain lost weight adequately.
- Assess and classify nutritional status and give appropriate treatment.
- Admit if the following are present:
  - A haemorrhagic rash
  - Stridor (from infection of the larynx and trachea; laryngotracheitis)
  - Pneumonia, dehydration, or severe under-nutrition
  - In ability to drink or breastfeed

**Prevention**

Immunization: Measles immunization is given to babies who are 9 months or above irrespective of whether they have suffered from measles/measles like illness. Measles immunization and Vitamin A should be given to babies 6 months to 9 months in the following circumstances:

- Siblings to a child with measles illness.
- Children living in crowded places, refugee camps, children'shomes.
- Children admitted to hospital for any condition (age 6–9months).
- Children in a locality with measles epidemic.
- HIV infected children

**Advice to Parents/Caregivers**

- Ensure their children are fully immunized.
- Child should attend under 5 years children clinic on discharge.
- Treat complications:
  - Conjunctivitis: With tetracycline eye ointment; review after 2days. If improving, ask mother to complete treatment. If not improved refer.
  - Acute otitis media: With amoxicillin. Review after 5 days.
  - Mouth ulcers: With gentian violet or nystatin if has thrush.
  - Pneumonia: See Section 24.2, on pneumonia.
  - Malnutrition: Commonly follows an infection of measles. It is precipitated by anorexia, stomatitis, fever, vomiting, diarrhoea, and other complications. Also important are frequent harmful cultural practices that impose fasting upon a child with measles.
- Nutritional followup is very necessary. Increasing the frequency of feeding (an extra meal per day over the usual feeding) after measles illness is very important to help the child regain lost weight adequately.

# 22. Meningitis

Meningitis is an acute inflammation of the pia and arachnoid coverings of the brain with spread into the cerebro-spinal fluid (CSF). It is important to diagnose and start treatment early in order to prevent complications. The predominant causative bacterial organisms (pyogenic meningitis) vary with the age of the child.
Haemophilus influenzae commonly affects children under 5 years, while streptococcus pneumoniae (pneumococcus) tends to be more common after age 5 years. Hib immunization, however, is reducing the incidence of meningitis due to H. influenzae. Viruses (aseptic meningitis), Tubercle bacilli (Tuberculous meningitis), and fungi (fungal meningitis) also cause meningitis. Neisseria meningitidis (meningococcus) tends to cause meningitis in epidemics and affects all ages.

**Predisposing factors for meningitis in children are:**
- Low immunity,
- Prematurity,
- Septicaemia,
- Infections in the nose, sinuses, ears, throat and lungs
- Penetrating injuries and fractures of the skull and spinal column
- Congenital malformations of the brain andspine.

**Clinical Features (Child >2 months)**
Fever, inability to drink, vomiting, repeated convulsions, irritability, altered level of consciousness, headaches, photophobia, neck stiffness and positive Kerning's sign. Young children may also have bulging anterior fontanelle and high-pitched cry. Signs of increased intracranial pressure include sutural diastasis, increased head circumference, unequal pupils, focal neurological signs, and irregular breathing.

Patients presenting late in the progression of the disease may have decerebrate rigidity or opisthotonos. For tuberculous meningitis, the onset is more gradual and non-specific. Child may complain of headache, vomiting, and poor feeding for several days before features of meningitis appear. Gradually the child becomes stiff and loses consciousness.

**Complications**
These include subdural effusion, hydrocephalus, blindness, deafness, secondary epileptic fits, mental retardation and cerebral palsy. The child may also have retardation in their physical development.

**Investigations**
- Lumbar puncture (after fundoscopy to rule outpapilloedema)
- CSF investigations: ZN, microscopy
- Full blood count
- Blood glucose
- Chest x-ray
- Mantoux test, if there is history of contact with TB or fever lasting >7days
- Indian ink staining in patients with HIV infection (cryptococcal infection)

- Cryptococcal antigen test
- HIV test if not known
- Blood smear for malaria

**CSF Characteristics**

- Refer to Table 22.1 for CSF characteristics.
- Always treat as pyogenic meningitis if the CSF is cloudy, blood stained, or cannot be obtained.
- Admit patient if meningitis is suspected. Initiate treatment immediately.

| Nature of CSF | Colour | Protein | Sugar | Cells | Diagnostic Features |
|---|---|---|---|---|---|
| Normal | Crystal clear | Below 0.4g/L | Above 2.5mmol/L | 0–5(x10/L) | |
| Pyogenic | Cloudy | High | Low or NIL | Hundreds to thousands, mainly polymorph | Gram stain: cocci and bacillarybacteria, confirmed on culture |
| Tuberculous | Clear OR opalescent | Moderately raised | Low | A few hundreds mainly lymphocytes | ZN stain to identify AAFBs, confirmed on culture and/or PCR |
| Viral | Clear OR opalescent | Moderately raised | Normal | A few hundreds mainly lymphocytes | PCR confirmation |
| Fungal | Clear or opalescent | Moderately raised | Normal | A few hundreds mainly lymphocytes | Indian ink microscopy/CRAG for cryptococcal meningitis |

**Management – General**

- Follow the patient's progress:
  - Maintain fluid and electrolyte balance.
  - Ensure child is passing urine well.
  - Continue anticonvulsant if there were convulsions.
  - Ensure adequate nutrition for age.
- Treat for malaria if in malarious area.

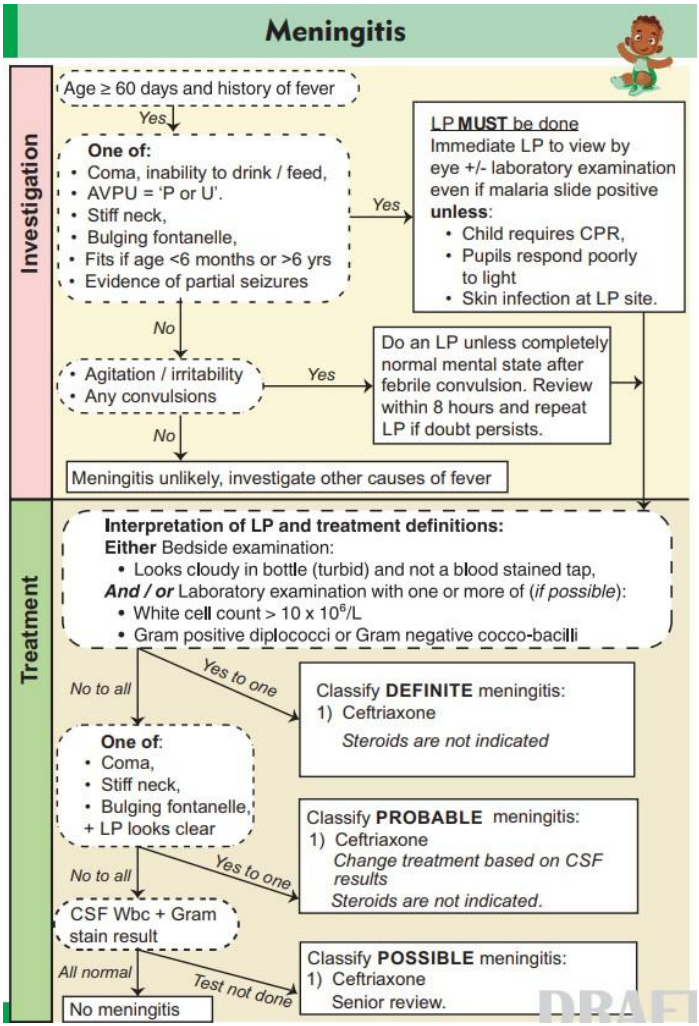
**Refer to Figure 22.1 for assessment and management of meningitis**

**Management – Pharmacological**

**Antibiotics – Pyogenic Meningitis**

- Ceftiaxone  50mg/kg IV every 12 hr for 10 to 14 days.
- Change treatment as per culture and sensitivity results.

**Figure 22.1: Flow chart for assessment and management of meningitis**




Source: Kenya Basic Paediatric Protocols, 2022

**Cryptococcal meningitis**

- Screening and primary prophylaxis in children are not recommended given the low incidence of cyptococal meningitis in this age group.
- Management:
  - Induction: Amphotericin B deoxycholate 1.0 mg/Kg IV daily) or liposomal Amphotericin B 5 mg/Kg/day plus Flucytosine (100 mg/Kg orally per day in four divided doses) for two weeks.
  - Alternative Regimen:
    Two weeks of amphotericin B deoxycholate (1.0 mg/kg/day) + fluconazole (12 mg/kg/day for children and adolescents up to a maximum of 800 mg daily).
  - Consolidation: Fluconazole (6 to 12 mg/Kg orally per day) for eight weeks.
  - Maintenance: Fluconazole for 12 months at 6 mg/Kg per day orally daily.

Re-evaluate, or consult if:

- The condition is deteriorating.
- Patient develops a wide spread skin rash,or easy bleeding before or during treatment.
- All children with complications as they will need specialized therapy according to the disability.
- After full treatment, child is brought back with fits with or without fever.

**Prevention of Meningococcal Disease**

*Neisseria meningitidis* (meningococci) and *Streptococcus pneumoniae* (pneumococci) are the most common causes of childhood meningitis (from 3 months of age). Before the widespread use of *H. influenzae* type b vaccine *Haemophilus influenzae* meningitis was a common cause of meningitis in infants and young children.

*Neisseria meningitidis* A, C, and W135 are the main subtypes involved in epidemics in the African meningitis belt.

Due to its infectiousness, it is recommended that close contacts of persons with meningococcal disease should receive chemoprophylaxis regardless of immunization status because they are at risk for infection.

**Prophylaxis for Meningococcal Infections**

- Ciprofloxacin
  - Children older than 12 years 500mg orally as single dose
  - Children aged 5-11 years 250mg orally single dose
  - Children younger than 5 years 30mg/kg orally as single dose max 125mg.
  - Rifampicin: Neonate–11 months: 10mg/kg/24hours; Children >1 year to 11 years 20mg/kg/24hours; Children 12 years and above maximum 600mg BD PO for 2 days.
- Vaccination in outbreaks: Purified capsulate polysaccharide vaccine is available to control outbreaks but it must be administered within 3–7 days of case identification to prevent an epidemic. The vaccine is not suitable for children <2years.
- Primary vaccination: Recommended for children at 9 months and then 1 year.

**— Notify the medical officer of health if meningococcal meningitis is diagnosed.**

# 23. Altered Consciousness or Convulsions

**Background and Aetiology**

Causes include infections (malaria, meningitis, encephalitis), trauma, tumours, cerebro-vascular accidents, complications of diabetes mellitus, epilepsy, liver failure, drug ingestion, poisoning and shock.

- A=Alert; V=responds to voice;P=responds to pain;U=unresponsive
- Children older than 5 years can be assessed using the Glasgow coma scale

**Assessment of altered consciousness**

This may be done using the AVPU scale or Glasgow Coma Scale

- AVPU: A=Alert; V=responds to voice; P=responds appropriately to pain; U=unresponsive
- Glasgow Coma Scale: Children older than 5 years can be assessed using the Glasgow coma scale

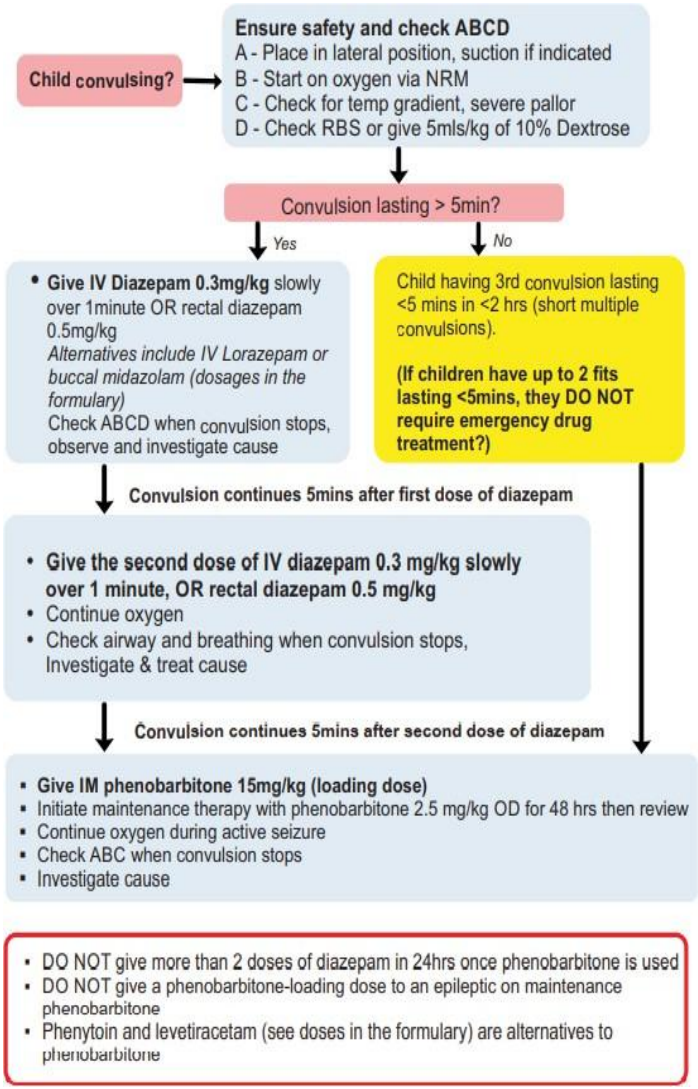
**Investigations**

- Full blood count
- Blood slide for malaria parasites
- Blood culture and sensitivity
- Blood sugar
- Lumbar puncture
- Urea electrolytes and creatinine
- Liver function tests if indicated
- Radiological investigations as appropriate
- Other tests according to suspected cause

**Management**

- A detailed history from parent or caregiver to establish the cause and duration is crucial. The convulsion should be described in detail.
- The child should be put on the lateral recovery position to avoidaspiration after the convulsion
- If the child is convulsing :
  - Assess airway, breathing, and circulation and manage the patient as per the flow chart.
  - Assess level of consciousness
  - Admit the child and examine all the systems fully to establish the cause of coma
- Continue monitoring vital signs and level of consciousness.
- Ensure adequate ventilation and circulation.
- Monitor fluid and electrolytes.
- Adequate nutrition.
- Treat the cause e.g. give antimalarials or antibiotics as indicated.
- Give antimalarials and antibiotic when indicated.
- Continue anticonvulsants if was convulsing.
- Turn patient 2 hourly to avoid pressure sores.
- Prevent contractures by regular daily passive exercises if condition becomes long-standing.

**Figure 23.1:Flowchart for management of convulsing child**




Source: Kenya Basic Paediatric Protocol, 2022

**Refer or consult if:**

- Patient does not respond to therapy or is deteriorating.
- Special investigation or treatment is needed that may not be available in your station.

**Treatment of Convulsions**

- Convulsions in the first 1 month of life:
  - Treat with phenobarbitone 20mg/kg IM STAT; a further 5–10mg/kg IM can be given within 24 hours of the loading dose (maximum 30mg/kg in 24 hours).
  - Give maintenance doses of 2.5-5mg/kg IM 24-hourly.

# 24. Respiratory Diseases

Acute respiratory infections are common and have varying severity. They are the commonest cause of mortality in children ages 1 month to 5 years.

## 24.1 Acute Upper Respiratory Tract Infections

### 24.1.1 COMMON COLD(ACUTE RHINITIS,CORYZA)

An acute, usually afebrile, viral infection of the respiratory tract within flammation of all the airways including the nose, paranasal sinuses, throat, larynx, and often the trachea and bronchi.

**Causes**

These include rhinoviruses, influenza viruses, parainfluenza viruses, respiratory syncytial viruses, corona viruses, adenovirus, and coxackie viruses.

**Clinical Features**

Nasal obstruction, watery rhinorrhoea, sneezing, sore throat, cough, watery red eyes, headache and general malaise. Most children with these features do not present to health facility. Young infants may have difficulty breastfeeding due to blocked nostrils.

**Management**

**Most colds resolve spontaneously in 7–10 days. The following is recommended.**

- Avoid aspirin, which may increase the risk of Reye's syndrome in children.
- Avoid cough and cold remedies in the form of antihistamines,cough suppressants, expectorants, and mucolytics.
- Treatment includes:
  - Analgesics and antipyretics e.g., paracetamol for pain and/or fever
  - Adequate fluid intake
  - Saline nasal drops/spray to decongest the nose

**Patient Education**

- The child should be kept warm and breastfed frequently; the nose should be cleared if it interferes with breathing or breastfeeding.
- The child should be brought back to the health facility if breathing is difficult or feeding becomes a problem.

**— Note: Antibiotics are of no value in viral infections.**

### 24.1.2 PHARYNGITIS AND TONSILLITIS

Acute inflammation of the pharynx and tonsils caused by streptococcus, viruses and occasionally diphtheria.

**Clinical Features**

Sore throat, painful swallowing, general malaise, fever, body aches, rhinitis.

In children vomiting and abdominal pain may be present. Tender cervical or submandibular lymph nodes usually indicates streptococcal infection. Look for pseudomembrane over the upper airways in suspected case of diphtheria.

**Complications**
Complications of streptococcal pharyngotonsilities include otitis media, rheumatic fever with or without carditis.

**Investigations**
- Full blood count
- Throat swab for microscopy, culture and sensitivity if possible
- Evidence of recent streptococcus infections – anti-dnase B, antistreptolysin 0 test (ASOT

**Management**
- Supportive measures – adequate hydration, antipyretics, analgesics.
- Amoxycillin 25mg/kg/dose every 12hr for 5-7 days.
- If patient is allergic to penicillin use erythromycin 30-50mg/kg/day in 3-4 divided doses; max: 2 g/day.

**Admit if:**
- Patient develops stridor, difficulty in breathing and profressive difficulty in swallowing.
- Exclude diptheria by a throat swab if there is a grey adherent membrane on tonsils and throat.
- Treat with crystalline penicillin 25,000 units/kg every 6 hours for 7 days.
- Add antitoxin in Diptheria management.

## 24.1.3 DEEP NECK INFECTION
These are infections (cellulitis or abscesses) in the potential spaces around the neck, e.g., peritonsillar space, retropharyngeal space, submandibular space and parapharyngeal space.

**Management**
Start on systemic antibiotics, e.g., amoxicillin or amoxicillin + clavulanic acid then refer as appropriate because of the risk of airway obstruction.

## 24.1.4 DISEASES OF THE ADENOIDS
### ADENOID HYPERTROPHY
Commonly occurs in children andmay lead to chronic upper airway obstruction.

**Clinical Features**
Nasal obstruction leading to mouth-breathing, hyponasal speech, difficulty in breathing and eating, snoring. “Adenoid facies” may later develop.

**Investigation**
Lateral soft tissue X-ray of the nasopharynx – shows narrowing of the nasopharyngeal air space.

**Management**
- Conservative treatment – for patients with mild symptoms
- Antibiotics in presence of infection as for acute tonsillitis (amoxicillin or a macrolide antibiotic incase of penicillin hypersensitivity).

- Adenoidectomy for patients with recurrent or persistent obstructive and infectious symptoms associated with adenoid hypertrophy.

## 24.1.5 SINUSITIS

**This is usually a complication following a URTI. It may be acute or chronic. It can be infective or allergic in origin.**

**Clinical Features**

- Pain over affected sinus
- Fever and headache
- Nasal blockage
- Thick nasal discharge
- Reduced sense of smell and taste
- Halitosis

**Investigations**

- CT scan or MRI
- Microscopy, culture and sensitivity of the nasal discharge
- Rhinoscopy

**Management**

- Analgesic/antipyretic
- Antihistamines for allergic symptoms
- For purulent nasal discharge administer oral amoxicillin or amoxicillin/clavulinc acid for 7 days
- If the nasal discharge is unilateral, exclude foreign body especially in young children.
- Refer to ENT if there is failure of treatment, onset of complications, or need for surgical intervention.

## 24.1.6 ACUTE EPIGLOTTITIS

A severe infection of the epiglottis and surrounding tissues that may be rapidly progressive and fatal because of sudden airway obstruction by the inflamed tissues. Haemophilus influenzae type B is almost always the pathogen. Group A beta haemolytic streptococci and other organisms have become more prominent aetiological agents following widespread vaccination against Haemophulis influenza type B Infection through the respiratory tract extends downwards to produce a supraglottic cellulitis with marked inflammation. The inflamed epiglottis mechanically obstructs the airway. The work of breathing increases; resulting in carbon dioxide retention and hypoxia. Airway obstruction may lead to fatal asphyxia within a few hours.

**Clinical Features**

**Onset is frequently acute with:**

- Sore throat with difficulty in speaking
- Difficulty in breathing
- Stridor
- Fever

- Drooling of saliva
- Difficulty or inability to swallow
- Children lean forward and hyperextend the neck
- Deep suprasternal, supraclavicular, intercostal and subcostal inspiratory retractions

**Management**

**A This is an absolute emergency! Speed in treatment is vital.**
- Admit immediately if the diagnosis is suspected.
- Keep the child calm and provde oxygen.
- Secure airway immediately (nasotracheal intubation ortracheostomy).
- Allow the child to remain in the position of comfort.
- Do not try to examine the throat to avoid precipitating obstruction.
- Avoid sedatives.
- Provide careful and skilled nursing care to remove secretions, which may cause obstruction even after intubation.
- IV cephalosporins to cover for haemophilus influenza type B and streptococcus (Ceftriaxone 80mg/kg every 24 hrs for 5 days).

**Direct visualization of the epiglottis by a designated trained person may reveal a beefy red, stiff, and oedematous epiglottis. An airway should be placed immediately!!**

**— Remember that manipulation may initiate sudden fatal airway obstruction.**

## 24.1.7 CONDITIONS PRESENTING WITH STRIDOR

**Stridor is a harsh sound heard during inspiration when there is narrowing of the upper airways, including oropharynx, subglottis, larynx, and trachea. Conditions presenting with stridor include:**
- Viral croup including that due to measles
- Retropharyngeal abscess
- Foreign body inhalation
- Diphtheria
- Pressure on the airways by masses in the neck or mediastinum
- Congenital laryngeal anomaly
- Epiglottitis

**Clinical Features**
- Viral croup: Barking cough, hoarse voice,respiratory distress if obstruction is severe (tachypnoea, supraclavicular, suprasternal, subcostal and intercostal inspiratory retractions, cyanosis). Fever in 50% of children. Signs of measles if it is thecause.
- Retropharyngeal abscess: Swelling in the neck, difficulty in swallowing, drooling, fever.
- Foreign body: History of choking, sudden onset of respiratory distress.
- Diphtheria: Severe neck swelling, membrane on throat and tonsils.
- Congenital anomaly: Stridor from birth.

- Pressure on airways: Obvious masses in neck or mediastinum on x-ray.

**Management**

- Mild croup can be treated at home. Encourage adequate intake of fluids and feeding according to age.
- Ask the mother to bring child back immediately she notices difficulty in breathing or feeding
- Foreign body: This may be life threatening if main airway is blocked. Action should be immediate if the child is to survive (see Section17.8,Choking,with the accompanying chart)
- Severe cases will need care in an intensive care unit:
  - Be prepared for intubation and/or tracheostomy
  - Administer O2
  - Nasotracheal intubation if signs of severe obstruction occur: Severe chest indrawing, agitation, anxiety (air-hunger) and cyanosis
  - Tracheostomy may be done if intubation is impossible.

## 24.2 Lower Respiratory Tract Infections: Pneumonia

### 24.2.1 CLASSIFICATION OF PNEUMONIA

Children aged 2 to 59 months with cough or difficulty breathing to abe assessed for pneumonia as follows:

- Severe pneumonia: Presence of any one danger sign or SP02 below 90% (central cyanosis) inability to drink, altered consciousness or severe respiratory distress such as grunting. Auscultation may reveal signs of complicated pneumonia such as pleural effusion, emphysema, pneumothorax.
- Non severe pneumonia: Presence of age specific fast breathing (ages 2 to 11 months) ≥ 50 breaths per minute, ages 12 to 59 months ≥ 40 breaths per minute) OR lower chest indrawing AND no signs of severe pneumonia.
- No pneumonia: Cough or cold. No signs of severe pneumonia and no chest wall indrawing and no fast breath breathing.
- All children with cough or difficulty in breathing should be assessed for wheeze and classified accordingly (See section on conditions with stridor).

**Management of pneumonia depends on age and severity.**

### 24.2.2 PNEUMONIA IN CHILDREN AGED BELOW 2 MONTHS

- Pneumonia, sepsis, and meningitis in infants less than 2 months of age can rapidly lead to the death of the infant.
- Pneumonia specific symptoms may be lacking.
- All children aged below 2 months with pneumonia should be admitted.

**Clinical Features**

**These conditions should be suspected if any of the following are present:**

- Stopped feeding well (if feeding well before)
- Convulsions
- Abnormally sleepy or difficult to wake
- Stridor in calm child
- Wheezing
- Fever (38°C or more) or low body temperature (below 35.5°C)
- Severe chest indrawing
- Fast breathing (60 per minute or more)
- Central cyanosis (of the tongue) SP02 of <90%
- Grunting
- Apnoeic episodes
- Distended and tense abdomen

**Supportive Management**

- Assess and manage the airway. Clear any mucus from nose and throat by gentle suction.
- Give oxygen by nasal prongs or catheter until signs of hypoxia disappear (cyanosis or severe respiratory distress) or oxygen saturations are above 90% on pulse oxymetry.
- Give paracetamol if temperature is >38$^{O}$C.
- Maintain fluid and electrolyte balance.
- Maintain adequate nutrition and use nasogastric tube if needed.
- Monitor vital signs 3 hourly.
- Reassess response twice daily.

**Definitive Management**

**For those under 2 months:**
**First line antibiotics:**

- Infants in first week of life: Benzyl penicillin 50,000 units/kg IM/IV Q 12 hr and gentamicin 5mg/kg 24 hourly IV.
- infants 1 week to 2 months old: Benzyl penicillin 50,000 units/kg IM/IV Q 6hr and Gentamicin 7.5mg/kg 24 hourly.
- Treat for at least 5 days. Continue for 3 days after child is well.
- If meningitis is suspected: Treat for at least 10–14 days with ceftriaxone (see meningitis section).
- If no improvement after 48 hours or suspect. Staphylococcal pneumonia use cloxacillin or flucloxacillin instead of penicillin.

**Second line antibiotics:**

- Ceftriaxone 50-80mg/kg/day OD.

## 24.2.3 PNEUMONIA IN CHILDREN AGED 2 MONTHS - 59 MONTHS

**Clinical Features**

**The following are important to find out about in history:**

- Duration of cough or difficulty inbreathing
- Chocking or sudden onset in a previously wellchild
- Exposure to someone with active TB within the last 2 years
- Known HIV infection
- Family history of asthma
- Presence and duration of fever
- Drinking or breastfeeding poorly

**— The following features are danger signs and their presence makes the illness very severe:**

- Inability to drink or breastfeed.
- The child had convulsions or is convulsing now.

- Abnormal sleepiness (lethargy) or difficult to wake (unconscious). AVPU scale.
- SPO2 below 90% or presence of central cyanosis.

Examination should be carried out in a calm child to determine the following:

- Respiratory rate (breaths per minute)
- Lower chest indrawing
- Stridor
- Wheeze
- Severe acute malnutrition

Evaluate carefully to make a diagnosis of the cause of cough or difficult breathing, which might be caused by a number of conditions, including the following:

- Pneumonia and its complications (pleural effusion, empyema, pneumothorax)
- Malaria
- Cardiac disease with cardiac failure
- Severe anaemia
- Foreign body aspiration
- Tuberculosis infection

**Investigations**

- Full haemogram
- Blood slide for malarial parasites
- Chest x-rays for suspected cardiac disease, suspected TB, any pneumonia that does not respond to antibiotics within 48hrs., suspected complications of pneumonia, children with HIV infectio
- Blood culture and lumbar puncture in young infant

See ARI/ pneumonia protocol for children aged 2 months to 4 years (Figure 24.1), and Table 24.1 for cut-off points for fast breathing.

NOTE: Presence or absence of either fever OR crepitations (rales) on auscultation are NOT reliable clinical features for diagnosing pneumonia in young children. The features listed above are more sensitive in identifying these diseases and facilitating their effective intervention.

**Table 24.1: Fast breathing cut off points**

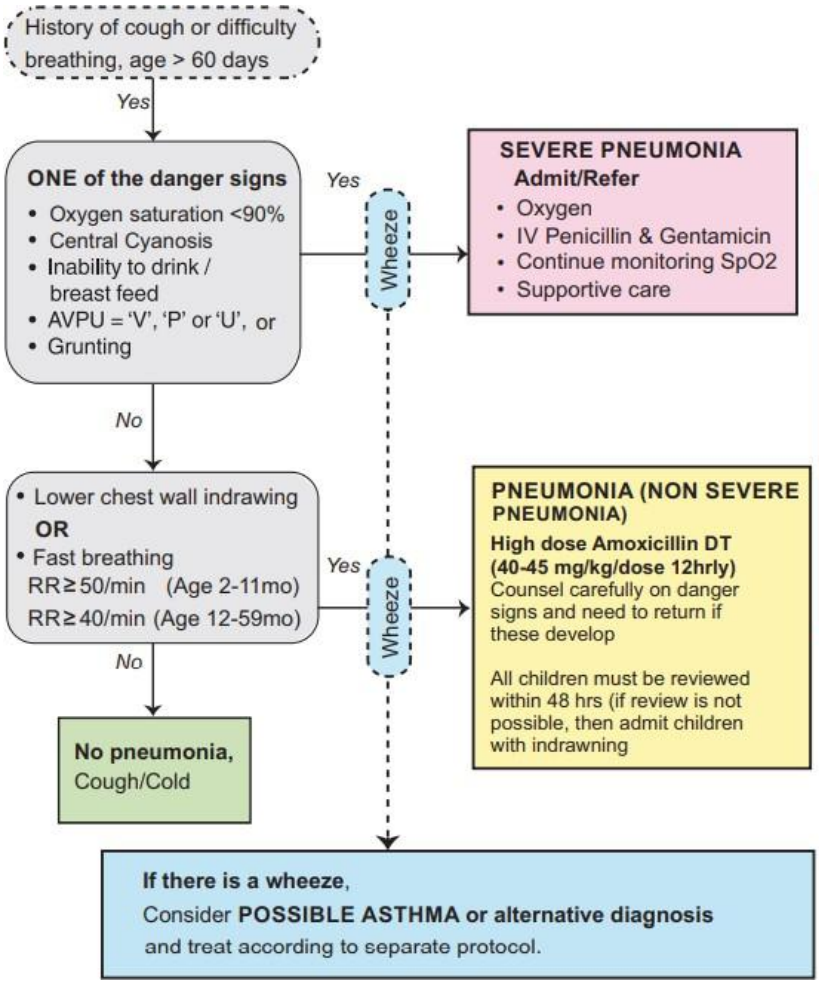
| Age | Fast breathing |
|---|---|
| Under 2 months (young infant) | 60 breaths per minute or more |
| 2months –12 months | 50 breaths per minute ormore |
| >12 months up to 5years | 40 breaths per minute ormore |

**Management of Non-severe Pneumonia**

- Treat as outpatient – use amoxicillin 40–45 mg/kg/dose 12 hourly
- Give first dose of antibiotic in clinic
  - Instruct mother on how to give the antibiotic for the five days at home.
  - Ask mother to bring the child for review after 2 days or earlier if child gets worse (see danger signs).
- Advice to mothers if child can be treated as outpatient:
  - Feed the child:
    - Feed the child during illness
    - Increase feeding after illness
    - Clear the nose if it interferes with feeding
  - Increase fluids:
    - Offer the child extra drink
    - Increase breastfeeding

- The following should be considered for **admission**:
  - if the mother is not able to bring the child for review after 48 hours
  - infants and children with features of severe disease
  - any child not responding to treatment.

**Figure 24.1:Pneumonia protocol for children aged 2-59 months without severe malnutrition**




Source: Kenya Basic Paediatric Protocols, 2022

**Management of Severe Pneumonia**
All children with severe pneumonia should be admitted

**Supportive management**
- Assess and manage the airway. Clear any mucus from nose and throat by gentle suction.
- Give oxygen by nasal prongs or catheter until signs of hypoxia disappear (cyanosis or severe respiratory distress) or oxygen saturations are above 90% on pulse oxymetry
- Monitor vital signs 3 hourly
- Give paracetamol if temperature is >38$^{o}$C
- Maintain fluid and electrolyte balance
- Maintain adequate nutrition and use nasogastric tube if needed
- Monitor vital signs 3 hourly.
- Reassess response twice daily.

**Definitive Management**
**For those under 2 months:**
- Benzyl penicillin 50,000 units/kg IM/IV Q 6hr and Gentamicin 7.5mg/kg 24 hourly for infants 1 week to 2 months old.
- Treat for at least 5 days. Continue for 3 days after child is well.
- If meningitis is suspected: Treat for at least 10–14 days with ceftriaxone (see meningitis section).
- If no improvement after 48 hours or suspect. Staphylococcal pneumonia use cloxacillin or flucoxacillin instead of penicillin.

**Second line antibiotics:**
- Ceftriaxone 50-80mg/kg/day OD.

**Treatment of complications:**
- Effusion, empyema,pneumothorax
  - Drainage is essential unless they are small and there is no respiratory embarrassment. If using syringe and needle, repeated aspiration will have to be done. It may therefore be preferable to insert a chest tube and do continuous under water drainage.

**Children with HIV or suspected HIV infection:**
- All children with unknown HIV status admitted with pneumonia should be tested for HIV infection to ensure adequate management.
- If proven HIV infection: First line therapy is penicillin/amoxicillin with gentamicin for10 days. Change to ceftriaxone if child does not respond within 48 hours.
- In addition to above, give cotrimoxazole (high dose 8mg/kgTMP/40mgS MZ) for 3 weeks to all infants aged 2–11 months and older children who have clinical or radiological signs of PCP.

**Counselling Parents**

All parents should be informed about child's illness and what to do to prevent recurrence.Parents should be encouraged to seek medical attention early in the disease to prevent te disease complications, which are associated with poor outcomes and are more difficult and costly to treat. Admit ALL infants under 2 months of age with suspected pneumonia, sepsis or meningitis

## 24.2.4 PNEUMONIA IN CHILDREN OLDER THAN 5 YEARS

Children aged 5 years and older are less likely to suffer from pneumonia than the younger children, unless they have another underlying condition. In a previously well child, the causative organism in this age group is usually pneumococci leading to consolidation of the lung parenchyma (lobar pneumonia). Organisms vary if a child is immunocompromised, has chronic lung disease, developed pneumonia operatively or after aspiration, or is debilitated.

**Clinical Features**

Breathlessness, cough with or without sputum (which may be rust coloured), fever, pleuritic chest pain, bronchial breathing, reduced chest movements, reduced breath sounds, tachypnoea, crackles, and percussion dullness.

**MANAGEMENT – NON SEVERE PNEUMONIA**

**Outpatient management:**

- Treat the child with a macrolide or penicillin
- Oral amoxicillin for 7 days.
- If penicillin allergy is present: erythromycin for 7 days
- Analgesics: Paracetamol

Child should be reviewed after 5 days.
Meanwhile, parents should be instructed to bring the child back to the health facility earlier if condition worsens or there is no response after 2 days.

**Admit the child if:**

- Cyanosis is present.
- Respiratory distress , chest indrawing and grunting
- Heart failure or pleural effusion is present.
- More than one lobe is involved.
- There is poor response as outpatient.
- Patient is dehydrated.
- Child has additional problems.

Investigate to identify any underlying cause.

**Inpatient management**

**First line antibiotic:**

- IV/IM Crystalline penicillin Q 6hr till response, then discharge and treat as outpatient.

**Second line antibiotic:**

- If failed outpatient treatment or immunocompromised – cefuroxime, ceftazidime or ceftriaxone. If staphylococcal pneumonia is suspected, consider cloxacillin or flucloxacillin.
- If atypical organisms are suspected (mycoplasma or chlamydia) add erythromycin or clarithromycin.
- Consider PCP if HIV infected.
- Give oxygen if indicated.
- Ensure adequate fluid, electrolyte and food intake.
- Manage underlying condition.
- Carry out further investigations like haemogram and HIV test.
-

## 24.3 Conditions Presenting with Wheeze

A wheeze is a high pitched sound during expiration due to narrowing of the small airways. Infections or allergic reactions can cause narrowing of the airways. Examples include asthma, bronchiolitis, foreign body, viral and bacterial infections.

**Conditions That Present with Wheeze**

- Secondary bacterial infections are common
- Bronchiolitis is a lower respiratory viral infection. The most common cause is respiratory syncytial virus. Its is typically most severe in young infants and is often associated with secondary bacterial infections.

**Management of Wheeze**

**For children with first episode of wheeze:**

- Give a rapid-acting bronchodilator – salbutamol via metered dose inhaler 2 puffs (100µg per puff) with or without a spacer according to age. Spacer can be made using a 1 litre plastic container (Figure 24.2). If inhaler is not available use nebulizer 2.5ml salbutamol.

- Assess response after 15 minutes. Signs of response are:
  - Less respiratory distress
  - Less lower chest retraction
  - Improved breath sounds
  - Manage according tothe cause and severity

**Figure 24.2: Inhaler with spacer. If unaffordable, use a plastic 750ml or 1 litre soft drink bottle**



**For children with Recurrent wheeze**

- Response to a rapidly-acting bronchodilator is an important part of the assessment of a child with recurrent wheezing to determine whether the child can be managed at home or should be admitted formore intensive treatment.
- Rapid acting bronchodilator should be given as above and the child's condition should be assessed 20 minutes later. If respiratory distress has resolved – the child should be treated with inhaler at home. The care giver should be taught how to use the inhaler.
  - Table 24.2 presents drugs and dosages for treating a child with wheeze.

**Admit the child if still distressed with or without cyanosis:**

- Give oxygen until cyanosis disappears or oxygen saturation >90%.
- Give first dose of prednisone 2mg/kg/day continue for 3-5 days. IV hydrocortisone 4mg/kg may be given if oral prednisone is not possible.

**Table 24.2: Treatment of child with wheeze**

| Rapid acting bronchodilator | | Oral salbutamol 3 times daily for 5 days | | |
|---|---|---|---|---|
| | | Age or weight | 2mg tablet | 4mgtablet |
| Subcutaneous epinephrine (adrenaline) (1:1,000 =0.1%) | 0.01ml/kg bodyweight | 2–12 mon (10kg) | 2 | ¼* |
| Salbutamolinhaler a spacer 750–1,000ml | 2 puffs per dose. 1 dose in10min. | 12 mon to 5 yrs.1 in (10–19kg) | | |
| Nebulized salbutamol 5mg/ml | | | | |
| Under 1 yr. | 0.5ml salbutamol in 2.0ml sterile water | | | |
| >1yr. | 1.0ml salbutamol in 2.0ml sterile water | | | |

Note:
(a) In all cases use of inhaler is better and cheaper than nebulizer or oral salbutamol.
(b) Steroids should be used early. Oral steroids are as effective as parenteral ones.
(c) When this is done aminophylline is rarely needed.
(d) Fluids should be limited to two thirds of the daily requirement.
(e) Antibiotics should be given only if there are clear signs of infection.
(f) Adrenaline is only used if use of inhaler is not possible.

- Repeat rapid acting bronchodilator (preferably salbutamol inhaler) at hourly intervals for 3 doses.
- If not improved IV aminophylline 5mg/kg can be given slowly over 20 minutes.
- Monitor vital signs every 3hrs. Signs of improvement are:
  - Less respiratory distress (easier breathing)
  - Less chest retraction
  - Improved breath sound especially in a previously quiet chest.
  - When the patient stabilizes, discharge child on inhaler or oral salbutamol.

## 24.3.1 BRONCHIOLITIS

Bronchiolitis is a lower respiratory viral infection. The most common cause is respiratory syncytial virus. Its is typically most common and severe in young infants and is often associated with secondary bacterial infections.

**Clinical Features**

- Wheeze that is not relieved by rapid acting bronchodilators.
- Hyperinflation of the chest, with increased resonance to percussion.
- Fever
- Lower chest wall indrawing, fine crackles and wheeze on auscultation
- Difficulty in feeding, breastfeeding or drinking owing to respiratory distress
- Nasal discharge, which can cause severe nasal obstruction
- Apnoea in neonates

**Treatment**

- The mainstay of management is maintenance of adequate hydration and oxygenation.
- Give oxygen to maintainSPO2 > 90
- Antiobitics - Amoxycillin 40-45mg/kg/12hr if there are features of pneumonia OR benzyl penicillin 50000 i.u and Gentamycin 7.5mg/kd every 24 hr.
- Fever more than 38°C give paracetamol.
- Maintain adequate fluid intake and encourage continued breasfeeding.
- Start feeding as soon as able to take orally, otherwise feed via nasogastric tube.
- Gentle nasal sunction to remove secretions.
- Monitor ever 6 hrs or every 3 hrs in severe disease.
- If the child fails to respond to oxygen or the condition worsen do a chest X-ray to assess for complications e.g., pneumothorax.
- Nebulization with Hypertonic saline
- Discharge when respiratory distress and hypoxemia have resolved and the infant is feeding well.



**Clinical Features**

This is a clinical diagnosis defined as increasingly severe asthma not responsive to usual drugs. Child is too breathless to feed or talk; there is severe chest retraction; tachypnoea.

**May have features of respiratory failure:**

- Altered consciousness
- Poor respiratory effort
- Silent chest
- Cyanosis

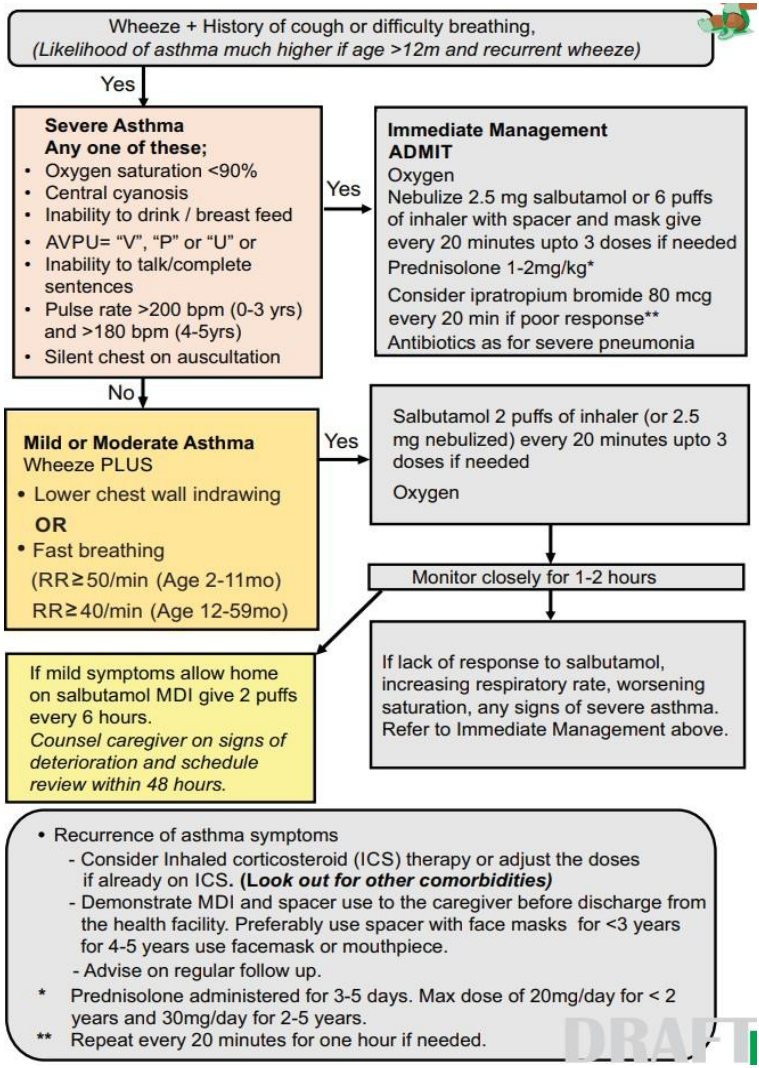
## 24.3.2 ASTHMA

Asthma is characterized by chronic airway inflammation with recurrent wheeze and good response to bronchodilators.

**It has 2 key defining features:**

- **A history of symptoms** such **as wheeze, shortness of breath, chest tightness and cough that vary over time and intensity**
- **Variable expiratory airflow limitation**

**Figure 24.3:Diagnostic Flowchart for Asthma in Clinical Practice**




Source: Kenya Basic Paediatric Protocols, 2022

**Management of Mild or Moderate Asthma**

- It is defined as wheeze wit Lower chest wall indrawing OR Fast breathing: RR ≥ 50/min - Age 2-11mo; RR ≥ 40/min - Age 12-59mo
- Administer two puffs of Salbutamol inhaler (or 2.5 mg nebulized) every 20 minutes upto 3 doses if needed Oxygen
- Monitor closely for 1-2 hours
    - If mild symptoms allow home on salbutamol MDI give 2 puffs every 6 hours. Counsel caregiver on signs of deterioration and schedule review within 48 hours.
    - If lack of response to salbutamol, increasing respiratory rate, worsening saturation, any signs of severe asthma. Refer to Immediate Management above

**Severe Asthma**

- Wheeze + History of cough or difficulty breathing with any one of these;
    - Oxygen saturation 200 bpm (0-3 yrs) and >180 bpm (4-5yrs)
    - Central cyanosis
    - Inability to drink / breast feed
    - AVPU= “V”, “P” or “U” or
    - Inability to talk/complete sentences
    - Pulse rate >200 bpm (0-3 yrs) and >180 bpm (4-5yrs)
    - Silent chest on auscultation

- Immediate Management
    - ADMIT
    - Oxygen
    - Nebulize 2.5 mg salbutamol or 6 puffs of inhaler with spacer and mask give every 20 minutes upto 3 doses if needed
    - Prednisolone 1-2mg/kg daily for 3-5 days. Max dose of 20mg/day for < 2 years and 30mg/day for 2-5 years
    - Consider ipratropium bromide 80 mcg every 20 min if poor response repeated every 20 minutes for one hour if needed
    - Antibiotics as for severe pneumonia

**STATUS ASTHMATICUS**

This is a clinical diagnosis defined as increasingly severe asthma not responsive to usual drugs.

**Cllnical Features**

Breathlessness, inability to feed or talk; severe chest retraction, tachypnoea, altered consciousness, poor respiratory effort, silent chest and, cyanosis

**Management**

- Admit
- Monitor vital signs every 15–30 minutes.
- Keep propped up in bed.
- Administer oxygen by intranasal catheter flow rate of 1–2 litres per minute.
- Treat as per acute attack of asthma (see section on asthma above).
- Look for and correct dehydration.
- Avoid antibiotics unless specifically indicated.
- Ventilate if necessary using bag and mask.

## 24.3.3 LONG-TERM AND HOME CARE OF ASTHMA

**Management of chronic asthma**

***Routine care for asthma patients***

Asthma is a chronic illness and therefore the clinical team and patients need to develop a long-term plan for the patient management. The patient – health provider partnership includes:

- Personalized education: ensure the following is included in the patient education.
  - Basic information about the disease
  - Medication including relievers and preventers.
  - Potential side effects of medicines.
  - Training on the medicine inhaler technique.
  - Recognition of worsening asthma and actions to be taken.
- Self-monitoring of asthma control.
  - If no response, consider investigation for tuberculosis.
  - Regular review to assess control and adjust treatment as may be necessary.
- Clear and preferably written instructions on how and when to use the inhaler at home.
- Regular assessment of patients for their symptom control
- Report immediately to a health facility when home treatment is ineffective
- Avoidance or reduction of triggers/allergens in the home.g.tobacco smoke, indoor or outdoor air pollution, medications including betablockers and NSAIDS.
- Child in school with exercise induced attacks should use the inhaler before exercise.

**Medication for chronic care**

- Long Acting β2 Agonists (LABA) have anti-inflammatory effects and are used in combinationwith inhaled corticosteroids (ICS) for the long-term control of asthma. Anti-inflammatory therapy (ICS) forms the backbone of asthma control.
- All asthma patients should be on inhaled corticosteroids. Bronchodilators should not be used without combining with ICS, since asthma is an inflammatory disease.

**For detailed information on Asthma Care refer to Global Initiative for Asthma - GINA (ginasthma.org).**

## 24.4 Children Presenting with Chronic Cough

Definition: Cough lasting 14 days or more

The following conditions are associated with chronic cough:

- Tuberculosis
- Asthma
- Foreign body aspiration, usually children under 5 years: Parents may not remember history of choking. Unilateral wheeze, or pneumonia with poor response to antibiotics suggests diagnosis
- HIV infection: In addition, these children have chronic chest signs with clubbing of fingers and toes but usually no cyanosis.
- Bronchiectasis: Purulent sputum, bad breath, finger clubbing.
- Lung abscess: Reduced breath sounds over affected part.
- Heart disease: Either due to congestive failure or recurrent pneumonias.

More details for respective clinical features are found in the respective sections for the diseases listed above.

**Investigations**

- HIVtest
- Mantoux test
- Chest x-ray

**Management**

Management is specific to the underlying disease.
Refer/consult as needed.

# 25. Poisoning

Accidental poisoning is common in children under 3 years of age. Usually a previously healthy child suddenly falls sick. For the older child, especially the adolescent, it may be intentional e.g a suicide attempt. Some common poisons include: Paracetamol, aspirin, pesticides (organophosphates), and kerosene (paraffin). Other poisons include drugs being taken by any member of the family.

**General Principles of Management**

Parent /caregiver is encouraged to try to identify the type of poison the child has taken. If possible, carry the container to the health facility. Do not give the child anything to drink and do not make the child vomit. The child to a health facility as soon as possible.

Note that most childhood poisoning is preventable by putting drugs and dangerous chemicals out of reach for children.

Take full history and try to identify the poisoning agent. Severe poisoning requires hospital admission for appropriate management.

**Decontamination**

- Stomach: Do not induce vomiting. A gastric lavage is possible if poison was ingested within an hour of presentation to the health facility. Activated charcoal if available can be given. Gastric decontamination is contraindicated in unconscious patients or those who have ingested corrosives or kerosene.
- Skin: Remove clothing and wash thoroughly
- Eyes: Irrigate with water or saline.
- Give specific antidote if indicated.

**Emergency and Supportive Management**

- Always have a resuscitation tray ready. Suction equipment (ambubag and mask, oxygen delivery equipment, IV fluids including dextrose; medicines – adrenaline and diazepam.
- Airway/breathing: Remove any airway obstruction. Start immediate treatment to restore or support breathing.
- Circulation: Restore circulating blood volume by giving 20ml/kg of Ringer's lactate or normal saline over 15 minutes. Repeat until return of pulse; this may be repeated up to 3 times.
- Convulsions: Give anticonvulsants if child is convulsing. Diazepam (IV or rectally) and then phenobarbital (IM) if no response to two doses of diazepam.
- Reasses every 2-3 minutes until stabilized following the same format– airway, breathing, circulation and intervene as needed.

## 25.1 Clinical Features and Specific Treatment of Common Poisonings

## 25.2 Paracetamol Poisoning

Paracetamol is a commonly used antipyretic/analgesic and is prone to accidental poisoning.

**Clinical Features**
There are four stages of paracetamol poisoning that are recognized if a child has ingested 140mg/kg or more

- Stage 1: First 24 hours – Anorexia nausea and vomiting
- Stage 2: 24–48 hours – Signs of hepatic dysfunction – jaundice,bleeding
- Stage3:72–96 hours–Peak liver dysfunction with possible hepatic encephalopathy
- Stage 4: 4 days–2 weeks – Resolution of liver dysfunction

**Investigations**
- Liver function tests
- Coagulation profile

**Management**
Treatment can only be done in hospital so admit all children.

**Treatment**
- **Activated Charcoal should be administered if ingestion is <u>less than one hour</u> and total dose ingested is greater than 150mg /kg.**
- Give N-acetylcysteine IV or oral within 8 hours of ingestion.
- Loading dose 150mg/kg in 3ml/kg 5% dextrose IV infusion over 15minutes.
- Then 50ml/ kg 5% dextrose over 4 hours. Then 100mg/kg of 5% dextrose over 16 hours.

### 25.2.1 KEROSENE (PARAFFIN)

Paraffin poisoning is the most common form of poisoning in children. Although death from Kerosene poisoning is rare, patients can develop severe respiratory complications. Clinical features depend on amount ingested and if there is aspiration. Aspiration results in coughing, gagging, chocking and respiratory distress. Severe respiratory distressmay lead to pulmonary oedema.
Absorbed kerosene may lead to CNS symptoms such as Headache, Lethargy, transient Euphoria and encephalopathy with varying degrees of altered consciousness. Cardiovascular symptom such as Syncope and Dyspnea may also result.

Other symptoms include nausea and vomiting.

**Management**

**Management is supportive as there are no antidotes**
**Admit all children and monitor as follows:**

- Vital signs
- Urine output (1–2ml/kg/hr.); catheterize if necessary
- Level of consciousness

### Supportive Management

- Chemical Pneumonitis- Oxygen supplementation.
- If arrhythmias are present, 347ssess magnesium and potassium levels and replace as appropriate.
- Watch for complications and treat accordingly.
- Activated Charcoal Not Beneficial.
- Gastric lavage, use of steroids or prophylactic antibiotics not recommended.

## 25.2.2 ORGANOPHOSPHATES

Organophophates are a diverse group of chemicals used in both domestic, agricultural and industrial settings. They include insecticides e.g. malathion and diazinon.

**Clinical Features**

Headaches, weakness, vomiting, colicky abdominal pain, profuse cold sweating, hypersalivation, muscular twitching, fasciculations, diarrhoea, tenesmus, convulsions, dyspnoea with bronchoconstriction, constricted pupils (meiosis), bilateral crepitations**.**

**Management**

- Admit all children
- Assess airway and intubate if necessary
- Assess breathing and supplement oxygen if necessary
- Obtain IV access and start IV fluids
- Decontaminate skin: Remove all contaminated clothing and wash exposed areas with soap and water, including the hair and under the nails. Irrigate exposed eyes with copious tepid water or saline
- Specific Antidotes:
  - IV atropine 0.05mg/kg over 15 min. Repeat every 15 min until bronchial sounds are fully eliminated, then maintain on SC atropine 4–6 hours x 24–48 hours.
  - Pralidoxime 30mg/kgIV infusion repeat 4 hourly, 12–24 hours depending on response.
- Refer/consult as necessary.

## 25.3 Prevention of Home Accidents and Poisoning

- Dangerous items including kerosene and drugs should be kept under lock and/or out of reach of young children.
- Medication should have child-proof caps and medication bottles should be kept tightly closed
- Avoid leaving small children unattended
- Keep household products in original bottles; never place poisonous products in food or drink containers
- Discard partially or unused medications appropriately, preferably return to a Pharmacy for proper disposal
- Do not store potentially poisonous compounds in soft drink bottles
- General Recommenations to caregivers:
    - Eye poisoning – Irrigate eye with plenty of water.
    - Skin poisoning – remove contaminated clothes and rinse skin with water
    - Swallowed poi.son – take away the item; Do not induce vomiting.

# 26. Neonate and Young Infant (0–2 Months)

## 26.1 Routine Care at Delivery

Dry the baby with a clean cloth. While drying observe breathing, muscle tone, and colour. If all normal, remove the wet cloth, wrap baby in a dry one and give to mother to initiate breastfeeding. Cover baby well to prevent over-cooling. If not breathing, initiate resuscitation. Refer to Figure 26.1 below.
For babies not requiring resuscitation do the following:

- Initiate breastfeeding as soon as possible
- Wrap in dry linen.
- Weigh the baby.
- Keep warm next to mother (skin to skin is the best way of keeping baby warm).
- Apply tetracycline eye ointment within 1 hr in both eyes and given only once.
- Examine carefully to exclude congenital malformation.

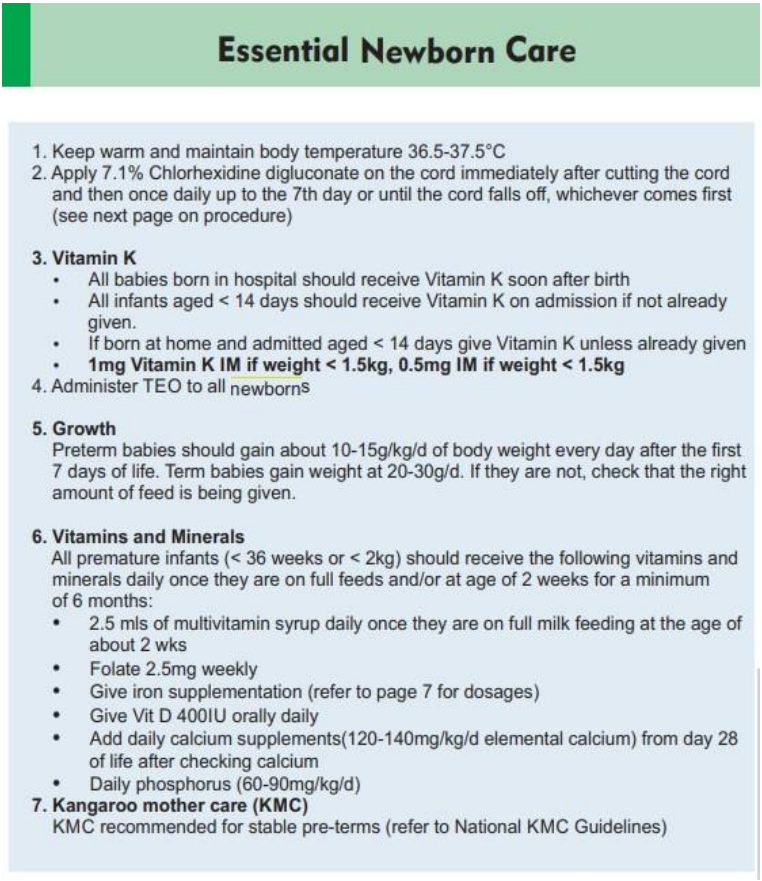
## 26.2 Postnatal Care of the Normal Newborn

**The infant should be given to the mother as soon as possible.**
**The following are important:**

- Breastfeeding should start as soon as possible to ensure good positioning and attachment
- Encourage exclusive breastfeeding (no water).
- Babies should be fed on demand at least 8–12 times in 24hours.

- HIVpositive mothers who have chosen not to breastfeed should be encouraged to cuddle their babies.
- Observe cord for bleeding and keep it clean.
- Administer OPV '0' and BCG.

**Figure 26. 1: Essential Newborn Care**




Source: Kenya Basic Paediatric Protocols, 2022

- On discharge: Counsel the mother on cord care and breastfeeding at home and tell her to bring the baby back immediately if she notices a problem,e.g., poor feeding or jaundice.

**What to Teach the Mother**

All expectant mothers should be taught about cord care. They need to know that babies often acquire infection through the cord. If they deliver in the community, cutting of the cord with clean instrument is needed. After delivery harmful practices need to be discouraged. Mothers should keep the cord dry until it drops off.



## 26.3 Neonatal Asphyxia and Resuscitation

A newborn who fails to establish regular breathing and appears blue and/or pale is likely to have asphyxia. Anticipate asphyxia in all high risk pregnancies or if there is irregular foetal heart, foetal bradycardia or tachycardia and meconium stained liquor during labour. Occasionally asphyxia occurs unexpectedly.

**All persons conducting deliveries should be able to resuscitate a baby at birth. Always be prepared to resuscitate.**

Refer to Figure 26.2 for the correct positioning of the baby's head for opening up the airway and of the caregiver's hands to cardiac massage.

**Figure 26.2: Positioning for neonate resuscitation**

Correct head position to open up airways and for bag ventilation. Do not hyper extend the neck.

**Management**

**Clinical Features**

- Assessment is best done following the ABC as in paediatric emergencies (see Figure 26.2).
- APGAR scoring (Table 26.1)can also be used for assessing the degree of asphyxia.

Correct position of hands for chest compressions in a neonate. The thumbs are used for compression of the sternum.

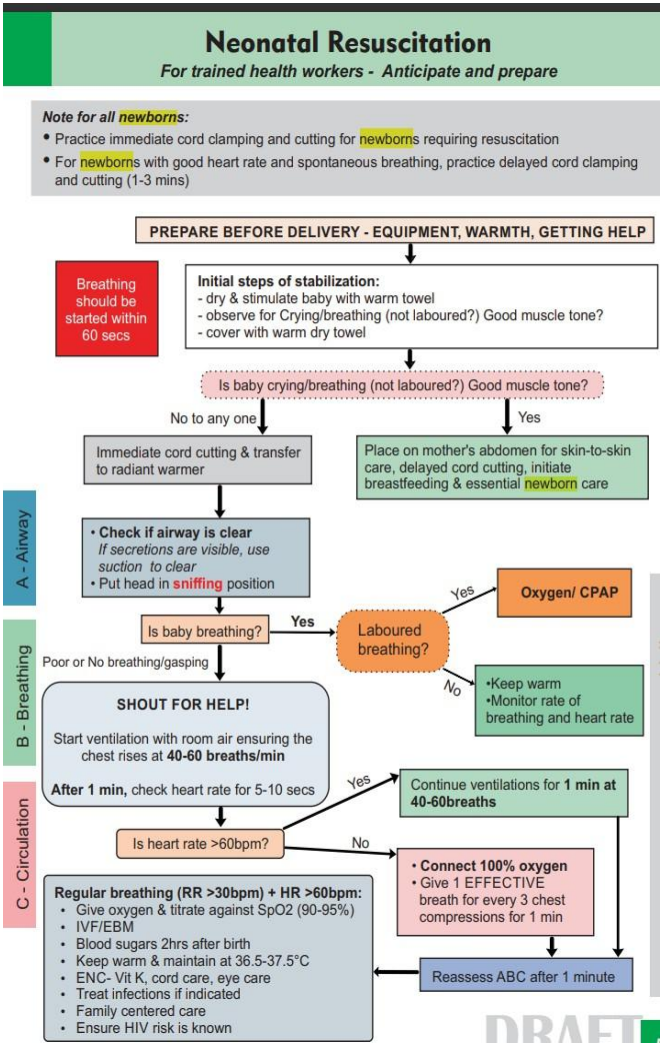
**APGAR Scoring**
A: Appearance or colour P
Pulse rate
G:Grimace or response to some stimulus A:
Activity (muscle tone)
R: Respiration

- Management is dependent on the APGAR of the baby. The management recommended at the various APGAR scores is indicated below:
  - APGAR score 7–10: None. Do not suction baby.
  - APGAR score 5–6: Give oxygen.
  - APGARscore0–4:Initiate resuscitation with bag and mask.
- If the mother had received pethidine: Give naloxone 0.01mg/kg/IV STAT.

**Table 26.1:APGAR scoring**

| Clinical features | Score | | |
|---|---|---|---|
| | 0 | 1 | 2 |
| Heart rate(per minute) | Absent | Less than100 | Over 100 |
| Respiration effort | Absent | Irregular,slow | Regular |
| Musclet one | Limp (floppy) | Some flexionof arms,legs | Well flexed, active motion |
| Reflex irritability (nasal catheter) | No response | Some emotion,grimace | Cries |
| Colour | Blue,pale | Pink body,blue extremities | Completely pink |

**Figure 26.3: ABC's of Neonatal Resuscitation**




Source: Kenya Basic Paediatric Protocols, 2022

Harmful practices in a baby who is not breathing include slapping the baby, holding baby upside down, and pouring cold water on it. Such practices should be avoided. The act of drying the baby is enough stimulation.
After resuscitation refer the baby to a facility that can deal with complications. Keep the baby warm throughout the journey by using Kangaroo Mother Care.

**Complications**
The following complications are known to occur:
- Convulsions
- Apnoea or irregular breathing
- Respiratory distress
- Poor feeding
- Floppiness
- Cerebral palsy if still neurologically abnormal at 1 week of age.

**Admit all babies with complications.**



## 26.3.1 MANAGEMENT OF COMPLICATIONS

- Convulsions:
  - These tend to be atypical and therefore easily missed. They may involve the face (e.g., chewing movements, facial twitches), or twitching of the limbs. They can partial or generalized. Most often they appear within 24 hours of birth.
  - Treatment: Give IM phenobarbitone. Loading dose 20mg/kg/dose. Maximum for 24 hours 30mg/kg. Always give maximum dose before giving another anticonvulsant if the convulsions are not controlled. Addition of phenytoin may sometimes be necessary given at 15mg/kg IV.
- Breathing problems:
  - Give oxygen as needed.
- Feeding:
  - Use NG tube if needed and baby is able to tolerate enteral feeds. Otherwise give IV fluids.
- Floppiness and neurological damage:
  - Start physical therapy as soon as baby stabilizes. Mother to be shown how to feed the baby, how to and stimulate and do passive movements. This may need to be continued after discharge(see cerebral palsy). Counsel the mother all the time. She needs a lot of support and understanding.

## 26.4 Birth Injuries

**Difficult deliveries may lead to birth injuries**

**Clinical features**

- Common injuries requiring no treatment include:
  - Caput succedaneum – Oedema over presenting part.
  - Massive oedema of scalp.
  - Conjunctival haemorrhage.Subgaleal/aponeurotic haemorrhage–Fluctuant swelling on the head not limited by suture lines. Can be extensive to cause anaemia and jaundice.
  - Cephalohaematoma–Firm but fluctuant swelling limited by suture lines. Takes very long to resolve.
- Nerve injuries
  - Erb'spalsy–Injury to the upper roots of the brachial plexus: Affected limb held extended at the elbow and forearm pronated.
- Fractures
  - Clavicle–Mother notes the baby cries on being lifted and after a fewdays swelling along the affected clavicle.
  - Femur or humerus–Affected limb swollen and very painful on movement. There is pseudoparalysis.
- **Less common but serious injuries** include the following:
  - Intracranial:Can be subdural or intracerebral haemorrhage. Baby is lethargic with signs of raised intracranial pressure and may have convulsions.
  - Intrathoracic: Presents with respiratory distress.
  - Intrabdominal: Usually ruptured liver either subcupsular or haemoperitoneum. If severe, baby shows features of hypovolaemic shock without obvious evidence of external bleeding; consider intrabdominal haemorrhage.

**Investigations**

- Full blood count if there is pallor
- X-ray of affected limb
- Ultrasound for cranial or abdominal injuries

**Management**

- Caput succedaneum: massive oedema of scalp: Do not need any special treatment.
- Severe scalp bleed: Requires no specific treatment; never aspirate as this predisposes to infection. If anaemia is severe, transfusion may be needed.
- Nerve injuries: include Klumpke's Paralysis, Erb's Palsy and Facial Nerve Palsy. Rest the baby for a few days then start passive movements. Most injuries will recover fully. Inform the mother and ask her to lift the baby carefully in order to prevent further injury. Involve an occupational therapist or physiotherapist.
- Fractures:Align the affected limb and immobilize. Usually healing occurs within 3weeks.
- Intracranial haemorrhage: Refer for drainage if subdural is present.
- Intrathoracic and intrabdominal injuries: Refer for surgery.

## 26.5 Born Before Arrival(BBA)

This is when a baby is born either at home or on the way to the health facility. During such times, most mothers will not have had a skilled attendant at delivery. Sometimes when the delivery was at night there may be several hours before presenting to the health facility.

**Management**
Weigh the baby and assess for danger signs (see below). Then do the following, if the baby is stable:

- Keep baby warm if cold.
- Ensure the cord is properly clamped and not bleeding.
- Do a thorough physical examination.
- Clean the cord with chlorhexidine.
- Apply 1% tetracycline ointment in both eyes once.
- Initiate breastfeeding unless the baby is unable to breastfeed.
- Treat any underlying condition



## 26.6 Organizing Care of Sick Baby 0–2 Months

**All small babies should not wait in the queue**

— Arrange for babies to be attended quickly
— Assess baby for danger signs before general administrative procedures
— Manage the danger signs

### 26.6.1 DANGER SIGNS AND THEIR MANAGEMENT

Place the baby in a warm environment, weigh the baby, establish IV access and manage accordingly:

**RESPIRATORY DISTRESS AND APNOEA**

- Not breathing (apnoea) or gasping (respiratory rate<20/minute):Start resuscitation immediately.
- Respiratory distress–rate>60/minute, chest retraction, grunting, central cyanosis: Give oxygen by nasal prong or nasal catheter.

**SHOCK**

Shock can be due to severe blood loss at birth, dehydration through failure to feed, vomiting or diarrhoea. Dehydration is covered in Section18, on diarrhoea. For the baby who has lost a lot of blood there will be severe pallor in addition to signs of shock. Signs of shock include cold hands and feet; capillary refill>3 seconds (this may be difficult to elicit in a baby with severe blood loss because of severe pallor); altered consciousness.

For both causes restore circulating blood volume by giving normal saline or Ringer's lactate at 20ml/kg intravenously as rapidly as possible. Reassess and if

still in shock repeat the dose. For the baby that has bled, get blood as quickly as possible and transfuse.

**ALTERED CONSCIOUSNESS AND /CONVULSIONS**

These could be due to serious bacterial infection, birth asphyxia, neonatal tetanus or bilirubin toxicity. Establish the cause through history and treat accordingly. Control convulsions using phenobarbital preferably IM 20mg/kg.

**INABILITY TO BREASTFEED**

Causes include: serious bacterial infection, birth asphyxia, or low birth weight (preterm baby). Give dextrose 10ml/kg IV or nasogastric tube to prevent or treat hypoglycaemia immediately. This can be followed by giving breast milk as soon as possible according to the condition of the baby.

**VERY OR EXTREMELY LOW BIRTH WEIGHT**

Refer or admit urgently for specialized care. If referring, "Kangaroo Mother" position can be used to keep baby warm during the journey; pass a nasogastric tube and give expressed breast milk to prevent hypoglycaemia.

All babies with danger signs will need admission to a unit that can treat them. Transfer by the quickest means available preferably by ambulance so that you can administer oxygen if the baby has breathing problems.

## 26.7 Serious Bacterial Infections and Meningitis

**Clinical Features**

There may be history of maternal fever, prolonged rupture of membranes, and foul smelling amniotic fluid. There may be danger signs and the infant may also have deep jaundice, abdominal distension, or extensive septic skin lesions.

**Note: Up to 30% of neonates with late onset sepsis will have meningitis without the obvious features of bulging fontanelle or neck stiffness.**

**Investigations**

- Full blood count; ask for a blood film and count of immature neutrophils. An immature to total ratio of >0.2 signifies infection.
- C-reactive protein (CRP) if available very useful for early diagnosis
- Blood culture
- Lumbar puncture
- Pus swab of any obvious septic area, e.g., umbilicus, skin
- Blood sugar
- Other investigations as required

**Management**

Admit the child.

**Supportive Care for All Babies**

- Thermal environment:
  - Keep dry and well wrapped; you may need extra heat either heating the room or keeping baby in incubator according to size of the baby (minimum room temperature 26°C).
- Fluid/nutrition:
  - Encourage breastfeeding if the baby is able or otherwise feed by tube. Volumes will depend on baby's weight and age.
  - If not able to tolerate enteral feed, then give IV fluid. On day1give 10% dextrose. Thereafter give maintenance electrolytes. Parenteral nutrition should be considered if baby is starving for longer than 3–4 days.
- Oxygen therapy
  - Give oxygen by nasal prongs or nasal catheter as needed. Do pulse oxymetry to monitor saturation. Oxygen can be discontinued once baby has saturations >90% in room air.
- High fever:
  - Avoid antipyretics; control the environment instead. Uncover the baby for short period then cover. If baby is in incubator reduce temperature.
- Convulsions:
  - Control if present; see section under convulsions

**Management–Specific**

- Give IV penicillin and gentamicin; use flucloxacillin instead of penicillin if there are skin lesions.
- If not improving in 2–3 days, change to secondline: ceftazidime or ceftriaexone with amikacin ***OR*** according to sensitivity of isolated organism
- Duration of therapy: depends on response can be 7–14 days of parental therapy. For meningitis treat for 21 days.

## 26.7.1 COMPLICATIONS OF NEONATAL MENINGITIS

**The following neurological sequelae occur:**

- Hydrocephalus
- Blindness
- Mentalretardation
- Hearing loss
- Motor disability
- Abnormal speech patterns

## 26.7.2 OTHER INFECTIONS

- Skin:
  - May have few to extensive septic skin lesions.
  - If few lesions, they tend to occur in flexures and are easily missed.
  - If few lesions, treat as outpatient with either amoxicillin or cloxacillin.
  - Admit if lesions are extensive;treat as for serious bacterial infection.
- Eye infection:
  - Treat with tetracycline eye ointment for 5 days.
- Umbilical sepsis:
  - Presents with pus discharge, foul smell and redness around the umbilicus.

- If child has no systemic signs treat as outpatient:

♦ Clean the umbilicus with antiseptic and show mother how to clean at home.
♦ Review baby after 5 days or earlier if systemic signs develop.

- If there is periumbilical redness, the baby needs to be treated with antibiotics. Give amoxicillin 50mg/kg/day for 5 days. If baby has systemic signs, refer for admission.

**Prevention**

**The following preventive measures are important:**

♦ Increased and improved prenatal care.
♦ Clean and atraumatic delivery.
♦ Regular cleaning and decontamination of equipment.
♦ Sound hand-washing principles by all personnel handling babies.
♦ Regular surveillance for infection.
♦ Early exclusive breastfeeding.

## 26.8 Respiratory Distress

Respiratory distress occurs when there is failure to maintain adequate exchange of oxygen and carbon dioxide by the lungs for a variety of reasons. It is characterized by: Respiration rate of 60/minute or more (tachypnoea), expiratory grunt, chest or subcostal recession, cyanosis, and flaring of alae nasi. The causes of respiratory distress include:

♦ Respiratory distress syndrome (RDS),
♦ Neonatal sepsis (Pneumonia)
♦ Aspiration of meconium or feeds,
♦ Transient tachypnoea of newborn,
♦ Congenital heart disease
♦ Congenital anomalies of the oesophagus, airways or diaphragm.

**Clinical Features That May Assist in Diagnosis**

♦ Respiratory distress syndrome (RDS) is most common in premature babies, but can occur in infants of diabetic mothers and following caesarian section.

♦ Neonatal sepsis: may be suspected with a history of prolonged rupture of membranes (more than12 hours) and maternal fever, offensive liquor, or vaginal discharge. These are features of sepsis in the mother.
♦ Meconium aspiration: Meconium stained liquor and staining of skin, nails, and cord.
♦ Transient tachypnoea of newborn: Difficult to differentiate from RDS but usually in term/near term babies. Resolves within 24 hours.
♦ Cardiac lesion: May or may not have murmurs depending on the defect.

**Investigations**

♦ Full blood count
♦ Bloodculture

- Chest x-ray
- Special tests according to suspected problem

**Management**
- Admit.
- Treat danger signs if present.
- Supportive therapy as in sepsis.
- Antibiotics: An infection cannot usually be excluded.
- Note: A baby who has pneumonia that does not respond to usual antibiotics could be having Chlamydia trachomatis infection. If this is so, the baby will respond to Erythromycin 50mg/kg/day given 6-8 hourly for 14 days.
- Reevaluate or consult if not improving within 2–3 days of treatment.
- Refer to specialists as needed to deal with any complex problem for further management.



## 26.9 Apnoeic Attacks

These are attacks caused by cessation of breathing for more than 10 seconds, or less than 20 seconds if accompanied by bradycardia. Apnoeic attacks are most commonly due to prematurity, but may accompany sepsis, hypoglycaemia, hypoxaemia, hypothermia, hyperthermia.

**Clinical Features**
Apnoea, bradycardia and cyanosis. Features of the predisposing condition.

**Investigations**
- Screen for sepsis–full blood count blood culture
- Blood glucose levels

**Management**
- Reestablish breathing by gentle stimulation. If poor response ventilate using bag and mask.
- For apnoea of prematurity give caffeine citrate 20mg/kg orally or IV over 30 minutes. If caffeine is not available, use IV aminophylline 10mg/kg over 15–30

minute. Maintenance doses should be given at 5mg/kg/day. Avoid rectal aminophylline; it may not achieve therapeutic levels. In all cases monitor heart rate.
- IV fluids according to the daily needs.
- Avoid oral feeding to prevent aspiration
- Treat the cause if known.
- If frequent give continuous oxygen by nasal catheter. Mechanical ventilation may be useful for frequent apnoeic attacks.
- In recurrent cases continuous positive airway pressure (CPAP)may be useful. This can be done in a neonatal intensive care unit(NICU)
- Monitor frequently.

## 26.10 Low Birth Weight and Preterm Infant

**Definitions**

- Low birth weight: Weight less than 2,500g at birth.
- Very low birth weight: Weight below 1,500g at birth.
- Extremely low birth weight: Weight below 1,000g at birth.
- Preterm: An infant born before 37 completed weeks of intrauterine life at birth.

**Problems Associated with Prematurity**

- Poor thermal regulation, hypothermia
- Respiratory problems: RDS, apnoeic attacks
- Feeding problems leading to hypoglycaemia
- Infections
- Hyperbilirubinaemia
- Anaemia of prematurity
- Congenital malformations

**General Management**

- Babies with weight 2,000–2,499g can be cared for as normal weight babies. Some of them may have feeding difficulties. Observe for a day or two before discharging from maternity ward.
- Babies of weight 1,750–1,999g need extra care. Kangaroo Mother care will provide enough warmth unless the baby has another problem. They should be able to breastfeed adequately, but some may tire quickly and may need tube or cup feeding.
- Babies with weight below 1,750g are at increased risk of respiratory distress, infection, apnoea, and hypothermia, and are usually not able to feed especially if very low birth weight. They need to be admitted to a specialized area that will cater for their needs. For these babies treat any intercurrent problem and when they stabilize, start Kangaroo Mothercare.

**Thermal environment**

- Keep baby dry and well wrapped and nurse away from open windows.
- Avoid unnecessary exposure.
- Keep the room warm (at least 25$^{o}$C).
- Kangaroo Mothercare.
- Incubators

**Note: Incubators are extremely expensive and thus not always available but useful for care of very sick babies needing oxygen and IV fluids. Kangaroo Mother care (KMC) is cheap and easy to carry out in many facilities. Use KMC when you have a stable LBW baby irrespective of weight**

**Kangaroo Mother Care (KMC)**
**KMC consists of:**

- Kangaroo position–Skin to skin contact between mother's breasts or those of any other adult female.
- Breastfeeding.

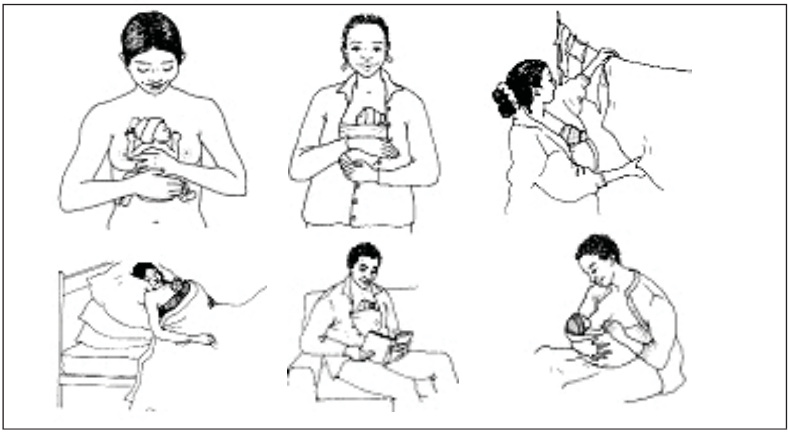
- Follow up to ensure adequate growth and development.

**Procedure for KMC (see Figure 26.1):**

- Mother wears a dress that opens at the front.
- Baby wears nappy/diaper, cap, and socks.
- Let the mother sit comfortably on a chair.
- Mother opens the dress.
- Place the naked baby in frog like posture on mother's chest between her breasts.
- Secure baby firmly but not too tight with a cloth round mother and baby.
- Breastfeed frequently. Top up with cup if baby is not able to suck adequately.
- Mother in recliner position during rest and sleep.

**Monitor growth at least 3 times per week.**

**Figure 26.4: Kangaroo mother care**



**Fluid and Feed Management**

- Encourage mother to breastfeed frequently if baby is able. Check positioning and attachment.
- Ensure adequate intake by calculating the requirement per day.
- Record all intake (oral and IV) and check every 6hrs to see if the desired intake is achieved.
- Feeding should be done within the first hour of birth to avoid hypoglycaemia.

- Introduce feeds as soon as possible; preferably no later than 24 hours after birth. Begin with 3ml for infants<1,500g and 6ml for those>1,500g. Increase by the same volume until the required volume for the day is reached. For infants on IV fluids, reduce gradually so that the total intake per day does not exceed daily requirements.

- Calculation of feeds/fluids: Start with 60ml/kg/day on day1. Increase by 20–30ml/kg per day to a maximum of 180–200ml/kg/day if using breast milk. For formula or IV fluid do not exceed 180ml/kg. Refer toTable 26.2 for amounts.
- Give micronutrients:
  - Multivitamins a preparation containing 400IU of vitamin D as soon as enteral feeding is established.
  - Iron supplement 6mg/kg/day after age of 4 weeks.
- Monitor weight atleast 3 times a week. Weight gain after the first week is 15g/kg/day.

**Table 26.2: Feeding chart for preterm and low birth weight babies:**

| Amount of milk to give every 3 hours (ml) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Birth weight** | **Age in days** | | | | | | | |
| (KG) | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 ormore |
| 1.0–1.4 | 8 | 10 | 15 | 20 | 25 | 30 | 30 | 35 |
| 1.5–1.9 | 10 | 15 | 20 | 25 | 30 | 40 | 45 | 50 |
| 2.0–2.4 | 15 | 20 | 30 | 35 | 40 | 50 | 55 | 65 |
| 2.5–2.9 | 20 | 25 | 35 | 40 | 50 | 60 | 70 | 75 |
| 3.0–3.4 | 20 | 30 | 40 | 50 | 60 | 70 | 70 | 75 |
| 3.5–3.9 | 25 | 35 | 45 | 60 | 70 | 80 | 80 | 80 |

Note: Introduce feeds as soon as possible; preferably no later than 24 hrs after birth. Monitor weight at least 3 times a week. Weight gain after the first week is 15g/ kg/day.

## 26.11 Anaemia of Prematurity

**This refers to anaemia occurring after the first week and often much later. It is due to a number of factors which include:**

- Deficiency of haematinics.
- Blood loss associated with repeated investigation.
- Intracranial haemorrhage.
- Erythropoietin deficiency.

**Management**

- Treat with iron and folic acid.
- Transfuse if:
  - Symptomatic: Poor weightgain, recurrent apnoea, congestive cardiac failure, or
  - Hb<8g/dl

**Prevention**

Limit blood loss; give prophylactic iron starting from 4 to 6 weeks of age.

# 26.12 Infants of Diabetic Mothers

**Clinical Features**

Size at birth will depend on the degree of diabetic control in the mother as well as the stage of foetal development. Hence the baby may be large, appropriate, or small for gestation.

**Complications**

**These include:**

- Perinatal asphyxia and injury,
- Hypoglycemia (most likely in babies who are either large or small fortheir gestation age),
- Hypocalcaemia,
- Hyperbilirubinaemia,
- Respiratory distress syndrome (RDS),
- Polycythaemia
- Feeding problems.

**Investigations**

- Blood sugar
- Bilirubin if indicated
- Haemoglobin or haematocrit if plethoric
- Others as indicated

**General Management**

**Diabetic mothers should deliver in hospital, where problems of the baby can be dealt with. Appropriate management of such mothers include:**

- Close cooperation between obstetrician and paediatrician.
- Maintenance of normoglycaemia in the mother [see diabetes in pregnancy].
- Decision on timing of delivery is made in consultation with the obstetrician.
- During delivery:
  - Manage as for routine care of all babies.
  - Obtain cord sample for blood sugar.
- In nursery:
  - Feed within an hour of delivery and then 3-hourly.
  - Monitor blood sugar at admission and then 3-hourly for 24 hours.
- Treat hypoglycaemia:
  - If blood glucose remains low (blood sugar <2.2mmol/L) despite feeding, establish an IV line and give 2ml/kg of 10% dextrose over 5 minutes and continue with 10% dextrose at the volume requirement per day. Repeat blood glucose after 30 minutes. If stabilized, then measure sugar 3-hourly. When the baby's blood glucose is normal on 2 or more readings, gradually reduce the infusion as you increase the feeds.
- Treat hypocalcaemia: Calcium levels should be determined at 6,12,24,and 48 hours if possible.

- If hypocalcaemic (serum calcium<7mg/dl),give 1-2 ml/kg of 10% calcium gluconate IVslowly.

♦ Treat anaemia: Check haematocrit levels at 1 and 24 hours.
- If haematocrit>65% do partial exchange transfusion 10–20 ml of fresh plasma/kg.

♦ Treat hyperbilirubinaemia: Estimate serum bilirubin levels at 24 and 48 hours.
- If bilirubin elevated, treat as needed (see Section 26.13, Neonatal Jaundice).

♦ Refer if congenital malformation(s) is/are present.

## 26.12.1 DISORDERS OF GLUCOSE METABOLISM

Hypoglycaemiais a common problem but there are no specific clinical features. Hypoglycemia should be suspected in low birth weight infants, infants born small for gestational age, infants of diabetic mothers, and any sick infant especially if the infant is not feeding well.

### NEONATAL DIABETES

This is rare but responds to continuous insulin infusion 0.02–0.125units/kg/hr, adjusted according to blood glucose levels. It usually resolves within 4–6 weeks.

### HYPERGLYCAEMIA IN PRETERMS

Usually iatrogenic when glucose infusions exceed 10mg/kg/hr. Baby develops polyuria and rapidly becomes dehydrated.

**Management**

♦ Reduce infusion rate to 6–8mg/kg/hr.
♦ Monitor blood sugar 3-hourly.
♦ Rarely insulin therapy, as in neonatal diabetes, may be needed.

## 26.12.2 HYPOGLYCAEMIA

There are no specific clinical features. Ideally all at risk neonates should have regular (3 hourly) blood sugar monitoring especially in the first 24 hours of birth. This condition is common in:

♦ Low birth weight infants
♦ Small for gestational age
♦ Infants of diabetic mothers
♦ Any sick infant especially if not feeding

**Prevention**

♦ Ensure early and adequate feeding for all babies.
♦ Give IV 10% dextrose 5ml/kg and maintain feeds/IV fluids to maintain normal glycemic levels. Repeat blood sugar levels in 30 minutes if still hypoglycaemic. Repeat the dextrose bolus. Continue feeding /IV fluids to help maintain normal levels.

# 26.13 NEONATAL JAUNDICE

Refer to Figure 26.5 for a guide to the assessment of neonatal jaundice.

## 26.13.1 PHYSIOLOGICAL JAUNDICE

Many babies have some jaundice in the first week of life. This is referred to as physiological jaundice and has the following characteristics:

- Appears on about the third day.
- Reaches peak levels 5–8mg/dl(85–135mmol/L) occur in term babies; reduces to normal in about a week.
- Reaches peak levels of 10–12mg/dl(170–205mmol/L)in preterm babies;falls to normal about 10 days.

**Serum bilirubin levels >12mg/dl in term babies and >15mg/dl(>255mmol/L) in preterms require investigation.**

**Management**

If a mother notices that her baby is yellowshe should bring the baby to a health facility as soon as possible for assessment. If jaundice is physiological, only observation is required. Ensure adequate feeding and hydration.

## 26.13.2 ACUTE NON-PHYSIOLOGICAL JAUNDICE

**This is common and is caused by:**

- ABO incompatibility: Mother group O, baby is A or B or AB
- Rhesus incompatibility: Mother Rh-negative, baby Rh-positive
- Sepsis

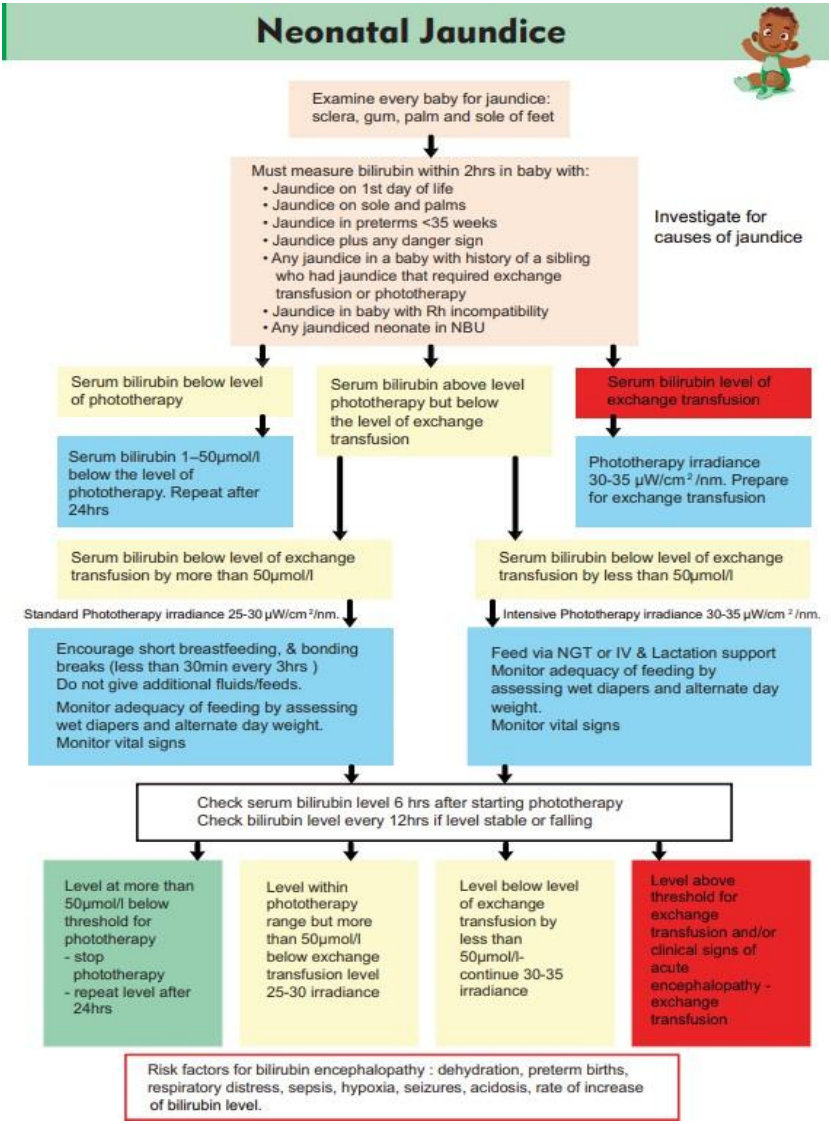
In ABO and Rhesus incompatibility, jaundice may appear from the first day, whereas in sepsis it may appear any day. It is most likely in babies who are large or small for their gestational age**.**

**Complications**

Bilirubin toxicity (Kernicterus): This is brain damage due to deposition of bilirubin in the brain. It presents with lethargy, poor feeding and vomiting, opisthotonos, seizures, and coma. Death may result from bilirubin toxicity. If the baby survives, mental retardation, cerebral palsy, hearing loss, and learning disorders are known sequalae. Factors that predispose to development of bilirubin toxicity include:

- Sepsis
- Prematurity
- Acidaemia
- Hypothermia
- Hypoglycaemia

**Figure 26.5: Assessment of neonatal jaundice**




Source: Kenya Basic Paediatric Protocols, 2022

**Investigations**

- Full blood count include peripheral blood film (PBF)
- Determine mother's and baby's blood group(s)
- Serum bilirubin levels; direct and indirect
- Appropriate cultures if sepsis suspected
- Coomb'st est

**Management**

All jaundiced babies with Rhesus incompatibility should be started on phototherapy. Exchange transfusion is indicated for high bilirubin levels as per the normogram in the basic paediatric protocols. The exchange transfusion should be carried out over 45–60 minutes period using aliquots of 20ml of blood in and out for term normal weight babies and 5-10ml for sick and premature infants. The goal should be an exchange of approximately twice the blood volume of infant (2x85ml/kg). Ensure aseptic environment. Refer to normograms in the basic paediatric protocols.



## 26.13.3 PROLONGED NEONATAL JAUNDICE

Prolonged neonatal jaundice is due to hepatitis or biliary obstruction. In obstructive jaundice the stools are pale and urine very dark. Hepatitis may be due to Hepatitis B viral infection, congenital syphilis, or cytomegalovirus, among other causative organisms. The baby may show features consistent with the specific infection.

**Investigations**

- Bilirubin
- Test for syphilis
- Hepatitis B surface antigen
- Serum transaminases
- Alkaline phosphatase
- Abdominal ultrasound

**Management**

Refer to a specialist urgently. For biliary atresia, surgery is best done within 6 weeks of birth to prevent hepatic damage.

# 26.14 Congenital Anomalies

## 26.14.1 HYDROCEPHALUS

This is an increase in the volume of cerebrospinal fluid (CSF) within the ventricular system and may be communicating or non-communicating.

**Clinical Features**

- Head enelargement
- Prominent dilated scalp veins
- Wide buldging fontanelle
- Cracked-pot sound on percussion (Macewen"s sign)
- Wide suturs (Sutural diastasis)
- Nystagmus is common and transillumination is positive later
- Other defects may be noted

**Investigations**

- Skull x-ray is useful
- Cranial ultrasound
- CT scan where possible
- Screen for congenital infections if necessary

**Management**

In order to prevent brain damage, early evaluation and diagnosis is essential. The baby therefore needs to be referred as soon as possible to a specialized unit.

**Management – Operative (Specialized Neurosurgical)**

- A shunt from the ventricle to the peritoneal cavity is inserted in a specialized centre.

## 26.14.2 NEUROTUBE DEFECTS

**Clinical Features**

These are the commonest CNS anomalies. The defect can occur in any part of the CNS starting from the head and down the spine. The abnormalities vary from the extreme anencephaly, through encephalomyelocoeleand encephalocoele, to spina bifida with or without myelocoele or meningomyelocoele.

### CRANIAL DEFECTS

Anencephaly is the complete absence of the brain apart from the brain stem, while encephalocoele and encephalomyelocoele are most commonly occipital but can be frontal.

## SPINE DEFECTS

**Spina Bifida**

This results from failure in the development of vertebral arches and is frequently associated with mal-developmentof the spinal cord and membranes. There are two main types: Spina bifida occulta and spina bifida cystica.

**Spina Bifida Occulta**

Many cases are asymptomatic and are undiagnosed. There maybe tell-tale signs on the back such as lipoma, dimple, tuft of hair (hypertrichosis), naevus, and telangiectasia. In other cases, the patient may present with nocturnal enuresis, foot-drop, persistent urinary tract infections due to neurogenic bladder, and recurrent meningitis due a communicating dermal sinus.

**Investigations**

- X-ray of full spine will show absent lamina on one side orbilaterally.
- Myelogram may be useful to rule out associated conditions such as diastematomyelia.
- A CT scan can also show the associated anomalies.

**Management**

This focuses on any complication noted. Excision of a communicating sinus is important in the prevention of recurrent meningitis.

**Spina Bifida Cystica**

In addition to the defect in the spine, there will be an obvious mass on the back. This may be a ameningocoele (a bulge of the meninges usually covered with skin), or meningomyelocoele (a bulge of the meninges that contain neural tissue). As a consequence, there is paralysis below the level of the lesion with or without incontinence of stool and/or urine.

**Investigations**

- Cranial ultrasound or CTscan
- Abdominal ultrasound to exclude intrabdominal anomalies especially the kidney
- Echocardiogram if indicated

**Management**

Management requires a multidisciplinary team approach including surgeons, paediatrician, and physical therapists. The patient should therefore be referred to a specialized centre for care. Appropriate sterile dressing of open lesions is necessary to prevent infection. Counsel the parents carefully so as to accept the child and be aware of what can be done.

**Prevention**

Pre-pregnancy folate supplementation is known to reduce chance of recurrence.

## 26.14.3 CLEFT LIP AND PALATE

- Cleft lip results from abnormal development of the medial nasal and maxillary processes.
- Cleft palate results from a failure of fusion of the two palatine processes. These again may be unilateral, bilateral, or median.
- Cleft lip and cleft palate may occur singly or in combination.
- Cleft lip and palate may also be part of syndromes such as Trisomy 15 or 18. In this case it is almost always associated with multiple congenital anomalies, with the prognosis depending on the associated anomalies. Effects on functions/ complications include:

**Clinical Features and Complications**

In this case it is almost always associated with multiple congenital anomalies, with the prognosis depending on the associated anomalies. Effects on functions/ complications include:

- This may present as unilateral, bilateral, or median cleft lip. The clefts may be complete or incomplete.
- Sucking and swallowing are greatly affected. This predisposes a child to malnutrition.
- Speech development is impaired.
- Hearing may be impaired because of recurrent acute or chronic otitis media.

**Management**

Counsel the care giver and family that the defects can be repaired and explain when repair will be done. If part of a syndrome counsel the care giver clearly with respect to the implications of the associated anomalies.

**— Refer all children with cleft lip to a specialist.**

**Timing of repair**

Operations for cleft lip may be done soon after birth or between 6 to 12 weeks. Cleft palate repair is best at 12–15 months. If repair is delayed it is important to ensure adequate nutrition. The baby with isolated cleft lip may be able to breastfeed but one with bilateral cleft lip and palate has difficulties in swallowing. Teach the mother how to feed the baby without choking using appropriate feeding devices.

Isolated cleft palate can be fitted with a prosthesis while waiting.

**Purpose of treatment**

The aim of treatment is to prevent or diminish complications and hence achieve:

- Normal appearance
- Well aligned teeth
- Normal sucking and swallowing
- Normal speech and normal hearing.

## 26.14.4 TRACHEOESOPHAGEAL FISTULA (TOF)

This is an anomaly in the development of the oesophagusin which there is usually a proximal atresia with a distal tracheoesophageal fistula. *This condition is an emergency*. It must be diagnosed within the first 24 hours of birth. Diagnosis is best done before the baby is fed to prevent aspiration of feeds.

**Clinical Features**

- Tracheoesophageal fistula is suspected:
  - When there is history of polyhydramnios.
  - When saliva drools continuously from the mouth.
  - Where there is respiratory distress
- For such a baby exclude TOF before feeding is initiated. If feeding is inadvertently started in such a baby:
  - Attacks of coughing and cyanosis (choking) are likely to occur.
  - The abdomen is likely to be distended especially at the epigastrium (due to swallowed air in the stomach).

**Investigations**

Insert nasogastric tube and with a tube in-situ, do x-ray that includes the neck, chest, and abdomen.

**Management**

**Once diagnosis is made, do the following:**

- DO NOT feed the baby enterally.
- Keep the baby warm.
- Institute intermittent suction/continuous drainage using the N/G tube to clear the secretions from the pouch.
  - Turning the baby to the side if possible to facilitate drainage.
  - Placing the baby in the head-up position to prevent gastric juice reflux.
  - Initiating intravenous infusion with 10% dextrose solution.
- Arrange for urgent transfer to a specialist centre that is equipped for this type of operation.
- Transfer the baby under the above circumstances. It is important to communicate on telephone with the respective surgeon before any movements are made.

Note: There are certain congenital abnormalities that are commonly associated with Tracheoesophageal fistula (TOF). These are vertebral, anal, trachial-oesophageal, and renal abnormalities, generally referred to as Vater syndrome.

Surgery may be carried out immediately after birth in a well baby. In some other cases gastrostomy is necessary to allow time for correction of intercurrent conditions. Adequately counsel the parents or guardians with respect to this.

## 26.14.5 ANORECTAL MALFORMATIONS

### ANAL ATRESIA (IMPERFORATE ANUS)

**Clinical Features**

This is when the child is born without an anal opening. This should be detected during the routine examination of a newborn. The mother may also report failure of the baby to pass stool. Congenital abnormalities are frequently multiple; a careful general examination of the baby is an important prerequisite.

**Investigations**

- It is urgent and important to determine whether the abnormality is high or low. Do an x-ray (Invertogram) 6 hours after birth (air has collected in the large intestine).This x-ray may have to wait for 24 hours for rectal gas to collect.
- Procedure for doing the Invertogram:
  - Strap a coin on the site of anus.
  - Hold the infant upside down for 3–5 minutes.
  - Put the thighs together and parallel to one another.
  - Take a radiograph and measure the distance between the metal coin and the shadow in the rectum. If the distance is over 2.5cm the abnormality is high; or draw a line on the radiograph from the tip of coccyx to the pubic crest (pubo-coccygeal line). If the gas shadow is above the line, the abnormality is high.

**Management**

**For high abnormalities, do the following:**

- Nasogastric suction
- Intravenous fluids
- Keep baby warm
- Refer to a specialized centre for surgery.

**Low abnormalities are easy to diagnose, simple to treat, and the outlook is good. There are 4 types of low imperforate anus:**

- The *stenosed anus:* The opening is in the normal position but very minute. The first treatment is careful dilatation with well lubricated dilators and there after digital dilatation. The mother is taught how to dilate the anus.
- The *ectopicanus:*The anus is situated interiorly and opens into the perineum in boys or vagina in girls.
- A careful search will reveal the low subcutaneous opening. This should be distinguished from the high vaginal opening or fistulae. The treatment is a pull through operation.
- The *covered anus:* The treatment is as for stenosed anus
- The *membranous anus:* Treatment is a cruciate incision.

# 27. Ear, Nose, and Throat Conditions

## 27.1 Acute Otitis Media

This is an acute inflammation of the middle ear, usually suppurative, occurring after an upper respiratory tract infection, rhinitis, or sinusitis. The commonest organisms are Streptococcus pneumoniae and H.influenzae.

**Clinical Features**
Acute Otitis Media is most common in children under 5 years. There is pain in the ear, loss or impairment in hearing, with or without ear discharge. There is also loss of appetite and fever. Examination shows signs of URTI, fever, and hyperemic oedematous tympanic membrane with loss of normal contours. Purulent discharge with perforation (central) may be present.

**Complications**
**These include:**
- Mastoiditis
- Meningitis

**Management**
**This includes:**
- Analgesics: Paracetamol 10mg/kg 8 hourly for 5 days.
- Antibiotics:Amoxicillin 25–50mg/kg 8 hourly for 5 days OR erythromycin 30–50mg/kg for 5 days.
- If there is perforation, treat as in chronic otitis media.
- Review after 5 days if not improved continue antibiotic for 5 more days.

**Admit if:**
- There are signs of complications (meningitis, mastoiditis) and treat according to the guidelines found in their respective sections.

## 27.2 Chronic Suppurative Otitis Media(CSOM)

**Clinical Features**
Discharging of pus from one ear or both ears for more than 2 weeks following untreated or unresolved acute otitis media with a central perforation. The discharge is usually not foul smelling. There is also impaired hearing. Recurrent ear discharge usually occurs after URTI. Secondary infection may be present with Gram-negative bacteria, yeast, and fungi. Complications include:
- Impaired hearing.
- Cholesteatoma.

**Investigation**
HIV test

**Management**
- If no antibiotics were administered recently, treat with antibiotics as in acute otitis media.
- Dry the ear by wicking. Show the mother how to dry the child's ear by wicking:
  - Roll a piece of clean absorbent cloth or cotton wool into a wick and insert it gently into the child's ear.
  - Roll the wick in the ear, then remove it and replace it with a clean wick.
  - Watch the mother repeat this until the wick is dry when it comes out.
- Tell the mother to continue to dry the ear by wicking at home atleast 4 times a day, until the wick stays dry and the perforation closes. Tell her that nothing should be left in the ear between treatments. The child should not go swimming until the ear heals.
- Reassess the child weekly. If the mother needs assistance in keeping the ear dry, reassess more frequently.
- Do not syringe such ears.
- Refer to ENT specialist if:
  - The patient develops mastoiditis.
  - There is no improvement after 4 weeks.
  - The patient has hearing impairment; they will benefit from tympanoplasty.
  - Patient complains of headache, earache, vertigo or facial paralysis:This indicates complications.

## 27.3 Mastoiditis

Mastoiditis is the infection of the mastoid air cells and mastoid bone occurring as a complication of acute otitis media or chronic otitis media.

**Clinical Features**
A painful swelling above the ear in children under 2 years of age. A painful swelling behind the ear in older children. There may be preceding otitis media and mastoid tenderness, with fever. There may be sagging of the posterosuperior meatal wall.

**Complications**
These include facial nerve palsy, meningitis, and brain abscess.

**Management**
- Admit.
- Give antibiotics:IV/IM chloramphenicol and benzyl penicillin till improvement, then discharge on oral chloramphenicol for total course of 10 days.
- Refer to ENT specialist if:
  - The swelling points and/or bursts to discharge pus.
  - The child develops a squint in the eye or facial palsy
  - The child develops signs of meningitis[see Chapter12.4,Meningitis]or brain abscess.

## 27.4 Otitis Externa

This is the inflammation of external ear most oftenly due to bacteria, but may also be due to fungi, e.g., Candida (whitish) or aspergilla (blackish) or Herpes zoster virus. It may also occur in generalized allergic and seborrhoeic states. The commonest bacterial organisms responsible are Streptococcus pneumoniae, Staphylococcus aureus, Ps. pyocyanea, B. proteus, and E.coli.

**Clinical Features**
Fever is uncommon. There is pain and tenderness accentuated by movement of the tragus. Pre or post auricular or cervical lymphadenitis may be present. Obliteration of the canal lumen may occur from the inflammation, causing deafness. There may be ear discharge with or without itching.

**Management**
- ♦ Admission is NOT necessary.
- ♦ Relieve pain; give analgesics such as paracetamol.
- ♦ In severe cases, e.g., a boil/furuncle, give antibiotics:
  - Benzyl penicillin 50,000 units/kg IM STAT followed by oral amoxicillin for 5 days.
  - Instil gentamicin ear drops or 2% acetic acid eardrops.
- ♦ Fungal otitis externa (otomycosis) is treated with fungicides, e.g., Clotrimazole 1% drops applied 8 hourly for at least 10 days.
- ♦ Allergic (eczematous) otitis externa is treated with antihistamine drugs and hydrocortisone ointment or drops:
  - Chlorpheniramine 0.4mg/kg/day BD in children.
  - Hydrocortisone ointment or drops apply BD.

## 27.5 Epistaxis

Epistaxis refers to bleeding through the nose (usually 90% from a plexus of veins in Little's areas) due to nose-picking, trauma (fall in games, assault, etc.), nasal and paranasal neoplasms, nasal infection, systemic derangements, e.g., acute fevers, hypertension, renal disease with uraemia, abnormalities of blood clotting, and foreign bodies in the nose.

**Clinical Features**
- ♦ Nosebleeding
- ♦ Anemia – if severe
- ♦ Other clinical features are of the underlying condition

**Investigations**
Usually none unless systemic disease is suspected.

**Management**
Assess airway, breathing and circulation and intervene as appropriate.
Trasfuse if in shock or severe pallor.

- Immediate: Sit the patient up (to avoid aspiration);pinch the nose for 10–20 minutes. This is usually sufficient to stop bleeding.
- Apply ice or cold packs on the bridge of the nose.
- To pack the nose, remove clots with suction catheter. Apply Lidocaine nasal spray then pack (preferably using Tiley's forceps) with ribbon gauze or narrow strip of gauze impregnated with liquid paraffin (mineral oil). Start packing from the floor of the nose towards the roof: The pack should fit lightly to be effective. Do not use adrenaline.
- Remove the paraffin pack within 24–48 hours.
- Admit if:
  - Bleeding is uncontrolled (patient may have a bleeding disorder).
  - Patient requires fluid replacement or blood transfusion.
  - Patient requires inpatient management of the underlying causative factor.

## 27.6 Foreign Bodies or Other Substances in Nose and Ears

Young children may push any object in the nose or ears. If a parent notices this, the best thing to do is not to struggle with the child as this may push the object even further in. Sometimes the object is noted several days after it was inserted, in which case there may be a nasal or ear discharge. Refer the child to a health facility that has health workers who are capable and equipped to remove the object without causing injury to the child.

### 27.6.1 FOREIGN BODIES IN THE EARS

The types of foreign bodies inserted include metallic pieces (hair clips, smooth pellets, needles, etc.), wooden pieces (e.g., match sticks), vegetable matter (e.g., seeds), or insects.

**Clinical Features**

There is obvious history of foreign body insertion into the ear, i.e., someone saw it happen. The child may have conductive deafness, ear pain, or discharging from the ear, and may experience disturbing noise (if insects involved) and bleeding from the ear (especially following traumatic insertion of a foreign body by the child). Complications include:

- Conductive deafness.
- Vegetable material is hygroscopic and leads to inflammatory reaction in the canal walls, leading to otitis externa. Beans, maize, and other seeds may sprout if they stay long enough.

**Management**

- Most foreign bodies can be removed fairly easily with crocodile forceps, a hook, an earprobe, or by suction and gentle syringing with warm,clean water.
- Rounded objects may be pushed further into the ear and rupture the eardrum.

- Do not attempt to remove a foreign body from the ear if you have difficulty doing so.
- Refer to ENT specialist if:
  - At any stage there is difficulty in removing the foreign body.
  - Perforation of the eardrum or foreign body in the middle ear is suspected.
  - The foreign body is deeply seated in the external auditory meatus.
- In general anaesthesia will be required:
  - In uncooperative patients.
  - When a foreign body is embedded in granulation tissue (easily bleeds).
  - When foreign body is posteriorly placed.
  - In suspected foreign bodies that cannot be easily be found.



## 27.6.2 FOREIGN BODIES IN THE NOSE

Occur usually in children. The foreign bodies include animate objects (e.g., maggots, regurgitated roundworms) and inanimate ones like vegetable (peas, beans, nuts), non-vegetable materials (pencils, paper, sponge, buttons, beads, pebbles, nuts, screws), traumatic objects (bullets, shrapnel, arrow heads).

**Clinical Features**

There may be pain, sneezing, and epistaxis or unilateral nasal discharge with nasal obstruction. There may also be pyrexia or headache especially with animate foreign bodies. Unilateral purulent nasal discharge in children should be regarded as due to foreign body until proven otherwise. Careful nasal examination is crucial for diagnosis of this condition.

**Management**

- For animate foreign bodies: remove with forceps.
- For in animate foreign bodies: if visible, attempt removal.
- Refer to ENT specialist if foreign body is difficult to remove.

## 27.6.3 WAX IN THE EAR

- Advise patients and parents to leave wax to come out of the ear on its own instead of attempting to remove with earbuds because these attempts may cause impaction of the wax in the ear.
- Rarely if the wax is causing impaired hearing it may need removal. Refer if hearing impairment occurs.

## 27.7 Foreign Body in the Oesophagus

The commonest objects are coins in children, and fish bones or meat in any age. Psychiatric patients may have many more types of foreign bodies in the oesophagus.

**Clinical Features**
Patients present with pain in retrosternal area and/or in the back, dysphagia, drooling of saliva in the mouth, regurgitation of food, dyspnoea, hoarseness if there is laryngeal oedema from compression by the foreign body, and localized tenderness in the lower part of the neck.

**Investigations**
Plain x-rays, anteroposterior and lateral views, may show opaque objects. Radiolucent objects are not seen on x-rays. However, an increase in the prevertebral soft tissue exceeding 1/3 of the anteroposterior distance of the patient's vertebral body is highly suggestive of the presence of a foreign body.

**Management**
Refer patient for oesophagoscopy and removal of the foreign body.

## 27.8 Laryngotracheal Trauma

These can be blunt or penetrating injuries. The priority is to secure and maintain an airway. Then refer urgently to an ENT specialist for endoscopy and repair.

## 27.9 Allergic Rhinitis

This is IgE-mediated rhinitis and is characterized by seasonal or perennial sneezing, rhinorrhoea, nasal congestion, pruritus, and often conjunctivitis and pharyngitis. Symptoms vary in severity from day to day or hour to hour.

**Management**
- Avoid the allergen (precipitating factor).
- Give the following medications to the patient:
  - Antihistamines: e.g., chlorphenamine 0.35mg/kg in children in 4 divided doses.
  - Sodium cromoglycate nasal sprays as prophylaxis given 4 hourly.
  - Topical steroids, which are safe and effective.
- Refer to specialist if:
  - There is gross nasal obstruction (hypertrophied inferior turbinates).
  - There are polyps.
  - There is sinusitis.
  - There is deviated nasal septum.

PAEDIATRICS

## 27.10 Parotid Masses

These may be parotid swellings (e.g., parotitis, parotid abscess, cysts, tumours, etc.) or pseudoparotomegalydue to swellings in nearby structures (e.g., hypertrophy of the masseter, jaw swellings, parapharyngeal masses, lymph node enlargement, facial nerve tumours, etc.). Parotid swellings may also occur in other systemic conditions (e.g., malnutrition, diabetes mellitus, HIV/AIDS). Infective masses may be associated with other features of infection like fever, pain, local inflammation, or discharge from the opening of the parotid duct. In children the commonest infection is mumps, which presents with pain and swelling.

**—Most parotid swellings are painless unless infected or malignant. Presence of facial nerve palsy is highly suggestive of a malignant process.**

**Investigations**

- Haematological tests,e.g.,WBC counts,ESR,serum protein,HIV,etc.
- Fine needle aspirate (FNA) for cytology.
- Open biopsy is contraindicated because of:
  - Risk of seeding of tumour in neoplastic conditions.
  - Risk of injury to the facial nerve or its branches.
- Should FNA report not be conclusive, then superficial or total parotidectomy (depending on suspected condition) is done to obtain the excisional biopsy.

**Management**

Viral parotitis may not require more than analgesics and bed rest. In the presence of bacterial infection, give amoxicillin. Refer the patient if:

- An underlying systemic disease is the causative factor for paroto-megaly.
- There are masses that may require surgical intervention.

## 27.11 ENT Manifestations of HIV/AIDS

In children chronic otitis media and parotid enlargement are the commonest manifestations. Other manifestations include:

- Infections: These can be viral, bacterial, or fungal, for example rhinitis, sinusitis,pharyngitis,glossitis,tonsillitis,laryngitis,parotitis,deep neckspace cellulitis, abscesses, otitis externa, otitis media, and labyrinthitis.
- Tumours: There is an increase in head and neck cancers associated with HIV/AIDS, especially Kaposi's sarcoma and lymphomas.
- Other manifestations: For example, adenoid hypertrophy, oropharyngeal and oral ulcers, atrophic rhinitis, lymphadenopathy, parotid cysts, otitis media with effusion, vertigo, deafness, tinnitus, and cranial nerve palsies.

**Management**

Is directed at the presenting condition, after confirming that the patient has HIV/AIDS infection.

## 27.12 Hearing Impairment

In the paediatric age group, pay special attention to children born prematurely, those with low birth-weight, difficult delivery, and yellowness of eye (neonatal jaundice). Further, look out for mothers who had febrile illness during pregnancy, and those treated for meningitis. Do not ignore parents' complaint of a child not hearing or a child who is slow to develop speech. Use changing voice intensity to assess grossly the state of hearing. If there is suspicion of hearing loss, refer at whatever age. A child who does not hear can be helped at any age but the earlier the better.

# 28. Infections (Selected) and Related Conditions

## 28.1 Septicaemia

This is suspected when there is fever with no localizing signs. Causes include infections by Staphylococcus aureus, Meningococcus and Salmonella group. Severity may vary and some children affected by this condition may be severely ill. Diagnosis is that of exclusion by doing the following investigations:

- Full blood count (shows leucocytosis and neutrophilia)
- Blood smear for malaria (negative)
- Urinalysis (negative)
- Blood culture (positive)
- CRP
- UECs
- Lumbar Puncture
- Blood sugar

**Management**

Give benzyl penicillin and gentamicin. In suspected Staphylococcus aureus infection, give flucloxacillin instead of penicillin. If there is no response after 48–72 hours consider using ceftazidime or ceftriaxone.

## 28.2 Septic Arthritis and Osteomyelitis

The above two are infections of the bone or joints often seen in children. They commonly follow septicaemia although occasionally may result from a penetrating injury. In children with sickle cell disease, more than one bone may be affected.

**Clinical Features**

The affected child looks sick and may be toxic. There is fever and limitation of movement of the affected limb. The affected limb is hot and extremely tender. The child may resist examination because of pain. Delay in treatment will result in bone or joint destruction. In the case of osteomyelitisa chronic discharging sinus may develop.

**Investigation**

- Full blood count
- Blood culture
- X-ray of affected limb
- Joint aspirate for culture and sensitivity

**Management**

- Admit.
- Give analgesics.
- Start antibiotic, penicillin and gentamicin, duration 4–6 weeks.

- Place limb in position of comfort.
- Refer to surgeon if
  - Need to aspirate under anaesthesia.
  - Chronic osteomyelitis if need to remove dead bone (sequestrum).

## 28.3 Salmonella Infections

The organisms Salmonella typhi and Salmonella paratyphi A, B, and C, commonly cause enteric fever or typhoid fever, while Salmonella enteritis causes gastroenteritis.

### 28.3.1 TYPHOID FEVER

This is a systemic disease and is caused by Salmonella typhi. Salmonella bacilli are shed in the faeces of asymptomatic carriers or in the stool or urine of those with active disease.

**Transmission**

Transmission of the Salmonella bacilli occurs via contaminated food or water. This may occur through:

- Direct contamination by faeces or urine
- Flies from faeces to food
- Through healthy carriers especially if they are foodhandlers
- Health personnel through inadequate hygiene when changing soiled linen.

**Clinical Features**

The patient may have high fever, headaches, anorexia, weight loss, diarrhoea, constipation, abdominal tenderness, changes in sensorium, splenomegaly, relative bradycardia and Rose spots (blanching lesions). A high index of suspicion is required when handling any patient with unexplained fever. The clinical picture tends to be atypical in infants, who may develop shock and hypothermia

**Complications**

The complications of typhoid fever include intestinal haemorrhage, convulsion, coma, shock, perforation with resultant acute abdomen, and “chronic carrier status”.

**Investigations**

- Full haemogram: Relative leucopaenia in relation to the fever
- Cultures: Positive in blood in first week, stool and urine cultures become positive in the third week.
- Widal test: A four fold rise of O antigen titres suggest S. Typhi infection significant. NB: Only titres of O antibody of 1:320 or more are significant. The gold diagnostic standard should be isolation of bacilli in cultures.
- Abdominal x-ray in suspected perforation: Erect/decubitus, which may show pneumoperitonium or multiple fluid levels.

**Management**

- Treat all patients for 14 daysusing:
  - Ciprofloxacin 15mg/kg/dose 12 hourly (max 1.5g/24 hours)
- Refer for
  - Surgical intervention if signs of perforation.

**Prevention**

**Preventive measures for typhoid fever include the following:**

- Using wholesome drinking water (water boiled for 10 minutes or chlorinated).
- Using pasteurized milk.
- Screening food handlers for typhoid and treating those infected, including healthy carriers.
- Ensuring proper hygiene while preparing or/and handling foods.
- Ensuring hygienic waste disposal.
- Vaccination.

## 28.4 Fever of Unknown Origin

This refers to fever of more than 3 weeks duration, the cause of which is still unknown in spite of at least 1 week of intensive investigations. Assessment of such a patient should include observation of the fever pattern, detailed history and physical examination, laboratory tests, and non-invasive and invasive procedures. This definition excludes common conditions of shorter duration and/or where the cause of the fever has already been determined within 3 weeks.

### 28.4.1 COMMON CONDITIONS MANIFESTING AS FEVER OF UNKNOWN ORIGIN

Most cases of prolonged obscure fever are due to well known diseases. Aggressive diagnostic effort is recommended as most of them are treatable. Do not just shift from one antibiotic to another as this confuses the picture even more. It may be better to stop every treatment and watch for a few days**.**

#### INFECTIONS

- Tuberculosis: This is the commonest cause of pyrexia of unknown origin in Kenya. Miliary tuberculosis may not be visible on chest x-ray until the disease is well advanced. Tuberculosis in other body sites like the central nervous system or abdominal lesion may be difficult to diagnose early.
- Infections due to some bacterial infections, without distinctive localizing signs, such as salmonellosis and brucellosis.
- Deep seated bacterial: Abscesses like intracranial,intra-abdominal,and hepatic abscesses may present as fever of unknown origin.
- Infective endocarditis.
- Some slow viruses, the commonest of which is HIV.
- Visceral leishmaniasis.

## NEOPLASMS

**Lymphomas are the commonest among the neoplastic causes of PUO. Diagnosis may be difficult if lesions are deep seated retroperitoneal nodes.**

## IMMUNOLOGICAL DISORDERS

These include:

- Juvenile idiopathic arthritis (JIA) especially systemic JIA
- Systemic lupus erythematosus and other systemic connective tissue disorders
- Auto-inflammatory disorders

**Investigations of PUO**

Routine investigations as set out under these immunological disorders, including a chest x-ray. In difficult cases it is worthwhile to consider the following:

- Repeated history taking and examination may detect new clinical features that give a clue or old clinical signs previously missed or overlooked.
- Other tests include:
  - Full haemogram and ESR
  - Blood culture
  - Immunological: Antinuclear antibody (ANA)
  - Ultrasound
  - Computerized axial tomography (CT) scans
  - HIV Test
  - TB-Gene Xpert, Mantoux
  - Echocardiography
  - Urinalysis
  - Bone marrow aspirate cytology and culture
  - Very rarely invasive procedure, e.g.,laparatomy
  - Specific according to diagnosis

**Management**

- Treat all diagnosed conditions in accordance with the diagnosis.
- Refer or consul tif:
  - Patient deteriorates rapidly.
  - Tests described above are not available in your centre.
  - Invasive procedure that needs more skill is required.

## 28.5 Guidelines for Use of Antibiotics in Bacterial Infections

Bacterial infections are a leading cause of morbidity and mortality. Accurate diagnosis and appropriate, cost-effective treatment are essential. Incorrect and over use of antibiotics facilitates the development and growth of drug resistant bacteria, which are difficult to treat. Specific treatments for the various infections are discussed under their respective headings. Generally the following should be taken into account:

- The organisms responsible for infections depend on the age of the victims.
- The management of the infections depend on their severity.
- Underlying conditions like immune depression determines the bacterial infections involved and the type of treatment required.
- The organisms and the treatment for community acquired infections differ from hospital acquired ones.
- Antibiotic dosage and the side-effects vary with age.
- Drug sensitivity to antibiotics is constantly changing.
- Treatments should be given using correct doses for the various conditions and compliance with drug administration should be encouraged for complete treatment.
- Leftover drugs should be disposed appropriately to avoid poisoning.
- Penicillin refers to narrow spectrum penicillin such as benzyl penicillin, procaine penicillin, and phenoxymethyl penicillin. Benzyl penicillin is used in moderate to severe infections where high blood levels are required, and because of its short half-life is given 4–6 hourly.
- Gentamicin doses should be adjusted according to renal function.

## 28.6 Paralysis (Acute Flaccid)

**Common differential diagnoses include:**

- Poliomyelitis
- Acute transversemyelitis
- Spinal cord injury
- Guillaine-Barré syndrome
- TB spine (not always acute)
- Neoplasms of spine or cord

**All of the above except poliomyelitis will have sensory loss.**

## 28.6.1 POLIOMYELITIS

**Clinical features**

- About 195 out of every 200 infections are asymptomatic.
- Abortive poliomyelitis: This presents as a brief febrile illness with malaise, anorexia, nausea, vomiting, sore throat, constipation, coryza, cough, and diarrhoea.
- Non-paralytic poliomyelitis: This form presents with the symptoms of abortive poliomyelitis with more intense headache,nausea, andvomiting,with bladder paralysis and constipation that are both transient.
- Paralytic poliomyelitis: Occurs in 0.5% of infections. The symptoms aresimilar to those of non-paralytic polio with additional weakness and pain of one or more muscle groups. Flaccid paralysis may involve one or more limbs as well as respiratory muscles. Transient bladder paralysis and bowel atony are common. Paralysis may be precipitated by IM injection.

**After the acute phase muscular atrophy ensues due to denervation. There is no sensory loss.**

**Investigations**

Stool specimen for viral detection and typing. The stool should be kept and transported to KEMRI laboratory under vaccine temperatures.

**Management**

**Avoid IM injections during epidemics or in suspected cases.**

- Admit all paralytic cases and give supportive therapy. During early phasegive:
  - Analgesics
  - Limb support for comfort and to prevent deformities
  - Respiratory support if bulbar or respiratory muscles are involved
  - Nutrition
- After acute phase (2 weeks):
  - Start rehabilitation: Initially gentle exercises of affected limbs. Continue even after discharge. Eventually child will need special shoes and calipers for mobility.

**Prevention**

- Immunization: On routine and National Immunization Days (NIDs)
- Active surveillance and mopping up
- It is hoped that polio will be eradicated in the near future with intensified childhood immunization combined with successful disease surveillance.

**For purposes of polio eradication, notify the local Medical Officer of Health of any acute flaccid paralysis.**

# 28.7 Tetanus

This is a neurological disorder characterized by muscle spasms due to endotoxin produced by Clostridia tetani. Tetanus occurs in several clinical forms including generalized, neonatal, and localized disease.

**Clinical Features**

These features include inability to open the mouth (trismus, or lock jaw), generalized muscle spasms initially on stimulation but may subsequently be spontaneous. There may also be opisthotonos (rigid arching of back muscles), dysphagia, laryngospasm with difficulty in breathing and there is no loss of consciousness. The port of entry for the infection in neonates is the umbilicus while in older children it can be thorn pricks, cuts or burns.

**Management**

- Admit urgently.
- On arrival maintain adequate airway (intubate if necessary).
- When airway and breathing are ensured, give IV diazepam.
- While the child is heavily sedated insert a nasogastric tube for nutrition and drug administration.
- Eliminate toxin production:
  - Crystalline penicillin 50,000IU/kg/day.Neonates Q12 hr, older children Q 6hr.
  - Clean the umbilicus thoroughly /surgical toilet of the wound.
- Neutralize toxin: Give neonates 500IU, older children 2,000IU of human tetanus immunoglobulin IM if available along site of wound. Horse serum is an alternative.
- Maintain fluid balance and nutrition, preferably enterally.
- Monitor for and treat intercurrent infections.
- Nurse in a dark, quiet isolation.
- Control spasms:
  - Diazepam is the drug of choice singly or incombination with phenobarbitone or chlorpromazine depending on the severity of the spasms.
  - Dose and frequency as shown in Table 28.1.
  - Give all medications IV to minimize frequent disturbance of the patient.
  - It may be necessary to give the drugs by infusion.
- Refer patient with refractory spasms needing admission to the ICU.

**Table 28.1: Guidelines for drug administration for tetanus**

| Drug tobeadministered | Time for drug administration in hours fromadmission | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 6 | 9 | 12 | 15 | 18 | 21 | 24 |
| Diazepam 0.5mg/kg/6 hourly | + | | + | | + | | + | | + |
| Chlorpromazine 5mg/kg/day6 hourly | | + | | + | | + | | + | |
| Phenobarbitone 6mg/kg/day 24 hourly | + | | | | | | | + | |

- Frequency of drug administration should be titrated against the clinical condition. Optimum level of sedation is achieved when patient remains sleepy but can be aroused to follow commands.

**Prevention**

- Against neonatal tetanus:
  - Pregnant mothers should receive tetanus toxoid 2 doses at least 4weeks apart as early as possible in pregnancy. They should then receive one booster dose at every subsequent pregnancy for a total of 5 doses.
  - Mothers with a baby with neonatal tetanus should be given neonatal toxoid immunization.
- People with open wounds should be given adequate surgical toilet and should also in addition receive 2 doses of tetanus toxoid at least 4 weeks apart.Only 1 dose of tetanus toxoid is given if patient was immunized during the last 3 years and adequate surgical toilet.
- All patients who recover from tetanus should be immunized.



## 28.8 Tuberculosis

Tuberculosis is caused by Mycobacterium tuberculosis, which is also referred to as acid-alcohol fast bacilli (AAFB) because of its staining properties. Transmission is by droplet infection through coughing and sneezing. Children almost always get infected from an adult living in the same household. The incidence of TB is on the increase and this is partly due to its association with HIV/AIDS, poverty, malnutrition, and overcrowding.

### 28.8.1 CLINICAL FEATURES OF TB

Most cases of tuberculosis in Kenya (80%) are pulmonary. Features of pulmonary tuberculosis include cough for 2 weeks or more, chest pain, fever, night sweats, weight loss and breathlessness. A persistent cough may be the earliest indication of TB infection.

Extra pulmonary tuberculosis is common in children. Its symptoms depend on the organs that are affected. Consequently the symptoms include TB adenitis(or lymphadenopathy), TB arthritis (with painful swollen joints),TB meningitis (with signs of meningitis), TB peritonitis (with ascites), and TB involving the pleura (with pleural effusion).

### 28.8.2 DIAGNOSIS OF TUBERCULOSIS IN CHILDREN

**The key elements for diagnosis of TB in children include:**

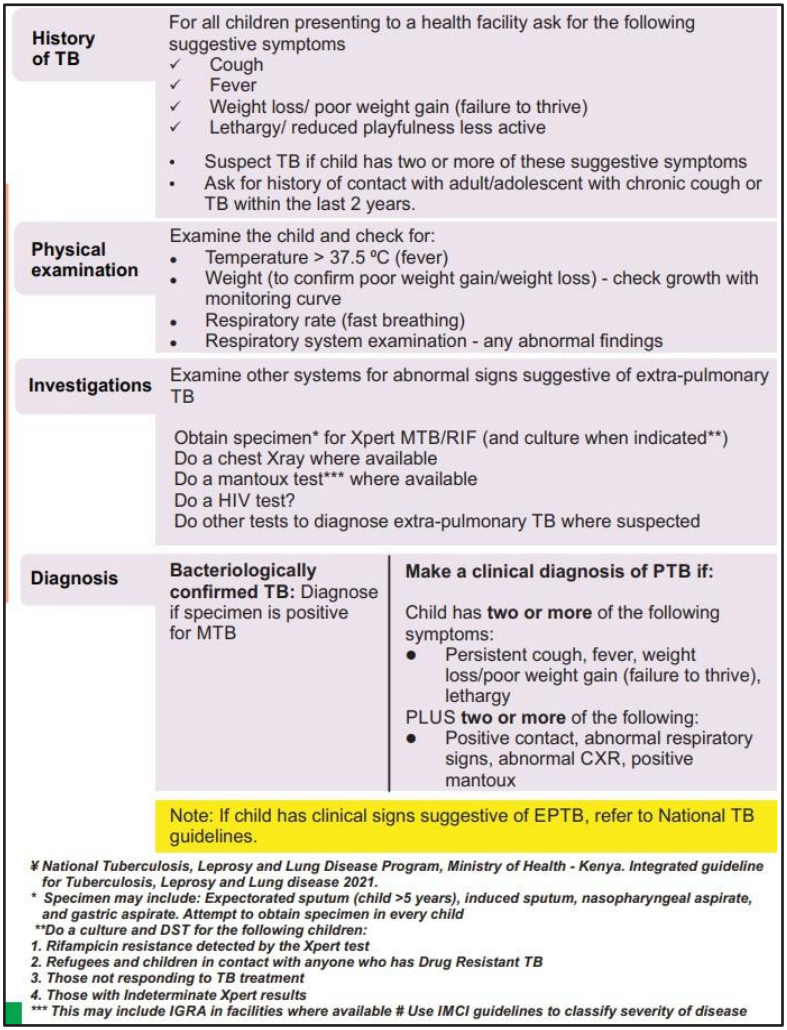
- A history of contact with an adult who has TB or a long-standing cough is useful.
- Smear microscopy (3 specimens – spot, early morning, and spot) for those children who can produce sputum. Sputum induction should be carried out for those who cannot.
- Sputum for AAFB culture and sensitivity (before the start of treatment in suspected resistant cases).

- Gastric lavage for AAFB in children (taken early morning).
- Tuberculin skin testing (Mantoux test)
- Chest x-ray
- HIV testing
- Lymph node biopsy

A high index of suspicion is important in diagnosing TB in children, as they seldom produce sputum and often have non-specific symptoms.

The diagnostic algorithm for TB in children is shown in Figure 28.1

**Figure 28.1: Diagnostic algorithm for TB in children**




Source: Kenya Basic Paediatric Protocols, 2022

## 28.8.3 PREVENTING TB IN CHILDREN

*BCG Vaccination:* Although not totally protective, BCG reduces the risk of severe/complicated TB.

**Preventing Tuberculosis in Exposed Children**

TB in children is always contracted from close contact with an adult. All children in households where an adult has been diagnosed to have TB should be screened for TB and appropriately managed. In addition, all adults from households where a child has been diagnosed to have TB should be screened for TB and appropriately managed.

A healthy newborn with a mother who is still sputum positive should be started on isoniazid prophylaxis immediately and the prophylaxis continued for 3 months. If a repeat sputum evaluation for the mother is found to be negative for TB, isoniazid should be stopped and the baby given BCG. If the sputum is found to be still positive, isoniazid prophylaxis should be continued for 9 months. It should be ensured that the mother is taking the drugs.

If a parent on treatment for tuberculosis has a child under 5 years of age, the child should have a Mantoux test carried out on them. If the Mantoux is positive, the child is infected and should receive full treatment for tuberculosis. If the Mantoux is negative, the child should be started on isoniazid prophylaxis at 10mg/kg body weight for 3 months. The Mantoux test should be repeated at 3 months. If the Mantoux test is more than 5mm, the child should receive prophylaxis for a further 3 months. If the test is negative, isoniazid prophylaxis should be stopped and the child given BCG vaccination after 3 days.

**Management**

The success of tuberculosis treatment depends on strict adherence to treatment. WHO's DOTS (directly observed treatment short-course) can be used if adherence is uncertain.

**General Guidelines on TB Management**

**The following are the general guidelines for TB Management:**

- Follow National Guidelines.
- Ensure adequate supply of drugs.
- Use correct regimens and dosages.
- Ensure regular patient attendance.
- Always supervise initial phase of treatment.
- Trace defaulters promptly.
- Maintain accurate patient information and clinic attendance records.

**Management - Pharmacologic**

In order to provide optimum treatment to patients with tuberculosis, such patients are classified into groups.

**Classification of TB Patients**

Patients are classified into the following groups for epidemiological and treatment purposes depending on the site, microbiology, severity of disease, and history of previous treatment. These classifications are also in the TB register for reporting.

- New (N): Patient who has never been treated before.
- Relapse (R): Patient who has received treatment and was declared cured but now has TB again.
- Transferred in (TI): Patient who was registered in another county/clinic initially and has now reported to continue treatment.
- Treatment resumed (TR): Patient who interrupted his/her treatment,and was declared "out of control", but is now resuming treatment.
- Other (O):Other types of patients e.g. failure cases put on re-treatment.

**Short Course Chemotherapy (SCC)**

SCC is given to all TB patients registered by the National Leprosy and Tuberculosis Programme (NLTP). Different SCC regimens are used for the different categories of tuberculosis patients. Treatment in the first 2 months (initial phase of treatment) should be administered under direct observation of either a health care provider in a health facility or a member of the household or community. Drugs and tools for registration and reporting should be available before treatment is started. Patient is admitted if very ill or DOTS cannot be ensured. The continuation phase (4–6 months duration) in principle is (or should be) available in all government and NGO health facilities. The patients should collect a supply of drugs enough for 4 weeks, for daily self-administration at home. The patient should return to the health facility for evaluation and supply of more drugs before the drugs run out, at 4-week intervals for self-administration at home.

**Treatment Regimens and Drug Dosages**

The drugs used for first line treatment of TB in children are:

- Rifampicin (R)
- Isoniazid (H) or (INH)
- Pyrazinamide (Z)
- Ethambutol (E)

**Table 28.2: Dosage of individual anti-TB drugs according to body weight**

| Drug | Recommendations Average dose in mg/kg | Range in mg/kg | Maximum dose |
|---|---|---|---|
| Isoniazid | 10 | 7-15 | 300mg |
| Rifampicin | 15 | 10-20 | 600mg |
| Pyrazinamide | 35 | 30-40 | 2.0mg |
| Ethambutol | 20 | 15-25 | 1.0mg |

The first 3 drugs have been combined into paediatric child-friendly fixed dose combinations which are dispersible in liquid, have a pleasant taste and are therefore easier for children to take.

The improved paediatric TB FDCs provide the correct dosing ratio of Rifampicin: Isoniazid: Pyrazinamide as follows: Rifampicin 75mg: isoniazid 50mg: pyrazinamide 150mg (RHZ 75:50:150) tablet Rifampicin 75mg: isoniazid 50mg (RH 75:50) tablet Ethambutol is available as a single drug paediatric tablet of 100mg (E 100).

**Table 28. 3: TB drug doses**

| Weight band (kg) | Number of tablets | | |
|---|---|---|---|
| | Intensive phase | | Continuation phase |
| | RHZ (75/50/150mg) | E (100mg) | RHZ (75/50mg) |
| < 2kg | ¼ | ¼ | ¼ |
| 2.0 - 2.9kg | ½ | ½ | ½ |
| 3.0 - 3.9kg | ¾ | ¾ | ¾ |
| 4.0 - 7.9kg | 1 | 1 | 1 |
| 8.0 - 11.9kg | 2 | 2 | 2 |
| 12.0 - 15.9kg | 3 | 3 | 3 |
| 16.0 - 24.9kg | 4 | 4 | 4 |
| > 25kg | Use adult dosage and preparation | | |

**Pyridoxine (Give through the whole course of treatment)**

| Weight (kg) | Number of tablets of pyridoxine (50mg) |
|---|---|
| 5-7 | Quarter tablet daily |
| 8-14 | Half tablet daily |
| 15 and above | One full tablet daily |

**Isoniazid Preventative Therapy (IPT):** *Refer to National TB Guidelines*

**Source: Kenya Basic Paediatric. Protocols, 2022**

**How to administer TB Treatment in children**

Treatment is given in two phases as follows:

- The Intensive phase – this takes two months. During this period, 4 medicines are administered daily to rapidly kill the bacilli in the body (bactericidal).
- The Continuation phase –This varies depending on the type of TB being treated. In this phase, 2 drugs are given daily to kill dormant and slowly multiplying bacilli that may linger after the intensive phase. All forms of TB are treated for four months in the continuation phase except TB meningitis, TB of the spine, bone and joint that are treated for ten months.

**Table 28.4: WHO recommended TB treatment regimen**

## Tuberculosis treatment

Treat for TB as follows:

- All children with **bacteriologically confirmed TB**
- All children with a **clinical diagnosis of TB**

NB : Children who do not have an Xpert result or their Xpert result is negative but they have clinical signs and symptoms suggestive of TB, should be treated for TB.

**Regimens and dosing**

| TB disease category | Recommended regimen | |
|---|---|---|
| | Intensive phase | Continuation phase |
| All forms of TB except TB meningitis, bone and joint TB | 2 months RHZE | 4 months RH |
| TB meningitis<br>Bone and joint TB | 2 months RHZE | 10 months RH |
| Drug-resistant TB | Refer to DR TB specialist | |

Steroid therapy should be given for; TB meningitis and other forms of intracranial TB, PTB with respiratory distress, PTB with airway obstruction by hilar lymph nodes, severe miliary TB or pericardial effusion.

- Give **Prednisone at 2 mg/kg (max 60mg/day) once daily for 4 weeks.** Taper down over 2 weeks (1 mg/kg for 7 days, then 0.5 mg/kg for 7 days)

**Tuberculous Meningitis**
Treatment in the initial phase consists of 4 drugs, including streptomycin.The duration of treatment is 9 months.

**Follow Up**
Review the patient 2 weeks after initiation of therapy and at end of intensive phase. Thereafter, monthly reviews should be carried out. Each review evaluates symptoms, adherence, side effect and weight gain. A child not responding should be referred for further evaluation.

## 28.8.4 TREATMENT OF TB IN HIV/AIDS PATIENTS

HIV increases a person's susceptibility to infection with Mycobacterium tuberculosis. In individuals infected with M. tuberculosis, HIV is a potent cause of progression of tuberculosis infection to disease. In HIV infected children, dissemination of TB is common. TB meningitis, miliary tuberculosis and widespread tuberculous lymphadenopathy occur. Diagnosis of TB in HIV

infected children can be very difficult as the clinical features of the two diseases are almost identical. There are several chest conditions that may mimic TB, and the tuberculin test may be negative despite TB infection. When in doubt, treat for TB but non responsemay mean it was not TB in the first place. Do not keep the child on TB drugs indefinitely.

**Check that drugs used for HAART are compatible with TB drugs.**

## 28.8.5 ACQUIRED DRUG RESISTANT TB

This is when an acquired drug resistance results from inappropriate use of one drug or poor adherence to treatment. This suppresses the growth of organisms susceptible to the drugs but encourages the multiplication of isolated strains with spontaneous drug resistance.

## 28.8.6 MULTIPLE DRUG RESISTANT TB (MDR-TB)

This is resistance to both rifampicin and isoniazid as a consequence of poor adherence to recommended treatment regimens by clinicians and patients. This resistance is further associated with increasing poverty and the HIV/AIDS epidemic. MDR-TB is confirmed on culture and sensitivity. MDR-TB can be prevented by:

- Strengthening TB programmes.
- Ensuring directly observed therapy whenever rifampicin is used.
- Using fixed dose combination tablets containing rifampicin.
- Referring all drug-resistant TB patients to a TB specialist for confirmation and management.

## 28.9 Rabies

Although not common, rabies is a devastating disease and is almost universally fatal once clinical features appear. It is therefore important to prevent onset of symptoms. The incubation period is 10 days to 1 year with an average of 1–2 months. This period is adequate to allow immunization.

**Clinical Features**
Initially, there is restlessness and paraesthesia at the site of the wound. Subsequently, the patient develops maniacal behaviour and may demonstrate violent behaviour; the patient also develops dysphagia and hydrophobia. Finally, repeated convulsions develop with hyperpyrexia and flaccid paralysis that ends in death in about 5 days from onset of symptoms.

**Management**
Rabies has no cure. The management is basically supportive and includes:
- Strict barrier nursing.
- Avoid bites from the patient.
- Sedation.
- Administration of fluids and feeding.

Since even supportive care cannot be given on an outpatient basis, the patient should be referred for admission to provide such management. Part I,Section 1.4.2, contains management details.

## 28.10 HIV Infection in Children

Approximately 6% of people living with HIV/AIDS are children aged 14 years and below. The majority of children acquire the infection from the mother either during pregnancy, delivery, or through breastfeeding (mother to child transmission). A few children are infected sexually through rape and a still smaller number through blood transfusion. The rate of progression of HIV in children once infection has occurred is in two forms. In one form, the disease progresses rapidly with the patients dying within 2 years from birth (rapid progressors) while the in other form progression of the disease is slow, over few to several years before becoming symptomatic (slow progressors).

HIV testing should be routinely offered to all clients visiting health facilities. People should also be encouraged to use community-based voluntary counselling and testing centres (VCTs). HIV self-testing is now available using saliva- based easy to use tests available in pharmacies and health facilities. Encouraging people to know their status allows appropriate interventions to be instituted at an early stage for those who are infected in order to reduce morbidity and mortality.

## 28.10.1 PREVENTION OF MOTHER TO CHILD TRANSMISSION (PMTCT)

Without intervention, 20–45% of mothers infected with HIV transmit the infection to their babies during pregnancy, delivery or through breastfeeding. However, appropriate intervention can reduce HIV transmission from mother to child to less than 1%. Prevention of HIV/AIDS centres around:

- Diagnosis of infection in the parents: Routine testing of both parents is recommended.
- Good quality obstetric care:
  - Pre-conception care – ensure viral load suppression for women known to be HIV positive (KPs), folic acid use, nutrition
  - Ensuring adequate maternal nutrition in pregnancy
  - ART should be initiated in all pregnant and breastfeeding women living with HIV regardless of WHO clinical staging.
  - Avoiding prolonged rupture of membranes (>4hours).
  - Ensuring a clean, a traumatic delivery.
  - Giving mother ARV during pregnancy and/or labour, and postnatally to the baby. The drugs currently in use are zidovudine and nevirapine. It is important to use the currently recommended ARVs.
- Counselling on feeding options for the baby. Counselling is best done antenatally to allow parents to choose the best option according to their socio-economic situation and other social factors.

## 28.10.2 FEEDING OPTIONS FOR HIV INFECTED WOMEN

### EXCLUSIVE BREASTFEEDING FOR 6 MONTHS

In this method of feeding, HIV infected mothers breastfeed their babies exclusively for 6 months. Mixed feeding before 6 months has been associated with increased risk of mother to child transmission of HIV as well as diarrhoeal and respiratory infections. It is therefore not recommended.

### REPLACEMENT FEEDING

This refers to mothers who are not breastfeeding but using another type of appropriate replacement milk exclusively for 6 months. They then introduce other feeds at 6 months while continuing the replacement milk. The current WHO recommendation is that when replacement feeding is acceptable, feasible, affordable, sustainable, and safe (AFASS), then mothers should avoid breastfeeding. If this is not possible mothers should be counselled on how to safely breastfeed. Recent studies from Africa, however, indicate that replacement feeding is associated with increased morbidity and mortality even when formula milk is provided by the government.

### COMPLEMENTARY FEEDING AFTER 6 MONTHS

Complementary foods should be introduced after 6 months. Breastfeeding can continue up to 24 months and beyond. Breastfeeding should only stop once a nutritionally adequate, safe and sustainable diet can be maintained.

## 28.10.3 CARE OF HIV EXPOSED INFANTS

**Care of an infant exposed to HIV consists of the following:**

- Initiating cotrimoxazole prophylaxis at 6 weeks.
- Continuing nutritional counselling at all visits.
- Ensuring immunization according to KEPI schedule.
- Giving vitamin A according to national guidelines.
- Monitoring growth: The growth curve should be evaluated: if the baby is not gaining weight appropriately despite nutrition counselling, the baby may have been HIV infected and should be referred to a facility that can carry out the tests to confirm infection status.
- All HIV-exposed infants should have DNA PCR at as soon as possible after birth with repeat test at 6 weeks and if negative repeated at 6 months and 12 months. A HIV antibody test should be done at 18 months and then repeated every 6 months during breastfeeding. The final antibody test should be performed 6 weeks after complete cessation of breastfeeding.

## 28.10.4 CARE OF HIV INFECTED CHILDREN

Unfortunately most mothers do not know their HIV status in pregnancy and consequently the diagnosis of HIV in children tends to be made late. Early signs of HIV infection are also often missed by the primary health care provider. Many of the severe illnesses that occur as complications of HIV/AIDS disease also occur in non-infected children and health workers may therefore not realize that they might be occurring as complications of HIV/AIDS.

## 28.10.5 DIAGNOSIS

The diagnosis of HIV infection can made by a rapid antibody test or an Elisa test for all children aged above 18 months. Diagnosis can also be made by virological test using PCR which is a confirmation test for infection in children below 18 months. Ideally, all children attending mother and child health (MCH) clinics should be tested for HIV to facilitate early intervention and appropriate management. All children requiring admission should be tested to avoid missing infected children and to facilitate optimum care of the infected. HIV infection should be suspected in the presence of the following cases:

- Chronic otitis media.
- Persistent parotid enlargement.
- Slow growth or weight loss that fails to respond to adequate nutrition.
- Non specific chronic rashes.

In more advanced disease, the following features are usually noted:

- Recurrent serious infections, e.g.,pneumonia.
- Persistent or recurrent fevers.
- Severe and recurrent oral thrush.
- Recurrent and persistent diarrhoea.
- Herpes zoster.
- Neurological dysfunction, either delayed or regressed milestones.
- Failure to thrive.

It is advisable to encourage all adults with HIV and on treatment to bring their children for testing even if they think the children are not infected.

It is necessary to refer the patient if:

- HIV infection cannot be confirmed.
- Child confirmed to be HIV infected, so that they can be taken care of in a comprehensive care centre, where CD4 count and viral load can also be done.

**Management**

The mother, child and any other infected family members should access care preferably in the same setting. If the clinic only caters for children then adult members must be referred to an appropriate clinic.

**Nutrition for Affected Children**

Ensure adequate diet for age of the child. Their energy needs are higher than those of non-HIV infected children. Many infected children have poor appetite, thus the parent or caregiver should vary and experiment on foods offered. Nutritional supplementation may be necessary, especially micronutrients.

## 28.10.6 HIV STAGING

Two approaches are taken to determining the phase or stage of HIV infection, i.e. WHO's clinical criteria given below, and an immunological approach. The immunological approach, based on age specific CD4 counts, is summarized in Table 28.7. The World Health Organization however recommends that all HIV – infected people including children be commenced on treatment regardless of their clinical, virologic or immunological stage.

**WHO Clinical Staging Stage 1:**

- Asymptomatic
- Persistent generalized lymphadenopathy

**Stage 2:**

- Skin eruptions that include recurrent/extensive lesions that may be infections due fungi or Molluscum contagiosum virus, or may be immunological like seborrheic dermatitis (eczema) and any non specific dermatitis.
- Herpes zoster
- Recurrent or chronic upper respiratory and/or ear infections
- Parotid enlargement
- Recurrent oral infections
- Hepatosplenomegaly

**Stage 3:**

- Moderate malnutrition (-2SD or Z score) not responding to therapy
- Unexplained persistent diarrhoea
- Oral candidiasis (outside neonatal period)
- Unexplained persistent or recurrent fevers
- Severe recurrent pneumonias (>2 episodes in 12 months)
- HIV related chronic lung disease
  - Symptomatic lymphoid interstitial pneumonitis
  - Pulmonary or lymph node TB
  - Systemic varicella infection
  - Unexplained anaemia, neutropaenia,thrombocytopaenia

**Stage 4:**

- For a child<18 months of age: 2 or more of the following:oral candidiasis, severe pneumonia, failure to thrive or sepsis
- For a child of any age:
  - Severe wasting, stunting, or malnutrition not responding to therapy
  - Pneumocystis jiroveci pneumonia (PCP)
  - Extra pulmonary TB
  - Candidiasis of oesophagus, trachea, or lungs
  - HIV associated cardiomyopathy, or nephropathy, or encephalopathy
  - Kaposi's sarcoma or other lymphomas
  - Unusual bacterial, fungal, or viral infection

## 28.10.7 PREVENTION OF MOTHER TO CHILD TRANSMISSION (PMTCT)

Without intervention, 20–45% of mothers infected with HIV transmit the infection to their babies. However, appropriate intervention can reduce HIV transmission from mother to child to 5% or even less. Prevention of HIV/AIDS centres around:

- Diagnosis of infection in the parents: Routine testing of all parents is recommended.
- Good quality obstetric care:
  - Ensuring adequate maternal nutrition in pregnancy.
  - ART should be initiated in all pregnant and breastfeeding women living with HIV regardless of WHO clinical staging.
  - Avoiding prolonged rupture of membranes (>4hours).
  - Ensuring a clean, a traumatic delivery.
  - Giving mother ARV during pregnancy and/or labour, and postnatally to the baby. The drugs currently in use are zidovudine and nevirapine. It is important to use the currently recommended ARVs.
- Counselling on feeding options for the baby. Counselling is best done antenatally to allow parents to choose the best option according to their socio-economic situation and other social factors.

**Table 28.5: Immunological stages:Based on age specific CD4 counts**

| Stage | <12 months (%) | 12–35 months (%) | 36–59 months (%) | 5 years & above (Cells/Cm) |
|---|---|---|---|---|
| Not significant | >35 | >30 | >25 | >500 |
| Mild | 30–35 | 25–30 | 20–25 | 350–499 |
| Advanced | 25–34 | 20–24 | 15–19 | 200–349 |
| Severe | <25 | <20 | <15 | <200 or <15% |

## 28.10.8 PREVENTION OF PNEUMOCYSTIS CARINII PNEUMONIA WITH DAILY COTRIMOXAZOLE

Pneumocystis carinii pneumonia (PCP) should be prevented in all HIV infected children by administering daily cotrimoxazole in the dosages shown in Table 28.8.

**Table 28.6: Daily cotrimoxazole dosages to prevent PCP**

| Weight (kg) | Syrup 240mg/5ml | Tablet 480mg | Tablet 960mg |
|---|---|---|---|
| 1–4 | 2.5ml | ¼ tab | - |
| 5–8 | 5ml | 2 tab | ¼ tab |
| 9–16 | 10ml | 1 tab | 2 tab |
| 17–30 | 15ml | 2 tabs | 1 tab |
| >30 | 20ml | 2 tabs | 1 tab |
| Adolescent/Adult | 2 tabs | 1 tab | |

## 28.10.9 TREATMENT OF INTERCURRENT CONDITIONS (OPPORTUNISTIC INFECTIONS)

Patients with any complications or co-existing disease should be treated for the condition using the recommended guidelines for the condition. Those more severely ill or with various complicating illnesses should be appropriately referred for management.

- Oropharyngeal candidiasis:
  Ketoconazole 3–6mg/kg in 2 doses for 7 days or fluconazole10mg/kg STAT then 3–6mg/kg per day for 2 weeks.
- Tuberculosis:
  - Refer to the national Tuberculosis treatment guidelines.
- Cryptococcal meningitis:
  - AmphotericinB 0.7–1mg/kg daily or fluconazole 3-6mg/kg daily for 6–10 weeks then half the dose OD for life.

### PNEUMOCYSTIS JIROVECI PNEUMONIA (PCP)

**Clinical Features**

The following clinical features are shown by children with Pneumocystis carinii pneumonia:

- Low grade fever
- Severe respiratory distress
- Normal auscultatory findings
- Poor response to standard antibiotics
- Severe hypoxaemia

**Management**

- Admit.
- Give oxygen.
- Give prednisone 2mg/kg/dayfor 7–14 days;taper off if treatment was over 7 days.
- Give cotrimoxazole(TMP/SMX) IV 20mg TMP/kg/day IV 6 or 8 hourly for 21 days. Use same dose orally if IV preparation is not available.

## TOXOPLASMOSIS

Children with this condition have features of encephalitis.

**Treatment**

- Give a combination of:
  - Pyrimethamine 2mg/kg/day (max 50mg) for 2 days then 1mg/kg/day (max 25mg).
  - Sulphadiazine 50mg/kg/ every 12 hours.
  - Folic acid 5–20mg 3 times per week.
- Treat for 1–2 weeks after resolution of symptoms.

## 28.10.10 ANTIRETROVIRAL THERAPY (COMPREHENSIVE CARE CENTRE)

This is indicted in all HIV-infected children irrespective of the clinical, virologic or immunological stage of infection,. Tables 28.9 and 28.10 summarize the treatment regimens for first and second line ARVs, respectivel Before starting the medication the child the investigations listed below should be done and the parents counselled on the use of the drugs.

**Before Starting ART**

Before ART is commenced, the following investigations need to be carried out:

- Full blood count
- Liver function tests (alanine transferase)
- Renal function (creatinine)
- CD4 count is desirable but not a pre-requisite test
- Viral load can be done after 12 weeks of treatment

**Before a child is started on ARVs, adherence counselling is done to help the parent or guardian understand:**

- The treatment that is required and side effects of the treatment.
- Correct administration of the drugs and the need to give the drugs everyday
- That treatment is for life.

**First Line ARV Therapy.**

In order to optimize paediatric anti-retroviral therapy, children and adolescents living with HIV are to be transitioned to Dolutegravir based regimens as summarized in Table 28.9, 28.10 and 28.11.

**Table 28.7: Initiation of ART among children and Adolescents Newly diagnosed with HIV (Less than 15 years)**

| Age | Preferred Regimen |
|---|---|
| Birth – 4 weeks | AZT + 3TC + NVP |
| 3 – 20 Kgs | ABC + 3TC + Ped DTG |
| 20-29.9 Kg | ABC + 3TC + DTG |
| > 30Kgs | TDF + 3TC + DTG |

NOTE: DTG will be double dose in children with Rifampicin based Anti-TB treatment

**Table 28. 8: Transition of children and adolescents currently on 1st line ART who are virally suppressed (<1,000 copies/mL)**

| Current Regimen | Optimized ART Regimen | | |
|---|---|---|---|
| | 3(≥ 4 Weeks < 20 Kgs) | 20-29 Kgs | ≥ 30 Kg |
| AZT + 3TC + EFV/NVP | ABC + 3TC + DTG | ABC + 3TC + DTG | TDF + 3TC + DTG |
| ABC + 3TC + EFV/NVP | ABC + 3TC + DTG | ABC + 3TC + DTG | TDF + 3TC + DTG |
| AZT + 3TC + LPV/r or RAL | ABC + 3TC + DTG | ABC + 3TC + DTG | TDF + 3TC + DTG |
| ABC + 3TC + LPV/r or RAL | ABC + 3TC + DTG | ABC + 3TC + DTG | TDF + 3TC + DTG |

**Table 28. 9: Transition of children and adolescents < 20 Kgs currently on 1st Line ART who are not virally suppressed (>1,000 copies/mL)**

| Current Regimen | Optimized ART Regimen |
|---|---|
| Contains ABC | Switch to AZT/3TC + DTG |
| Contains AZT | Switch to ABC/3TC + DTG |

## TREATMENT FAILURE

Treatment failure can only be considered when a child has been on treatment for at least 6 months (24 weeks).

**The following features constitute treatment failure:**

- Clinical: Poor growth or weight loss after gaining, recurrence of severe infections, neuro-developmental delay or regression.
- Immunological:Drop in CD4 count below level for age,>50% peak or below baseline.
- Virological: Failure to achieve significant suppression load or progressive increase in viral load after significant suppression.

**It is important to note the following:**

- Second line therapy should not be introduced in a rush.
- Adherence to the first line drugs should always be determined.
- First line therapy should not be discontinued before second line drugs are available.

**DISCONTINUATION OF ART**

Sometimes it may be necessary to stop ART. This may be required in the following situations:

- When adherence is a problem despite repeated counselling.
- When there is drug toxicity.

# 29. Nutrition, Growth, and Development

All children from conception, require adequate nutrition for their growth, development and normal function. Under or over nutrition are undesirable and may lead to disability. Currently 31% of Kenyan children aged below 5 years are stunted. There is little information on nutritional status of children 5–18 years of age. What is known is that poor nutrition leads to poor school performance.
Nutritional needs vary according to the rate of growth, and both are highest in utero, followed by the first year and gradually reducing until the adolescent growth spurt.

**— Notably, stunted children will result in stunted adults. Further, damage that occurs in foetal and early childhood is irreversble later in life.**

## 29.1 Foetal Nutrition

Foetal nutrition depends on the mother's nutrition hence good nutrition of the mother contributes to good nutrition of the foetus. Preferably, a well nourished mother before conception should continue getting adequate nutrition through pregnancy and lactation. Foetal under-nutrition predisposes to adulthood diseases such as diabetes and obesity, while micronutrient deficiency predisposes to congenital defects. It is therefore essential to ensure adequate maternal nutrition. All programmes of maternal and reproductive health should have a component on maternal nutrition.

## 29.2 Infant and Young Child Feeding

This is centred on exclusive breastfeeding for 6 months with timely and adequate complementary feeding with continued breastfeeding up to 24 months. All infants should be breastfed unless there is medical contraindication. These national guidelines need to be followed to ensure prevention of malnutrition, which is the main underlying cause of death in children aged below 5 years. Community support for appropriate breastfeeding is needed. Figures for 2003 indicated that only 2.6% of women at that time practised exclusive breastfeeding for the recommended 6 months. Although the recommendations for feeding in this section are strictly for ages 0–2 years, they can be extended to older children up to 3 years. Mother should be prepared and counselled for breastfeeding during antenatal and postnatal periods.

Compliance with the feeding recommendations for infants and young children can be achieved by the help of support groups, which could have a number of additional activities on other aspects of health in the community. Children aged 2- 5 years are often on an adult diet and this may not be sufficient for their needs.

Consequently,families need to know how to feed these children adequately.

Some of these children may have started nursery school and may thus fit in the existing early childhood development (ECD).

## 29.2.1 RECOMMENDED FEEDING FOR YOUNG CHILDREN

| **Age** | **Type of Feeding Recommended** |
|---|---|
| Birth to 6 months | Exclusive breast milk. Breastfeed as often as the child wants day and night, at least 8 times in 24 hours.<br>There should be no other food or milk or fluid offered (including water) for healthy babies except medicines including ORS when indicated. |
| **6–12 months** | Breastfeed on demand. If not breastfeeding, give 500ml of milk. Introduce enriched complementary foods like *uji* mixed with milk, sugar, or oil,<br>Along with mashed green vegetables and proteins (plant or animal sources).<br>Also give fresh fruit juice or mashed fruit.<br>Feed 3 times a day if breastfed and 5 times a day if not breastfed. |
| **13–24months** | Breastfeed on demand.<br>Continue energy rich foods, giving at least 5 times a day**.** |

## 29.2.2 NATIONAL POLICY ON INFANT AND YOUNG CHILD FEEDING PRACTICES

**Summary Statement**

**Every facility providing maternal and child health (MCH) services should:**

1) Adhere to the National Infant Feeding Policy, which should be routinely communicated to all health staff and strategically displayed.
2) Train all health care staff in skills necessary to implement this policy.
3) Provide information to all pregnant and lactating mothers and their partners on the benefits and management of breastfeeding.
4) Assist mothers to initiate breastfeeding within the first 30 minutes of birth.
5) Give newborn infants no food or drink other than breast milk unless medically indicated(see specific guidelines on infants of HIV infected mothers).
6) Show mothers how to breastfeed and to maintain lactation even if they should be separated from their infants.

Practice rooming-in,allow infants to remain together with the mother 24 hours a day

7) Encourage breastfeeding on demand.
8) Encourage and actively promote exclusive breastfeeding for infants upto 6 months.
9) Provide information and demonstrate to mothers how to introduce and prepare appropriate and nutritious complementary foods for their infants after 6 months.
10) Encourage mothers to breastfeed for atleast 24 months (see guidelines for HIV infected mothers).
11) Foster the establishment of breastfeeding supportgroups and other support groups and refer mothers to them on discharge from hospital or clinic.
12) Not accept any free samples and supplies of breast milk substitutes.
13) Not allow any publicity by the manufacturers or agents of breastmilk substitutes.
14) **Not give any feeds using bottles or teats.**

## 29.2.1 HIV AND INFANT FEEDING PRACTICES GUIDELINES

All infants irrespective of HIV status, should be exclusively breastfed for the first 6 months of life. There should be a timely introduction of appropriate complementary foods after 6 months and continues breastfeeding up to 24 months and beyond.

Figures 29.1 and 29.2 illustrate the links between voluntary counselling and testing (VCT) and infant feeding. Figure 29.1 shows the various types of information that VCT opens up, while Figure 29.2 provides a schematic representation of the value of VCT as it affects mothers' infant feeding options. When mothers know their status, they can make informed choices about how to feed their babies.

**Table 29.1:Information links between VCT and infant feeding**

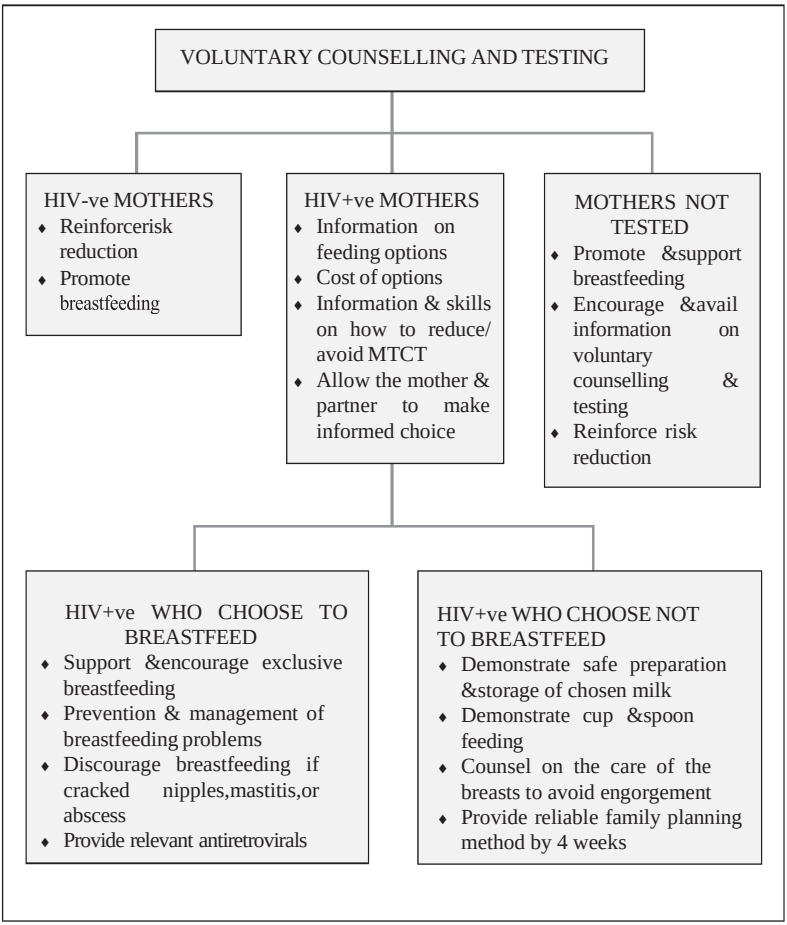
| ALL PARENTS | | |
|---|---|---|
| Information on benefits of breastfeeding<br>Prevention and management of breastfeeding problems*<br>Appropriate complementary feeding | Promote good maternal nutrition and self-care<br>Provide Vit. A supplements, iron, folic acid, and zinc<br>Counsel on child spacing<br>Treat infections promptly | Reduction of HIV infections<br>Risk of mother to child transmission (MTCT) of HIV<br>Information on voluntary counselling and testing<br>Reinforcing risk reduction to couple |

* Breastfeeding problems include abscess, mastitis, breast and nipple disease.

## Healthy Feeding through Childhood

Eating habits are established during the first 2 years. At 2 years, the child is eating family foods. Encourage child to eat, but respect the child's appetite without forcing them to eat. Adequate and balanced diet need to be followed. Up to age 5 years nutritious snacks are essential.

**Figure 29.1: VCT and the HIV-positive mother**




* For women who have features of clinical AIDS, manage as HIV positive.

Organized feeding through the school years may help to prevent hunger, which can affect the child's learning. In low cost schools, parents may offer food or service for the children. In high cost schools, food and snacks sold in school shops/canteens should be healthy. Parents and teachers are responsible for this.

## 29.3 Growth Monitoring and Growth Promotion

- ***Rates of growth:*** Rate of growth is highest in the first year of life and gradually reduces thereafter until child reaches puberty when there is another growth spurt that lasts 2–5years.

- ***Weight gain:***Term neonate aged 0–2 months gains 30g per day;an infant aged 2–6 months gains 20g per day. A child doubles birth weight at 5–6 months and triples birth weight at 12 months.
- ***Increase in height:*** Infants increase their height by about 25 cm in first year and 10cm in second year.
- ***Head growth:*** Head is measured by head circumference. At birth head circumference ranges between 33 cm and3 7cm. Thereafter,it increases by 2cm per month for the first 0–3 months; by 1cm per month from 3 to 6 months of age; and lastly by 0.5cm per month from 6 to 12 months of age. These increments add to a total of 12cm at the end of the first year. Eighty percent of the brain growth occurs in the first 2 years of life.
- ***Interpretation of changes in weight and height:*** Weight loss leads to wasting and is usually a sign of recent food shortage or illness. On the other hand, inadequate gain in height or length leads to stunting and is a sign of chronic lack of food or illness.
  - Body mass index (BMI) = weight in kg/(height in metres)$^2$.Children with BMI above the 85th percentile are overweight, while those above the 95th percentile are obese.
  - Head circumference below that expected for age is microcephaly and above expected is hydrocephaly or macrocephaly.

### 29.3.1 GROWTH MONITORING

As part of the Maternal and Child Health (MCH) programme, serial weight, height measurement and recoding should be done. All children have their individual growth curve. However, if they deviate from the curve the reason should be investigated.

Growth monitoring after 9 months is generally inadequate as parents and health care providers tend to associate clinic attendance with immunization. So after the measles vaccine at 9 months few mothers see the need to come to clinic unless the child is unwell. Also as the child grows bigger and may be the mother has a new baby, the older child is no longer a priority. Growth monitoring at community level has been in existence for a long time in Kenya, but is probably not widespread.

It is necessary to make growth monitoring an important community activity. This is because growth monitoring can help in detecting not only failure to grow well but also features of over-nutrition which can lead to obesity. Poor growth is detected by the regular use of the growth chart. As soon as a slowing growth is detected, action must be taken. The advice given to a mother depends on the age of the child.

The advice must be practical and the mother must do what she is told.The community health workers can be trained and supported to do this. Together with the parents, the health worker needs to visualize the growth of children and seek help if the child is not growing appropriately. All children up to age 5 should be weighed regularly – preferably monthly. To do this, they need weighing scales and tools for length/height measurement. Currently, charts are readily available only for children up to 5 years.

**When a Child Does Not Grow Well: Assess Nutritional Status**

The following classifications are important for parents to know about their children to assist them to avoid malnutrition:

**Table 29.2: When a child does not grow well: Assess nutritional status**

| Classification | Signs |
|---|---|
| Normal | No low weight for age and no other signs of malnutrition |
| Malnutrition | Very low weight Very low weight for age<br>Poor weight gain |
| Severe malnutrition | Visible severe wasting, "baggy pants"sign<br>Oedema of both feet |

**When a child does not grow well:**

- Assess the child's feeding.
- Ask what the child is fed on.
- Ask how many times the child is fed in a day.
- Counsel the mother on feeding. Review the recommendations in Table 29.3 against the child's growth chart, and discuss with the mother about any necessary changes.

**Follow up programme for child:**

- Review the progress of the child in 5 days.
- Re-assess feeding.
- Counsel mother about any new or continuing feeding problems.
- If child is very low weight for age, ask the mother to return 14 days after the initial visit to monitor the child's weight.
- Encourage the mother to continue the feeding programme until the child gains appropriate weight for age if after14 days the child is no longer very loW weight for age. And then advise her to maintain feeding the child an adequate nutritious, well balanced diet.

**Table 29.3: Feeding recommendations children with poor growth or lack**

| | Of growth | |
|---|---|---|
| **Age** | **Growth chart shows** | **Recommendations** |
| 0–6 months | Poor or no weight gain for 1 month | Breastfeed as many times as possible, day and night. Check that mother is breastfeeding properly and that her diet is adequate. |
| | Poor or no weight gain for 2 months | As above. In addition, the mother should be encouraged to eat and drink enough. Refer child for investigation. Child may have hidden illness. |
| 7–12 months | Poor or no weight gain | Breastfeed as often as child wants. Give adequate servings of enriched complementary feed at least 3 times a day if breastfed and 5 times if not breastfed. |
| 13–24 months | No/poor weight gain for 1 month | Continue breastfeeding. Check diet composition and how much child takes. Advise on how to enrich the food. Feed 3 main meals. Give snacks at least 2 times between meals. |
| | Poor or noweight gain for 2months | Continue feeding as above. Take history and refer. |
| 24 months and over | Poor or no weight gain | Child should eat half as much food as the father. Child should be encouraged to eat with other children, but should have an adequate serving of food served separately. Take history and refer. |

**Refer all children for further evaluation if:**

- Weight has not increased in the last 2 months eventhough the mother/ caregiver says they are following the advice on feeding practices.
- Sick children are not gaining weight adequately. (Sick children may need to be referred immediately for other reasons).
- Child continues to lose weight (consider TB, HIV infection among other problems).
- Child's weight is well below the bottom line on the chart.
- Child has any sign of swelling of the feet and face (Kwashiorkor) or severe wasting (marasmus).

**Advice to mothers should be:**

- Well babies less than 6 months old need no other milk or food apart from breastmilk.
- Adding oil,margarine,or sugar,and milk,egg,or mashed groundnuts makes uji and other foods energy rich and helps young children grow well.
- Feed often – like 5 times a day: small children have small stomachs.
- Feed older children at least 5 times aday.
- Feed sick children atleast one extra meal per day and continue for 1–2 weeks after they recover.
- Continue to take interest in what the child feeds on even in the school years.
- Mothers should know that the children are likely to have poor school performance if not fed well.
- Avoid overfeeding and limit nonnutritious snacks, especially if the child is overweight.

## 29.4 Development

Besides nutrition, children need appropriate stimulation in order to reach their development potential. Parents and health workers need to know the normal developmental milestones.

**Table 29.4: Developmental milestones**

| Milestones | Normal Limits |
|---|---|
| Social smile/follows a colourful object dangled before their eyes | 0 - 2 months |
| Holds the head upright / follows the object or face with their eyes / turns the head or responds in any other way to sound / smiles when you speak | 2 - 4 months |
| Rolls over / reaches for and grasps objects with hand / takes objects to her mouth / babbles (makes sounds) | 4 - 6 months |
| Sits without support / moves object from one hand to the other/ repeats syllables (bababa, mamama) | 6 - 9 months |
| Takes steps with support / picks up small object or string with 2 fingers / says 2-3 words / imitates simple gestures (claps hands, bye) | 9 - 12 months |
| Walks without support / drinks from a cup / says 7-10 words / points to some body parts on request | 12-18 months |
| Kicks a ball / builds tower with 3 blocks or small boxes / points at pictures on request / speaks in short sentences | 18 - 24 months |
| Jumps/ undresses and dresses themselves / says name, tells short story/ interested in playing with other children | 24 months and older |

In Kenya, 45% of children under 5 years are at risk for poor development. It is important to assess all risk factors that may contribute to such poor development such as undernutrition, maternal mental health and stress, hearing impairment, lack of safety and security, infections and metabolic illness.

Any child whose milestones are delayed needs careful assessment to identify the cause and offered appropriate therapy. Often, this requires referral to various services such as nutrition, social, ear and hearing care, occupational and paediatric clinical services. It is important to maintain a multidisciplinary

approach to the care or children with developmental delays as the causes are often multifactorial.

Counselling for care for child development should be done at every child welfare visit using the counselling guide provided in the Mother to Child Health Booklet. Children need simple culturally appropriate toys to play with. Parents can be taught how to make simple toys with materials available in the home. Encourage caregivers to spend time with their young children. Encourage caregivers to talk to their children often, including babies who may not understand the words, but will learn about interaction. Male parental involvement should always be encouraged.

# 30. Nutritional Disorders

## 30.1 Micronutrient Deficiencies

### 30.1.1 IRON DEFICIENCY

The commonest sign of iron deficiency is anaemia, which is discussed in Section 30.1. Iron deficiency negatively affects cognitive function. A school going child performs poorly at school long before iron deficiency anaemia manifests. Iron deficiency also increases risk of infection.

**Prevention**

Diet should consist of iron rich foods like dark green leafy vegetable (whose iron is poorly absorbed), meat, liver, and other animal sources (whose iron is easily absorbed).

### 30.1.2 IODINE DEFICIENCY

Iodine deficiency leads to deficiency of thyroxine since iodine is involved in the production of thyroxine. The thyroid gland may become enlarged in an effort to produce more thyroxine, leading to goitre.

**Prevention**

Consumption of iodized salt is adequate prevention against iodine deficiency.

### 30.1.3 VITAMIN A DEFICIENCY

Vitamin A is a retinol ester that can be either ingested or synthesized within the body from plant carotene. It is important in maintaining the integrity of skin and membranes, immunity, and night vision. Deficiency of vitamin A results in increased rate of infection, as well as increased mortality. In Kenya, about 75% of children aged below 5 years have vitamin A deficiency. Worldwide, vitamin A supplementation has been shown to result in 23–34% reduction of all childhood mortality (6–59 months), 50% reduction in measles mortality, and 33% reduction in diarrhoeal disease mortality. Vitamin A deficiency is a major cause of illness and blindness among poor communities worldwide.

**Eye Manifestations of Vitamin A Deficiency**

- Early signs include reversible dry cornea and night blindness.
- Later signs include irreversible damage of cornea – rupture and scarring, Bitot's spots (white areas on lateral parts of the sclera) and blindness also develops as a consequence of vitamin A deficiency.

**Prevention of Vitamin A Deficiency**

- Encourage families to consume vitamin A rich foods, which include:
  - Animal products, for example liver, milk and kidneys.

- Plant products, for example dark green leafy vegetables, yellow fruits and vegetables (carrots, pumpkin,pawpaw).

♦ Give vitamin A supplementation together with immunization.
♦ Give vitamin A supplementation routinely in the presence of the following conditions:
  - Malnutrition
  - Diarrhoea
  - Malaria
  - Tuberculosis
  - Pneumonia
  - Worminfestation
  - Fever
  - Measles

— **For children aged below 5 years, it is important to ensure that they have not received vitamin A in the last 1 month.**

**Treatment for Xerophthalmia**
Affected children are given vitamin A on day 1 and 2 and a third dose 1–4 weeks after second dose. Children suffering from measles should be treated as if they have xerophthalmia.

## 30.1.2 VITAMIN D DEFICIENCY

Although there are no data from national surveys, vitamin D deficiency is common in many parts of the country. Usually, it starts during the second half of the first year. For children who were born premature, the deficiency is diagnosed much earlier.

**Clinical Features**
Children present with poor growth, delayed or regressed milestones, recurrent pneumonias, widening of the wrists, and prominence of costo-chondral junctions (rickety rosary).

**Investigation**
♦ X-ray wrist – cupping of radius and ulna
♦ Serum calcium, phosphate
♦ Alkaline phosphatase
♦ Urine to exclude renal causes

**Management**
♦ Give vitamin D2 at 2,000–5,000 IU per day for 6–12 weeks or D3 at 0.05µg/kg/day.
♦ Supplements of calcium and phosphate will also be beneficial. Advise parents to expose their children to sunshine as a preventive measure against rickets.

**Prevention**
Children should be exposed to sunlight with minimal clothing for 30 minutes a day. For infants born preterm, supplementation with vitamin D at a dose of 4,000 IU/day is recommended. In addition, there should be provision for calcium and

phosphate in the diet, which is usually adequate from milk for the infant and young child.

## 30.2 Macronutrient Malnutrition

Macronutrient malnutrition presents as protein energy malnutrition (PEM). PEM is a common disorder which covers a wide spectrum of deficiency in nutrition ranging from mild or underweight to severe forms like marasmus and kwashiorkor. The first sign of PEM is poor weight gain.

**Clinical Features**

The clinical features of the two severe forms of malnutrition, kwashiorkor and marasmus, are itemized in Table 30.1. Each of the features varies from mild to severe. A child may have combination of features for both kwashiorkor and marasmus, and then be diagnosed to have marasmic kwashiorkor.

**Table 30.1: Clinical features of the two severe forms of malnutrition**

| Kwashiorkor | Marasmus |
|---|---|
| Pedal oedema | Very low weight forage |
| Low weight | Gross loss of subcutaneous fat |
| Apathy | "Wise old man look" |
| Poor appetite | Good appetite (if no complications) |
| Muscle wasting | Severe muscle wasting |
| Flaky paint dermatosis | |
| Hair changes (thin,sparse) | |

**Classification**

"Weight for height" rather than "weight for age" is now used for classifying malnutritionfor the sake of deciding on management options because weight is affected by stunting. It is known that a child who is less than 60% for their "weight for age" may be so mainly because of stunting and such a child does not need hospital treatment. Mid upper arm circumference (MUAC) can also add value.

Consequently, the following classifications are available for children with macronutrient malnutrition:

- Mild malnutrition: Child <5 yrs who is failing to gain weight for 2 months.
- Moderate malnutrition: Weight for height Z score between >-3 SD and <-2SD, MUAC >11.0cm and <12.5cm.
- Severe malnutrition: Weight for height Z score <-3SD,MUAC <11.0cm with or without oedema. If weight for height is not available,"visible severe wasting" is used to make a judgement.

Children with macronutrient malnutrition may have the following additional features or complications in varying degrees and combinations:

- Anorexia
- Lower respiratory infections

- Fever
- Hypothermia
- Vomiting
- Diarrhoea with or without dehydration
- Altered consciousness
- Severe anaemia

**Investigations**
- Mantoux test
- HIVtest
- Bloodsugar
- Haemogram
- Chest x-ray

**Management**
If clinically "well", that is, has good appetite and is alert, treat as outpatient with ready to eat therapeutic food. Advise mother to keep the child warm. Teach her how to feed the child at home. Review weekly until weight for height Z score is >- 2, MUAC >11.0cm, and there is no oedema. If not well or if any of the complications listed above are present, admit urgently for inpatient care. Specific management issues for the different classifications of malnutrition are given below.

**Mild malnutrition:**
- Advise the mother to bring the child to the clinic fortnightly for nutrition counselling and growth monitoring.
- Treat any intercurrent problem, e.g., diarrhoea, pneumonia,malaria.
- Check HIVstatus.

**Evaluate carefully if:**
- There is no change after 2 months. The child may have an underlying cause.
- Admit if the child develops moderate to severe malnutrition.

**Moderate malnutrition:**
Patients with this degree of malnutrition can be treated as an outpatient with food supplementation and nutritional counselling.

**Severe malnutrition:**
Such children should be assessed for the presence of complications: dehydration, shock, severe anaemia, hypoglycaemia, hypothermia, malaria, pneumonia, septicaemiaand mouth ulcers. If the children do not have any of these complications or problems, and have good appetite and are alert, they should be treated as outpatients with ready to eat therapeutic food. They should be reviewed weekly until weight for height Z score is > -2, MUAC >11.0cm and no oedema. If the children have the complications mentioned and/or have poor appetite and/or are not alert, look for other intercurrent problems like the presence of oedema that signifies kwashiorkor or marasmic kwashiorkor, and appropriately manage.

**Plan of Care (See Flow Chart, Figure 30.1, and Table 30.2)**

- Advise the mother to keep child warm.
- Ensure sufficient staff to provide feeds during day and night. Death often occurs at night because of hypoglycaemia.
- Initiate feeding within 2 hours of admission and feed every 2 or 3 hours throughout the 24-hour period until the child is out of danger. The child may need tube feeding in the first days of admission.
- Give all children with severe PEM a broad spectrum antibiotic.
- Update immunizations.
- Keep any skin ulcers clean; you can use antiseptic washes.
- Mouth ulcers: Clean mouth with normal saline (or salt water) and apply gentian violet.

**Feeding Regime**

- Initial phase:100kcal/kg/day; protein1–1.5g/kg/day; liquid 130ml/kg/day ***OR*** **100ml/kg/day if severe oedema.**
- After stabilization: Gradually increase intake to 150–200kcal/kg/day;protein2–4g/kg/day.
- Correct micronutrient deficiencies:
  - Multivitamins
  - Folic acid
  - Zinc
  - Vitamin A
  - Ferrous sulphate 3mg/kg/day after child has started gainin gweight

**Monitoring Response to Therapy**

- Weigh child daily:
  - Child with oedema: Weight loss initially, then weight gain of >10g/kg/day is expected. If weight gain is less than that, check feeding, re-examine for possible missed infection.
  - Child without oedema: Should gain weight as soon as good feeding is established.
  - Calculation of weight gain: Child's weight 3 days ago 6,000g; current weight 6,300g; weight gain=300g; daily weight gain=100g. Divide 100g by 6kg to get g/kg/day.
- Check for intercurrent problems daily.

**Figure 30.1: Symptomatic severe malnutrition**

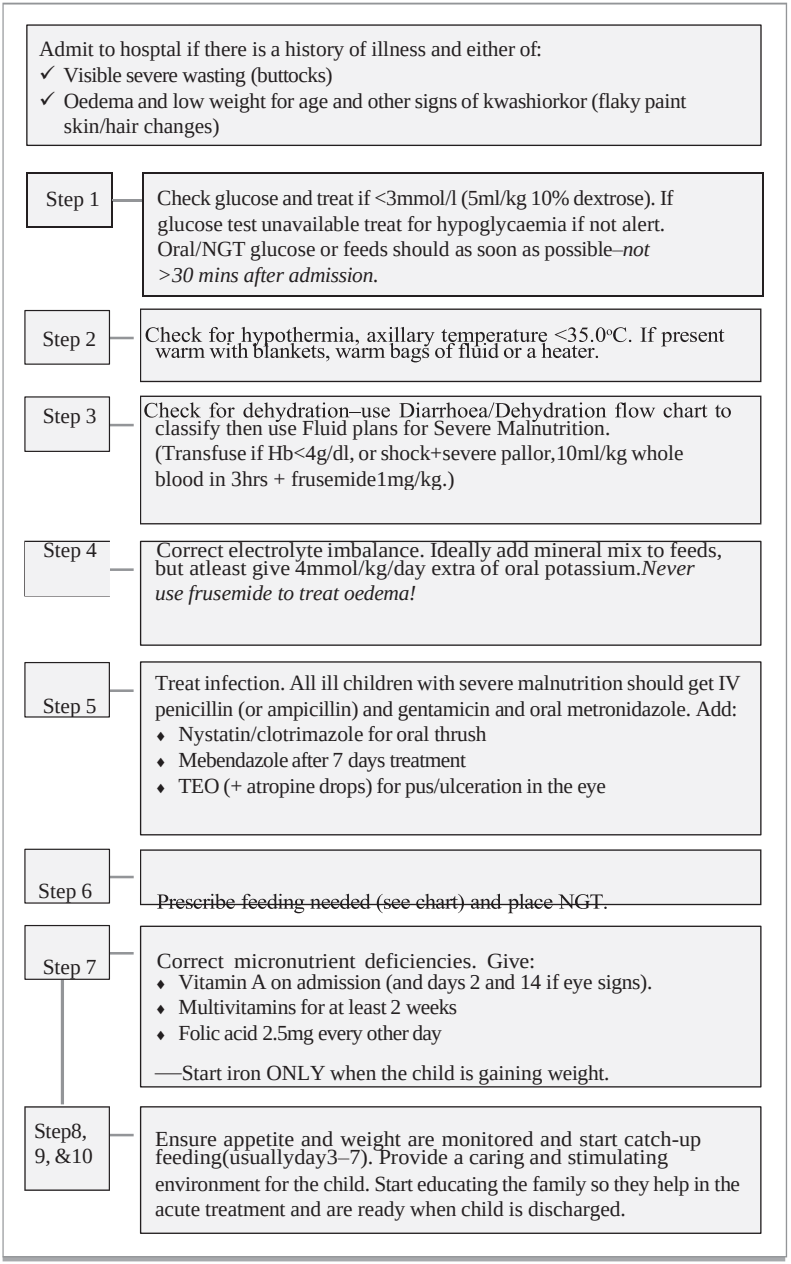


**Table 30.2: Time frame for care of seriously malnourished child**

| Arrival at health facility : Triage for danger signs and initiate treatmen t then admit | Stabilization | | Rehabilitation |
|---|---|---|---|
| | Days1–2 | Days 3–7 | Weeks 2–6 |
| 1. Hypoglycaemia | € | | |
| 2. Hypothermia | € | | |
| 3. Dehydration | € | | |
| 4. Electrolytes | € | | € |
| 5. Infection | € | € | € |
| 6. Micronutrients | noiron | with iron | € |
| 7. Initiate feeding | € | € | |
| 8. Catch up growth | | | € |
| 9. Sensory stimulation | € | € | € |
| 10. Counsel on feeding | € | € | € |

**Advice to Mothers**

- Explain the problems and involve the mother in the care of the child.
- Show the mother how well the child is doing on the weightchart.
- Nutrition counselling: Advise mother on how to mix nutritious food from the 3 food groups.
- Show her how to provide sensory stimulation once child is over acute phase and takes interest in surroundings.

**Prevention**

Preventive strategies for macronutrient malnutrition include the following:

- Appropriate nutritional advice in the MCH clinic (breastfeeding and complementary feeding), with emphasis on how to mix nutritious food from the 3 food groups.
- Showing mothers how to provide sensory stimulation to their children.
- Use of growth chart in the MCH clinic for all children aged below 5years.
- Health education to parents attending all health facilities and in the community on appropriate child rearing and feeding practices.
- Advocating for good hygiene in food preparation.
- Advocating for environmental sanitation.

**Admit to hospital if there is a history of illness and either of: visible and severe wasting (buttocks), or oedema and low weight for age and other signs of kwashiorkor (flaky paint skin/hairchanges).**

# 31. Children with Special Health Needs

## 31.1 Failure to Thrive

A child whose physical growth is significantly below expected for age is said to have"failure to thrive". Failure to thrive is placed in two categories,non-organic and organic.

### 31.1.1 NON-ORGANIC FAILURE TO THRIVE

In this category, the child is usually less than 5 years with no underlying medical condition. The failure to thrive may be due to maternal emotional problems e.g. the child may have been unwanted or there may be severe poverty. This form of failure to thrive could be a form of child abuse.



**Clinical Features**

Besides the size, child is often unkempt, has delayed social motor and speech development, and there is poor parent–child interaction.

### 31.1.2 ORGANIC FAILURE TO THRIVE

The child in this category of failure to thrive has an underlying medical condition that is usually a chronic illnesses, for example a chronic infection like TB, HIV, or kalaazar; major congenital malformations; or an endocrine or metabolic disorder.

A complete history including nutritional, social and growth monitoring is essential. In non-organic FTT the mother's history may be inconsistent, orshow no concern for the child. A thorough physical examination for all forms of failure to thrive is essential.

**Investigations**

- Stool for ova and cysts
- Haemogram and blood film
- Urea and electrolytes creatinine
- Urinalysis
- Mantoux test
- CXR – to rule out chronic chest infections
- HIVtest
- Additional tests as indicated

**Management**

- Feed the child depending on the degree of malnutrition.
- Treat the cause if known and treatable and counsel the mother on how to cope and manage at home.
- Counsel the mother in case of non-organic FTT to try to resolve the underlying issues.

## 31.2 Child Abuse and Neglect

Child abuse is maltreatment of children or adolescents by parents, guardians, or other caregivers. Early recognition is very important for prompt intervention. About 90% of child abusers are related caretakers who tend to be lonely, unhappy, angry and under heavy stress. Many of these caregivers might have experienced child abuse of one form of another during their own childhood. Abused children may have certain provocative characteristics, negativity, difficult temperament, offensive behaviour, or disability.

**Types of child abuse are in various forms, as itemized below:**

- Physical abuse (non-accidental trauma): This is the commonest form of child abuse. It manifests as physical injuries that include bruises, burns, head injuries, and bone fractures. Their severity can range from minor bruises to fatal injuries.
- Emotional abuse: This type of abuse is characterized by intentional verbal acts, criticisms, and lack of nurturing. This type is very difficult to prove.
- Nutritional neglect or deliberate underfeeding: This is associated with failure to thrive.
- Sexual abuse: This usually occurs with family members and is the most overlooked (or under reported)form of abuse. Types of sexual abuse include molestation, sexual intercourse, and rape.
- Others: These include intentional drugging (or poisoning) or neglect of medical care.

### 31.2.1 CLINICAL PRESENTATION

Physical abuse may manifest as unexplained inconsistent injuries and delay in seeking medical help for the injuries. Sexual abuse may remain concealed for fear of reprisal from the perpetrator. Oftenly, the victim (in this case the abused child) does not know what to do, where to report and how to go about it. Most victims report to a health facility due to acute stress or vaginal bleeding, STIs, UTI, enuresis, encompresis (faecal incontinence in absence of organic defect) or pregnancy. Children with nutritional neglect present with failure to thrive, poor hygiene, delayed immunizations, delayed development in speech, mental status and social interaction. Most abused children are shy with expressionless faces and tend to avoid eye-to-eye contact.

### 31.2.2 INVESTIGATIONS

For children who are suspected to be abused, the following are recommended:

- Thorough history and examination for all types of abuse, indicating who accompanies the child to the health facility.
- In physical abuse, total skeletal survey (x-ray–may find fractures at various healing stages) is recommended.
- Sexual abuse: Examine for sperms, acid phosphatase and infections, e.g., gonorrhoea. Rape cases may require examination under GA to determine the type and extent of genital injury.
- Nutritional neglect: Must rule out all other causes of failure to thrive.

## 31.2.3 MANAGEMENT

Admit the child for the following reasons:

- The diagnosis may be unclear, admission may be important for the child because of consideration for immediate safety, or the state of the child might require medical or surgical intervention.
- The need to remove the child from the source of the abuse in order to protect the child until the evaluation of the family with respect to the safety of the child is completed.
- The needs of the perpetrator for psychiatric evaluation and care
- The need to involve the police and the social worker for more effective management of the child.

**For children who experience rape or sodomy, the following needs to be done:**

- Sedate as necessary with phenobarbitone 5–8mg/kg/day or diazepam at 0.1–0.25mg TDS.
- Give prophylaxis for HIV/AIDS (see under HIV/AIDS).
- Carry out surgical repair of injuries (sphincter injury,which may require colostomy with secondary repair).
- Counsel the child.

## 31.2.4 PREVENTION

Health workers should have a high index of suspicion on the likelihood of abuse. Older children should be encouraged not to keep “secrets” and to refuse any enticement to what could potentially be sexual abuse. Children who are in a high-risk situation should be removed from that environment and not left there.
Referral for these children is necessary for long-term psychological and psychiatric care.

# 32. Gastrointestinal Conditions Other than Diarrhoea

## 32.1 Infestation with Worms

The focus here is on nematodes although worm infections comprise a large group of parasitical cestodes, schistosomes, flukes, nematodes, and filarial worms. Among these are hookworm disease, ascariasis, enterobiasis, trichuriasis, trichostrongyliasis, anisakiasis, capillariasis, and gnathostomiasis. Even so, only a few of these are included. Table 33.1 summarizes the most common worm infections with their clinical features and the method of detection, and Table 33.2 presents the preferred drugs and dosages for treatment of worm infestations.

**— De-worm children above 2 years at least every 6 months with albendazole 400mg STAT.**

**Prevention**

Appropriate prevention depends on the particular worm. In general, the following measures should be instituted:

- Providing safe water
- Washing hands and trimming finger nails
- Changing inner wear and sheets frequent
- Using latrines

**Table 32.1: Specific worm infestations, their clinical features, and investigations required for diagnosis**

| Worms | Clinical features | Investigations |
|---|---|---|
| Ascaris lumbricoides (roundworms): Large round, cream coloured worms that live in the small intestines | ◊ Infection by swallowed embryonated eggs<br>◊ Loeffler'ssyndrome<br>◊ Mild bouts of recurrent colic<br>◊ The mother has seen the worm in stool or vomitus<br>◊ Complications such as obstruction, vomiting may occur | Stool for ova |
| Hookworms | ◊ "Grounditch"<br>◊ Features of anaemia (iron deficiency) | Stool for ova<br>Haemogram |
| Trichuris trichiura (whipworms) | ◊ Diarrhoea with blood<br>◊ Rectal prolapse<br>◊ Anaemia<br>◊ Wasting | Stool for ova<br>Worms may be seen adhering to rectal mucosa |
| Strongyloidess tercoralis | Most infections are asymptomatic but the following may occur:<br>◊ Larva currens (buttocks)<br>◊ Soiling of inner wear with stool<br>◊ Hyperinfection syndrome<br>◊ Diarrhoea<br>◊ Gram-negativesepticaemia<br>◊ Bacterial peritonitis<br>◊ Encephalitis | Direct stool microscopy (motile larvae, adult worms |
| Enterobius vermicularis oxyuriasis (pin worm) Synonyms: Threadworm, pinworm, seatworm.<br>The worm is 4mm long and is just visible to the human eye | Mode of spread<br>*Auto-infection:*<br>◊ Direct anal to mouth transfer via thefingernails<br>◊ Retro- infection; eggs may hatch into larvae at the anal-rectal area. Then larvae move retrogradely to thecaecum.<br>*Cross infection:*<br>◊ Contamination of fingers by cloth-ing, objects, toilet seats,etc.<br>◊ By inhaling and swallowing eggs in the dust<br>◊ Main presentation: perianal and perineal itching. Migrating larvae may cause:<br>◊ Vaginitis, vulvitis, salpingitis, and peritonitis<br>◊ Irritation, insomnia may occur | Stool for ova<br>Ova can be obtained from the perianal region by use of adhesive tape |
| Taenia saginata (beef tapeworm) | ◊ Non-specific symptoms,irritability<br>◊ Segment may be passed with stools<br>◊ Egg in stools | Stool for ova (motile proglottides) |

**Figure 33. 1: Drugs and their dosages for worm infestations**

| Worms | Adults | Children |
|---|---|---|
| Ascaris lumbricoides (Roundworm) | Levamisole 2.5mg/kg as a single dose *OR* Albendazole 400mg STAT | Levamisole 2.5mg/kg as a single dose *OR* Albendazole 200mg STAT for children under 2 years |
| Hookworm | Levamisole 2.5mg/kg as a single dose *OR* Albendazole 400mg STAT | Levamisole 2.5mg/kg as a single dose Albendazole 200mg STAT for children under 2 years + ferrous sulphate |
| Trichuris trichiura (whipworm) | Albendazole 400mg STAT | Albendazole 200mg STAT for children under 2 years |
| Strongyloidesstercoralis | Albendazole 400mg BD x 3 days *OR* Thiabendazole 25mg/kg x 3 days | Albendazole 200mg BD x 3 days *OR* Thiabendazole 25mg/kg x 3 days |
| Enterobius vermicularis (pinworm) | Albendazole 200mg STAT for children 12 months up to 24 months and 400mg STAT for children above 2 yrs | Albendazole 200mg STAT for children 12 months up to 24 months and 400mg STAT for children above 2 years |
| Taenia saginata (beef tapeworm) | Niclosamide 2g; 1g before breakfast, 1g 1 hour after breakfast | >6 years 1g before & 1g after breakfast<br>2–6 years 500mg before and 500mg after breakfast<br>< 2 years 250mg before and 250mg after breakfast |



## 32.2 Amoebiasis

This is an infection usually of the colon by Entamoeba histolytica. Most of the people infected by Entamoeba histolytica are asymptomatic cyst carriers.

### Clinical Features

**Two diseases that are caused by Entamoeba histolytica are amoebic** dysentery and amoebic liver abscess.

- Amoebic dysentery: This presents as bloody diarrhoea, and depending on the severity of infection there may be varying degrees of dehydration.
- Amoebic liver abscess: This presents as intermittent fevers, night sweats,and tenderness in the right hypochondrium. Some patients may have difficulty breathing. The abscess may rupture into the chest, causing empyema or into the abdomen causing peritonitis.

### Investigations

- Stool for microscopy – Trophozoite with ingested RBC in amoebic dysentery
- Full haemogram – Liver abscess (leucocytosis, mild anaemia)
- Chest x-ray – Elevation of the right hemidiaphragm liver abscess
- Abdominal ultrasound will show abscess in liver

**Management**

- Amoebic dysentery: See Section 18, on diarrhoea.
- Admit if liver abscess is suspected and start treatment for amoebic liver abscess:
  - Metronidazole 30–50mg/kg/day in 3 divided doses for 7–10 days
  - Aspiration or surgical drainage of pointing liver abscesses is indicated to prevent spontaneous rupture in pointing abscesses.
- Asymptomatic cyst carriers:
  - Treat cyst carrier in food handlers only. Use diloxanide furoate-metronidazole (e.g.,entamizole).

**Prevention**

- Provision of safe drinking water and sanitary disposal of faeces are important preventive measures.
- Regular examination of foodhandlers and appropriate treatment when necessary are needed, including in schools.

## 32.3 Schistosomiasis

This is an infection with blood flukes of the genus Schistosoma, which may cause chronic disease of intestines, liver, and genitourinary tract. Adult flukes are white worm-like creatures that inhabit parts of the venous system of man. All the worms need molluscan intermediate host. Important species of schistosomiasis in Kenya are Shistosoma haematobium and Shistosoma mansoni. Adult worms live and copulate within the veins of the mesentery. The sexually mature worms are mainly found in the intestinal veins for Shistosoma mansoni, while those of Shistosoma haematobium are mainly located in the venous plexus of the genitourinary tract. Eggs that are laid penetrate the intestinal or bladder mucosa, pass into the lumina, and are passed in faecesor urine. Once passed, the eggs hatch in fresh water, liberating cercariae that multiply in snails (the intermediate host) and produce thousands of cercariae. These penetrate human skin within a few minutes after exposure and transform into schistosomes, which develop into sexually active adult worms in the intestinal veins or venous plexus of the genitourinary tract depending on the species.

An adult worm's lifespan ranges from 3 to 37 years. Shistosoma haematobium is common along the coastline, especially along Tana River, Kwale and Lamu. Shistosoma mansoni, on the other hand, is widespread, and occurs particularly in Machakos, the rice schemes, parts of Nyanza, and even Nairobi.

**Clinical Features**

Acute dermatitis and fever after exposure is a rare presentation. Occasionally transverse myelitis and convulsions may occur. Chronic schistosomiasis is the main presentation in Shistosoma mansoni, manifesting with portal

hypertension, splenomegaly, anaemia, and oesophageal varices. On the other hand, Shistosoma haematobium may present with terminal haematuria and dysuria and may progress to obstructive uropathy; bladder cancer has been noted as a late complication in some patients. Metastatic eggs can be found in other organs such as the spinal cord and the brain. It has also been noted that Salmonella infection, presenting as recurrent pyrexia, is difficult to eradicate until schistosomiasis has been treated.



**Investigations**

- For Shistosoma mansoni:
  - Stool for ova, use concentration or Kato technique rectal snip.
  - Barium swallow and endoscopy to demonstrate oesophageal varices
  - Abdominal U/S
  - Liver biopsy if indicated
- For Shistosoma haematobium:
  - Urine for RBC and for ova of S.Haematobium hatching test
  - X-ray of lower abdomen may show calcified bladder (sandy patches)
  - Intravenous urogram when obstructive uropathy is suspected

**Management**

Shistosomiasis should be treated with praziquantel 20mg/kg BD for one day (effective against all types). Patients should be examined for living eggs and if positive given another course of treatment.

**Refer to specialist if:**

- There are features of obstructive uropathy.
- There are features of portal hypertension.

**Prevention**

Preventive strategies against schistosomiasis include the following:

- Avoid contact with contaminated water.
- Give mass chemoprophylaxis to school age children in endemic areas.
- Improve environmental hygiene, for example by advocating for the use of toilets by communities.
- Eradicate snails, which are the intermediate hosts.

## 32.4 Gastrointestinal Bleeding

**Clinical Features**

Gastrointestinal bleeding may present as blood in vomitus or in stool. In either case, there may be frank red blood or altered blood that would appear as coffee grounds or there may be black stool. Bleeding could occur from the upper or lower gastrointestinal tract. The amount of bleeding varies depending on the cause of bleeding. Massive bleeding can present with features of shock.

Among the common causes of features of upper gastrointestinal bleeding are:

- For the newborn
  - Swallowed maternal blood: In this situation the baby looks well.
  - Stress ulcers often following birth asphyxia.
  - Coagulopathy: DIC associates with asphyxia, sepsis, vitamin K deficiency.
  - Necrotizing enterocolitis (NEC)– more common in sick preterm infants.
- Infants and children
  - Swallowed blood following epistaxis (history of epistaxis).
  - Gastritis.
  - Oesophageal varices.
  - Gastric/duodenal ulcers.

**For all ages, the common causes of lower gastrointestinal bleeding include the following:**

- Anal fissure
- Infectious diarrhoea (including NEC in neonates,shigella,campylobacter, salmonella, amoebiasis, and schistosomiasis).
- Coagulopathy due to bleeding disorders that include liver disease and DIC.
- Intussusception that is more common in infants and young children.

**Investigations**

- Full blood count and blood film
- Group and cross match if excessive bleeding
- Stool for occult blood
- Stool culture or microscopy as indicated
- Specific tests according to suspected cause of bleeding:
  - Endoscopy
  - Barium swallow or meal or enema
  - Septic screen
  - Abdominal x-ray for neonate with suspected NEC
  - Coagulation screen
  - Liver function tests
  - Abdominal ultrasound

**Management**

- Initiate treatment for shock (refer to Section 17.6).
- Monitor vital signs half hourly until bleeding stops.
- Transfuse as soon as blood is available.
- Use nasogastric suction to assess blood loss and monitor continued bleeding.
- Be ready to give more blood when needed.
- Investigate and treat the underlying condition.

## 32.5 Vomiting

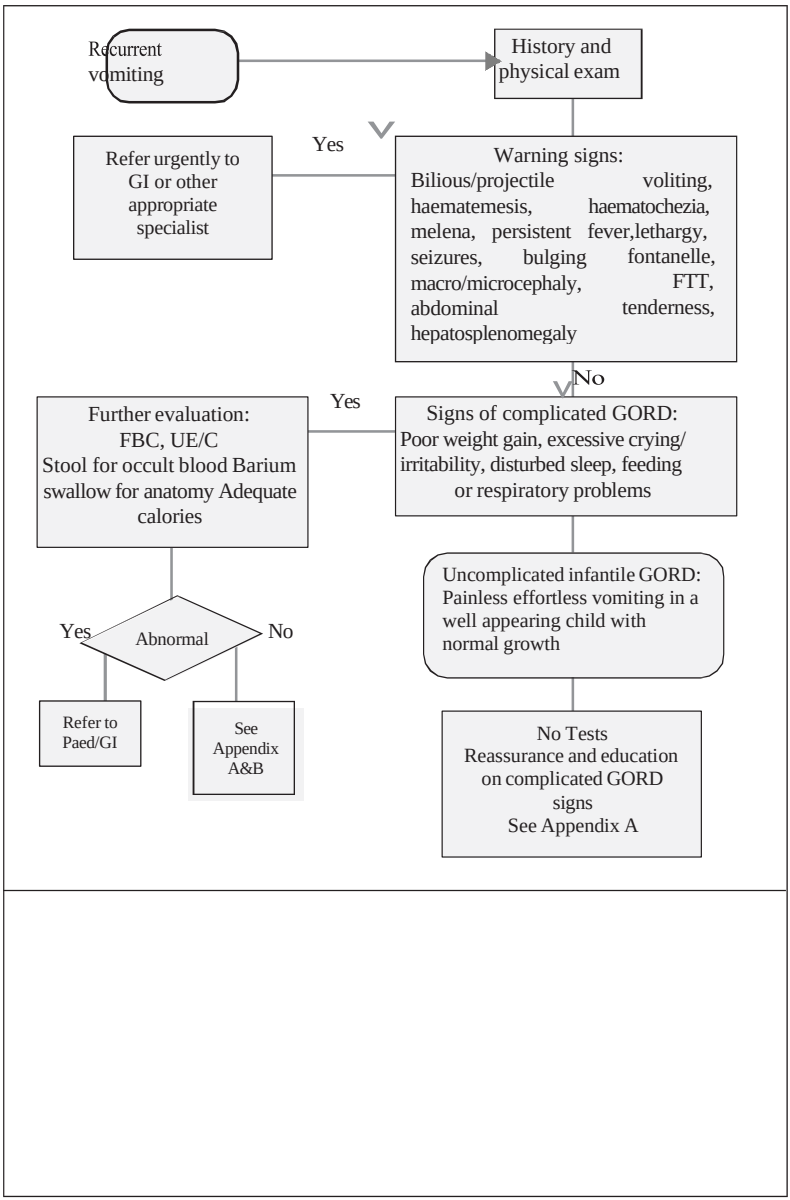
**Clinical Features**

Vomiting in children may be due to a systemic infection or may accompany diarrhoea, as it often happens. It should be noted that some normal babies regurgitate milk regularly and are clinically normal with normal growth. These are not considered to be having a vomiting problem. Vomiting may also be due to upper gastrointestinal tract obstruction, and may be the primary presentation for this condition.

**The common causes of vomiting include the following:**

- For early infancy
  - Gastro-oesophageal reflux disease (GORD), which initially presents as painless and persistent vomiting (see Figure 32.1).
  - Pyloric stenosis that presents with projectile vomiting and with a mass palpable in the right upper abdominal quadrant in the affected children.
  - Congenital upper gastrointestinal obstruction.

**Figure 33.2: Gastro-oesophageal reflux disease (GORD)**



- Later infancy/early childhood
  - Intussusception that presents with intermittent acute pains and blood in the stool. A mass maybe palpable in the abdomen.

**Investigations**
- Full haemogram
- Serum electrolytes
- Plain abdominal x-ray supine and erect or dorsal decubitus views
- Abdominal ultrasound
- Upper GI series
- Endoscopy

**Management**
- Avoid antiemetics.
- Treat non obstructive causes appropriately.
- Initiate rehydration according to degree of dehydration, using normal salin ein the acute phase.
- Arrange to transfer to surgical unit urgently all children suspected to have gastrointestinal obstruction and gastro-oesophageal reflux disease syndrome.

**Reflux precautions:**
- Head up 30 degrees, side position to sleep
- Upright after feeding for 30 minutes
- Minimal handling after feeding
- Small frequent feeds
- Thicken feeds

**Medications:**
- Acid suppression: Omeprazole 0.5 – 1.0mg/kg/day (maximum 30mg/day)
- Prokinetic: Domperidone 0.1mg/kg/day

**Refer if symptoms persist despitetreatment.**

## 32.6 Peptic Ulcer Disease

This refers to ulceration of gastric or duodenal mucosa that tends to be chronic and/or recurrent.

**Clinical Features of Duodenal Ulcer**
Duodenal ulcer has the following features:
- Presents with epigastric pain that is typically nocturnal and also when the patient is hungry.
- May present for the first time with complications as described later int his section.
- There is a wide individual variation in presenting symptoms and in the foods that give pain or discomfort when eaten.
- 95% of duodenal ulcers are caused by Helicobacter pylori (H.pylori).

**Clinical Features of Gastric Ulcer**

Gastric ulcer presents with:

- Epigastric pain that is worse after eating food.
- Other symptoms are similar to those for duodenal ulcers.

**Investigations**

- Stool for occult blood
- Barium meal
- Upper GIT endoscopy, where available and biopsy gastric mucosa for H.pylori

**Complications**

Chronic blood loss may lead to iron deficiency anaemia, and acute bleeding results in haematemesisor melaena stool**.**

**Management**

- Avoid any foods that to the patient's experience give pain.
- Avoid obviously acidic foods, e.g., Cola drinks.
- Avoid gastric irritating drugs (NSAIDs).
- Give magnesium-based antacids or combined magnesium-aluminium compounds, liquid preferred. Adjust dose to limit pain.
- Eradicate H. pylori by triple therapy:
  - Omeprazole start at 1mg/kg/day PO once daily or divided 12 hourly (max 20mg/d) 14 days.
  - Clarithromycin 7.5mg/kg/dose PO 12 hourly for 14 days or metronidazole 15-20mg/kg/day divided 12 hourly
  - Amoxicillin 25–50mg/kg 12 hourly 14 days 14 days
- Refer/consult if there is severe haemorrhage.
- Make sure you stabilize the patient before transfer.
- Infuse fluids/blood to maintain normal pulse.
- Continue to assess for any further loss of blood as evidenced by:Persistent tachycardia, postural hypotension, continuing haematemesis.

— **Note:Eradication of H.pylori leads to healing and most patients will not need long-term treatment. Complications will also be avoided.**

## 32.7 Constipation and Encopresis

**Clinical Features**

- Constipation is failure to open bowels regularly and is often accompanied by painful passage of hard stool. It may be associated with soiling of pants.
- Encopresis is intermittent leakage of soft/watery stool in a child with chronic constipation.
- Constipation may be caused by obstructive lesions (these include congenital or acquired defects), neurological or endocrine abnormalities (hypothyroidism), or they may be functional.

Note: Exclusively breastfed infants may take several days without passing a stool. But when they do the stool is soft. This should not be confused with constipation.

**Investigations**
- Abdominal x-rays in suspected obstructive lesions
- Barium enema when indicated
- Thyroid function tests when indicated
- Investigate and treat nonfunctional lesions

**Management**
Children with perceived constipation are often treated at home with herbs and even enemas. Such treatment may make it difficult to diagnose this condition and may lead to some complications. However, the inclusion of bananas or pawpaw in the diet may be beneficial, especially in increasing fibre intake.
Treatment of functional constipation is in three stages:
- Disimpaction (2–5 days): Mineral oils taken orally is the preferred treatment but daily enemas using magnesium salts can also be used. In general tryto avoid enemas.
- Sustained evacuation (about 3 months): this aims to restore normal bowel function. Child is encouraged to use toilet at regular intervals with positive rewards; diet is gradually modified to a low diary, high fibreone once disimpaction is achieved. Occasionally fibre supplement (1g/yr/day) may help. Encourage good water intake. Laxatives and stool softeners are used in conjunction with diet.
- Gradual withdrawal of medication while maintaining bowel habits and diet.

**Refer**
- All children with suspected non functional lesions
- Any child that fails to respond to above treatment
- Children in need of psychological counselling

# 33. Disorders of the Liver and Spleen

## 33.1 Hepatosplenomegaly

Liver enlargement is reported to have occurred when the liver measures more than 3cm below the costal margin or has a liver span greater than normal for age. Enlargement of the spleen, on the other hand, is reported to have occurred if the spleen is "just palpable". Causes of these conditions are summarized in Table 33.1

**Investigations**

- Full blood count and bloodfilm
- Liver biopsy when indicated
- Bone marrow if needed
- Specific tests will depend on the suspected cause listed in the table

**Table 33.1: Causes of hepatosplenomegaly**

| Category of causes | The specific causes associated with hepatomegaly | The specific causes associated with splenomegaly |
|---|---|---|
| Infections | Malaria kala azar<br>Schistosomiasis<br>Infectious hepatitis<br>Amoebic hepatitis/abscess<br>Brucellosis | Malaria/tropical splenomegaly<br>HIV<br>Kala azar (leishmaniasis)<br>Schistosomiasis<br>Infectious hepatitis<br>Brucellosis<br>Other infections, like SBE, typhoid fever, infectious mononucleosis |
| Blood | Haemolytic anaemia<br>Leukaemia | Haemolytic anaemia, e.g., sickle cell anaemia in child <3 years autoimmune haemolytic anaemia Leukaemia |
| Nutrition | Kwashiorkor | Iron deficiency |
| Congestion | Cardiac failure | Portal vein thrombosis |
| Metabolic disorders | Gaucher's disease | Gaucher's disease |
| Other | Liver tumours | Liver cirrhosis (portal hypertension) |
| | Displaced rather than enlarged liver | Juvenile idiopathic arthritis,SLE |

**Management**

- Make sure you identify the cause and treat accordingly
- Admit
  - If patient is severely anaemic– may need transfusion
  - If patient is febrile
  - For invasive diagnostic tests

## 33.2 Jaundice after the Neonatal Period

- Definition: Yellow discolourationof skin and mucous membranes due to excess bilirubin. It is also referred to as hyperbiliribinaemia, usually with serum bilirubin at that time of >2mg/dL (34.2mmol/L).
- Jaundice is a clinical feature and not a diagnosis. Any patient with jaundice should be carefully evaluated to determine the cause of the jaundice so as to institute appropriate management.
- Hyperbilirubinaemia is categorized according to the location of the abnormality in the metabolism and excretion of bilirubin:pre-hepatic,hepatic,or post-hepatic:
  - Pre-hepatic: This is due to excess intravascular release of bilirubin, often by haemolysis)
  - Hepatic: This is due to hepatocyte dysfunction with faulty uptake, metabolism, or excretion of bilirubin.
  - Post-hepatic: This is due to blockage of bile and its constituents so that they do not exit from the biliary system; this may result from common bile duct obstruction or intrahepatic cholestasis).
- The common causes of hyperbilirubinaemia include viral hepatitis,haemolytic anaemia (e.g., sickle cell, malaria), cirrhosis, biliary obstruction, hepatoma, drug induced reactions.

**Clinical Features**

Meticulous history and physical examination are important before ordering investigations. The history should include exposure to hepatotoxic drugs, known history of haematological disorder, history suggestive of viral hepatitis(anorexia, nausea,and aversion to fattyfoods), history suggestive of obstructive jaundice (of dark urine, pale stool and pruritus).

Physical examination should look for features suggestive of cirrhosis (spider naevi, gynaecomastia, loss of axillary hair, parotid gland enlargement and ascites) or features suggestive of parenchymal liver disease or haemolytic jaundice (splenomegaly). In hepatic encephalopathy in children the early signs may be mild and easy to miss. Child becomes slow and may have disturbed sleep-wake pattern. This progresses through drowsiness, arousable sleep to unconsciousness.

**Investigations**

- Full haemoglobin – Polymorphonuclear leucocytosisis found in infections including leptospirosis. Sickle cells maybe seen in the peripheral blood smear
- Reticulocyte count–Increased reticulocyte count indicates a haemolytic anaemia.
- Blood slide for malaria parasites–***Jaundice in a patient with malaria is a medical emergency.***
- Urine –Bilirubin:
  - Absence of bilirubin in a patient suggests haemolytic anaemia.
  - Presence of bilirubin suggests hepatobiliary jaundice.
- Urine –Urobilinogen:

- Excessive urobilinogen suggests haemolysis. Urobilinogen is absent in obstructive jaundice.

♦ Liver function tests:
  - Gamma Globulin Transaminase (GGT)–Elevated levels suggest primary liver disease.
  - Alkaline phosphatase – Elevated levels suggest obstruction.
  - SGOT (AST) – Elevated levels suggest hepatocellular damage.
  - SGPT(ALT)–Elevated levels suggest hepatocellular damage more specific than AST.
  - Serumproteins:
  - Albumin – Low levels in chronic liver disease such as cirrhosis.
  - Globulins–Hyperglobulinaemia is found in chronic active hepatitis, cirrhosis.

♦ If above investigations are not diagnostic consider:
  - HBsAg,HAV-Ab.TORCHES in young infants.

♦ Ultrasound: Useful in obstructive jaundice, gall stones, differentiating between abscess and tumour.

♦ Alpha-foetoproteins: Substantial elevations of alpha-foetoproteins are found in malignancy.

♦ Paracentesis of ascitic fluid: Protein content <3g% is found in cirrhosis. Protein content >3g% is found in tuberculosis, peritoneal tumours, peritoneal infection or hepatic venous obstruction. Blood stained ascites usually indicates a malignant disease – cytology is mandatory.

♦ Liver biopsy is important in diagnosis of chronic hepatitis,cirrhosis,and hepatocellular malignancy.

**Management**

♦ Patients with history and physical findings suggestive of viral hepatitis can be managed as outpatients requiring advice on bed rest and should be given multivitamins.

♦ Conduct diagnostic evaluation and manage according to cause.

♦ Admit if patient shows signs of encephalopathy. Refer/consult if not able to manage.

♦ Consider hepatic encephalopathy in any patient who has jaundice and mental complaint. Early treatment of hepatic encephalopathy may reduce mortality.

## 33.3 Obstructive Jaundice beyond Neonatal Period

This refers to jaundice caused by obstruction of bile in the biliary tree. It can be due to intrahepatic or extrahepatic causes.

**Clinical Features**
**These include the following features:**

- Jaundice and pruritus,which can be severe, with steady increase in jaundice
- Distended gall bladder
- Anorexia
- Troublesome diarrhoea with pale, foul smelling stool.
- Dark urine with a history of flatulence.

**The causes of obstructive jaundice include the following:**

- Those that are intraluminal include gallstones,which can dislodge from the gallbladder and get impacted in the common bile duct (CBD), and helminthiasis, especially ascaris and liver flukes.
- Those within the wall or mural include primary sclerosing cholangitis.
- Those acting outside the wall or extramural include enlarged lymph nodes of any cause, and neoplasms.
- Other causes include iatrogenic trauma to the ducts during surgery (especially cholecystectomy).

**Investigations**

- Full haemogram
- Liver function tests
- Prothrombin time index
- Plain abdominal x-rays may show stones
- Abdominal ultrasound and CTscan

**Management**
Appropriately manage the conditions diagnosed and refer those that require surgical management.

# 34. Haematologic Conditions

## 34.1 Anaemia

Patients with anaemia have a reduction in total red blood cell mass, decreased, concentration of red blood cells (RBC) and reduced haemoglobin (Hb) in the peripheral blood, resulting in a corresponding decrease in the oxygen carrying capacity of the blood. The average normal haemoglobin levels for the various ages in childhood are shown in Table 35.1.

**Table 34.1: Average normal haemoglobin levels in childhood**

| Age category in childhood | Average haemoglobin level |
|---|---|
| Newborns | 14g/dl |
| Children aged under 5 years | 10g/dl |
| Children aged 5–9 years | 11g/dl |
| Children aged 9 years and above | 12g/dl |

**Anaemia except in the newborn may therefore be classified as follows as follows:**

- Severe anaemia being haemoglobin below 5g/dl.
- Moderate anaemia being haemoglobin between 5g and 8g per decilitre.
- Mild anaemia being haemoglobin above 8g/dl but below normal for age category.

**The common causes of anaemia in Kenya are the following:**

- Haemolysis of red blood cells caused by infections like malaria or congenital abnormalities like haemoglobinopathies exemplified by sickle cell disease.
- Iron deficiency as a result of chronic blood loss due to bleeding or loss following parasitic infestation like hook worm or nutritional deficiency of iron.
- Reduced production of red blood cells by the bone marrow due to depression of its function by chronic illness, infection, infiltration or just failure to produce blood cells (aplasia).

**Clinical Features**

- Meticulous history and examination are essential in order to identify the cause of the anaemia.
- Pallor of the palms is a useful indicator of anaemia and is classified into two categories:as “some pallor” for mild to moderate anaemia and “severe pallor” for severe anaemia.
- Other features of severe anaemia include irritability, listlessness, anorexia, easy fatigability, heart failure, and shock. Other clinical features depend on the underlying cause of the anaemia.

**Investigations**

- Full haemogram include reticulocyte count if haemolysis is suspected
- Thin blood film examination for cell morphology and blood parasites
- Stool for ova of helminths, occult blood
- Sickling test/Hb electrophoresis if indicated.

- Bone marrow
- Urinalysis.
- Others depending on suspected cause

**Management**

- Admit all patients with severe anaemia or those who fail to respond to treatment as outpatients for appropriate investigation and management.
- For anaemia due to malaria, manage the malaria according to the guidelines given in Section 20. In addition, do the following:
  - Folic acid: Give to all patients who have malaria and anaemia
    - Below 2 years of age 2.5mg daily for 3 months
    - Above 2 years of age 5mg daily for 3months
    - Continue with the doses once weekly as for malaria prophylaxis above
- For anaemia due to iron deficiency, do the following:
  - If severely anaemic admit and manage appropriately
  - If the anaemia is mild or moderate and the child is not severely malnourished give iron and folate orally.
  - Review the child every 2 weeks.
  - If severely malnourished, delay giving iron until recovery phase of malnutrition.
- For anaemia due to worm infestation, de-worm using albendazole or mebendazole.
- Continue iron therapy until normal haemoglobin is achieved, usually after 3 months of treatment (1 month's treatment corrects the anaemia while the other 2 months treatment is used to build up iron stores). The dose of iron is usually 6mg/kg/day of elemental iron (or 30mg of ferrous sulphate, which contains 6mg of elemental iron) to a maximum of 200mg 3 times a day.
- If patient is not able to tolerate oral iron or if compliance is poor, consider iron dextran as total dose infusion:
  - Dose of dextran iron in mg=(normalHb–patient'sHb)×1,000.Give as total dose infusion. This also replenishes body stores of iron.

— **Do not give iron in the presence of sickle cell disease, so as to avoid excessive iron load in the body, which might result in toxicity.**

- Advise mothers to give a balanced and adequate diet to all children. Iron and folate containing foods include meat, fish, eggs, dark green leafy vegetables, and fruits.
- Refer and consult if:
  - Anaemia is not improving after treatment for a month.
  - No cause for anaemia has been identified.

## 34.2 Sickle Cell Anaemia (Disease)

This is a chronic haemolytic anaemia found mainly in Nyanza, Western and Coast regions. It is characterized by sickle-shaped red blood cells as a result of homozygous inheritance of Haemoglobin S. Because sickled red blood cells are fragile and cannot withstand the trauma of being squeezed through capillaries during circulation, haemolysis occurs in the small blood vessels. These abnormal red blood cells are also destroyed within the spleen.



**Clinical Features**

Symptoms of sickle cell disease or anaemia usually start around the age of 6 months and include the following:

- Pain and swelling of the hands and feet (hand and foot syndrome).
- Anaemia and mild jaundice.
- Impaired growth and development.
- Susceptibility to infections (including malaria, Haemophilus influenza, Streptococcus pneumoniae).
- Hepatosplenomegaly.
- Acute splenic sequestration of blood with resultant cardiovascular collapse
- As the child grows pain predominate, being experienced as:
  - Bone pain, involving the long bones, the back, and thehead.
  - Severe abdominal pain with vomiting.
- Acute chest syndromes (sudden onset of fever, chestpain,leucocytosis and pulmonary infiltrates on x-ray) that may be fatal.

**Other features of sickle cell disease include:**

- Aplastic crisis
- Priapism (painful erection of the penis)
- Hyperhaemolytic crisis
- Impaired renal function
- Avascular necrosis of the femoral head is common
- Occlusion of major intracranial vessels that may lead to hemiplegia cranial nerve palsies and other neurological deficits
- Bossing of the skull that might be “Tower shaped”skull.

**Investigations**

- Newpatients
  - Full haemogram to include peripheral smear.
  - Sickling test
  - Hb electrophoresis
- At other times these will be determined by type of presentation
  - X-ray: To exclude osteomyelitis,pneumonia
  - Full haemogram
  - Blood cultures
  - CTscan
  - Renal function

**Management**

Management options for sickle cell disease include:

- Maintaining adequate diet to prevent growth failure due to malnutrition.
- Ensuring adequate hydration, therefore avoiding dehydration by encouraging the child to drink as much as possible.
- Avoiding exposure of the child to precipitating conditions,e.g.,exposure to cold.
- Allowing activity according to tolerance.
- Seeking medical care early.
- Giving prophylaxis for malaria.
- Giving supplementary folic acid but avoiding administration of iron.
- Ensuring adequate immunization including that of pneumococcal vaccine if possible.
- For severe SCD only: Pain >3 episodes/yr; stroke; transfusion ≥ 2/ yr; acute chest syndrome); Child 2-12 years initially 10-15mg/kg once daily, increased every 12 weeks in steps of 2.5 - 5 mg/kg daily according to response; usual dose 15 - 30 mg/kg daily (max. 35 mg/kg daily)

**Management of Crises**

- For all patients with sickle cell crisis the following should be done:
  - Intravenous or oral fluids should be given and their intake monitored carefully
  - Infections should be treated vigorously and promptly
- For patients with thrombotic(vaso-occlusive,painful,or ischaemic)crisis,the following should be done:
  - Assess severity of pain carefully and give appropriate analgesia at all times
    - For mild pain give paracetamol, diclofenac or ibuprofen
    - For moderate pain use give dihydrocodeine, codeine phosphate
    - For severe pain give strong analgesia eg morphine and refer appropriately
- Admit all patients with aplastic, sequestration, and haemolytic crises for appropriate management.

**Transfusion in SCD**

Children with the following complications may need transfusion:

- Aplastic crisis
- Hyperhaemolytic crisis
- Acute splenic sequestration
- Acute chest syndrome
- Acute stroke especially if recurrent.
- Priapism

In some of these cases exchange transfusion may be helpful. In general, avoid blood transfusion unless patient develops cardio-respiratory distress (nasal flaring, intercostal or subcostal retractions, heart failure,grunting),or has severe anaemia (Hb well below patient's usual level).

**Refer to an appropriate specialist if**

- Patient is not responding to treatment
- Surgery is indicated

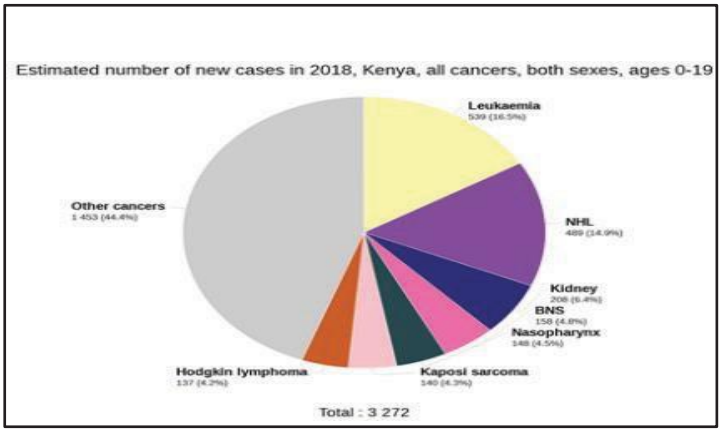
# 35. Neoplasms in Childhood

Neoplasms can occur in any age group. In general, most neoplasms require referral to higher level for treatment. All suspected malignancies or those for which the diagnosis is unclear should be referred early to facilitate appropriate evaluation and management. Early treatment of malignancies carries the best prognosis. Clinical features, useful investigations, and management of common childhood malignancies are summarized in Table 35.1

## 35.1 Burden of Childhood Cancer in Kenya

In Kenya, GLOBOCAN estimated that 3,272 new cancer cases were diagnosed in 2018
(Figure 35.1).

**Figure 35.1: Estimated number of all Chilhood Cancers in Kenya**




Source: GLOBOCAN, 2018

**Table 35.1: Common childhood malignancies, their clinical features, useful investigations, and line of management**

| Tumour | Clinical features | Investigations | Management |
|---|---|---|---|
| Leukaemias | Anaemia bone pains, haemorrhagic tendencies, epistaxis and gum bleeding. Repeated infections | Haemogram Bone marrow Cytochemistry Flowcytometry | Refer to haematologist/ oncologist for specialized care for chemotherapy |
| Burkitt's lymphoma | Usually a jaw tumour May also present as an abdominal mass or central nervous system tumour | Biopsy of the mass; haemogram, bone marrow, x-ray, ultrasound scan CT scan, PET scan Lumbar puncture | Refer for specialized care |
| Hodgkin's disease | Lymph node enlargement, usually cervical Splenomegaly abdominal masses | Haemogram Chest x-ray Lymph node biopsy for histology and immunohisto-chemistry Bone marrow | Refer for specialized care for chemotherapy with or without radiotherapy |
| Nephroblastoma (Wilms' tumour) | Average age 2 years: Embryonal tumour Early childhood Painless loin mass (abdominal mass) Fast growing | Full haemogram U/E in normal IVU (intravenous urography) shows displaced calices FNAC shows malignant embryonal tumour cells CXR for metastasis | Refer to specialized care Chemotherapy Surgery – nephrectomy with post surgical chemotherapy has good prognosis |
| Neuroblastoma | Embryonal tumour Abdominal mass in loin region. Markedly elevated blood pressure. Fast growing often crossing midline. Child is sick looking | Full haemogram IVU shows caudally displaced normal kidney. FNAC – malignant embryonal cells. Ultra sound showssupra renal tumour withnormal kidney. CXR – look for metastasis, 24 hr urine– VMA grossly elevated | Refer to specialist centre. Chemotherapy Surgery NB: Challenging anaesthesia, has poor prognosis |
| Dysgerminoma | Commonest midline tumour in neonatal period Commonest in ovary, testis, thymus, sacrococcygeal (most dramatic – teratoma). Presents with pressure symptoms. May ulcerate especially when malignant. | Plain x-ray may show calcification U/S – defines extent/site of tumour. Foetoprotein tumour marker | Surgical excision; if benign, leave alone; if malignant, chemotherapy. Good prognosis |

*Continued*

**Table 35.1,*continued***

| Tumour | Clinical features | Investigations | Management |
|---|---|---|---|
| Rhabdo-sarcoma/ rhabdomyo-sarcoma | Tumour of muscle;can occur anywhere commonest in pelvis, bladder,vagina may present with a fungating mass (sarcoma botryoid). May ulcerate and bleed | Good physical examination: Full haemogram U/S, CXR CT scan when available Biopsy FNAC | Surgery Chemotherapy Poor prognosis |
| Retinoblastoma | Age usually below 3 years. Inherited through chromosome 13. May be unilateral or Bilateral. Yellowish whitish reflex | Skull x-ray Urine catecholamines Fundoscopy CT scan – head | Refer to ophthalmologist and oncologist for specialized treatment |
| CNS tumours | Headache, convulsions, vomiting Papilloedema Disturbance of gait & vision | X-ray skull CT scan MRI scan | Refer to neurosurgeon Surgery, radiotherapy, chemotherapy |

## 35.2 Prevention of Childhood Cancer

- Vaccination – HPV, Hep B Vaccines reduce risk of future cervical, hepatic and other cancers
- Screening for Childhood Cancers

  1. Majority of childhood cancers are not amenable to screening apart from retinoblastoma and some cancers associated with inheritable conditions. Further, many childhood cancers are not associated with lifestyle.
  2. Screening is recommended for;

  a. Retinoblastoma.

  b. Children with genetic disrepair syndromes such as Blooms disease, Fanconis anaemia. Ataxia Telengiectasia, Xeroderma pigmentosum etc

  c. Cancer survivors.

  3. However, awareness among the healthworkers and the community is important to aid in early diagnosis as some cancers are amenable to cure if diagnosed early.

# 36. Blood Transfusion

## 36.1 General Principles

- Use blood only when required to save life.
- Do not transfuse on the basis of haemoglobin alone, but also on the clinical status of the patient.
- For all transfusions in the neonatal period, cross match blood against the mother of the neonate as well as the baby, especially if the baby is jaundiced.
- Never use blood that has not been screened.
- Do not used blood beyond expiry date.
- Rate of transfusion for small babies must not exceed 15ml/kg/hr.
- Use packed cells whenever possible except in acute blood loss.
- Remove the bag of blood from the blood bank refrigerator just before transfusion. Never transfuse blood that has been out of the refrigerator for more than one hour.
- Re-evaluate the patient immediately prior to transfusion to ensure that blood is still required to save life.
- Use only blood that has been properly grouped and crossmatched and is in the correct bag labelled for the patient.
- Give blood immediately at the time that it is needed.
- Give frusemide (1mg/kgSTAT) IV at the beginning of the transfusion (but only if the patient is not actively bleeding, or dehydrated). If patient has heart failure, give frusemide immediately; do not wait until blood is available.
- Give antimalaria drugs (full course) to all patients having blood transfusion.

- Determine the volume of blood to be transfused (V) by use of the formula:
  *Volume = Wt in kg x Hb deficitx 6 if whole blood is used (OR: x 3 if packed red cells are used) (Hb deficit = desired Hb minus current Hb)*

  *Alternative Formula: Whole blood 20ml/kg; Packed red cells, Platelets - 10ml/kg*

- Note: When the above formula is used, the volume of the blood needed may be too much to give in one transfusion. In this case it may be necessary to give the volume in divided aliquots over two or more days. In general avoid giving more than 20ml/kg/session unless the patient is bleeding.

## 36.2 Indications for Transfusion

- When there is acute blood loss with signs of shock and/or signs of continuing bleeding.
- When there is severe anaemia:
  - Transfuse as soon as possible if:
  - Neonate Hb <10g/dl in 1st week and <8g/dl thereafter
  - Child Hb <4g/dl
  - Child Hb 4–6g/dl with following: dehydration, shock, heart failure, very high malaria parasitaemia
- So as to provide plasma and platelets for clotting factors when specific components are not available.
- In exchange transfusion for neonate with severe jaundice, or in older patient when indicated.

## 36.3 Transfusion Reactions

**If the patient develops fever, skin rash or becomes ill, then:**

- Stop blood transfusion immediately.
- Give chlorpheniramine 0.4mg/kg STAT IV or IM (max 5mg).
- Return blood to the bank with a fresh sample of patient's blood.
- Monitor urine output.
- Monitor cardiovascular and renal function.
- If hypotension develops start IV fluids.

## 36.4 Other Transfusion Management Issues

### 36.4.1 REFER/CONSULT

**Refer the patient or consult for appropriate management if:**

- You cannot give blood transfusion for any reason.
- Anaemia is due to persistent or recurrent bleeding that cannot be easily controlled.
- Anaemia has not improved after 1 month of supervised treatment (Hb should increase by 2–4g/dl in one month).
- Anaemia recurs within 6 months of full treatment.

### 36.4.2 ADMIT PATIENTS

**Patients with the following conditions should be admitted:**

- Severe anaemia.
- Active and severe bleeding.
- Anaemia and/or jaundice and aged below 2 months.
- Anaemia (any degree of severity) accompanied by pneumonia, heart failure, dizziness, confusion, oedema, severe malnutrition.

### 36.4.3 ADVICE TO MOTHERS

Give balanced and adequate diet to all children. Iron and folate containing foods include meat, fish, liver, eggs, dark green leafy vegetables, and yellow fruits.

# 37. Cardiovascular Diseases in Children

Most cardiac diseases in young children are congenital. In older children, cardiovascular diseases may be acquired or congenital. The heart may also be affected by systemic disorders like pneumonia, anaemia, electrolyte imbalances and malnutrition.

**Clinical Features**

The clinical features depend on the severity of the lesion or defect in the heart. Minimal lesions or defects may only be discovered on routine examination, but major ones may lead to functional disability. Easy fatigability and difficulty in breathing are prominent features of cardiac dysfunction, while frequent interruptions of breastfeeding accompanied by sweating may be a manifestation in infants. Other features include poor weight gain and poor growth. The affected children have stature and nutrition that is usually below the average for the age as well as frequent respiratory infections.

Physical examination that consists of evaluation of pulses in all limbs and of blood pressure,apex beat,and heart sounds, and inspection of the precordium is likely to detect the specific cardiac lesion. The presence of a murmur indicates presence of a defect but does not indicate its size. Cyanosis and digital clubbing are often noted in children with cyanotic heart diseases.

Parents can usually notice that the affected child has a problem, although they may not be able to localize the problem. A young baby who gets tired quickly or who has to pause many times while breastfeeding, who looks breathless or is not growing well, or who has a darkish bluish tinge on the lips and tongue should be suspected to have a heart problem and should be taken to a health facility for examination. Innocent murmurs occur at any age, but are commonest among neonates.

## 37.1 Heart Failure (Congestive Cardiac Failure)

Heart failure occurs when the heart is unable to supply output that is sufficient for the metabolic needs of the tissues in the face of adequate venous return. Any severe cardiac condition, severe pneumonia, or anaemia can lead to heart failure.

**Signs of Cardiac Failure**

- Among infants and young children, cardiac failure manifests as feeding difficulties and excessive sweating, rapid weight gain,tachycardia,gallop rhythm, respiratory distress, and tender hepatomegaly.
- Among older children, cardiac failure manifests in addition with raised jugular venous pressure, dependent oedema, orthopnoea, fatigue, exercise intolerance, and basal crepitations.

**Investigations**

- Chestx-ray: May show cardiac enlargement as well as evidence of other cardiac or pulmonary lesions
- Haemogram
- Urea and electrolytes
- Electro-cardiogram(ECG)
- Echocardiography

**Management –General**

- Let the child regulate physical activities when out of hospital.
- Order bed rest in cardiac position.
- Give oxygen by nasal prongs or catheter for child in severe failure.
- Restrict salt intake, control fluid intake and measure urine output.
- Take daily weight if admitted.

**Management – Pharmacological**

**Infants and young children:**

- Diuretics: Give frusemide:IV 1mg/kg per dose(max 2mg/kg/dose).
- Digoxin:In all cases give ½ total digitalizing dose (TDD) initially, then ¼ TDD after 8 hours, then ¼ TDD after another 8 hours. Daily maintenance dose,¼ TDD, given in 1 or 2 divided doses. Total digitalizing doses are:
  - Premature babies: 0.03mg/kgPO
  - Full term newborn: 0.03–0.05mg/kg PO
  - Infants less than 2 years: 0.05–0.06mg/kg PO
  - Children 2–10 years: 0.04–0.05mg/kg PO
- After load: Captopril (ACE inhibitors) – Begin initially 0.5mg/kg/24 hours 8 hourly, increase by 0.5mg/kg/24 hours every 24–48 hours until dose reaches 3–8mg/kg/24hours,neonates 0.03–2mg/kg/24hours. Or use enalapril0.1mg/kg/day with gradual increase as needed (max 0.5mg/kg/day up to 40mg/24 hours).

- Note: Electrolytes should be monitored during therapy with diuretics and digoxin.
- Treat anaemia and sepsis or pneumonia concurrently.

**Older children (over 10 yrs):**

- Diuretics:Frusemide 0.5–2mg/kg/dose(max6mg/kg/dose)IV or PO OD;use higher doses in patients who were already on it.
- Digoxin:0.01–0.015/kg/24hours. Maximum should not exceed adult dose. Divide dose as for the younger child.
- After load: Captopril 0.3–0.5mg/kg/dose increase gradually to maximum of 6mg/kg/day in 2 or 3 doses per day. Or enalapril dose as in young children.
- Potassium supplements: Advise patient to eat fruits,e.g., bananas or oranges.
- Treat underlying causative factor.
- Maintenance therapy: All children will need maintenance diuretic, digoxin and ACE inhibitors which are continued on outpatient basis.
- Refer to specialist:
  - Patients who fail to respond to therapy or deteriorate despite therapy.

- Children with CHD or heart failure of uncertain origin.
- For definitive treatment of underlying cause.

## 37.2 Pulmonary Oedema

Pulmonary oedema is the accumulation of fluid in the alveoli due to an increase in pulmonary capillary venous pressure resulting from acute left ventricular failure. *This is an acute emergency.*

**Clinical Features**

Breathlessness,sweating,cyanosis, frothy blood-tinged sputum,respiratory distress, rhonchi, and crepitations.

**Investigations**

Chest x-ray: Loss of distinct vascular margins, Kerley B lines, diffuse haziness of lung fields.

**Management**

- Initiate treatment urgently and admit.
- Prop up patient in bed.
- Administer needed drugs:
  - IV frusemide0.5–2mg/kg/dose,maximum 6mg/kg/dose. Infusion–0.05mg/kg/hour.
  - Digitalize if not already on digoxin.
  - IV aminophylline 6mg/kg over 15 min then 0.9mg/kg/hour.
- Give oxygen by nasal prongs or catheter.
- Start on oral medication as soon as possible.
- When the patient has stabilized, investigate to identify the cause.

- Manage the underlying cause.
- Refer to specialist:
  - If patient fails to respond to above therapy.
  - For definitive treatment of underlying cause

## 37.3 Congenital Heart Disease

### 37.3.1 CONGENITAL HEART DISEASE WITH CYANOSIS

The congenital cardiac abnormalities that are associated with cyanosis involve shunting of blood from the right side of the heart to the left side. These include the following cardiac abnormalities:

- Tetralogy of Fallot
- Pulmonary atresia with ventricular septal defect (VSD)
- Transposition of the great vessels
- Truncus arteriosus (associated VSD is always present)
- Eisenmenger syndrome
- Hypoplastic left heart syndrome

**Those abnormalities manifesting in the neonatal period have a poor prognosis.**

**TETRALOGY OF FALLOT**

This is the commonest of the cyanotic group because of a slightly better prognosis in infancy, allowing more of infants to survive longer. Classically, Tetralogy of Fallot consists of pulmonary stenosis, ventricular septal defect, dextroposition of the aorta, and right ventricular hypertrophy.

**Specific Clinical Features**

Cyanosis is a major feature. It may not be present at birth, butdevelops later during first year. Other features include dyspnoea on exertion, to which the affected child responds by assuming a squatting position for a few minutes after such an exercise. Affected children also tend to have paroxysmal hypercyanotic attacks often referred to as "blue" spells. The pulse may be normal but a systolic thrill is felt along the left sternal border in 50% of cases. Clubbing of fingers and toes occurs after a long time.

**The following complications are associated with Tetralogy of Fallot:**

- Cerebral thrombosis due to polycythaemia,
- 453rain abscess (usually after 2 years of age) presenting with headache,fever, nausea and vomiting with or without seizures,
- Bacterial endocarditis,and
- Congestive heart failure.

**Investigations**

- Full blood count or haematocrit
- CXR – Boot shaped heart oligaemic lung fields
- Electrocardiogram
- Echocardiogram

- Blood culture if suspect endocarditis
- CT scan if cerebral thrombosis or abscess is/are suspected

**Management**

- For children with "blue"spells, administer oxygen;child should be in knee- chest position.
- Prevent/correct dehydration in these children at all times.
- Provide supportive therapy:
  - Venesection: Maintain haematocrit at 55–65% but avoid iron deficiency.
  - Intravenous or oral propranolol.
- Referall children to a specialist unit for definitive treatment by interventional closed repair or open heart surgery.

### 37.1.1 CONGENITAL HEART DISEASE WITHOUT CYANOSIS

The commonest in this group of conditions are ventricular septal defect, patent ductus arteriosus, and atrial septal defect.

**VENTRICULAR SEPTAL DEFECT (VSD)**

This is the most common cardiac malformation, accounting for 25% of congenital heart diseases. The magnitude of the left to right shunt is determined by the size of the defect and the degree of the pulmonary vascular resistance.

**Clinical Features**

Small defects with minimal left to right shunts are the most common. Patients are often asymptomatic. The patients may have a loud, harsh or blowing left parasternal pansystolic murmur, heard best over the lower left sternal border on auscultation. Large defects with excessive pulmonary blood flow and pulmonary hypertension are characterized by dyspnoea, feeding difficulties, profuse perspiration, recurrent pulmonary infections and poor growth. Physical examination reveals prominence of the left precordium, cardiomegaly, a palpable parasternal lift and a systolic thrill, besides a systolic murmur.

**Prognosis and Complications**

Spontaneous closure of small defects occurs in 30% to 50% of cases. A large number remains asymptomatic and a significant number with large defects get repeated infections and congestive cardiac failure. Infective endocarditis is a complication in VSD while pulmonary hypertension may develop as a result of high pulmonary blood flow.

**Investigations**

- CXR–Usually normal but some show minimal cardiomegaly and increased pulmonary vasculature
- ECG – May suggest left ventricular hypertrophy
- Electrocardiography
- Echocardiography
- Cntrol congestive cardiac failure if present.
- Refer the affected child to the specialized unit

**PATENT DUCTUS ARTERIOSUS (PDA)**

The pulmonary arterial blood is shunted through the ductus arteriosus into the aorta during foetal life. Functional closure occurs soon after birth when pulmonary pressure falls. Gradual anatomical closure takes place over several days. This process is slower in the preterm infant. Patent ductus arteriosus occurs when ductus fails to close and the blood continues to shunt through it to the aorta.

**Clinical Features**

On auscultation one frequently hears a systolic or machinery murmur over the entire precordium, axilla, and back. The patient also has bounding peripheral pulses. The affected child may also be in congestive cardiac failure with its typical clinical manifestations. There are three types of patent ductus arteriosus:

- Anatomical defect: This type is the typical ductus that occurs in term and preterm babies and treatment is surgical management.

- PDA of prematurity: This is basically a “functional” problem in which the ductus remains open when there is tissue hypoxia,e.g.,in respiratory distress or anaemia, and is contributed to by fluid overload. The ductus normally closes spontaneously or by use of drugs and sometimes surgery may be required.
- PDA accompanying other abnormalities: Other congenital cardiac abnormalities may be present and may be the only communication between the right and left side of the heart. In such cases closure of the patent ductus may lead to death unless the accompanying defects are also corrected.

**Investigations**
As for VSD

**Management**
- Medical management of CCF if present
- Referall children to specialized unit for confirmation of diagnosis and management
- Medical closure in preterms– indomethacin or ibuprofen

### 37.3.2 GENERAL MANAGEMENT OF CONGENITAL HEART DISEASE

The following general principles should guide the management of congenital heart disease:
- Parents should be counselled on what can and what cannot be done depending on the heart lesion.
- Evaluation and close followup of affected children are vital for appropriate and effective management.
- The majority of patients having mild CHD require no treatment. Such patients are expected to live normal lives and should not have any exercise restriction. The parents of the child should be made aware of this.
- Good nutrition should be maintained with adequate immunization and prevention of anaemia.
- Children with severe disease will tend to limit their own exercise, but if dyspnoea, headache,and fatigability in cyanotic patients occur,their exercise and other activities should be limited.
- Bacterial infections should be treated vigorously.
- Prophylaxis against bacterial endocarditis should be given before dental procedures, urinary tract instrumentation, and lower GIT manipulation.
- Cyanotic patients should be observed for polycythaemia and dehydration avoided.
- Venesection with volume replacement should be carried out for polycythaemic when haematocrit goes above >65% and maintain it between 55–65%.

## 37.4 Acquired Heart Disease

### 37.4.1 ACUTE RHEUMATIC FEVER

This is an acute, systemic connective tissue disease in children related to an immune reaction to untreated group A beta haemolytic streptococcus infection of the upper respiratory tract . The significance of this disease is the rheumatic heart disease complication that may result from it, which may cause severe heart valve damage. Rheumatic heart disease is the commonest form of heart disease in Kenyan children. The initial attack of acute rheumatic fever occurs in most cases between the ages of 3 and 15 years.

**Clinical Features**

For the sake of diagnosis, clinical features related to rheumatic heart disease are categorized into major and minor criteria.

- The major criteria include migrating polyarthritis, carditis (manifested by signs of cardiac failure, persistent tachycardia, pericardial rub or heart murmurs), Sydenham's Chorea, erythema marginatum, and subcutaneous nodules.
- The minor criteria include past history of rheumatic fever, raised ESR, fever, and arthralgia.

**Diagnosis of rheumatic fever occurs when 2 major criteria and 1 minor criteria are present, or 1 major criteria and 2 minor criteria.**

**Complications**

The main complication of rheumatic fever is rheumatic heart disease.

**Investigations**

- Anti-streptolysin-0-titre (ASOT) – Titre of 1:300
- Throat swab for culture
- ESR
- Chest x-ray – Features of cardiomegaly
- ECG if available
- Admit for strict bed rest until symptoms resolve.
- Eradicate streptococcal infection from the throat:
  - Give penicillin or amoxicillin for children 25–50mg/kg in divided doses TDS for 10 days.
  - If allergic to penicillin ***OR*** amoxicillin, give erythromycin 30–50mg/kg QDS for 10 days.
- Give aspirin: 75–100mg/kg/day in 4–6 divided doses. Treatment continued until fever and joint inflammation are controlled and then gradually reduced over a 2-week period.
- Treat heart failure if present.
- Treat chorea, if present, with haloperidol 25 micrograms/kg(0.025mg/kg) TDS.

**Prevention**

- Reduction of overcrowding among populations as much as possible.
- Early treatment of streptococcal sore throat with appropriate antibiotics (benzathinepenicillin 25,000–50,000 units/kg/dose STAT;maximum 1.2 mega units dose ***OR*** phenoxymethylpenicillin 25–25mg/kg/24 hour TDS for 10 days).

**Long-Term Prophylaxis**

Parents should be made aware of the necessity for long-term prophylaxis. Children with previous acute rheumatic fever without carditis should be given benzathine penicillin 1.2 mega units monthly for 5 years or up to the age of 18 years, whichever is longer. Patients allergic to penicillin should be given erythromycin 125–250 mg BD for 5 years. On the other hand, the children with previous acute rheumatic fever with carditis should be given benzathine penicillin 1.2 mega units *OR* erythromycin 125–250mg/dose BD for those sensitive to penicillin for life.

## 37.4.2 RHEUMATIC HEART DISEASE

Rheumatic heart disease is the inflammatory damage of the heart valves, as a complication of acute rheumatic fever. The mitral valve is the most commonly involved valve, although any valve may be affected. The inflammatory damage in rheumatic heart disease results in stenosis or incompetence of the valves, either singly or in combination with other valves. Patients' rheumatic heart disease may be asymptomatic with the lesion only discovered during routine examination. However, some of the patients may present with congestive cardiac failure. Heart murmurs are over the precordium on auscultation, but the murmurs depend on the nature of the damage (whether incompetence or stenosis) and on the specific valves involved.

**Complications**

The complications for rheumatic heart disease include congestive cardiac failure, pulmonary oedema and bacterial endocarditis.

- Chest x-ray
- ECG
- Echocardiography

**Management**

- Treat underlying complication, e.g., heart failure, pulmonary oedema.
- Continue prophylaxis against recurrent rheumatic fever.
- Advise that infective endocarditis prophylaxis is indicated before or during dental procedures, urinary tract instrumentation, and GIT manipulations.
- Refer to a specialist
  - All patients with significant heart murmur for evaluation.
  - All patients with increasing cardiac symptoms.

**Long-Term Prophylaxis**

- Rheumatic fever: All patients with a history of rheumatic fever should be given prophylaxis for life.
- Endocarditis prophylaxis in addition to rheumatic fever prophylaxis the following should be done:
  - For dental procedures, give amoxicillin 50mg/kg PO 2 hours before procedure and 25mg/kg PO 6 hours after the initial dose. If the patient has penicillin allergy give erythromycin 1g PO 2 hours before procedure then half the dose 6 hours after the initial dose.
  - For lower gastrointestinal and genitourinary procedures give amoxicillin 50mg/kg IM 30 minutes before procedure and 6hours after the initial dose as well as gentamicin 1.5mg/kg IM 30 minutes before procedure and 8 hours after the initial dose.

**Patient Education**

The need for follow up should be strongly emphasized.

### 37.4.3 INFECTIVE ENDOCARDITIS

The most common pathogens are bacterial, although they may also be fungal. Any child with a heart condition can get endocarditis but it can occasionally affect normal valves.

**Clinical features**

The clinical features include fever, splenomegaly, petechiae, and new murmurs.

**Investigations**

- Full blood count
- Urinalysis
- Blood culture
- Electrocardiogram
- Echocardiography
- Give penicillin 100,000–400,000U/kg/24 hour IV 4–6 hourly,and gentamicin 6–7.5mg/kg/24 hours.
- Monitor carefully for response.
- If patient is not improving, consider changing antibiotic after results of culture.
- Refer to specialist if:
  - No or poor response to therapy.
  - Patient deteriorates.

## 37.5 Pericardial Disease

Diseases of the pericardium are difficult to detect unless one has a high index of suspicion. Clinical features may be vague or very dramatic, as when one has cardiac tamponade. A review of some of the diseases affecting the pericardium is given below.

## 37.5.1 ACUTE PERICARDITIS

This is caused by bacterial infections, but it may also be due to viral pathogens.

**Clinical Features**

Patients may present with fever, chest pain and dyspnoea and may have pericardial friction rub on auscultation.

**Investigations**

- Full blood count
- Chest x-ray
- ECG
- Echocardiogram
- If pericardiocentesis is done, send fluid for microscopy, protein estimation,and culture.

**Management**

Bed rest

Penicillin and gentamicin if bacterial infection is suspected.

## 37.5.2 PERICARDIAL EFFUSION

This may be due to bacterial infection resulting in collection of pus in the pericardium (exudates) or to some non-infective inflammation with collection of serous fluid in the pericardium (transudate), for example in rheumatoid arthritis.

**Clinical Features**

This condition may be asymptomatic if it is due to non infective cause and is a small effusion. Otherwise, there may be chest pain that is acute or a dull ache depending to the cause of effusion. On examination, the apex beat may be difficult to palpateand the heart sounds may be distant if the amount of fluid in the pericardium is large.

- Full blood count
- Chest x-ray
- ECG
- Echocardiogram
- If pericardiocentesis is done, send fluid for microscopy,protein estimation,and culture.

**Management**

- Pericardiocentesis may be diagnostic as well as therapeutic.
- Treat according to type of fluid.

### 37.5.3 CARDIAC TAMPONADE

Cardiac tamponade occurs when cardiac filling is severely limited by the presence of a large amount of pericardial fluid.

**Clinical Features**

The affected patient presents with severe dyspnoea, cold extremities with decreased capillary refill, raised jugular venous pressure (JVP), tachycardia, pulsus paradoxus, and inaudible heart sounds.

**Investigations**

- Full blood count
- Urinalysis
- Blood culture
- Electrocardiogram
- Echocardiography

**Management**

- Admit urgently and do pericardiocentesis.
- May need to have a temporary drainage catheter.
- Treat any underlying condition.
- Needs ICU care.

### 37.5.4 CONSTRICTIVE PERICARDITIS

This tends to be chronic and is often due to tuberculosis. The pericardium becomes thick and inelastic leading to poor filling of the heart.

**Clinical Features**

**The patient presents with cough and dyspnoea, small volume pulse, ascites, hepatomegaly, and raised JVP.**

**Investigations**

- Chest x-ray–Heart size normal or small. There maybe calcification in the pericardium.
- Electrocardiogram
- Echocardiogram
- Surgical removal of pericardium.
- Treatment of TB if it is the cause.

## 37.6 Hypertension in Children

This is defined as elevation of systemic blood pressure beyond the 95th blood pressure centile for age (or above the upper limit of normal). The blood pressure varies with age and gender and stature and these are found in normograms for blood pressure for children. A simplified version of normogram that considers only age is shown in Table 38.1. In order to record blood pressure accurately, a

correct size cuff for the child is needed; such a cuff is expected to cover about two-thirds of the arm.

**Table 37.1: Upper limits of normal blood pressure values for both sexes at differentages (inmmHg)**

| Averageage | 12 months | 8years | 9years | 10 years | 12 years | 14years |
|---|---|---|---|---|---|---|
| Systolic blood pressure | 80 | 120 | 125 | 130 | 135 | 140 |
| Diastolic blood pressure | 50 | 82 | 84 | 86 | 88 | 90 |

**The following are the common causes of hypertension at different ages:**

- For neonates and infants: Renal artery thrombosis or stenosis and coarctation of the aorta.
- From1 year to10 years: Renal parenchyma disease and coarctation of the aorta.
- From11 years to18 years: Renal parenchyma disease, essential hypertension.

**Clinical Features**

Essential hypertension may initially be asymptomatic. Coarctation of the aorta in a neonate may present with sudden collapse or features suggesting sepsis. Others will present with clinical features of the underlying disease or target organ system – hypertensive encephalopathy, pulmonary oedema.

**Investigation**

- Urinalysis
- Urea and creatinine
- Chest x-ray
- Special investigations as indicated for the suspected cause

**Management – General**

- Maintain blood pressure at or slightly below the 95th centile for age (blood pressure should not be reduced by more than 25% in the acute phase).
- Determine and treat any underlying cause of hypertension.
- Advise aerobic exercise, salt restriction, weigh treduction.

**Management – Pharmacological**
**This is summarized in Table 38.2.**

**Table 37.2: Summary of plan for care in hypertension**

| Severity of hypertension | Drugs to be used |
|---|---|
| Mild: | HCTZ* ***OR*** propranolol/atenolol ***OR*** HCTZ* + propranolol/atenolol Moderate: HCTZ*+propranolol/atenolol+hydralazine***OR***HCTZ*+methyldopa***OR*** HCTZ* + nifedipine/captopril |
| Severe: | HCTZ* + propranolol/atenolol + hydralazine/captopril ***OR*** HCTZ* + propranolol/atenolol + nifedipine/captopril ***OR*** HCTZ* + propranolol/atenolol+ methyldopa |

Note:
*HCTZ = Hydrochlorothiazide; bendroflumethiazide, frusemide, or other appropriate diuretics may be substituted
Beta-blockers: Propranolol oral 1–8mg/kg/24 hours on 3 divided doses ***OR*** Atenolol oral 0.1–0.5mg/kg/24 hours in 2 divided doses, maximum 20mg per day
Calcium channel blockers: Nifedipine oral 0.2–1mg/kg/24 hrs in 3–4 divided doses (6–8 hourly)



**Hypertensive Crisis**

- Defined as systolic or diastolic pressure above the 95th percentile by 50% or when signs of hypertensive encephalopathy or pulmonary oedema occur.
- Congestive heart failure

**Management**

- Admit urgently.
- Monitor closely: This is mandatory – may require ICU care.
- Aim to lower BP by 20% over 1 hour, by one-third over 6hrs, and return to baseline levels within 24–48 hrs.
- Administer nifedipine sublingual 0.2–0.5mg/kg dose 4 every 4–6 hours (max 10mg/dose).Watch:precipitous fall in BP may occur.***OR***
- Hydralazine IM/IV 0.1–0.8mg/kg/dose every 4–6 hours (max. 20mg/dose). Be careful not to cause uncontrollable hypotension. ***OR***
- Sodium nitroprusside IV continuous infusion 0.5–8ìg/kg/minute.Requires ICU setting.
- Monitor blood pressure during infusion, titrate dose according to response.

# 38. Urinary Tract and Renal Condition

## 38.1 Features of Renal Disease

**Clinical Features**

The clinical features of renal disease include the following:

- Changes in urine output that include reduced urinary output (oliguria, anuria), increased urinary output (polyuria), increased frequency without increased volume.
- Oedema of the body, usually facial initially but later involving legs and generalized.
- Haematuria that ranges from microscopic to gross. Haematuriais a serious sign of disease and should be aggressively investigated. Causes include infections (urinary tract infection, tuberculosis, schistosomiasis), acute glomerulonephritis, trauma, meatal ulcers, blood disorders (bleeding disorders, leukaemia, purpura, sickle cell disease), tumours, scurvy, congenital abnormalities.
- The blood pressure may be raised in some conditions or it may be a terminal manifestation in some conditions.
- Renal masses maybe palpable,for example if the patient has nephroblastoma, polycystic kidneys, horse-shoe kidneys, neuroblastoma, and hydronephrosis.

**Laboratory Findings**

The following laboratory findings may be found in renal disease:

- Pyuria of >10 cells/$mm^3$ in uncentrifuged urine specimen.
- Casts of renal tubules formed by red blood cells (RBC), white blood cells (WBC), epithelial cells. The casts may be granular or hyaline.
- Proteinuria that may vary from minimal to gross.
- High blood urea or blood urea nitrogen (azotaemia, BUN) that accompany renal failure.
- Raised blood creatinine levels that accompany renal failure.
- Hyperkalaemia:Usually,there are no clinical consequences until the levels rise to 6mmol/L and above. Clinical features of hyperkalaemia include muscle weakness, abdominal distension, tingling of the face and of the muscles on the hands and feet, and irregular pulse, heart block, and increased amplitude of the T-wave on the ECG.

## 38.2 Urinary Tract Infections (UTI)

Urinary tract infection is commonly caused by the following bacterial organisms: Eschericchia coli (75%), Klebsiella, Proteus vulgaris; less commonly by Streptococcus faecalis and some Pseudomonas species; and rarely by a Staphylococcus species.

**Clinical Features**

- In children it is not easy to differentiate upper from lower urinary tract infections, but loin (lumbar) pain and tenderness suggest upper urinary tract infection.
- In neonates and early infancy, boys are affected more often than girls because of the occurrence of the higher incidence of congenital urinary tract malformation in boys than girls that is noted at that age. Affected children present with fever, failure to thrive, irritability, poor feeding, and vomiting.
- In older infants and children, girls are affected more often than boys because of their anatomically shorter urethra than that found in boys. Affected children present with anorexia, vomiting,fever,abdominal pain,frequency,enuresis in a previously dry child, and dysuria. For the younger child, the mother may report that the child cries when passing urine.
- For all male children, ask about the nature of the stream of urine when they are passing it. In those with urinary tract obstruction, the urinary stream is poor.
- Recurrences of urinary tract infection are common.

**Investigations**

The following investigations are recommended for a child with urinary tract infection:

- Full blood count
- Urinalysis:>10WBC/cubic$^3$ in uncentrifuged urine midstream or catheter specimen
- Urine C&S(midstream,suprapubic puncture or catheter specimen). Bacterial colony count: Most reliable providing urine has been plated within 1 hour of voiding. Interpret results as follows:
  - <10,000:Nonspecific contaminants; significant if suprapubic specimen.
  - 10,000–100,000: Doubtful significance. Repeat cultures and evaluate clinical symptoms.
  - 100,000: Diagnostic of UTI.
- The urine specimen should reach the laboratory within 2 hours of voiding or be refrigerated at 4°C for a period not exceeding 24 hours.
- Further evaluation include:

Micturating cystourethrogram – urethral valves and reflux.

  - Abdominal ultrasound best done when child is febrile to demonstrate acute pyelonephritis.
  - Intravenous urography.
- When associated with haematuriaor proteinuria, pyuria is suggestive of parenchymal renal disease such as glomerulonephritis or interstitial nephritis.
- Sterile pyuria is often due to TB – do cultures for TB.

**Management**

- Encourage a lot of oral fluid.
- Give amoxicillin 25mg/kg/dose 8 hourly ***OR*** Cefuroxime 10-15mg/kg 12 hourly for 7-14 days;
- nitrofurantoin can also be used.
- Important: Clear infection in order to prevent chronic pyelonephritis.
- Repeat urine culture 1 week after treatment.
- Put children with recurrences of reflux on prophylaxis.
- Refer to specialist if:
  - Patient is an infant.
  - Recurrent attacks occur more than 3 in one year.
  - Sick child showing features of upper UTI.
  - Deranged renal finction
  - UTI which is not e coli
  - Less than 2 years of age
  - Recurrent (>3 episodes)

# 38.3 Glomerular Disorders

## 38.3.1 ACUTE GLOMERULONEPHRITIS (AGN)

This is an inflammatory renal disease commonly following streptococcal infection of skin and tonsils.

**Clinical Features**

The patient presents with smoky or tea coloured urine as a result of haematuria, with oedema that manifests as puffiness of the eyes, more noticeable in the morning. The oedema is seldom severe or generalized. The affected children also experience back pain, hypertension – commonly presenting as headaches, visual disturbance, and vomiting. Occasionally the patients may present with pulmonary oedema with dyspnoea or convulsions and coma due to hypertensive encephalopathy. There may be evidence of primary streptococcus infection, most often as an acute follicular tonsillitis with cervical adenitis and less often as skin sepsis. In the initial stages of the illness there is oliguria that is followed by diuresis (oliguric – diuretic phases).

**Investigations**

- Urinalysis: RBC,RBC casts and WBC. Granular and hyaline casts, mild to moderate proteinuria.
- Blood urea: Moderately high in oliguric phase; otherwise normal.
- Antistreptolysin O titre: Increased except in those with a skin primary cause where it remains normal.
- Throat and skin swab where indicated, but culture may be negative. Streptococcus may be cultured.
- Complement c3, c4, ANCA
- May need renal biopsy if has been deteriorating renal function/proteinuria of 3+, normal complement levels.

**Management**

- Admit the child.
- Give penicillin or amoxicillin for 10 days.
- Monitor fluid intake, urine output, weight, and BP daily.
- Restrict fluid input in oliguric phase: child <5 years 300ml/day and child >5 years 500ml/day in addition to urine output.
- Order a high calorie, low salt and protein diet in oliguric phase.
- Treat hypertension if present [see hypertension].
- Monitor electrolytes, urea, and creatinine daily especially in the oliguric phase.
- Refer to specialist if
  - in acute renal failure
  - If ASOT negative
  - Renal failure
  - Normal c3 or c4

## 38.4 Nephrotic Syndrome

**Causes of nephritic syndrome include the following:**

- Idiopathic or unknown for the majority of children with nephritic syndrome.
- Congenital nephritic syndrome, which may be to congenital syphilis.
- Secondary nephritic syndrome, which is due to post acute glomerulonephritis, plasmodium malaria, other infection and infestations, allergy following bee stings, heavy metal poisoning (e.g., mercury and lead), urinary tract infection.

**Clinical Features**

**The clinical features of nephritic syndrome include the following:**

- Oedema that is marked to massive and maybe accompanied by ascites and/or pleural effusion
- Marked proteinuria
- Hypoproteinaemia, mainly low serum albumin in blood
- Hyperlipidaemia
- Children with nephritic syndrome who have haematuria with hypertension are categorized as nephritic nephrosis.

**Investigations**

- Urinalysis
- 24-hour urine for protein
- Serum protein
- Urea and electrolytes
- Serum cholesterol
- Complement c3 c4
- Chest Xray and mantoux to rule out TB

**Management**

- Normal protein diet: 1-2gm/kg
- Low salt diet (no salt added to food)

- Frusemide administered carefully to induce diuresis only if needed and if not hypotensive.
- Prednisone 2 mg/kg daily for 6 weeks then 1.5 mg /kg alternate day for 6 weeks and taper off over 6 weeks (maximum 60mg). The responses to prednisone are generally divided into steroid responders and non steroid responders:
  - Response usually occurs within 2 weeks demonstrated by no protein in urine.
  - Relapses are treated the same way
  - If there is continuing proteinuria after 1 month the child is steroid resistant.
  - If proteinuria returns after the steroids are stopped or is off high dose steroids the child is steroid dependent and may require continuation
  - Repeated relapses or steroid dependants who develop steroid toxicity can be treated with steroid sparing regimens with oral cyclophosphamide or rituximab. Cyclosporin or levimazole maybe better alternatives in future.
  - Steroid resitant cases may benefit from ACE inhibitors even in the absence of hypertension. Requires biopsy and start on calcineurin inhibitor like cyclosporine or tacrolimus ; diuretics are used to control oedema.
- Antibiotics are used if there are clinical signs of/or suspected infections. Possibility of urinary tract infection should always b econsidered.
- Refer to specialist patients:
  - With persistent gross haematuria
  - With hypertension
  - Who develop chronic renal failure
  - Who relapse or do not respond.
  - age less than 2 yrs and older than 10 yrs

## 38.5 Tubular Disorders

Tubular disorders can be congenital or be the result of shock or toxins. Congenital variety tends to be associated with acidosis (renal tubular acidosis – RTA) and renal rickets.

**Investigations**

- Urine pH: Suggestive if it is low (<5.8).
- Serum electrolytes: Low bicarbonate, low potassium and high chloride suggestive.
- Specific tests of tubular function may be needed to identify the abnormality.

**Management**

- For acute tubularnecrosis:
  - Maintain intravascular volume.
  - Monitor urine output.
  - Correct any electrolyte or acid base disturbances.
  - Order dialysis if due to dialysable toxin.
- For renal tubular acidosis:
  - Correct acidosis using oral sodium bicarbonate or sodium citrate.
  - Use potassium citrate if patient is hypokalaemic.
  - Give high dose vitamin D and calcium if child has rickets.
- Refer/consult specialist as required.

## 38.6 Acute Kidney Injury

Acute kidney injury is an acute or sub-acute decline in the glomerular filtration rate and/or tubular function characterized by rapid accumulation of nitrogenous waste products, for example urea and creatinine, in the blood.

**Aetiologies of Acute Kidney injury**

The causes of acute kidney injury divided into pre-renal, renal and post-renal groupings:

- Pre-renal acute kidney injury: This group of diseases includes the following:
  - Diarrhoea and vomiting with severe dehydration,
  - Burns,
  - Inappropriate diuretic treatment,
  - Peritonitis,
  - Pancreatitis,
  - Heart failure,and
  - Liver disease with ascites.
- The renal grouping includes the following:
  - Diseases of the renal arteries and veins that include:
    - Direct trauma to renal vessels
    - Dissecting aortic aneurism
  - Intrinsic renal problems that include:
    - Glomerulonephritis
    - Acute interstitial nephritis
    - Acute tubular necrosis
    - Intratubular obstruction
  - Post-infectious glomerulonephritis:
    - Renal damage related to drugs for example methicillin, ibuprofen, and gentamicin
    - Following volume depletion and also as a result of toxins
    - Rhabdomyolysis
    - Uric acid nephropathy
- The post-renal grouping includes the following:
  - Obstruction of the collecting system:
    - Bladder outlet obstruction,
    - Bilateral ureteral obstruction,
    - Ureteral obstruction,and
    - A single kidney.

**Clinical Features**

- Low or no urinary output (sometimes it may be normal)
- Oedema
- Heart failure
- Hypertension
- Hyperkalaemia
- Acidosis
- Rising blood urea and creatinine
- Diagnostic workup including history and physical examination, as well as:

- Careful review of medical records and medications (e.g.,gentamicin).
- Presence of swelling and oedema of muscles, which may indicate rhabdomyolysis
- Abdomen or flank pain, which may indicate obstruction to urine flow or inflammation of the kidneys

**Investigations**

- Full blood counts
- Urinalysis and urine culture and sensitivity
- Urea and electrolytes
- Serum creatinine.
- ECG if hyperkalaemic

**Management**

- Hypovolaemic patients: Give 10 ml/kg of normal saline over 30 minutes for three doses after fixing a urethral catheter – patient should pass urine in the next 2 hours. Replace fluid as completely as possible in patients who have vomiting, diarrhoea or burns.
- Non hypovolaemic patient: Restrict fluid to 300 ml/m2 plus previous days output
- Do not give drugs that may further damage the kidneys,e.g.,gentamicin, tetracycline, sulfonamides, NSAIDs, nitrofurantoin
- If the blood pressure is normal or high and the patient is not dehydrated, give a trial of intravenous frusemide in a dose of 1–5mg/kg.
- Treat the hypertension if indicated.
- Treat hyperkalaemia, as indicated below:
  - For mild to moderate hyperkalaemia (K =6–7mmol/L):
    - Do not give potassium containing fluids or food.
    - Give oral potassium retaining resins.
  - Severe hyperkalaemia (K>7mmol/L):

- Give 1ml/kg 50% glucose with insulin 1 unit/5g of glucose over 30 minutes.
- Repeat after 30–60 minutes if hyperkalaemia persists.

- If there are ECG changes, give IV 10% calcium gluconate 1ml/kg/dose to be injected over 5–10 minutes.
- Refer to centre with facilities for dialysis if:
  - If hyperkalaemia is persistent.
  - Anuria is present for more than 24 hours ***OR*** oliguria for more than 48 hours.
  - Metabolic acidodis
  - Fluid overload
  - Pulmonary edema
  - BUN >30 mmol/l
  - Features if uremia like seizures

## 38.7 Chronic Renal Failure

Chronic renal failure describes the situation in which there is advanced, irreversible, and usually progressive renal failure. Chronic renal failure is commonly caused by chronic glomerulopathies, hypertension, chronic interstitial nephritis, and diabetes mellitus. The following are important manifestations of chronic renal failure:

- There is poor growth.
- At biochemical level in the blood, there is acidosis, hyperkalaemia, elevated blood urea and elevated serum creatinine.
- At cardiovascular level there is pulmonary oedema, hypertension, pericarditis and cardiac tamponade and heart failure.
- At skeletal level, there is bone pain and bone fractures (rare).
- At nervous system level, there is encephalopathy (confusion, convulsions)and peripheral neuropathy.
- At haematological system level there is anaemia, excessive bleeding,e.g., from gums, skin, nose.
- At the skin level, there is scratching (pruritus) and darkening of skin.

**Chronic renal failure should be suspected in the presence of the following:**

- A previous history of renal disease e.g. acute nephritis, nephrotic syndrome.
- A known history of hypertension.
- A known history of diabetes mellitus.
- High blood urea and serum creatinine.
- Some of the systemic manifestation listed under “manifestations of chronic renal failure”.

**Management**

- Monitor clinical state regularly: This includes BP measurement and nutritional status (growth monitoring).
- Monitor laboratory parameters: BUN, creatinine, alkaline phosphatase.
- Adjust diet: High energy intake above recommended for age; protein1.5g/kg/day, preferably high quality types; watch out for micronutrient deficiency and correct when needed; sodium restriction (1–4mg/kg/24 hours) if oedema or CCF.
- Watch potassium intake, especially when child needs dialysis.
- Treat hypertension if present.
- Do not transfuse blood unless HB is<6g/dl. Use packed cells preferably leucocyte depleted and erythopoetin
- For renal osteodystrophy, give high doses of vitamin D preferably the active form.
- Phosphate binders to keep phosphate and PTH normal for age
- Avoid drugs that may worsen the problem, and adjust dosing according to degree of renal failure (creatinine levels).
- Refer to specialist if

- Chronic renal failure is diagnosed.
- End stage renal failure.

## 38.8 Hypokalaemia

Hypokalaemia is said to have occurred when serum potassium levels are persistently below 3.5mmol/L. Causes of hypokalaemia include inadequate dietary intake (rare), gastrointestinal fluid loss (vomiting, diarrhoea, fistulae), renal loss (diuretics, uncontrolled diabetes mellitus), systemic metabolic alkalosi, and dialysis.



**Clinical Features**
Clinical features for hypokalaemia include the following:
- Muscular weakness
- Tetany
- Fatigability
- Thirst

- Polyuria
- Paralyticileus
- Cardiac arrhythmias
- Low serum potassium
- Elevated serum bicarbonate
- Low serum chloride
- ST segment depression and appearance of V waves on ECG

**Investigations**
- Urea and electrolytes
- ECG

**Management**
- Treat cause where possible.
- If necessary give oral potassium (SlowK),80–100 mmol daily or intravenous (at a rate of infusion not to exceed 25 mmol/hr).
- Care must be taken in patients with renal failure to avoid hyperkalaemia.

**— Never give potassium IV as a bolus. The patient will have cardiac arrest.**

## 38.9 Genito-Urinary Anomalies

The genito-urinary anomalies include undescended testes, hypospadias, ectopia vesicae, patent urachus, and urachal cyst, as well as recto urethral fistula in males with imperforate anus. The important one to note is the obstructive type – urethral valves and obstructed ureters. Early identification and relief of the obstruction will prevent renal damage. Management of these conditions is complex and the patients need to be referred appropriately for management.

**Investigations**

- Urine pH: Suggestive if it is low (<5.8).
- Serum electrolytes: Low bicarbonate, low potassium and high chloride suggestive.
- Specific tests of tubular function may be needed to identify the abnormality.

**Management**

- For acute tubularnecrosis:
  - Maintain intravascular volume.
  - Monitor urine output.
  - Correct any electrolyte or acid base disturbances.
  - Order dialysis if due to dialysable toxin.
- For renal tubular acidosis:
  - Correct acidosis using oral sodium bicarbonate or sodium citrate.
  - Use potassium citrate if patient is hypokalaemic.
  - Give high dose vitamin D and calcium if child has rickets.
- Refer/consult specialist as required.

# 39. Central Nervous System

## 39.1 Seizure Disorders

A seizure is defined a transient occurrence of signs and/or symptoms due to to an abnormal excessive or synchronous neuronal activity in the brain. Seizures can result from structural brain lesions, infections, metabolic disorders, immune, genetic causes but sometimes the cause is unknown. Seizures can be epileptic or non-epileptic. Epilepsy is defined as recurrent unprovoked epileptic seizures.

**Clinical Features**

The clinical features depend on the type of seizure. The various forms of seizures are:

- Focal onset seizurese:
  - Simple partial seizures– Can be motor, sensory and sensory-motor (consciousness not impaired).
  - Complex partial seizures–Starting with an aura (later impairment of consciousness) and often accompanied by automatic behaviour.
  - Partial seizures becoming progressive (jacksonian seizures) or generalized.
- Generalized onset seizures, which include:
  - Absences, which are brief lapses of awareness that last for about 30 seconds and are uncommon below 5 years of age.
  - Tonic seizures, which manifest with sustained muscle contractions.
  - Myoclonic seizures, which are repetitive symmetrical muscle contractions whose distinctive forms are:
    - Benign myoclonus of infancy disappear by age 2 years.
    - Early childhood type, whose onset starts at about 2 years and has a relatively good prognosis.
    - Complex type, whose onset starts in the first year of life commonly following birth asphyxia, with a poor prognosis.
    - The juvenile form that begins at age 12–16 years, among children that are neurologically and has a good response to treatment.
  - Clonic seizures, characterized by rhythmic jerking.
  - Tonic-clonic seizures characterized commonly by an aura with loss of sphincter control and postictal deep sleep.
  - Atonic seizures characterized by sudden loss of muscle tone.

- Infantile spasm, characterized by their initiation at age 4–8 months, sudden symmetrical contraction of all parts of body, and whose prognosis is poor if there is identifiabe underlying pathology but good if there is not identifiable underlying pathology.
- Meticulous history from parents and reliable witnesses is critical in diagnosing a seizure disorder. It is important to find the details of the prodromal phase, aura, and the type,duration,frequency,and age of onset of seizures. Details about the post ictal phase are important. It is also important to determine the underlying pathology, for example birth asphyxia, neonatal jaundice, or infection of the central nervous system.
- A careful and thorough physical examination is necessary to detect associated neurological dysfunction or abnormality. Evaluation of blood pressure, head circumference in those aged less than 2 years, and fundoscopy are important in the examination of such children.



**Investigations**

- If child has fever
  - Full haemogram
  - Malaria parasites
  - Lumbar puncture if meningitis
- When metabolic conditions are suspected,do
  - Blood sugar
  - Urea and electrolytes and creatinine
- Electroencephalography (EEG)
- CT scan of the head in the emergency setting.
- Magnetic resonance imaging (MRI), is the preferred imaging modality for a seizure disorder as it provides a detailed image of the entire brain.

**Management**

During a seizure, the following should be observed:

- Ensure the patient's safety by moving them away from danger and any objects that may cause injury.
- Place the patient on the left lateral position with the head turned to the same side;
- Loosen or remove tight fitting clothing around the neck.
- Do NOT attempt to insert any instrument into the mouth to avoid tongue biting, as this may have already happened.
- Shield the patient from being surrounded by too many eager observers.
- Allow seizure to complete its course without physically attempting to hold down the patient. Most seizures are likely to stop in less than five minutes. If a seizure continues for more than three minutes, start arrangements for transfer to a hospital.

**General Management of Seizures**

For a child with seizure, the following should be observed:

- Treat any underlying diagnosed condition.
- For most patients with epilepsy, start on therapy as out patients.

- Counsel parents and patient that treatment is usually long term.. Therapy may be discontinued after a seizure-free period of at least two to three years if the patient has no significant risk factors for seizure recurrence.

Reduce dose gradually over many months. Sudden discontinuation of drugs may precipitate status epilepticus. Seek expert advice for complex seizure patient.

**Pharmacological Management**

- Refer toTables 40.1 and 40.2 for a summary of the drugs of choice for common seizures and the appropriate paediatric dosages, respectively.
- Start long-term therapy if patient has had 2 or more unprovoked seizures at least 24 hours apart. Start therapy with 1 appropriate first line anti-seizure medication, . Increase the dose gradually and titrate with response.
- If side effects appear and seizures are still not controlled, introduce other drugs and taper off the first drug.
- Admit for evaluation if underlying metabolic cause is suspected or raised intracranial pressure is present.
- Refer to specialist if:
  - Seizures are not controlled with maximum drug dose.
  - Raised intracranial pressure is suspected.
  - Space occupying lesion is suspected.

**Table 39. 1: Drugs of choice for common seizures**

| Main classifica- tion of convulsive disorder | Subclassification of the main convulsive grouping | Preferred drug of choice for treatment | Other drugsthat can be used for treatment |
|---|---|---|---|
| **Focal seizures** | With awareness | Carbamazepine | Carbarmazepine, Valproic acid Phenotoin |
| | With impaired awareness | | |
| | Secondarily generalized | Sodium valproate | Phenotoin |
| **Generalized seizures** | Absence | Sodium valproate Ethosuximide. | Valproic acid Clonazepam |
| | Tonic-clonic, clonic, tonic,atonic | Sodium valproate | Carbamazepine Phenytoin |
| | Myoclonic | Clonazepam | Nitrazepam, Valproic acid,Phenobarbitone |

**Parent and Patient Education**

The following is important for the education of the patient and the parent:

- Medication should be taken regularly and it should not be assumed that the child is healed when the seizures are controlled. Treatment in most cases is life long.
- Ensure normal activity for the age of the child including school.
- Child should avoid dangerous activities like climbing trees.
- Protect child from falling intofires.
- The patient should never swim alone and all precautions should be taken when swimming.
- The parent should not to be over protective for the child.

**Table 39. 2: Paediatric dosages of common drugs for convulsive disorders**

| Drug | Dosage | Frequency | Remarks |
|---|---|---|---|
| Phenobarbitone | 3–8mg/kg children | Once daily | May cause hyperactivity insome |
| Phenytoin | 4–7mg/kg | Once daily | Causes gumhypertrophy |
| Carbamazepine | 10–30mg/kg/day | 2-3 divided doses | |
| Sodium valproate | 20–40mg/kg/day | 2-3divided doses | May precipitate, absence statusif given with clonazepam. Also causes transient alopecia. |
| Ethosuximide | 20–40mg/kg/day | 2–3 divided doses | |
| Clonazepam | 0.01–0.03mg/kg/day | Once daily | May precipitate absence statusif Given with sodium valproate |

NB: Sodium valproate is the most broad spectrum anticonvulsant, but it is very costly and is better used as a second line drug. If seizures are not controlled, drugs used at maximum recommended dose should be withdrawn gradually as another one is introduced.

## 39.2 Status Epilepticus

**Clinical Features**

A succession of seizures without regaining consciousness between attacks or one prolonged convulsion lasting 30 minutes or more. Status epilepticus can occur with partial, complex partial, absence, tonic-clonic, or clonic seizures and may result in respiratory embarrassment with cyanosis and hypoglycaemia.

**Management**

The following is recommended in stabilizing the child with status epilepticus:

- For the airway and breathing:
  - Establish the airway.
  - Give oxygen.
  - Provide ventilation.
- With regard to circulation and disability
  - Establish intravenous access.
  - Give 10% dextrose 5ml/kg.
  - Give diazepam intravenously or rectally.

**Management – Pharmacological**

- In the first 5–15 minutes:
  - Give diazepam: 0.3mg/kg IV over 1–3 minutes or 0.5mg/kg rectally (max 10mg in 1–3 years and 15mg in 3–15 years). Repeat after 5–10 minutes if not controlled.
- In the next 15–45 minutes:
  - If seizure persist: Use phenobarbitone or phenytoin
    - Phenobarbitone: Loading dose 15–20mg/kg IV in 5 minutes.
    - Rate of infusion not exceed1mg/kg/min.
    - Additional 5mg/kg/dose can be repeated every 15–30 minutes to maximum of 30mg/kg.
    - **IV Phenytoin (with glucose-free solution). Loading dose 15–20mg/kg over 20 minutes. Infusion not to exceed 1mg/kg/minute. Monitor the blood pressure due to risk of hypotension.**
- In the next 45–60 minutes:
  - If all these do not control the convulsion, or severe respiratory depression results from the drugs, child needs ICU care where ventilation can be done.
  - When patient is stable look for the cause and treat as needed.

## 39.3 Febrile Convulsions

Ordinarily seen in childhood, these are generalized tonic-clonic seizures with the following characteristics:

- They occur in children aged between 6 months and 5 years,
- There is fever at the time of the attack (usually greater than 38°C),
- They are of brief duration (always less than 15 minutes),
- They occur in the absence of central nervous system infection,and
- There is absence of neurological abnormalities in the inter-ictal period.

**Investigations**

Evaluate to find the cause of the fever if not determined by physical examination:

- Blood slide for MPs
- Full blood count
- Lumbar puncture and CSF examination–Strongly recommended in all infants or children who have received antibiotics.
- Blood for culture

**Management**

- Emergency care:
  - Give paracetamol to reduce the temperature.
  - Reduce child's clothing to a minimum to facilitate lowering of temperature.
  - Give rectal diazepam if child is convulsing at the time of presentation.
  - Treat identified cause.
  - Check blood sugar levels and correct with 5ml/Kg 10% Dextrose if low.
- Subsequent care:
  - Educate parents that recurrences are common but that they can be reduced by administration of antipyretics as soon as child becomes febrile. .

- Prophylactic antibiotics are not required for simple/typical febrile seizures. Recurrent complex (atypical) febrile seizures may require further evaluation by a paediatrician or neurologist.

**Seizures in the neonate are covered under neonatal care.**

## 39.4 Cerebral Palsy

Cerebral palsy (CP) is defined as a non-progressive disorder that consists of motor and other neurological problems resulting from a defect or lesion of a developing brain. The aetiological factors associated with cerebral palsy are:

- Prenatal causes include rubella, syphilis, toxoplasmosis, and asphyxia.
- Perinatal causes include birth asphyxia as the main factor, being responsible for about 50% of the cases.
- Postnatal causes include bilirubin encephalopathy,meningitis,encephalitis, intracranial haemorrhage,hydrocephalus.

**Clinical Features**

Spastic paralysis is the commonest variety. It involves one or more limbs and the trunk. Posture is that of hyperextension with tendency to contractures. Deep tendon reflexes are increased. The choreoathetoid type of cerebral palsy is less common and is characterized by involuntary movements and abnormal posture. Cerebral palsy may also present as ataxia with low muscle tone and lack of balance. Abnormalities associated with cerebral palsy include deafness, visual defects, speech difficulties, mental retardation, convulsions, and growth retardation. If the problem dates from birth, neonatal reflexes may persist. Malnutrition can result from neglect of the child or from difficulties associated with feeding the child.

**Management**

All children should, if possible, be seen once by a doctor with some experience of cerebral palsy children for correct diagnosis. The nature of the motor dysfunction, its distribution and all related abnormalities should be noted and a decision made on what could be offered to the child.

**Symptomatic Therapy**

Occupational therapy is the mainstay of management of these children. Such therapy should be started as early as possible. The main aim is to prevent contractures and abnormal patterns of movement, to train other movements, and build coordination.

Depending on the degree of disability, the child can be trained by an experienced therapist to attain some degree of independence. Home training programme for the parents is the most important part. Anal sphincter control may be assisted by administration of stool softeners and enemas where necessary.

Anticonvulsive drugs should be given if there are convulsions, and any accompanying problem should be dealt with appropriately. A multidisciplinary approach is recommended for the management of children with cerebral palsy.

**Support of Family**

Parents are encouraged to bring their children early for care and not hide them from the public. The diagnosis should be discussed with the parents in an open and honest manner, explaining that there is no cure for the condition but that physical therapy contributes significantly to the wellbeing of the affected child.

## 39.5 Intellectual Disability

Children whose neuromotor and cognitive development is delayed are considered to have intellectual disability (formerly mental retardation). The degree of impairment in the mental retardation varies from mild to very severe. Intellectual performance is below average, as expected, and the severely retarded child is not able to adapt to daily demands and thus may not be able to lead an independent life. Intellectual disability may also be part of a condition like Down's Syndrome. It is necessary to exclude deafness and cerebral palsy because hearing impairment retards the child's ability to learn at normal pace, while children with cerebral palsy may have normal intelligence but are physically impaired to perform.

**Management**

- Proper assessment is needed so that the child can be placed in an appropriate school.
- Counselling of the parents and their involvement is essential for success of care school.
- Special school may be necessary.

## 39.6 Hydrocephalus

Hydrocephalus is excessive enlargement of the head because of accumulation of cerebral spinal fluid in the cerebral ventricles as a result of the blockage of its flow or rarely, excessive production. Hydrocephalus can be congenital or acquired, as indicated below:

- Congenital isolated hydrocephaly occurs due to blockage of flow of CSF. Commonest area is the Aqueduct of Sylvius. It may also be part of neuro-tube defect.
- Acquired hydrocephalus is usually due to complications of meningitis or to a tumour. In both situations, the flow of the cerebral spinal fluid is blocked.

**Clinical Features**

For those aged 0 to 2 years there is enlargement of the head, bulging fontanel, sunset eyes, and large veins on the head. Depending on the cause and the severity, there may be neurological signs as well. For those over 2 years there is headache, vomiting, and papilloedema. There may also be focal neurological signs.

**Investigations**

- Cranial ultrasound in the young child with open fontanelle
- CT scan of the head and/or MRI Brain.
- Lumbar puncture if indicated
- Special investigations according to suspected cause

**Management**

- Treat underlying cause if treatable.
- Place ventriculo-peritoneal shunt to relieve the pressure.
- Refer as indicated.

# 40. Skin Diseases

## 40.1 Eczema

### 40.1.1 ATOPIC ECZEMA

Atopic eczema is a chronic relapsing skin inflammation associated with dry skin and pruritus. It has a genetic predisposition, with a strong personal or family history of asthma and allergic rhinitis. The onset of this condition is usually in the first 1 to 3 months of life.

**Clinical Features**

- Pruritus is the cardinal feature of eczema.
- Acute changes include erythema, papules or vesicles, crusting and secondary infection.
- Subsequently, there is scaling, hypopigmentation, or hyperpigmentation.
- Distribution of the lesions varies with age. In infants it tends to be on the scalp, face, and extensor surfaces, while in older children it tends to be in flexures, and skin creases.

**Management**

The management of atopic eczema consists of the following:

- Educate parents on the disease and its natural history and advise them to avoid any precipitating factors,e.g.,
  - Synthetic clothing, pets, detergents,
  - Any food substance that is noted to aggravate the eczema
  - Allowing the skin to dry excessively, e.g., by using harsh soaps. One should use the normal toilet soaps. No need to use medicated soaps.
  - Any of the petroleum jelly products for those who react against them (Vaseline, Ballet, Valon, Ideal,etc.)
- Keep the skin moist by using emulsifying ointment and other moisturizers.
- Use antihistamines like chlorpheniramine maleate to alleviate the itch.
- Use mild topical steroids, hydrocortisone, for few days, <7 days. Note that infants can absorb steroids through the skin easily.
- Treat any intercurrent infection (bacterial, or fungal).
- Refer to the skin specialist if the body surface area involved is extensive (e.g., 50% and over) or if lack of response to treatment.

### 40.1.2 CONTACT DERMATITIS

- Acute or chronic inflammation produced by substances contacting the skin and causing toxic (irritant) or allergic reactions.
- Primary irritants include acids, alkalis, soaps, detergents, and acetone.
- Allergic contact dermatitis may be caused by topical drugs, plants, shoes, clothing, metal compounds, dyes and cosmetics.
- The lesions in contact dermatitis may be acute vesicles or may consist of weeping subacute erythema, dry scaly papules. Chronic lesions may be lichenified (thickened), excoriated, and hyperpigmented.

- The distribution of the lesions may take the shape of offending item or area of its contact, for example shoes,watch,and gloves,or may be asymmetric or have other forms.

**Management**

The following management is recommended for children with contact dermatitis:

- Identify and remove the causative agent.
- Drain large blisters, but do not remove their tops (roofs).
- Apply gauze or thin cloths dipped in water or normal saline.
- Apply topical 1% hydrocortisone ointment to dry lesions and cream to wet ones.

### 40.1.3 SEBORRHOEIC DERMATITIS

This is an inflammatory scaling disease of the scalp, face, and occasionally other areas with high density of oil glands (axilla, upper chest, anogenital areas).

**Clinical Features**

Symptoms develop gradually as:

- Dry or greasy diffuse scaling of scalp (dandruff) with pruritus.
- Yellow- red scaling papules in severe cases found along the hairline, external auditory canal, the eyebrows, conjunctivae, and naso-labial folds. The lesions are not accompanied by hair loss.
- Cradle cap (thick, yellow crusts on scalp) in newborns.
- Severe seborrhoeic dermatitis, found in neurologic disorders (Parkinson's disease) and HIV infection.

**Management**

The following management is recommended for children with seborrhoeic dermatitis:

- Apply 2% salicylic acid in olive oil to control scaling.
- Remove dandruff by applying shampoos containing selenium sulphide, sulphur and salicylic acid, or tar daily (more recently ketaconazole shampoo is excellent).
- Apply topical steroids
- Treat superimposed bacterial, fungal, or viral infections, which are especially prevalent in patients with HIV.
- Refer to specialist if patient does not respond to treatment.

## 40.2 Bacterial Infections

### 40.2.1 IMPETIGO CONTAGIOSUM

This is a contagious intradermal infection caused by streptococcal or staphylococcal organisms. This condition is commonly associated with poor hygiene, crowded living conditions, and neglected minor trauma. The condition frequently complicates eczema, scabies, purpura urticaria, and insect bites. Impetigo contagiosum may presents as bullous lesions that rupture and crust on the face, arms, legs, and buttocks.

**Management**

The recommended management of this condition comprises the following:

- Local treatment for minor lesions consisting of cleaning the lesion with normal saline.
- Topical antibiotic like mupirocin for few lesions.
- Systemic treatment for extensive lesions consisting of administration of systemic antibiotics (amoxicillin/cloxacillin or erythromycin).

### 40.2.2 BULLOUS IMPETIGO

- This condition is common in neonates (pemphigus neonatorum), although any age can be affected. It is caused by an infection by staphylococcal bacteria, involving mainly the axilla and the groin.
- The skin lesions are usually large bullae containing pus and clear serum,and may rupture easily leaving raw areas. Crusting is not a feature in this condition.
- The patient should be admitted if toxic with suspicion of septicaemia, or if there are extensive lesions especially in the neonate.

**Patient Education**

The patients and their guardians need to know the following about bullous impetigo:

- It can spread easily, especially in schools.
- Affected children should be isolated and treated.
- Topical antibiotics for mild and systemic medication- cloxacillin or erythromycin for extensive disease
- Towels and bath facilities for those affected should be kept separate.

## 40.3 Fungal Infections

The fungal infections of the skin include dermatophyte (genus microsporum, trichophyton, and epidermophyton) infections, which thrive on non-viable keratinized tissue of the skin (stratum, comeum, hair, nails). Sources of infection include other persons, animals such as puppies or kittens, and more rarely the soil.

The nomenclature for the infection is “tinea” followed by the Latin name of the appropriate part, for example, Tinea pedis for athlete’s foot, which is manifested by scaling or maceration between the toes particularly the fourth interspace.

This is caused by Tinea rubrum and/or Tinea interdigitalae. Predisposing factors include hot humid weather and occlusive footwear. Tinea cruris is an erythematous and scaly rash with distinct margin extending from groin to upper thighs or scrotum. It is common in males and itching may be severe.
Tinea corporis (body ringworm) forms characteristically annular plaques with raised edges and central clearing and scaling with variable degrees of itching.
Tinea capitis (scalp ringworm) is mainly a disease of children and show spontaneous recovery at puberty. It manifests commonly with scaling, itching, and loss of hair, often referred to as “Mashillingi” in Kiswahili. Scarring, alopecia may result from the infection.

Tinea anguum involves the nails and presents with nail discolourationand subungual hyperkeratosis (friable debris).

**Investigations**
These are not usually necessary but in doubtful cases do direct microscopy of skin scale in 20% potassium hydroxide mounted on a slide to demonstrate hyphae.

**Management**
The management for fungal skin infections comprise of the following;
- Apply gentian violet paint, 0.5% concentration, daily to wet lesions (that are in skinfolds).
- Administer fluconazole 6mg/kg/day for a month, grisofulvin 10-20mg/kg in divided doses for the scalp infection for 8-12 weeks
- Apply clotrimazole or terbinafine or miconazole cream for 14 days for tinea corporis, tinea cruris, tinea pedis
- Administer ketoconazole shampoo for scalp lesions weekly until lesions clear.
- Avoid mixed, combination cream or steroid creams for fungal infections.

# 40.4 Parasitic Infestations

## 40.4.1 SCABIES

Scabies is caused by the human itch mite, Sarcoptes scabiei, and spreads through intimate personal contact, facilitated by overcrowding and poor hygiene. Transmission via bedding or clothing is infrequent, partly because the mites do not survive for a day without host contact.

**Clinical Features**
The clinical features of scabies include the following:
- Intense itching worse at night or after hot shower.
- Skin papular rashes associated with burrows, which occur predominantly on the finger webs, the wrists flexor surfaces, elbow and axillary folds, and around the areola of the breasts in females and the genitals especially male, along the belt line, and on the buttocks. In young children rash may be generalized and may affect the face, palms and soles of feet.

- Secondary infection causes that manifest themselves as urticarial papules, crusts and pustules.

**NB: The burrow is a fine, wavy scaly line (0.5–1cm long) with a small papule/ vesicle at the end.**

**Diagnosis**
- Diagnosis is made by demonstration of typical burrows on the skin;these may be difficult to demonstrate.
- Microscopy of skin scrapings (avoid KOH) and demonstrate the mite,ova,or faecal pellets.

**Management**
The following is recommended for management of scabies:
- Apply to the entire skin (from the neck down) a 25% benzyl benzoate emulsion (use 12.5% in children) on days 1 and 2 without bathing. On day 3 bathe and apply again.
- Apply 5–10% sulphur ointment.
- Use nonspecific measures, which include the following:
  - Maintaining good personal hygiene
  - Using antihistamines for pruritus
  - Putting the clothing used by the affected individually, including bedding and mattresses, in the sun.
- Treat secondary bacterial infections using cloxacillin in severe cases.
- Treat the whole family for scabies at the same time.

## 40.4.2 JIGGERS/TUNGA PENETRANS

Diagnosis of jiggers is not a problem. However, educating the community on treatment is mandatory. The following is recommended:
- The jigger should be extracted with clean pin.
- The jiggers should be suffocated by soaking the affected feet in Lysol,liquid paraffin, or kerosene.
- The topical application of a two-component dimeticone with a defined viscosity, as found in treatments for headlice, is highly effective.
- Tetanus toxoid vaccination.

**Prevention**
The following preventive measures are recommended:
- Smoothening the walls and floors with mud or cowdung.
- Dusting of the earthen floors with insecticide powders.(Ensure that any such insecticide is safe for human contact.)
- Promoting personal hygiene for affected populations.

## 40.5 Pellagra (Niacin Deficiency)

Pellagra is a dietary deficiency that may occur in starvation, isoniazid therapy, diarrhoea, and liver cirrhosis.

**Clinical Features**
This condition presents with characteristic dermatitis, diarrhoea, and dementia, and may result in death if appropriate treatment is not given. Weight loss, anorexia, fatigue, malaise, pruritus with burning sensation, dysphagia, nausea, diarrhoea, vomiting, impaired memory, confusion, and paranoid psychosis may occur. Skin lesions are limited to areas exposed to the sun, e.g., the face, neck, hands, and feet. Mucous membranes may be involved, manifesting as scarlet stomatitis and scarlet red tongue.

**Management**
Management of pellagra involves the following:
- Administer high protein diet.
- Administer multivitamin tablets o rsyrup.
- Administer niacin 50–100mg/dose, 3 times a day.

## 40.6 Dermatological Emergencies

### 40.6.1 STAPHYLOCOCCAL SCALDED SKIN SYNDROME(SSSS) OR RITTER'S DISEASE

This is a toxin-mediated epidermolytic condition leading to detachment of the superficial epidermal layers to resemble scalding. Affected children may look like they have been immersed in a basin of hot water and sustained burns.

The condition mainly occurs in children under 2 years of age, and varies in severity and distribution from a localized form (bullous impetigo) to a generalized form of epidermolysis. This condition is also found in immuno-compromised patients and in those with renal failure.

**Clinical Features**
The clinical features in this condition comprise the following:
- Flaccid vesicles that shear off, leaving raw areas, when gentle lateral pressure is applied to them.
- Focus of infection may be in the nose, umbilical stump, purulent conjunctivitis, otitis media. or nasopharyngeal infection.

**Investigations**
Pus swab for culture and sensitivity is essential

**Management**
- Admit.
- Practise barrier nursing – isolate.
- Maintain meticulous fluid and electrolyte balance as in burns.
- Ensure adequate nutrition.
- Give parenteral antibiotics, cloxacillin or flucloxacillin preferred. Change antibiotics according to culture and sensitivity results.

- Maintain skin care:
  - Topical care baths with normal saline.
  - If widespread and weeping lesions are present treat
- Refer/consult severe cases unresponsive to available treatment.
- Do not give corticosteroids.

## 40.6.2 ERYTHEMA MULTIFORME SYNDROME

This condition is now a common problem because of the increased prevalence of HIV/AIDS. It is characterized by an infiltration by mono-nuclear cells into the dermoepidemial junction, leading to the formation of vesicles, which are generally found in the extremities, palms, and soles in the mild form of disease. In severe forms of the disease, widespread mucosal involvement occurs, with typical features of Stevens-Johnson syndrome, and may last 1–2 months, being accompanied by a high mortality.

The following is known about its aetiology:

- About 50% of occurrences are idiopathic, with no known cause.
- Administration of drugs like sulphonamides, phenytoin, barbiturates, penicillins, and thiacetazone have been known to lead to its occurrence.
- Viral infections like herpes simplex and bacterial infections like streptococcal and infections with mycoplasma have been associated with the development of the condition.
- Underlying malignancies have been known to be associated with this condition.

**Clinical Features**

The clinical features of this condition include the following;

- In severe form the mucous membranes are always involved, with extensive bullae formation and systemic symptoms of fever and prostration.
- There may be cheilitis and stomatitis, which interfere with feeding, with vulvitisin females and balanitis in males, leading to difficulties in micturition.
- There may be conjunctivitis that leads to keratitis.
- There may be epidermal necrolysis, that may be lifethreatening.

**Management**

- Admit all cases.
- Stop offending factor – Minimize drug therapy.
- Give intravenous corticosteroids, which are the current therapy for Stevens-Johnson syndrome.
- For skin care, clean with normal saline.
- For eye care, administer 1% tetracycline eye ointment. Refer to ophthalmologist.
- For mouth care, use antiseptic wash.
- Keep patient warm.
- Practise cradle nursing, single room/bed.
- Give IV fluids until able to feed orally.
- Refer urgently to specialized centres.

## 40.6.3 EXFOLIATIVE DERMATITIS (EXFOLIATIVE ERYTHROMA SYNDROME, ERYTHRODERMA)

### 40.6.4

This is a serious, life threatening skin disease characterized by generalized and confluent redness with scaling of the skin, associated with systemic toxicity, generalized lymphadenopathy, and fever. The disease manifests as an acute illness and may also manifest as a chronic illness. More than 50% of patients with this condition have a history of pre-existing dermatosis, commonly eczema. Other conditions, psoriasis, serborrhoic dermatitis, pityriasis rubra pilaris, contact dermatitis, blistering disorders, mycosis fungoides, drug reaction, leukaemia, and lymphoma. In up to 10–20% no possible cause can be identified.

Constitutional symptoms of the condition include fatigue, weakness, anorexia, weight loss, malaise, feeling cold (shivering), red appearing skin that is thickened and scaly, and commonly without any recognizable borders for the lesions.
Oedema of lower legs and ankles may occur. The palms and soles may be involved with resultant thickening and fissuring. There may be alopecia (although this is not a constant finding), with shedding of the nails. Erythroderma may be purely secondary to HIV infection.

**Prognosis**

This is a very serious disease with many complications in a number of body systems. The highest level of skill and facility are necessary for its management, and the prognosis is guarded.

**Investigation**

- Confirm primary skin disorder by skin biopsy.

**Management**

- Bath soaking
- Bland emollients: liquid paraffin, emulsifying ointment
- Nursing care in a single room and keep warm
- Systemic management
  - Supportive – fluid, electrolyte, protein replacement
  - Systemic steroid used under specialist care are prednisone or prednisolone
  - 0.5mg/kg/day in 2 divided doses
- Note: Erythroderma may be purely secondary to HIV infection.

# 41. Endocrine System Conditions

## 41.1 Diabetes Mellitus

Diabetes mellitus is recognized by persistent elevation of the concentration of glucose in the blood (hyperglycaemia).

**Clinical Features**

The clinical features of diabetes mellitus include polyuria, polydipsia, and polyphagia. The affected child also has weight loss and experiences recurrent infections. In severe uncontrolled diabetes with ketoacidosis, there may be altered consciousness and coma.

**Classification**

Diabetes mellitus is classified into type 1 and type 2 diabetes mellitus.

- Type 1 (which is insulin dependent diabetes mellitus) usually occurs in children and young adults and in the absence of appropriate therapy is associated with keto acidosis. These patients require insulin to sustain life.
- Type 2(which is non-insulin dependent diabetes mellitus) usually afflicts adults, although it is increasingly being seen in obese children.

**Investigations**

The following investigations are recommended:

- Evaluation of plasma glucose: Fasting venous plasma glucose of more than 7.8mmol/L on more than one occasion or random plasma glucose of more than 11.1mmol/L in symptomatic patients is indicative of diabetes mellitus. HBA1c of more than 6.5%, if available.
- Urinalysis for protein, sugar, and ketones is useful for making a diagnosis.
- Serum urea and electrolytes.

**Management**

Management of this condition aims at the following:

- Abolition of symptoms of diabetes
- Correction of hyperglycaemia, and glycosuria
- Prevention and management of complications.

**For children with diabetes mellitus, the following is also important:**

- Maintaining normal weight, growth, and development.
- Improving quality of life.
- Keeping the urine free of ketones.

**General Management**

Dietary modification is important in both types of diabetes mellitus. The hospital nutritionist should be consulted so as to carry out appropriate dietary modification that is preferably individualized.

The following food composition is recommended:

- Carbohydrate: 50–60% in complex form; should be based on the staple for the family and refined products should be avoided.

- Protein: 10–20% that should incorporate vegetable protein sources including soyabeans, lentils(dengu), and beans. Animal products should be included if possible.
- Fat: 25–30% of energy intake that should be preferably polyunsaturated types
- There should be adequate fibre in diet, because fibre can prolong absorption of sugar. Fibre containing foods include most unrefined staple foods, beans, legumes, bran, fruits and vegetables
- Strict adherence to meals schedule should be maintained.

## 41.1.1 TYPE 1 DIABETES MELLITUS

This form of diabetes usually presents with diabetic ketoacidosis (DKA). Patients with type 2DM can also present with DKA, especially insituations of stress such as infection or neglect of therapy.

**Clinical Features**

The clinical features include intense polydipsia, polyuria, and polyphagia. In young children the condition may present with enuresis in a previously dry child. The child may also present with abdominal pain, vomiting, dehydration, acidotic breathing, and altered consciousness or coma. The child has weight loss in spite of having a good appetite.

**Investigations**

- Urinalysis: Ketonuria and glycosuria
- Blood sugar: Hyperglycaemia
- Urea and electrolytes

**Management**

- *Management of diabetic ketoacidosis is a medical emergency.* Some patients with DKA present without coma.
- Admit the patient.
- Rehydrate the child if dehydrated using normal saline in line with management guidelines is recommended (Table 41.1). After the initial resuscitative rehydration, transfer the child to higher level for appropriate management.

**Table 41.1: Fluid replacement in a child with diabetic ketoacidosis**

| Age | Amount |
|---|---|
| <24 months | 100ml/kg |
| 2–4 years | 85ml/kg |
| 5–10 years | 70ml/kg |
| >10 years | 20–30ml/kg |

Note: Use the same basic principles for rehydration in children, with fluid volume The working assumption is that child has lost 10% of weight due to dehydration. Intravenous infusion of normal saline is initiated. Total fluid to be given should be 100ml/kg/24 hours, with additional fluid for maintenance. The child should receive 20ml/kg of fluid in the first hour and then receive the rest of the rehydration over 24 hours. Cerebral oedema may occur during the rehydration phase.

- Initiate fluid replacement with normal saline then change to 5% dextrose alternating with N/S when blood sugar is between 12.0–14.5mmol/L. If severely dehydrated continue N/S and 5% dextrose together. Continue intravenous fluids until fluid losses have been corrected and ketonuria has disappeared.
- Insulin therapy:
  - After bolus fluid, give short acting (soluble)insulin at 0.1 IU/kg/hr as a continuous IV infusion.
  - Continue IV insulin until blood glucose is 10mmol/L and base deficit is<5.
  - Change to maintenance SC insulin regime when patient is conscious, cooperative, and able to eat.
- Potassium replacement:
  - Hypokalaemia is a common feature. Confirmation should be through ECG and electrolytes. If present supplement as indicated in Table 41.2.
  - Potassium replacement should commence immediately after the first dose of insulin and 20ml/kg of fluids. Potassium can safely be given at the rate of 10–20mmol/hr (10ml of 15% KCL=20mmol K) in an infusion. ***Never give potassium as abolus.***
- Acidosis:
  - Correction of acidosis is not always necessary unless pH is<7.0 and serum potassium is >4mmol/L or <7.1 and not improving after initial rehydration. Give NaHCO3 8.4% (diluted to 4.2%). Use the following formula:

*Base excess x 0.3 x weight in kg. Give 25% over 1 hour and reassess (1ml NaHCO3 8.4% = 1mmol HCO3)*

**Use NaHCO3 with caution: May cause paradoxical CNS acidosis.**

**Table 41.2: Potassium replacement**

| Serum potassium (mmol/L) | Potassium supplements (mmol/L of fluid) |
|---|---|
| <3 | 40 |
| 3–4 | 30 |
| 4–5 | 20 |
| 5–6 | 10 |
| 6 | None |

- Monitoring:
  - Two-hourly plasma potassium (while potassium infusion is being given).
  - Hourly blood sugar estimations are mandatory in the first few hours (use glucose oxidase reagent strips).
  - Urine output; if no urine after 3 hours catheterize patient.
- Nasogastric suction should be done in comatose patients to prevent aspiration.
- Oral intake is initiated after ketoacidosis has been corrected.

- Some patients do not have DKA at presentation. For these:

- Admit patient for insulin therapy.
- Start patient on soluble insulin 0.1unit/kg subcutaneously half an hour before meals TDS. The severity of hyperglycaemia will aid in selection of the dose.
- Plasma glucose should be monitored before meals and at bed time.
- Maintain plasma glucose in the range of 8.3–13.4 mmol/L in the hospital to avoid hyperglycaemia at home.
- Gradual adjustment of insulin dosage is essential when blood glucose is near the desired range.
- When blood glucose level is between 8.3 and 11.0 mmol/L, change to an intermediate-acting insulin.
- The dose of intermediate-acting insulin is 2/3 of the total daily soluble insulin requirement. Alternative strategy is to base control on 2 doses of intermediate acting insulin, 2/3 in the morning and 1/3 before supper.

♦ Maintenance of insulin therapy:
  - Maintenance insulin for pre-pubertal children is in the region of 0.6–0.8 unit/kg/day and at pubertal 1.5–2unit/kg/day. Total daily dose 2/3 in the morning and 1/3 in the evening. Adjust to prevent excess weight gain. Optimum control at home is blood sugar 4–9 mmol/L : 4–7mmol/L before meal and <9mmol/L after food.
  - Maintain blood glucose: HbA1c<6.5–7.5%.
  - Short-acting (soluble) insulin is injected 15–30 minutes before a meal
  - Insulin dose should not increase or decrease by more than 2 units at a time
  - Sites of subcutaneous injection:
    - Upper outer areas of the arms
    - The front and sides of the thigh
    - The upper outer surface of the buttocks and the abdomen (except the areas close to the navel)
♦ Avoid hypoglycaemia–Teach caregiver or child to recognize features of hypoglycaemia
♦ Adjust doses: Increase during infection, surgery, and reduce during exercise, renal and hepatic impairment.

**Hypoglycaemia should be considered in all diabetic patients who present with altered consciousness or coma. Take blood for glucose and give 5ml/kg of 10% dextroseimmediately.**

**Parent/Patient Education**

The parent of, or a child with, diabetes mellitus should receive the following information to enhance management of the condition:

♦ The parent or child (if old enough) should be taught how to give insulin at home and how to look after the insulin: how to measure insulin, technique of injection, care of syringe,
♦ The parent or child (if old enough) should be taught how to recognize and manage hypoglycaemia.
♦ Child with any infection should always be taken to a health facility for immediate treatment.
♦ Such a child should seek medical advice for any injury, however minor.
♦ Patients with diabetes should take their meals regularly, even at school.
♦ Teachers should be made aware of child's diabetic status.

- Patients should carry sweets or glucose and chew them if they experience any symptoms of hypoglycaemia.
- Patients should always carry a “Diabetic Alert” card with them and inform all health workers when they present to clinic with any problem.
- Patients should be encouraged join support groups for diabetes mellitus.

## 41.1.2 TYPE 2 DIABETES MELLITUS

This form of diabetes occurs in obese children usually over age of ten years and can also present as ketoacidosis. Children whose BMI is >85% for age 10 and above should be screened for this condition, especially if there is family history of diabetes.

**Management**

The primary management of Type 2 diabetes mellitus is based on manipulation of the diet and use of exercises. The following is recommended:

- Manage as outpatient, preferably in the hospital’s diabetic or specialist paediatric clinic if there is such a clinic.
- Consult hospital nutritionist for dietary modification.

**Pharmacological Management**

Oral hypoglycaemic drugs should be used only if the diet and exercise regimen failsand should be strictly under guidance of specialist.

- Use metformin for a child over10 years 500 mg once a day. Adjust at intervals not less than 1 week to maximum of 2g per day.
- Use Tolbutamide child >12 years 0.5–1.5g (max 2g) daily after food.

**Avoid use of glibenclamide and chlorpropamide in children.**

- Insulin is indicated in Type 2 DM if
  - Oral hypoglycaemic drugs are not effective,e.g.,persistent polyuria, hyperglycaemia.
  - Ketonuria occurs.
  - Infection occurs.
  - Other complications, e.g., renal failure,occur.
  - Patients undergoing surgery.

## 41.1.3 COMPLICATIONS

The following complications occur among children with diabetes mellitus**:**

- Hypoglycaemia
  - This occurs when blood glucose falls lower than 4mmol/L.
  - Clinical features include:
    - Sudden onset of sweating,
    - Tremours,
    - Hunger,
    - Mental confusion and drowsiness,and
    - If hypoglycaemiais prolonged,coma.

- Management includes the following:
  - Non-pharmacological management: Give sugar-containing soft drinks, snacks, or sweets. These can be given at home if patient or caregiver notices signs of hypoglycaemia.
  - Monitor blood sugar every 15 minutes until blood glucose is 6–8mmol/L.
- Pharmacological management:
  - Give IV10% dextrose bolus 5ml/kg (do not use 50% dextrose in children).
  - Give 5 or 10% dextrose fluid as a continuous infusion until normal blood glucose is achieved, then change to oral feeding.

**In recurrent episodes,**

- Reduce insulin doses and instruct child to take snacks before exercises.
- In refractory hypoglycaemia, give IM/IV glucagons: for neonates give 20µg/kg; for child < 30kg, give 0.5µg stat dose; for child >30kg give 1mg STAT dose; give continuous infusion1–10µg/kg/hour.
- Infections: Treat with broad spectrum bactericidal antibiotic while awaiting results of cultures where applicable.
- Nephropathy: This is very rare in children. However, all children over 12 years should be screened for microalbuminuria.
- Refer all children with complications to specialists.

## 41.2 Thyroid Diseases

### 41.2.1 GOITRE

This is the enlargement of thyroid gland usually caused by lack of iodine or defects in synthesis of thyroxine hormone. Children may demonstrate features of hyperthyroidism or hypothyrodism.

**HYPERTHYROIDISM**

This condition is due to excessive levels of the thyroid hormone.

**Causes**

In the neonatal period it is a manifestation of Graves' disease in the mother. In older children it may be a manifestation of Graves' disease in the child or subacute thyroiditis.

**Clinical Features**

The clinical features for this condition include tachycardia, cardiac failure, arrhythmias, tremors/jitteriness, lid lag, exophthalmos, sweating, and failure to thrive. If the child has a goitre, there may be pressure symptoms on trachea like stridor and difficulty in swallowing.

**Investigations**

- Thyroid function: TSH, T3, T4
- Thyroid ultrasound
- ECG

**Management**

**Treatment must be done by a specialist:**

- Antithyroid drugs – dosage adjusted according to response; given till child becomes euthyroid
  - Carbimazole
    - Neonate 250–500µg/kg every 6–8 hours up to1mg/kg/day
    - Child 1 month –12 years 250µg/kg every 6–8 hours (max 30 mg per day )
    - Child 12-17 years 30 mg daily
  - Propylthiouracil
    - Neonate Initial Dose 2.5–5mg/kg 12 hourly (max10mg)
    - **Child 1–11 months** Initial Dose 2.5 mg/kg/dose 8 hourly
    - 1–4 years Initially 25 mg 3 times a day
    - 5–11 years Initially 50 mg 3 times a day
    - **Child 12–17 years** Initially 100 mg 3 times a day

  Beta blockers propranolol or atenolol given for control of cardiac symptoms

  Digoxin may be necessary if there is cardiac failure.
- Duration of therapy
  - Neonatal: 8–12 weeks
  - Older children surgery or radioactive iodine may be used depending on response to above medication or when compliance is difficult.

**HYPOTHYROIDISM**

This condition is due to deficiency of the thyroid hormone.

**Classification**

Hypothyroidism can be classified into the following 5 categories:

- Congenital failure of thyroid development (complete or partial)
- Endemic cretinism due to iodine deficiency
- Iatrogenic (after thyroidectomy,radio-iodinetherapy,pituitary ablation,drug induced)
- Auto-immune thyroiditis
- Pituitary gland damage, e.g., cranial pharyngeoma

**Clinical Features**

The deficiency ranges from mild with minimal or unrecognized clinical manifestation to severe mental retardation (cretinism).

In congenital hypothyroidism, most neonates appear normal at birth. Prolonged neonatal jaundice, feeding difficulty, lethargy and somnolence, apnoeic attacks, constipation, large abdomen, umbilical hernia, macroglossia, failure to thrive, delayed physical and mental development, slow pulse rate, dry skin, sparse and dry hair, and hoarse voice are some of the clinical features of such children.

Ideally, diagnosis should be based on neonatal screening tests and not abnormal physical signs. Since such tests are not routinely carried out in the health

services, the clinical features listed and a high index of suspicion continue to play an important role in picking up such children, who can then undergo appropriate laboratory investigations to confirm the diagnosis.

**Investigations**

Hormone levels assay:

- T4 TSH suggests deficit in thyroid gland (mostcases)
- T4 TSH suggests deficit above level of thyroid gland
- T4 suggests thyroid hormone unresponsive (goitre is also present in most patients).

**Management**

Treatment should be done by a specialist

- Give thyroxine
  - Neonates:10µg/kgODPO; adjust dosage in steps of 5µg/kg every 2 weeks until usual dose of 25–37.5µg/day for life.
  - Child 1 month to12years: Start 5–10µ/kg/day with increments of 25mcg daily every 2–4 weeks till normal metabolism.
  - Child 12–18 years: 50–100µg/day then increments of 50µg/day every 3–4 weeks. Usual dose 100–200µg daily
- Adjust dosage to T4,TSH levels,growth,and neuro-development assessments.

**Prevention of Endemic Hypothyroidism**

Iodization of salt has helped to reduce the incidence of endemic goitre in our country.

# 41.3 Adrenal Disorders

## 41.3.1 ADRENAL INSUFFICIENCY

**Causes**

The following situations have been associated with adrenal insufficiency:

- Congenital adrenal hyperplasia
- Long term use of steroids
- Addison's disease
- Pituitary hypofunction

**Clinical Features**

Congenital deficiency may be associated with ambiguous genitalia and precocious puberty. Other manifestations of deficiency include hypoglycaemia, hyponatraemia, hyperkalaemiaand hypotension, Addison's Disease with increased skin pigmentation, hypoglycaemia, muscular weakness, craving for salt and hypotension. Adrenal crisis is associated with cardiovascular collapse.

**Investigations**

- Serum electrolytes for salt losing type
- Blood sugar
- Urinary 17-ketosteroids
- Serum cortisol and ACTH
- Abdominal ultrasound to detect the gonads
- Buccal smear for Barr body to determine sex of the baby

**Management**

- This should be under a specialist
- Acute stage:
  - Give IV hydrocortisone neonate 10mg/kg STAT, then 100mg/$m^2$every 6–8 hours until stable. Older child 2–4mg/$m^2$ every 8 hours. After 4–5 days, change to oral maintenance therapy.
  - Maintenance therapy:
  - Oral hydrocortisone 4–5mg/$m^2$
  - For salt losing type use fludrocortisone 50–100µg once daily.
  - Prednisolone 5mg/$m^2$ per day in 1–2 doses can also be used.
  - High sodium intake may be needed to maintain balance in the salt losing variety
- For all children, adjust doses to maintain normal growth but avoid hypertension. Therefore monitor blood pressure and electrolytes regularly.
- For children on long-term steroid use, if withdrawing always do it very gradually to allow adrenal gland to recover. Reduce prednisolone by 5mg/$m^2$daily.

**Parent/Patient Education**

The parent of, or a child with, diabetes mellitus should receive the following information to enhance management of the condition:

- The parent or child (if old enough) should be taught how to give insulin at home and how to look after the insulin: how to measure insulin, technique of injection, care of syringe,
- The parent or child (if old enough) should be taught how to recognize and manage hypoglycaemia.
- Child with any infection should always be taken to a health facility for immediate treatment.
- Such a child should seek medical advice for any injury, however minor.
- Patients with diabetes should take their meals regularly, even at school.
- Teachers should be made aware of child's diabetic status.
- Patients should carry sweets or glucose and chew them if they experience any symptoms of hypoglycaemia.
- Patients should always carry a "Diabetic Alert" card with them and inform all health workers when they present to clinic with any problem.
- Patients should be encouraged join support groups for diabetes mellitus.

# 42. Musculoskeletal Conditions

## 42.1 Arthralgia (Non-Specific)

This condition presents as joint pain without features of inflammation.

**Clinical Features**
The clinical features include general malaise, joint pains without affecting joint mobility and without features of inflammation (redness, warm, tenderness), although the joint might be slightly tender. The arthralgia is usually a feature of another illness and careful systemic examination is likely to reveal the responsible disease.

Common causes in children include benign hypermobility syndrome, benign nocturnal pain of childhood (growing pains) though in the latter, pain is usually diffuse over the long bones and not in the joints.

**Investigations**
There is no specific investigation besides that to identify the responsible disease.

**Management**
Paracetamol should be administered at 40mg/kg/day given 4 times a day.

## 42.2 Juvenile Idiopathic Arthritis (JIA)

**Clinical Features**
JIA is a diverse group of disorders presenting with arthritis beginning at or before the age of 16 years and persisting for more than 6 weeks, that is not due to any other disease. It affects large and small joints and may interfere with growth and development. Refer to Table 43.1 for a summary of characteristics and the clinical classification.
Children manifesting joint or bone pain especially severe nocturnal pain or pain with reluctance to weight bear must be evaluated for haematological malignancies and infections before making a diagnosis of JIA.

JIA is classified into 7 groups: Systemic JIA, Oligoarticular JIA (<5 joints), Polyarticular Rheumatoid Factor (RF) +ve, Polyarticular RF-ve, Psoriatic JIA, Enthesitis Related Arthritis (ERA), and undifferentiated JIA.

**Management**
- Supportive treatment including Ibuprofen at 10mg/kg every 8 hours; physiotherapy and occupational therapy.
- Intraarticular steroid injection for inflamed joints with methylprednisone acetate or Triamcinolone acetonide 40mg for large joints and 20mg

for small joints.

- Concomitantly, start disease modifying antirheumatic drugs (DMARDs) as soon as diagnosisis is confirmed. These are methotrexate 0.5mg-1mg/kg once weekly, ***OR*** sulfasalazine 30-50mg/kg daily in 2 divided doses to maximum of 2g per day, hydroxychloroquine 5mg/kg/day maximum 200mg once daily. These may be given as single agents or in combination.
- For severe disease not responding to the conventional DMARDs above after 3-6months, start on biologics: etanercept, adalimumab or tocilizumab.
- For systemic JIA disease, tocilizumab may be considered first line treatment (these children should be referred to a facility with a rheumatologist for therapy initiation due to the highly specialized nsture of treatment with these drugs).

**Prognosis**

- Complete remission occurs in 10–60% ofpatients depending on subtype. However, overral, JIA is a chronic disease and majority of children will have active disease into adulthood.
- Those with oligoarticular, ANA+ disease are at high risk for vision threatening chronic anterior uveitis.
- Generally, it is recommended that all children with JIA have routine ophthalmologic evaluation (3-6 monthly) for early detection and management of any case of anterior uveitis.
- Those with polyarticular and rheumatoid factor positive have a less a less favourable prognosis.

- NB: For osteomyelitis and septic arthritis see Orthopaedics section.

**Table 42.1: Summary of juvenile idiopathic arthritis (JIA)**

| Characteristic | The clinical classification of JRA observed | | |
|---|---|---|---|
| | **Systemic** | **Pauciarticular** | **Polyarticular** |
| Percentage | 20% | 40% | 40% |
| Rheumatoidfactor | -ve | -ve | +ve/-ve |
| Antinuclearfactor | -ve | +ve 75% | -ve |
| HLAB27 | -ve | -ve/+ve | -ve |
| **Clinical presentation** | | | |
| Number of Joints | 1 or more | Arthritis in <5 Joints | >4 joints |
| High fever, | Yes | No | No |
| rash, | Yes | No | No |
| rheumatoid Factor | -ve | -ve | -/+ve |
| Serositis, | +ve | -ve | -/+ve |
| Acute painful uveitis | -ve. | +ve | -/+ve |
| Risk chronic uveitis | Low | High | Low |
| Lymphadenopathy, Hepatosplenomegaly, | +ve | -ve | -/+ve |

## 42.3 Juvenile Systemic Lupus Erythematosus

This is a multisystem disease that manifests with symptoms in virtually all parts of the body. The clinical and laboratory features are listed below and form a diagnostic criteria as per the American college of rheumatology (ACR) and European Leugues against Rheumatism (EULAR).

Clinical features and diagnostic criteria are shown in table 42.2 below

**Investigations**
Generally, children with suspected SLE should be referred to a centre with a rheumatologist for investigations and initiation of management. Investigations for suspected lupus include the following (see table 42.2 on the investigations necessary for disease diagnosis and classification).

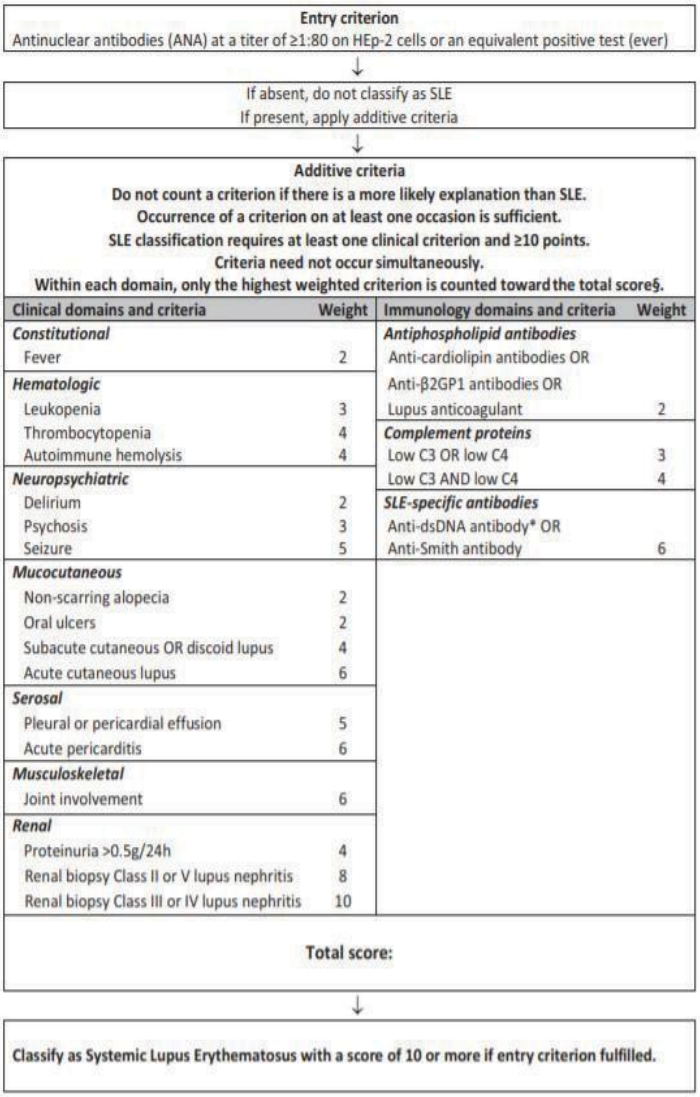
- Antinuclear antibodies (ANA).
- Antibodies specific to SLE: Anti-double stranded antibodies, anti-smith antibodies.
- Antiphospholipid antibodies: anti-beta 2 glycoprotein 1; anti-cardiolipin and lupus anticoagulants.
- Complement levels: C3, C4.
- Direct coomb's test.
- Full haemogram.
- Othe auto-antibodies such as anti-SSA, antui-SSB.
- Urinalysis

**Management**
Once confirmed, a diagnosis of SLE should be communicated to the family in a way that enables the family to cope with and support the child adequately.

- Counselling/ psychosocial support is key to management and includes issues of drug adherence.
- Supportive care includes analgesia as needed.
- Specific pharmacotherapy includes:
  - Hydroxychloroquine for all cases unless specifically contra-indicated.
  - Oral steroid therapy.
  - For severe disease, pulse steroid therapy and induction therapy with either i.v cyclophosphamide or mycophenolate mofetil (600mg/M2/dose every 12 hours)Maintenancetherapy after induction with either mycophenolate mofetil at above doses or azathioprine (2mg/kg/day).

**Complications/ prognosis**
The main determinant of morbidity and mortality is renal failure. Indeed, SLE is a major cause of chronic kidney disease in children.

**Figure 42.1: European League Against Rheumatisim/American College of Rheumatology Classifaction Criteria for Systemic Lupus Erythematosus**



**Entry criterion**

Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

↓

If absent, do not classify as SLE
If present, apply additive criteria

↓

**Additive criteria**

**Do not count a criterion if there is a more likely explanation than SLE.**
**Occurrence of a criterion on at least one occasion is sufficient.**
**SLE classification requires at least one clinical criterion and ≥10 points.**
**Criteria need not occur simultaneously.**
**Within each domain, only the highest weighted criterion is counted toward the total score§.**

| Clinical domains and criteria | Weight |
|---|---|
| ***Constitutional*** | |
| Fever | 2 |
| ***Hematologic*** | |
| Leukopenia | 3 |
| Thrombocytopenia | 4 |
| Autoimmune hemolysis | 4 |
| ***Neuropsychiatric*** | |
| Delirium | 2 |
| Psychosis | 3 |
| Seizure | 5 |
| ***Mucocutaneous*** | |
| Non-scarring alopecia | 2 |
| Oral ulcers | 2 |
| Subacute cutaneous OR discoid lupus | 4 |
| Acute cutaneous lupus | 6 |
| ***Serosal*** | |
| Pleural or pericardial effusion | 5 |
| Acute pericarditis | 6 |
| ***Musculoskeletal*** | |
| Joint involvement | 6 |
| ***Renal*** | |
| Proteinuria >0.5g/24h | 4 |
| Renal biopsy Class II or V lupus nephritis | 8 |
| Renal biopsy Class III or IV lupus nephritis | 10 |

| Immunology domains and criteria | Weight |
|---|---|
| ***Antiphospholipid antibodies*** | |
| Anti-cardiolipin antibodies OR | |
| Anti-β2GP1 antibodies OR | |
| Lupus anticoagulant | 2 |
| ***Complement proteins*** | |
| Low C3 OR low C4 | 3 |
| Low C3 AND low C4 | 4 |
| ***SLE-specific antibodies*** | |
| Anti-dsDNA antibody* OR | |
| Anti-Smith antibody | 6 |

**Total score:**

↓

**Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.**

Source: Arthritis & Rheumatology Vol.71, No.9, September 2019

# 43. Mental Disorders

Childhood mental dysfunction is not uncommon. However, it is often overlooked especially in busy clinics with a lot of sick children with somatic illnesses. These illnesses depend on recognition by the parents and, to some degree, the teachers especially if the children are in school. Assessment of such children requires a friendly and a non-threatening environment. It is important to observe as the child plays, relates to the parent and the environment as well as to the clinician depending on the age of the child. The older child with mental illness is able to relate and talk to the clinician. Early recognition of children with mental illness and their referral to a mental specialist is important.

## 43.1 Vegetative Disorders

These include eating (pica, bulimia and anorexia nervosa) and elimination (enuresis and encompresis) disorders. Encompresishas already been dealt with.

### 43.1.1 ENURESIS AND ENCOPRESIS

**ENURESIS (BED WETTING)**

Most children by 5 years will be dry at night. Enuresis is more common in boys. It may be a feature of diseases like renal diseases, cardiac diseases, diabetes mellitus, and seizure disorders. Enuresis is categorized as primary when a child has never been dry, and secondary when a child has been dry for at least 1 year before starting to bed-wetting again. Secondary enuresis is usually due to some stressful event(s) in a child's life. However, it is important to rule other diseases that have been mentioned earlier.

**Management**

The general management of enuresis involves the following:

- Getting the cooperation of the child and parent.
- Avoiding punishment and humiliation to the child.
- Limiting evening and night fluid intake.
- Giving low dose imipramine, which may help in difficult cases.
- For secondary enuresis, dealing with the causative factors.

### ENCOPRESIS

Refers to passage of feaces into inappropriate places after chronologic age of 4 years. It includes encopresis associated with constipation and overflow incontinence. It may be primary if the child has never been toilet trained or secondary if it occurs in a child that was previously toilet trained. The disorder is more common in males.

**Clinical Features**

- Assess for fecal retention by rectal exam or abdominal Xrays.
- Abnormal anal sphincter function which is often associated with chronic constitipation.

- Primary encopresis is associated with global developmental delays while secondary encopresis is often associated with social stressors.
- School performance and attendance may be affected as the child may be subject of scorn and ridicule by the school mates.

**Treatment**
- Clearance of impacted stool material.
- Short term use of mineral oil or laxatives to prevent further constipation.
- Behavioral therapy – compliance with regular post-prandial toilet sitting and adoption of high fibre diet.

## 43.2 Anxiety Disorders

Anxiety disorders are the commonest psychiatric disorders in children and adolescents. It may be difficult to distinguish between an anxiety disorder and normal anxiety, but it is very important to make such a distinction. The three types of anxiety disorders are:
- Separation anxiety, in which the affected child shows excessive distress when separated from home and may refuse to go to school or sleep away from home.
- Phobia, where there is persistent fear of social situations, school, animals, and other phenomena.
- Post-traumatic stress disorder, which is related to a traumatic or a life threatening event.

**Management**
- Counselling the child and family
- Behaviour treatment – teaching the child coping mechanisms
- Play therapy

## 43.3 Mood Disorders: Depression

**Clinical Features**

The clinical features depend on the age of the child, as illustrated below:
- In infants, there is panic behaviour and irritability initially looking for a parent/ care giver. This is followed by the child losing interest in every body. Child becomes inactive, apathetic with sad facies.
- Affected children have a sad face, are withdrawn, have poor feeding and poor sleeping, with poor school performance.
- In adolescence, there is fatigue for no apparent reason, lack of interest in normal activities, poor school performance, and suicidal tendencies.

**Management**

- ♦ General:
  - Address problem area: social skills, school performance, family issues, and any event that may have precipitated the depression.
  - Psychotherapy such as cognitive behavioral therapy especially in adolescents.

- ♦ Pharmacological:
  - Mood stabilizers: Lithium, carbamazepine, valproate.
  - Selective Serotonin reuptake inhibitors are effective in reducing depressive symptoms.
  - Tricyclic antidepressants are less efficacious in children.



## 43.4 Conversion Syndromes (Hysteria)

These are mental disorders in which there is a psychogenic disturbance of either motor or sensory function in some parts of the body.

**Clinical Features**

Patients with this condition may present with paralysis of a part of the body, tremors, blindness, deafness, seizures, or aphonia. The severity of disability fluctuates, and the patient fails to exhibit the seriousness the disability accords.

**Management**

- ♦ Good psychiatric history to reveal the source of conflict.
- ♦ Thorough physical examination to exclude an organic problem.
- ♦ Counselling and behaviour modification.

## 43.5 Disruptive Behaviour Disorders

### 43.5.1 ATTENTION DEFICIT/HYPER ACTIVITY DISORDER

**Clinical Features**

The onset of this condition is usually before the age of 7 years. The child is permanently on the move during the waking period, leading to poor sustained attention, and as a result finds it difficult to complete tasks and is inattentive. Very often the child is labelled as being stubborn by the parents and has poor school performance.

**Management**

- ♦ General
  - Use behaviour modification approaches at home and school.
  - Provide a structured learning environment at school and home.

- Pharmacological–Drugs are given in the morning. Another dose in the afternoon can be given if needed:
  - Methyphenidate: start with 0.3mg/kg/dose or 2.5–5mg/dose increase by 0.1mg/kg/dose to a maximum dose of 2mg/kg/day.
  - Atomoxetine: 0.5/kg/day to a maximum of 1.2mg/kg/day.

## 43.5.2 CONDUCT DISORDERS

- These are defined as repetitive and persistent behaviours that violate societal norms.
- Children present with truancy, drug abuse, defiance of authority, stealing, excessive lying, running away from home, aggressiveness, and involvement in criminal activities. Such children often have a background of family disharmony.

**Management**

- Behaviour modification
- Mentorship recreation programmes
- Involvement family and other relevant authorities.
- Sometimes it may be necessary to resort to legal sanctions

## 43.5.3 PERVASIVE DEVELOPMENT DISORDER

The conditions appear early in life and affects the child's social, cognitive, and language development. These disorders include autistic disorder, Asperger's disorder, and Rett's disorder.

**AUTISTIC DISORDER (AUTISM)**

Children with this condition show marked impairment of social and emotional interaction with the people around them. The onset is in the first year of life. There is lack of language development, the child is inflexible and may have ritualistic behaviour. It is important to exclude medical conditions like cerebral palsy and hearing impairment.

**Management**

- Clear diagnosis by an experienced specialist
- Family behavioural therapy
- Special school

## 43.5.4 CHILDHOOD PSYCHOSIS

Childhood schizophrenia, bipolar mood illness, and depression may present with psychotic features similar to those in adults. The age of onset is usually after 12 years, rarely before that age. Those with very early onset may be difficult to diagnose; they are often mistaken for having some conduct disorder and have poor school performance.

**Management**

Refer to a psychiatrist.

## 43.5.5 SUBSTANCE ABUSE RELATED DISORDERS

A substance is defined as any chemical with brain altering properties. The disorders that result from repeated maladaptive use of these substances is the focus here. Substance abuse disorders are characterized by significant impairment in psychological, social, and occupational functioning as observed over a12-month period.

Commonly abused substances in Kenya include tobacco, Cannabis sativa (bhangi), khat (miraa), opioids (heroin), cocaine including crack cocaine, and solvents (glue, petrol, wood varnish). Substance-related syndromes include intoxication, dependence, withdrawal, psychosis, mood disorders, anxiety, sleep disorders, and sexual disorders. Those at high risk include children aged 12–20 years and patients with primary mental disorders.

**Management**

- Substance specific detoxification
- Patient/family education/counselling
- Alternative leisure activities
- Work/school rehabilitation
- Involvement of community agencies, e.g., religious organizations, Alcoholics Anonymous, Narcotic Anonymous where available.
- Refer for long-term management by psychiatrist.

## 43.5.6 SUBSTANCE ABUSE BY THE ADOLESCENT

Such patients usually present with self-neglect, slovenliness, deteriorating school performance, excessive sleeping, rough appearance, increasing and unexplained demand for money from care givers, involvement in petty crime (pilfering), and running away from home – in addition to aforementioned substance-related disorders.

**Management – General Principles**

- Substance specific detoxification
- Patient/familyeducation/counselling
- Alternative leisure activities
- Work/school rehabilitation
- Involvement of community agencies, e.g., religious organizations, Alcoholics Anonymous, Narcotic Anonymous where available.
- Refer for long-term management by psychiatrist.

**Management – Pharmacological**

- For agitation,use: Diazepam 0.2–0.8mg/kg/dose. Max 0.6mg/kg/dose PO daily to be tapered off in 10 days.
- For the parasympathetic upsurge,use:Clonidine5–7µg/kg/24hour, maxdose 0.9mg/24 hour PO daily for 10 days.
- For any assaultive behaviour, use: Haloperidol 0.05–0.15mg/kg/24 hour; children over 12 years 2–5mg/dose TDS PO;***OR*** chlorpromazine 2.5–6mg/kg/ 24 hour 4–6-hourly TDS as necessary.
- For pain, use: Paracetamol 20–40mg/kg/24 hour 4–6-hourly PO as necessary.

**Management of Selected Substances of Abuse**

- ***Opioid detoxification:*** Opioids abused include heroin, morphine, dihydro-codeine, and pethidine. Tolerance develops rapidly and withdrawal features include agitation, lethargy, sweating, goose flesh, running nose, shivering, musculo-skeletalpains,diarrhoea,and abdominal cramps. These effect speak at 48 hours and subside over a period of 10 days. Owing to the highly addictive nature of opioids, admission to hospitals is necessary.
- ***Cannabis dependence:*** Chronic users may develop psychosis, anxiety, mood disorders, and a withdrawal state. Admission is usually necessary for initiating abstinence. Treatment of the psychiatric complication is the same as for the primary syndromes.
- ***Khat (miraa) dependence:*** Chronic users ("2 kilos" or more per day) may develop anxiety, mood disorders, and schizophrenia-like psychosis. Abstinence is to be encouraged. Treatment of the related psychiatric disorders is the same as for the primary syndromes.
- ***Solvent abuse:*** Solvents have powerful euphoriant properties. They are mainly abused by street children and the homeless. Chronic users may develop organ damage (liver, heart, kidney), apart from neurological damage. Patient education is vital. Involve family and relevant authorities in rehabilitation if possible.

## 43.5.7 SUICIDE ATTEMPTS

Suicide is an unsuccessful attempt to end one's own life. It is more common in adolescents following severe social problems or stress. A suicide attempt is used as a desperate attempt at conflict resolution, But it may also be due to depression, schizophrenia, or influence of alcohol/drugs.

**Management**

Refer to higher level for appropriate management, which would include admission. The following are general principles to observe for such patients:

- Admit.
- Urgently restore physical fitness, which is important for the person's wellbeing.
- Once patient's life is out of danger, take a full history without accusing the patient.
- Treat the patient with understanding and respect. An emphatic approach is very important if you are to win the confidence of the patients so that they will be able to volunteer the true story.
- ***Regard every suicide attempt as serious.*** The next attempt may be successful. Do not regard an attempt as just attention seeking.
- Explore the underlying cause and provide counselling.
- Involve the parents/ family.

# 44. Child Health

All parents are encouraged to take their children for immunization immediately after birth. Presentation of the child health card at every visit to a health facility helps to detect children who missed previous vaccinations. Health workers can also check on health cards of children in the community.



**— It is necessary that informed consent be obtained before any vaccination, from either the parent or the patient.**

**Vaccine Administration**

**The following is important for vaccine administration:**

- The vaccine dose should always be checked against the instructions on the vaccine.
- Site for vaccine administration (refer to table 44.1 for individual vaccines).
- Simultaneous administration of uncombined live vaccines must be given at different sites.
- Minimum interval between vaccine doses should be 4 weeks.

**Age at Vaccination**

Vaccines are given at specific ages, in accordance with the national immunization schedule, shown below. The list includes vaccines not currently on the national vaccination schedule but indicates when such vaccines could be given.

- Vaccination of the preterm baby follows the chronological age rather than weight.
- Pre-term infants and low birth weight infants (<2500g) should receive the BCG vaccine at the time of discharge from hospital irrespective of the weight at discharge.

Vaccines currently given as part of the routine national immunization schedule are listed in table 45.1 below.

- Refer to table 45.1 for the full immunization schedule which includes other vaccine antigens that are available in the country but are not yet part of the routine national immunization schedule.

**Specific Instructions**

The following are general instructions with respect to immunization:

- A slight fever and/or other minor illness should not prevent you from immunizing a child.
- Children should be vaccinated during recovery from a serious illness if they had missed the vaccine.
- Mothers/child-caregivers should be informed about possible side effects of each of the given vaccines.
- All vaccinations should be recorded on tally sheets and on the Child Health Immunization cards and mothers should be instructed to always bring the cards along with them when taking children to a health facility.

- Mothers should be instructed to return the child for the next immunization on the date indicated on the card.
- The disposal of used sharp syringes should be handled appropriately to prevent injury and spread of diseases like HIV.
- To ensure appropriate cold storage of the vaccines, follow the recommended cold-chain instructions for each of the vaccines carefully. All the vaccines and diluents must be kept cold. DPT, HB, and TT vaccines are damaged if kept below 0°C and therefore should never be frozen. Always check the Vaccine Vial Monitor (VVM). The cold chain should be maintained because vaccines are easily destroyed by heat and rendered ineffective.
- Hands should be washed before and after handling vaccines.

**Contraindications**

— Anaphylaxis after previous dose or severe allergy to vaccine component is a contraindication to further doses of the same vaccine.
— In pregnancy, live vaccines including OPV, Measles-Rubella, Yellow Fever and HPV vaccines.
— Severely immunocompromised; Live vaccines including BCG, OPV, Measles-Rubella and Yellow Fever.

***https://vaccine-safety-training.org/contraindications.html***

## 44.1.1 IMMUNIZATION IN SPECIAL SITUATIONS

**Immunization in Immunocompromised Host**

- HIV/AIDS infection:
  - HIV exposed and asymptomatic children infected with HIV should receive all standard Kenya Expanded Programme on Immunization (KEPI) vaccines.
  - BCG vaccination should not be repeated if there is no scar formation.
  - live and live attenuated vaccines are to be avoided in symptomatic HIV infected children, as well as in children with unknown HIV status but who are symptomatic for HIV infection.
  - The National Vaccines and Immunization Program does not recommend testing for HIV before giving vaccinations.
  - The immunization program recommends a supplemental Measles vaccine dose at age 6 months for asympromatic HIV infected children and all children from age 6 months during measles outbreak response.
- Oncology patients, patients on immunosuppressive therapy and primary immunodeficiencies:
  - Live vaccines are generally contraindicated in severely immunocompromised individuals.
  - Severely immunocompromised patients include patients on Immunosuppressive therapy including high dose Corticosteroid

therapy (more than 0.5mg/kg/day or >10mg per day of prednisolone equivalent):
  - For those on immunosuppressive therapy, Live vaccines may be given after cessation of immunosuppressive therapy .
- Children with Immune Mediated Inflammatory Disorders (IMIDs)
  - Immune mediated inflammatory disorders include Juvenile idiopathic arthritis, Systemic Lupus Erythematosus, Diabetes mellitus, inflammatory bowel disease, psoriasis and other autoimmune and autoinflammatory diseases.
  - It is recommended that children with IMIDs follow the normal routine immunization schedule.
  - Live vaccines are generally to be avoided in children with IMIDs and should be given before start of or after stopping treatment with immunosuppressive drugs.
  - The non-live vaccines can be given safely at any time to age-appropriate children before, after or during immunosuppressive treatment.
  - All children with IMID should receive a single booster (4th) dose of pneumococcal (PCV) vaccine at the age of 2 years or later and varicella vaccine (if not previously given and no history of having contracted chicken pox provided the child is not on immunosuppressive treatment).
- Pregnancy:
  - Generally live vaccines are contraindicated during pregnancy unless the risk of disease outweighs the risk of vaccine, e.g.during yellow fever epidemic.

**Side Effects and Adverse Reactions to Vaccinations**
**The side effects range from mild to severe for various vaccines.**

- BCG vaccine: These include injection abscess, regional or widespread
- lymphadenitis, osteomyelitis, and disseminated BCG infection. These should be treated with anti-tuberculosis drugs.
- Oral polio vaccine: Adverse reactions rarely occur.
- Measles vaccine: Adverse reactions include fever, mild rash, and rarely convulsions and encephalitis.
- DPT-HepB-Hib (Pentavalent): Most adverse reactions are attributed to the pertussis component. Minor reactions include pain at the injection site and fever. Major reactions are persistent crying, high pitched cry, excessive somnolence, convulsions, encephalopathy, and coma.
- Recombinant DNA Hepatitis B vaccine: Side effects include pain, fever, and swelling at the site of injection.

## 44.1.2 IMMUNIZATION TYPES AND SCHEDULES

Kenya's national immunization schedule specifies both the schedule of vaccines (Table 44.1), and the dosage and mode of administration (Table 44.1).

**Table 44.1: Kenya Childhood Immunization Schedule**

| | Age of child | Vaccine Antigen | Dosage | Route |
|---|---|---|---|---|
| 1. | At birth or at first contact | BCG | 0.05 ml (<1 year)<br>0.1 ml (> 1 year) | Intradermal, Upper outer aspect of the left forearm |
| 2. | At birth or at first contact (within the first 2 weeks of life) | OPV birth dose (bivalent) | 2 drops | Oral |
| 3. | At 6 weeks or 1st contact after 6 weeks | OPV I | 2 Drops | Oral |
| | | DPT-HepB+Hib 1 | 0.5 ml | Intramuscular into the upper outer aspect of left thigh |
| | | PCV10- I | 0.5 ml | Intramuscular into the upper outer aspect of Right thigh |
| | | Rotavirus-1 | 1.5 ml | Oral |
| 4. | At 10 weeks or 4 weeks after OPV I | OPV 2 | 2 Drops | Oral |
| | At 10 weeks or 4 weeks DPT-HepB-Hib 1 | DPT-HepB+Hib 2 | 0.5 ml | Intramuscular into the upper outer aspect of left thigh |
| | At 10 weeks or 4 weeks PCV10 - 1 | PCV10- 2 | 0.5 ml | Intramuscular into the upper outer aspect of Right thigh |
| | At 10 weeks or 4 weeks Rota-1 | Rotavirus-2 | 1.5 ml | Oral |
| 5. | At 14 weeks or 4 weeks after OPV 2 | OPV 3 | 2 Drops | Oral |
| | | IPV | O.5 ml | Intramuscular into the upper outer aspect of right thigh, 2.5cm from PCV -3 site |
| | At 14 weeks or 4 weeks DPT-HepB-Hib 2 | DPT-HepB+Hib 3 | 0.5 ml | Intramuscular into the upper outer aspect of left thigh |
| | At 14 weeks or 4 weeks PCV10 - 2 | PCV10- 3 | 0.5 ml | Intramuscular into the upper outer aspect of Right thigh |
| 6. | At 6 months | Vitamin A | 100,000 iu | Oral |
| | At 12, 18, 24, 36 months | Vitamin A | 200,000 iu | Oral |
| 7. | At 6 Months | Measles-Rubella (MR) | 0.5 ml | subcutaneous right upper arm (deltoid muscle) during measles-rubella outbreak or HIV infected infants without severe immunosuppression |
| 8. | At 9 months or first contact after 9 months | Yellow fever (High Risk counties) | 0.5 ml | Subcutaneous into the Left upper arm (deltoid muscle) |
| | | Measles-Rubella (MR1) | 0.5 ml | Subcutaneous into the right upper arm (deltoid muscle) |
| 9. | At 18 months or first contact after 18 months | Measles-Rubella (MR2) | 0.5 ml | Subcutaneous into the right upper arm (deltoid muscle) |
| 10. | At 10 years (girls), | HPV-1 vaccine | 0.5 ml | Intramuscular left deltoid muscle |
| | At 10 years 6 months or 6 months after HPV1. | HPV-2 vaccine | | |

## 44.1.3 VACCINES AVAILABLE BUT NOT YET IN KEPI PROGRAMME

**The following are beneficial, though not yet mandated.**

- Hepatitis B monovalent vaccine: Given as soon as possible at birth; andalso recommended in health workers and other high-risk groups in three doses at 0, 2 and 6 months.
- MMR (measles, mumps, rubella): Given at 12–15 months.
- Influenza vaccine: Inactivated seasonal influenza vaccine. Given in 2 doses for children aged 6 months or older and subsequently, annually.
- Meningococcal vaccine: Polysaccharide type for age >2 years is often used to control epidemics.
- Hepatitis A: Given at the age of 12 months. Improving socioeconomic status is shifting the infection age to older children who are at a higher risk of severe Hepatitis A including fulminant hepatitis. • Routine childhood vaccination should be considered. • Travellers from low endemic areas should be considered for vaccination.
- Rabies vaccine (see national immunization guidelines and policy.
- Varicella vaccine (live attenuated): Can be given simultaneously with MMR. Can be given either routinely to all children, or post exposure to high risk groups–immunocompromised patients without history of having had varicella infection. For cancer patients it is best given during remission.
- Malaria vaccine: protein based recombinant vaccine recommended at age 6,7, 9 and 24 months.
- Typhoid polysaccharide vaccine: recommended at age 2 years and booster every 3 years. Prioritization of vaccination to those at highest risk of contracting or transmitting the disease. (Food handlers, especially those employed in institutions and hotel as well as laboratory staff and employees of sewerage and treatment works).
- Cholera: Inactivated oral cholera vaccine is available in the country and is recommended for people aged 2 years or older. Pre-emptive vaccination with oral cholera vaccines should be undertaken in epidemic prone regions of the country once the risk of cholera becomes significant due to events such as flooding and emergency displacement of communities.

## 44.1.4 IMMUNIZATION SCHEDULE FOR ALL WOMEN AND PREGNANT MOTHERS WITH TETANUS-DIPTHERIA TOXOID(TD2+)

1st dose at first contact.
2nd dose – 4 wks after first dose
3rd dose – 6 months after 2nd dose
4th dose – at least 1 year after the third dose 5th
dose – at least 1 year after the fourth dose

A total of 5 doses is recommended during a woman's reproductive age.
For pregnant women who have not received the 5 doses, give the 1st and 2nd during the first pregnancy (or first contact). The 3rd, 4th, and 5th doses can be given in subsequent pregnancies. Immunizing a pregnant mother ensures protection of her newborn baby against tetanus.

## 44.1.5 VITAMIN A SUPPLEMENTS

Vitamin A supplementation is recommended for all children at age 6 months and every 6 mmonths subsequently thereafter.

## 44.1.6 IMMUNE GLOBULINS (PASSIVE IMMUNIZATIONS)

**These may be nonspecific or specific and are given either IM or IV.**

- Nonspecific immunoglobulins: Can be used as replacement in individuals with antibody deficiency disorders.
- Specific immunoglobulins: Prepared from donors known to have high antibody to specific antigens or specific sources. Very useful in post exposure prophylaxis. Examples include rabies, varicella, and RhO(D) immuneglobulin (anti D).

## 44.1.7 RABIES

Any mammalian may carry rabies. Saliva from a rabid animal contains large numbers of the rabies virus, which is inoculated through a bite or any laceration or break in the skin. For more details refer to 1.4 in Part I of these guidelines. Pre-exposure prophylaxis vaccine is recommended for anyone above 1 year of age at increased risk e.g Veterinarians, wildlife officers and visitors to high rabies-enzootic areas. The vaccine is given intramuscularly on days 0 and 7. Post exposure prophylaxis (PEP): This is given following suspected rabid bites. For individuals who are not previously vaccinated, the vaccine is given on days 1,3,7,14 & 28. For previously vaccinated individuals, give post exposure Booster on Days 0 and 3.

**Management**

Emergency care for a suspected rabid bite includes the following:

- Thorough irrigation of bite with copious amounts of saline solution
- Cleansing the bite with a soap solution
- Debridement of the bite area
- Administration of antibiotic
- Administration of tetanus toxoid
- Delayed suture or skin grafting
- Infiltrate the wound with rabies immunoglobulin

**Indications for rabies vaccine are the following:**

- Bites from wild animals
- Bites from UNPROVOKED domestic animal
- Bites from a sick looking domestic animal, whether immunized or not
- Laboratory findings of Negri bodies in the brain of the involved animal
- Persons at high risk of exposure

**Always refer as soon as possible to a centre that can vaccinate (with the vaccine and immunoglobulin as necessary)**

## 44.1.8 SNAKE BITES AND ENVENOMATION

- All snake bites are to be treated as poisonous and patients administered the highest valency anti-snake venom available.
- The dose of anti-snake venom is the same for children and adults.

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# PART III: Surgery and Related Disciplines

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# 45. Anaesthesia and Critical Care

## 45.1 Preoperative Patient Evaluation

A patient for elective surgery needs thorough evaluation not only for suitability for general anaesthesia but also for possible complications related to or arising from the operation (e.g., a toxic goitre, chronic cough in a hernia patient). Surgical services should ideally be carried out only at level 4 facilities and above.

**Note: The staff needs to appreciate the abilities of the facility so that high risk procedures are referred to facilities with an ICU available unless they are urgent.**

### 45.1.1 HISTORY

A thorough history must be taken. This should include a history of chronic illnesses, a drug history, and a history of previous surgical encounters.

### 45.1.2 EXAMINATION

- Conduct a thorough physical examination and in particular check for:
  - Anaemia
  - Jaundice
  - Level of hydration
  - Fever
  - Lymph node enlargement
  - Respiratory and cardiac function
- Assess psychological preparedness for surgery.
- Take and record vital signs
- For any major operation an observation chart needs to be kept for atleast 24 hours before surgery. Specific charts are available for certain disease conditions, e.g., diabetes, hypertension, asthma, etc.

### 45.1.3 BASIC INVESTIGATIONS

**These should include the following:**

- Urinalysis.
- Full haemogram.
- Urea and electrolytes.
- Blood sugar.
- A chest radiograph (where applicable)
- **Additional relevant investigations** (As per the affected systems or regions)
  - Urine for culture and sensitivity.
  - An intravenous urography in most urological operations.
  - Liver function tests and prothrombin time index (PTI) in hepatobiliary disease.
  - Creatinine clearance in renal patients.
  - Electrocardiogram (ECG) in hypertensive and known heart patients.
  - A thyroid profile may be necessary before thyroid surgery.
  - CT Scan – Head (where applicable)

### 45.1.4 TREATMENT–SUPPORTIVE BEFORE SURGERY

**These include the following:**

- Correction of conditions that are identified in the preoperative evaluation as necessary.
- Correction of volume and electrolyte imbalance.
- Control of blood pressure.
- Control of thyrotoxicosis.
- Control of diabetes mellitus (and any other metabolic disease).
- Correction of anaemia and malnutrition.
- Prophylactic antibiotics where indicated (see appropriate section for details).
- Preoperative physiotherapy.
- Counselling the patient and family.

### 45.1.5 PREMEDICATION

These are prescribed to surgical patients in order to achieve the following objectives:

- Relieve anxiety (oral/parenteral benzodiazepines)
- Antiemetics like metoclopropamide (plasil 5–10mg).
- Reduction of secretions (anticholinergics like atropine 0.6mg hyoscine 10–20mg).
- Preoperative analgesia (pethidine 1mg/kg).
- Amnesia (benzodiazepines).
- Reduce gastric fluid pH and volume – Ranitidine 50mg IV

## 45.2 Use of Blood Transfusion in Surgery

The golden rule of blood transfusion should be that no transfusion is given unless the benefit of the transfusion outweighs the risks. Before all blood transfusion, therefore, there must be a balance among the risks associated with transfusion, the indications for transfusion, and the availability and benefit of using alternatives to conventional transfusion. The following is a listing of risks, indications and alternatives to traditional transfusion.

- Risks associated with transfusion
  - Viral infections
  - Bacterial infections
  - Compatibility complications
  - Heamodynamic complication
- Indications for transfusion
  - Transfuse blood intra-operatively for preoperative haemoglobin less than 6.0g/dl.
  - Transfuse blood for haemoglobin of 6–10gm% with obvious continuing blood loss or obvious morbidity like heart disease.
  - Transfuse blood for blood loss of 10% of blood volume or more.
  - Avoid “topping-up” anaemic patients prior to surgery and use altenatives to conventional transfusion (haematinics).
  - Autologous donation is frequently used in patients for elective surgery. A. pint of blood is removed every 7 days prior to surgery and is re-transfused at the

time of surgery. This blood can safely be stored for 21 days. It is important to liaise with the blood donor bank to ensure that the patient gets own blood.

- Intraoperative haemodilution where a unit is withdrawn and replaced with saline. This can be set aside for re-transfusion as needed.
- Another alternative, for level 5 and 6 facilities, is the use of cell savers during surgery as for example during abdominal aortic aneurysm surgery.
- Strictly observe all precautions that appertain to blood transfusions.



## 45.3 Antimicrobial Prophylaxis in Surgery

Antimicrobial prophylaxis can decrease the incidence of infection, particularly wound infection after certain operations, but this benefit must be weighed against cost, risks of toxic or allergic reactions, and emergence of resistant bacteria. The administration of antibiotic agents to prevent infection cannot be substituted for either sound surgical judgment or strict aseptic technique.

Surgical wounds may be designated as clean, contaminated, or dirty, as described below:

- **Clean wounds:** Chemoprophylaxis has no place in clean operative procedures.
- **Contaminated wounds:** This category includes operations such as the interior of respiratory, urinary, or gastrointestinal systems. Chemoprophylaxis may be useful in such situations.
- **Dirty wounds:** These include most traumatic wounds, which are highly contaminated. In such situations, a thorough surgical debridment is necessary, apart from chemoprophylaxis. Other highly contaminated wounds involve operations on the large intestines and severe burns.

Other risk factors in the development of infection include the development of infection secondary to malnutrition, inadequate blood supply, obesity, old age, and immunodeficiency states.

### 45.3.1 OTHER INDICATIONS FOR PROPHYLAXIS

**These include the following:**

- Operative procedures of long duration, such as cardiac and vascular procedures, orthopaedic, and neurosurgical procedures.
- In clean surgeries for insertion of a prosthesis or graft material.

### 45.3.2 PROPHYLACTIC TREATMENT

- A single dose of parenteral antimicrobial given with induction of anaesthesia before an operation usually provides adequate tissue concentrations for several hours.
- Or 3 doses cover of the same antibiotic for 24 hours.

## 45.4 Post-operative Care

**The aims of postoperative care are to:**

- Monitor the patient's postoperative period to detect and correct any anomalies.
- Keep the patient comfortable and give adequate analgesia. Rectal diclofenac 100mg (adults), paracetamol for children or pethidine 1mg/kg TDS.
- Offer supportive feeding.
- Restore normal health and independence.

To achieve the above, the surgeon must give legible, concise, and clear postoperative instructions and involve other team members like physiotherapists in the management of the patient.

### 45.4.1 IMMEDIATE POST OPERATIVE RECOVERY PHASE

This period normally lasts about 1–2 hours from theatre to that period in the recovery ward, where facilities allow. During that period, the following are carried out:

- Keep the patient in semi prone (recovery position) with extended neck and flexed limbs.
- Maintain clear airway using oropharyngeal airway and provide supplemental oxygen till fully awake.
- Monitor vital signs ½ hourly.
- Keep in recovery ward till fully awake (arousal).

### 45.4.2 TRANSIT FROM THEATRE TO WARD

- During this process, keep airway clear to avoid upper airway obstruction and aspiration pneumonitis. Use recommended trolley with siderails.

### 45.4.3 POST OPERATIVE CARE IN FIRST 24 HOURS

**This involves the following procedures:**

- Continue observing vital signs 4 hourly or as often as individual case demands.
- Relieve pain with analgesia,e.g.,pethidine 50–100 mg every 6 hours in adults and in children 1mg/kg in divided doses or use infiltration method at the operative table as for example intercostals infiltration in thoracotomies.
- Transfuse if necessary.
- If not feeding, give intravenous fluids, Hartmann's, normal saline, or 5% or 10% dextrose about 4 litres in 24 hours for a 70 kg adult. Titrate against state of hydration.
- Maintain an input and output chart (urine output 1–2ml/kg/hour).
- Watch for airway obstruction, reactionary bleeding,etc.
- Attend to drains if in situ and make sure they are draining.
- Offer general nursing care, e.g., keep patient warm, turn in bed, and change wet linen to avoid bedsores.
- Carry out appropriate wound care.

### 45.4.4 POST OPERATIVE PERIOD 72 HOURS –7 DAYS

During this period, the following procedures are carried out:

- Mobilize out of bed after about18–72 hours to avoid static pneumonia and thrombosis.
- Encourage independence, e.g., self feeding, attention to calls of nature.
- Give oral medication as appropriate.
- Take observations every 6 to 12hours.
- Carry out wound care asappropriate.



## 45.5 Theatre Etiquette

In order to attain optimal results, a doctor operating in theatre and the anaesthetist is expected to maintain the following "rules":

- Be involved in the preoperative selection and preparation of patients including obtaining of informed consent.
- As the team leader, maintain strict time discipline and other aspects of good leadership.
- Be familiar with any problems with the theatre set up for the day well in advance.
- Accord respect to all team members.
- Observe the required sterility requirements int heatre.
- Observe the required health and safety rules while operating.

**— In our environment it is mandatory to wear eye goggles and aprons to avoid the dangers associated with blood splashes.**

- Operate in the safest environment and do not allow attention to wander during surgery.
- Observe silence and as much as possible avoid discussions not directly related to the surgery.
- Make sure to write own operating notes, or if delegated, make it a point to confirm what has been documented.
- Ensure anaesthetic notes are complete and accurate.
- Do not leave the theatre before the patient is handed over to the ward team. unless care has been delegated to another competent team.
- Follow up the patient till the time of discharge.
- Conduct regular assesments for quality improvement purposes.

## 45.6 HIV/AIDS and the Surgeon

The HIV/AIDS disease in Kenya is currently affecting all age groups, including teens. It is important for a surgeon to appreciate the varying surgical presentations this disease has. Strict surgical barriers must be implemented at all times to avoid spreading of the disease between the patient and the surgeon. This can be achieved through

community dissemination of knowledge, individual or group counselling, knowledge skills, and the use of physical barriers like double gloving, gowns, and surgical goggles.

The surgeon should always be on the look out for signs of possible HIV infection in the patient; these include:

- Wasting
- Enlarged nodes
- Skin lesions like Kaposi's sarcoma or herpetic rash
- Oral and oesophageal lesions
- Pulmonary lesions
- Other gastrointestinal lesions

Counselling of both the patient and the relatives is important and an assessment of the suitability of the patient for surgery. The CD4 cell count is a useful indicator of perioperative risk.

NB: This topic is dealt with in detail the chapters for Internal Medicine, Paediatrics and Obstetrics and Gynaecology.

# 46. Abdominal Injuries

**Summary**
The spleen, liver, retroperitoneum, small bowel, kidneys, bladder, colorectum, diaphragm, and pancreas tend to be the most commonly injured organs. Their proper clinical assessment is vital. Abdominal injuries can be masked by injuries elsewhere, e.g., fractured limbs, fractured ribs or spinal cord, and head injuries, and may also develop slowly. If a patient has multiple injuries, assume the abdomen is involved until this is ruled out. Organomegaly makes the involved organs more vulnerable to abdominal trauma, so be cautious with children with pretrauma splenomegaly.

**Unexplained shock in a trauma patient should point towards an intra-abdominal bleed.**

**Clinical Features**
- Vital signs (pulse rate, blood pressure, respiratory rate, temperature, SP02)
- Obvious bruises
- Abdomianl wall wounds
- Pain
- Loacalised tenderness
- Rigidity of the abdominal wall (indicates the most likely site of injury)
- Abdominal distension (could be due either to gas leaking from a ruptured viscous or from blood from injured solid organ(s) or to torn blood vessels. This is a serious sign)
- Haematurea (occurs in bladder injuries).
- Haematochezia (rectal injuries)

**NOTE: The absence of bowel sounds or sustained shock despite resuscitation mandates urgent surgical intervention.**

**Investigations**
- X-rays of the abdomen and chest x-rays may show existing fractures, foreign bodies, gas under the diaphragm, gas under the anterior abdominal wall, or bowel loops in the chest.
- Ultrasound or CT scans of the abdominal wall (where applicable).
- Total blood counts are useful for serial assessments.
- Blood group and cross-match blood if intra abdominal bleed is suspected

**Note:**
- Bloody nasogastric aspirate may indicate upper gastrointestinal tract injuries.
- Peritoneal lavage is indicated in the following patients:
  - Patients with spinal cord injury.
  - Those with multiple injuries and unexplained shock.
  - Confused patients with a possible abdominal injury.
  - Intoxicated patients in whom abdominal injury is suggested.

**Management**

- Maintain airway and breathing.
- Circulation - Is your patient in shock? (Has low BP, high pulse rate, cold clammy extremities).
- Secure a wide bore (Gauge 18 in adults) intravenous cannula.
- Take blood sample for grouping and cross matching.
- Start intravenous fluid appropriately.
- Cathetarize the patient appropriately.
- Clean, stitch, and dress small superficial wounds, but do not let this adversely delay referral. (Management at level 2 and 3 is limited mainly to patient resuscitation in order to stabilize)
- Give tetanus toxoid 0.5ml intramuscular STAT as per expamded program of immunization (EPI) schedule.
- Start antibiotics appropriately.
- Keep patient warm and comfortable.
- Closely monitor BP, pulse rate, respiratory rate, temperature, and urine input and output.
- Measure abdominal girth, as this may prove useful in follow up of patients' progress.

**NOTE**

- If not sure of wound depth, explore the wound directly under local anaesthesia.
- Explore penetrating wounds early.
- In blunt trauma, manage according to clinical findings and how they evolve overtime. Mild symptoms are managed conservatively, while deterioration is managed by abdominal exploration.
  - Indications for laparotomy in blunt trauma include:
    - Persistent abdominal tenderness and guarding.
    - Persistent unexplained shock
    - Paralytic ileus
    - Positive peritoneal lavage or positive ultrasound findings pneumo-
  - Manage specific organ injuries at laparotomy.
- Inform receiving facility prior to referral as trauma needs urgent attention on arrival.
- At discharge, provide adequate documentation to be sent back to referring facility.

# 47. Animal and Snake Bites

These include bites by humans, dogs, and other domestic and wild animals, as well as wild animal bites.

**Management**
This will depend on the extent of tissue loss and the site of injury and the type of animal. Most bites consist of cuts and simple lacerations. Other animals (hippopotamus and crocodiles) inflict major tissue destruction (lacerations, avulsions, and amputations).

**Immediate Care**
If not already acted upon at lower health facilities, stop all bleeders by pressure and ligature while preparing for thorough debridement. Administer a pain reliever.

**Local Care**
- Clean cuts and lacerations thoroughly with normal saline (hydrogen peroxide is indicated for septic wounds only). .
- Dress with povidine solution appropriately.
- Give tetanus toxoid 0.5ml IM STAT as per expanded programme (EPI).
- Give analgesia as appropriate.
- Give antibiotics appropriately.
- Give rabies vaccine where indicated (Section 1.4.2, on rabies management.)
- Give antivenom for snake bites in appropriate cases.

**NOTE**
- Consider urgent referral if rabies vaccine or antisnake venom is not available in facility within 24 hours. Ensure adequate documentation and availability of resuscitation equipment during the actual referral phase.
- For large bites, carry out surgical debridement under anaesthesia.
- DAILY dressing is advised and later skin graftingor flap repair is performed.
- Open chest injuries will require closure and underwater seal drainage.
- Open abdominal wounds will necessitate an exploratory laparotomy.
- In the case of amputated extremities, carry out debridment and stump refashioning where necessary followed by appropriate rehabilitation and appropriate assistive device.
- Should the patient be in shock, treat aggressively with saline infusions, blood transfusions, and vasopressor agents.
- In major tissue destruction, administer appropriate antibiotics.

# 48. Burns

The majority of burns are caused by heat, which may be open flame, contact heat,and hot liquids (scalds). Others are chemical, electric, friction, sunburns, and irradiation. Extreme cold can cause tissue injuries (i.e.,frostbite).

Intervention in burns patient should aim to prevent the following complications

- Airway obstruction
- Fluid and electrolyte imbalance
- Acid base balance
- Infections
- Hypothermia
- Joint stiffness/contractures
- Anaemia
- Muscle protein catabolism
- Compartment syndrome

## 48.1 Initial Management of Burn Cases

### 48.1.1 FIRST AID MEASURES

**If not acted on at lower level, initiate the following management plan:**

- Airway: Ensure patient has a clear airway.
- Breathing: Ensure patient is breathing and receiving oxygen by mask if need be.
- Circulation:
  - Ensure adequate intravenous access and availability of intravenous crystalloids;
  - Assess peripheral circulation and look out for compartment syndrome
  - Group and cross match blood.
- Give tetanus toxoid and analgesics.

### 48.1.2 QUICK ASSESSMENT OF THE EXTENT OF BURNS

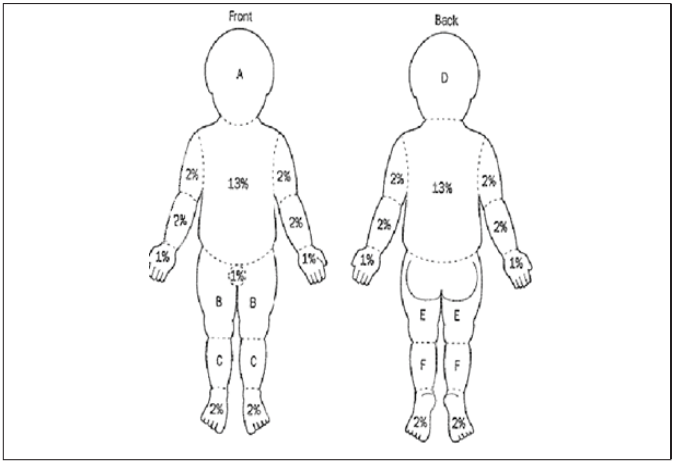
- Degree of burn:
  - First degree: Epidermis only involved.
  - Second degree: Epidermis and portions of dermis involved.
  - Third degree: All skin layers, including the subcutaneous tissue are involved.
- Special sites of injury (note facial, perineal, hands, and feet).
- Look out for circumferential burns on extremities.
- Look out for other injuries (for example: fractures, head injuries, chest injuries,
  abdomen, etc)
- The Wallace Rule of Nines (see Figure 48.1) is used to estimate the extent of burns
- Admit if meets admission criteria.

**— Initiate fluid management schedule.**

## 48.1.3 CRITERIA FOR ADMISSION

- Extent of burns: Are >10% body surface area. If the extent is >25% of body surface area, transfer to special burns unit.
- Degree of burns:
  - Hands and feet
  - Face and neck
  - Perineum
  - Circumferential
  - Joints and other associated injuries
- Inhalational burns.
- Chemical and electricburns.
- In the presence of other known pre-existing diseases, e.g., diabetes mellitus.
- Other burns requiring hospitalization: full thickness burn, circumferential burns.

**Figure 48.1: Body surface area estimation in children**




It is safer to overestimate body surface area than to underestimate it. A useful rough guide is to estimate the palm of the hand excluding the fingers as being approximately 1%. The total body area is critical to the fluid management of the burn patien

**Table 48.1: Change in body surface area with growth**

| Body area | < 1 yr (%) | 1 yr (%) | 5 yr (%) | 10yr(%) | 15 yr (%) |
|---|---|---|---|---|---|
| Head (A/D) | 10 | 7 | 7 | 6 | 5 |
| Thigh (B/E) | 3 | 3 | 4 | 5 | 5 |
| Leg (C/F) | 2 | 3 | 3 | 3 | 3 |

## 48.1.4 AMOUNT OF FLUIDS TO BE ADMINISTERED

**Calculation Using the Parklands Formula**

- 4 x Total body surface area burnt x weight in Kg = ml of fluids to be administered within the first 24 hours from the time of the burns.
- The total fluids calculated should be administered as indicated below:
  - First 8 hours from the time of burns=1/2 total calculated fluid
  - Next 8 hours = ¼ total calculated fluid
  - Next 8 hours = ¼ total calculated fluid
- As an example, for a 80kg man with 20% burns, total fluid (80kg x 20% x4) ml = 6400 ml. Administer as follows:
  - 3,200 ml within the first 8 hours
  - 1,600 ml next 8 hours
  - 1,600 ml over the next 8 hours

**Other Fluid Management Considerations**

**For management of a person with burns, the following is necessary:**

- Types of fluids to use these should either be normal saline or Hartman's solution
- Monitoring should be carried out for vital signs, urine output (maintain atleast 1–2ml/kg/hr) and packed cell volume.
- Care of the burn surface includes the following:
  - Cleaning with normal saline , antiseptics or normal saline
  - Applying antiseptic cream like silver sulphadiazine when nursing wounds.
  - Cover using a cradle.
  - Using a moist plastic bag for burns of the hands and feet after antiseptic cream application.
  - Early surgical debriding of dead, burned tissue and skin grafting for extensive burns.
- Pregnant mothers are more prone to the effects of burns than non pregnant females. For pregnant women with burns observe thefollowing:
  - Prompt and aggressive fluid management is essential.
  - Pregnancy is associated with a 50% increase in intravascular volume as well as a 43% increase in cardiac output.

— These factors in addition to others make ***pregnant women more prone to fluid loss associated with burns***. As a result, a pregnant woman will not likely conform to the Parklands fluid replacement formula and may need up to twice this volume. In fluid resuscitation for these patients' variables like urine output, heart rate, central venous pressure, and mean arterial pressure are more reliable indicators of successful resuscitation.

- Types of fluids used for treating burns include the following:
  - Crystalloids
  - Normal saline
  - Ringer's lactate solution (use with caution in patients who have associated metabolic acidosis following burns)
- The type of monitoring required for patients with burns includes the following:
  - Vital signs
  - Urine output (maintain at least 1–2ml/kg/hr)
  - Urea and electrolytes
  - Packed cell volume
  - Blood gas analysis for severe burns.

## 48.2 Special Burns

### 48.2.1 TYPES OF BURNS

- Circumferential burns:Do an early escharotomy to prevent compartment syndrome.
- Elevate the affected limb and appluy crepe bandage appropriately to reduce oedema
- Inhalational burns: Should be suspected if there are burned lips and/or burned nostrils, especially in cases of open fires land smoke. Give humidified air and oxygen, bronchodilators. and appropriate antibiotics. Intubation may be necessary.
- ***For pregnant females with burns,*** early intubation and mechanical ventilatory support is strongly recommended if inhalation burn injury is suspected due to risk of tracheal oedema. Both the functional residual capacity and the residual volume are decreased by 20% in pregnancy, making ventilatory support particularly important.
- Electrical burns: These are deep burns and require specialized care.
- Chemical burns:To manage these types of burns, irrigate with plenty of water and soap.

### 48.2.2 MANAGEMENT OF ELECTRICAL BURNS

Low voltage electrical injury tends to be associated with electrocution (cardiac arrest), while high voltage burns are associated with extensive tissue destruction rather than electrocution. The body tissues most vulnerable to electrical injury are peripheral nerves and skeletal muscles.

- Injury from electrical burns occurs through two main avenues:
  - Electric shock resulting in cardiac arrhythmias and muscle spasm.
  - Thermal injury resulting in muscle destruction.
- Diagnosis of electrical burn can be made on the basis of:
  - History of contact.
  - Presence of 2 contact injury points on skin.
  - Presence of cardiac arrhythmias and respiratory disturbances.
  - Presence of skeletal fractures secondary to muscles spasms.
- Management at levels 4 to 5:
  - Resuscitate as appropriate.

- Maintain a fluid balance and urine output.
- Initiate or continue analgesia. For severe burns, give morphine10mg IV 6 hourly.
- If more specialized treatment is needed refer to burns unit.

♦ At level 6 (burns unit):
- Cardiovert if needed. Cardioversion may be electrical or chemical.
- Maintain adequate urine output, more than 50ml/kg/hr, but raise to more than 2ml/kg/hr to avoid renal failure secondary to myoglobinaemia.
- Give anti arrhythmic drugs if needed.
- Administer tetanus toxoid as in all burns.
- Look out for compartment syndrome.

♦ The rest of the treatment plan will follow in a similar fashion to other burns.
♦ Skin grafting shortens the duration of hospital stay and should be performed early when necessary.
♦ Start physiotherapy and occupational therapy early.



## 48.3 Mortality Risk from Burns

Improvements in wound care and the use of antibiotics have had influence on survival following burns. However, the risk of mortality is directly related to the body surface area that is burned. Other relevant independent factors influencing mortality are the immune status of the patient and the presence of respiratory burns.

# 49. The Multiply Injured Patient

A patient injured in more than two body systems is defined as multiply injured. This situation commonly occurs in road traffic accidents, falls from a height, blast injuries among others.The approach to a patient with multiple injuries has to be systematic in order to identify all the injuries and prioritize their sequence of attention.

## 49.1 Resuscitation Required and Its Order

- Airway: Position the head and with finger or suction, clear blood, mucus, and foreign bodies. Take care to avoid causing cervical injury and apply cervical collar or use the jaw lift manoeuvre. Use log rolling procedure if it is necessary to reposition the patient in anyway.
- Breathing: Check respiratory rate and air entry into the chest. If need be use na ambu bag to maintain air flow.
- Circulation: Stop active bleeding and monitor pulse rate and blood pressure. Fix a large intravenous cannula (gauge 18) preferably in the antecubital area.
- Dysfunction of CNS: Assess neurological status, consciousness level and spinal cord.
- Drugs, including fluids: Use these to correct acid base and volume imbalance.
- Exposure fo rexamination: Disrobe the patient entirely and carry out a complete physical examination.

  Look for:
  - Chest injuries: For example, haemopneumothorax from whatever cause takes priority.
  - Head injuries: Require setting of baseline observations.
  - A patient in shock from non-obvious causes: This points towards the abdomen,suggesting visceral injury. It may be very unapparent and can be fatal.
  - Peripheral bone fracture: This may need stabilization initially and proper attention later.
- After resuscitation and stabilization: Carry out frequent and more thorough examinations.
- Give attention to:
  - Continued bleeding–Arrest the bleeding and transfuse appropriately; haemopneumothorax may need underwater seal drainage.
  - Persistent shock from unexplained source–May necessitate an exploratory laparotomy.
  - Fractured limb will need splinting. Spine fractures need bed rest with fracture boards. X-rays of a patient with multiple injuries should be taken after adequate resuscitation. Exceptions are in the chest and cervical spine, which should be taken after initial resuscitation.
  - Acute gastric distension–Managed by nasogastric tube and suction of the same; the patient will require feeding to counter the catabolism associated with multiple injuries.

- Some of the injuries may require referral for more specialized care.This referral is executed ***after*** adequate resuscitation.

## 49.2 Chest Injury

### 49.2.1 PENETRATING INJURY

Common objects causing injury are knives, arrows, spears, and bullets. The objective of management is to restore normal anatomy and/or physiology resulting from the stab injury.

**Investigations**

- CXR: for the majority of cases, chest radiograph alone is adequate.
- Specialized investigations are ordered where more detail is required e,g CT scan.

**Management**

- Clean wounds and apply clean dressing to wounds.
- Give tetanus toxoid 0.5ml STAT.
- Give analgesics and sta antibiotics treatment.
- NOTE: Make sure resuscitation measures continue during transportation.
- If the instrument used during stabbing is still in situ, DO NOT remove. It is advisable to remove only in a controlled setting like in theatre. For referral, stabilize this by surrounding with heavy dressing or other cloth like material.
- If available, insert chest tube with under water seal drainage system.
- Drain pleural collection using chest tube, which will suffice for most injuries.
- Conduct surgical intervention to stop bleeding that continues, or correct significant anatomical or physiological anomalies.(This is applicable for only about 5% of cases.)

### 49.2.2 SIMPLE RIB FRACTURES

This is a break in the continuity of a rib(s). Could be traumatic or pathological. Types of fractures can be crack fracture(s), single or multiple fractures with fragment displacement, and segmental fracture(s).

**Clinical Features**

- There is history of trauma. Pain on breathing or movement. Evidence of chest trauma. Crepitus at the fracture site or tenderness. May have signs of associated with haemo-pneumothorax, subcutaneous emphysema.
- Caution:The chest injury may be associated with splenic or liver injury, especially with higher and lower ribf ractures.

**Investigations**

- Physical examination
- Chest radiograph; Antero-Posterior oblique views are necessary

**Management**

- **Oxygen**:Supplement if signs of respiratory distress are present.
- **Analgesia:** Administer intramauscular diclofeanac and 2% lidocaine 2–5 ml directl into fracture site; repeat once daily or after 3 days
- **Chest drainage:** Insert tube as indicated. Admit patient for observation if fractures of the first rib and those of the 8$^{th}$ rib and below are present.

- **Antibiotics:** Give antibiotics appropriately. Antibiotics given because of the associated atelectasis.
- **Mucolytic drugs:** to aid in expectorations of chest secretions e.g., carbocisteine 750 mg TDS for adults; children 2–5 years 62.5–125 mg QID, while 6–12 years 250mg TDS.
- Manage associated conditions.
- Initiate chest physiotherapy.

## 49.2.3 FLAIL CHEST

This occurs when multiple fractures are sustained with more than one site per rib. The main danger is that the patient may lapse into respiratory failure.

**Clinical Features**

The features include the following:

- Chest pain
- Paradoxical chest movement
- Dyspnoea may be present
- Evidence of fractured ribs
- Haemothorax or pneumothorax or both

**Investigations**

- Chest radiograph

**Management**

- Splint the flail segment using kinesiology tape.
- Administer analgesia. Make sure no neurological deficit is present.
- Restrict fluids to avoid development of adult respiratory distress syndrome.
- If there is no respiratory failure continue conservative management. If it develops transfer patient to ICU for intubation and positive airway breathing. If no respiratory failure results, continue with conservative management in general wards.
- At referral centre if referred to ICU: Carry out intubation with positive end expiratory pressure (PEEP) applied.
- Best managed at facilities where ICU is available

## 49.2.4 PNEUMOTHORAX

This occurs when air enters the plural space, which causes lung collapse on the affected side. Causes include spontaneous development following staphylococcal pneumonia due to chronic obstructive pulmonary disease. Pneumothorax may also be caused by blunt trauma with rib fractures and or lung contusion, penetrating injuries, stab wounds, and missiles.

**Note: Tension pneumothorax is a clinical diagnosis and not a radiological diagnosis. Ordering a chest radiograph may result in patient death before active treatment can be implemented.**

**Clinical Features**

- Shortness of breath
- Tightness of the affected chest
- Chest pain
- Tachypnoea and tachycardia
- Sweating
- Cyanosis
- Reduced air entry on auscultation
- Hyperresonant chest is noted on percussion
- Reduced chest expansion

**Investigations**

Chest radiograph: Shows various degrees of lung collapse.

**Management**

- If more than 5% pneumothorax, institute tube thoracostomy drainage (under water seald rainage); ***maintain absolute sterility while performing the procedure.***
- Chest tube may be removed when the lung is fully expanded and remains fully expanded after test clamping the chest tube for a number of hours.
- Tension pneumothorax needs more rapid treatment with immediate insertion of a wide bore cannula drainage or underwater seal drainage under local anaesthesia.

**Note:** Tension pneumothorax is a clinical diagnosis and not a radiological diagnosis. Ordering a chest radiograph may result in patient death before active treatment can be implemented.

- An associated frail chest leads to paradoxical breathing and may require assisted ventilation (i.e., intermittent positive pressure ventilation), if features of respiratory failure develop.

## 49.2.5 HAEMOTHORAX

This occurs when blood collects in the pleural space. Haemothoraxmay vary in amount from small to massive collections. Causes include trauma, post surgical bleeding, and tumours of the chest cavity and chest wall.

**`. Clinical Features**

Depending on the magnitude of the blood collection, there could be hypovolaemia from massive bleeding, or symptoms similar to those associated with pneumothorax, except for the percussion note, which is dull for haemothorax. However, haemopneumothorax is the more common presentation following chest trauma.

**Investigation**

- Resuscitation if needed.
- Chest radiograph (erect, posteroanterior view and lateral views).
- Look for fractured ribs, collapsed lung(s), fluid collection in the pleural space (air- fluid level), position of mediasternum, and diaphragm.
- Specialized tests as needed.

- Other tests relevant to primary underlying cause of the haemothorax.

**Management**
- Resuscitation if needed
- Small haemothorax (blunting of the costophrenic angle),will resolve spontaneously. Conservative management with daily reviews.
- Large haemothorax will require underwater seal drainage.
- Physiotherapy as needed.
- For large clotted haemothorax, perform thoracotomy to drain clot or refer to a more specialized unit.
- Look at the primary problem
  - For a fracture of rib, inject 2% lidocaine about 2–5ml intercostal block.
  - Advanced malignant disease with recurrent pleural effusion. Do chemical pleurodesis using tetracycline powder etc.

## 49.3 Head Injury

This describes a series of injuries that can occur to the scalp, skull, brain, underlying tissue and blood vessels in the head. Early and proper management is critical in order to avoid death or long-term morbidity. Especially with a high prevelance of road trffic accidents and assaults, this is a fairly common injury.

**Investigations**
- Skull X-ray
- CT scan usually more informative than simple skull radiograph.

**Management**
- Initiate resuscitation measures.
- Document accurately the neurological status with the Glasgow Coma Scale or other reliable scale.
- Ensure adequate oxygenation and monitor fluid balance. Avoid over hydration.
- Review regularly every 15 to 30 minutes.
- Arrange immediate referral to a Specialized unit.
- Admit patient for hourly neurological observations
- Record hourly neurological observations to include:
  - Glasgow Coma Scale
  - Blood pressure, pulse and respiratory rate
  - Pupil size and reaction
  - Limb movements (normal, mild weakness, severe weakness, spastic flexion, extension, no response).
- Check for peripheral deep tendon reflexes.
- Carry out surgical intervention as needed.
- Rehabilitate as appropriate: Physiotherpay, occupational therapy and counselling.

**Note:** Regular neurological assessments performed less often than hourly are of no use for interpretation.

**Note:**

- If there are signs of an intracranial haematoma developing (declining conscious level, pupil signs, onset of confusion) send for an urgent CT-scan of the head.
- Compound skull fracture:
  - Thorough wound debridement and haemostasisas an emergency
  - Cover with a broad spectrum antibiotic.
- Depressed skull fractures:
  - If it involves more than one table of the skull bone, it requires surgical elevation in theatre.
- Basal skull fracture:
  - Bloody CSFcoming from the ear or nose is indicative of a basal skull fracture unless other external source of bleeding are seen.
- Give antibiotics to cover for bacterial meningitis appropriately.

- **Note: Do not give narcotic analgesics to head injury patients. Use paracetamol**
- Convulsions must be rigorously controlled by giving anticonvulsants appropriately.

## 49.4 Spinal Injury

Spinal injury could involve soft tissues (muscles and ligaments), bones (vertebrae and discs), and neural tissue (spinal cord and nerves). It is important for primary assessment to establish the presence of an injury and initiates immediate treatment to avoid worsening either the primary or the secondary injury.

### 49.4.1 CAUSES OF SPINAL INJURIES

- Road traffic accidents
- Assault
- Blunt injury
- Penetrating injuries: sharp objects like knives, spears and firearms
- Sports injury
- Falling from a height

Bone injury could be stable (involving only one column) or unstable (involving two or more columns) and could be associated with neurological manifestation like paraplegia or quadriplegia depending on the level of injury. The injury could be a compression fracture with retropulsion of bone fragments into the spinal canal, causing spinal cord compression or complete transection of the cord.

**Clinical Features**

- Condition may present as part of the multiply injured patient and caution is needed not to overlook this condition.
- Neurogenic shock may be present and this refers to the haemodynamic triad of
  - Hypotension
  - Bradycardia

- Peripheral vasodilatation
  resulting from autonomic dysfunction and the interruption of sympathetic nervous system control in acute spinal cord injury.

Spinal shock is defined as the complete loss of all neurological function, including reflexes and rectal tone, below a specific level that is associated with autonomic dysfunction.

**Investigations**

- Plain spinal radiographs.
- **Note:**It is critical to maintain cervical stability during transfer and examination hence apply a cervical collar
  - Also see chest injury guidelines in this document and manage appropriately
  - Do a CT scan or MRI in facilities where they are available.

**Management**
**For levels 4 and 5:**

- Give anti-inflammatory analgesic.
- If open wound: tetanus toxoid as per EPI schedule and appropriate antibiotic
  - Care of the spinal column should be observed with application of a cervical collar or a hard board. ***Practice log rolling procedure at all times.*** Spinal immobilization should be provided during transportation. Resuscitation should continue during transportation.
  - Where facilities for surgical toilet for associated injuries are available, this may be performed prior to referral.
  - Refer to a level 6 for acute treatment and thereafter spinal injury unit for rehabilitation. Transfer should be made even if the clinical manifestations of spinal injury are minor.

**For level 6:**

- Bone injuries to be addressed through orthopaedic surgeries.
- Spinal decompression and stabilization are managed as per the individual case appropriately.
- Skin, bladder, and bowel care should be maintained appropriately
- Rehabilitation with physiotherapy, occupational therapy, prosthetic and orthotic fittings, etc.

# 50. General Surgery

## 50.1 Abdominal Conditions

### 50.1.1 ACUTE ABDOMEN

Acute abdomen is a clinical term used to describe a syndrome that usually incorporates unusual symptoms and signs in the abdomen. Central to the syndrome is acute severe abdominal pain. The term “acute abdomen” is a symptomatic diagnosis and not a definitive one. It is critical in these patients that a variety of conditions be suspected and diagnosed or clearly excluded before definitive treatment is initiated.

The common causes of abdominal pain which should be considered as differentials are:

- Medications (NSAIDs – gastric wall ulcerations),
- Gastro- enteritis,
- Peptic ulcer disease,
- Acute erosive gastritis,
- Appendicitis,
- Acute cholecystitis,
- Acute pancreatitis,
- Acute intestinal obstruction,
- Renal colic,
- Diverticulitis,
- Ectopic pregnancy,
- Ruptured or twisted ovarian cyst,
- Mittelschmerz,
- Urinary tract infection,
- Pelvic inflammatory disease.

**Clinical Features**

Meticulous history and physical examination are very important in establishing the diagnosis. The clinical features include

- Abdominal pain
- Adominal distension,
- Abdominal guarding and rigidity, a
- Altered bowel sounds,
- Alteration of bowel habits.

There should be a high index of suspecison that should be made of signs and symptoms of

- GIT disease
- Genitourinary disease
- Hepatobilliary disease
- Respiratory disease
- Metabolic disorder (diabetes mellitus, porphyrias)

- CNS diseases (neuropathies)
- Haematological diseases (for example, thrombotic cirisis in sickle cell disease) and
- Cardiovascular disease.

**Note: As a result of organ displacement associated with pregnancy, clinical examination of the abdomen for abdominal pain in a pregnant female should be ruled out.**

**Investigations**
- Total blood count
- Urea and electrolytes
- Urinalysis
- Plain abdominal radiograph (erect and dorsal decubitus)
- Chest radiograph
- Ultrasound in suspected appendicitis, cholecystitis, liver abscess or pelvic inflammatory disease among others.

**Management**
Details of the patient's history and condition, as well as an accurate documentation of events are important. Ensure the following:
- Order nil by oral.
- Conduct nasogastric suction.
- Prepare wide bore intravenous line or other form of secure intravenous access.
- Catheterize and initiate an input-output chart.
- Perform portable radiological investigations.
- Use analgesia appropriately and document.
- Transfer to a suitable surgical unit or facility as soon as possible.
- Maintain resuscitation during transfer, nasogastric suction, fluids, and input output chart.
- Manage conservatively if found appropriate: Nil by mouth, nasogastric suction, correct fluid and electrolyte imbalance by intravenous fluids
- Re-evaluate with the appropriate investigations.
- Initiate specific treatment of the underlying cause, e.g., surgery for perforation, peritonitis, ruptured ectopic pregnancy, etc.
- Group and cross-match blood for all laparoscopies.
- Initiate specific treatment of the underlying cause, e.g., surgery for perforation, peritonitis, ruptured ectopic pregnancy among others

**Note:** Organize post discharge follow up as indicated**.**

## 50.1.2 INTESTINAL OBSTRUCTION

**Clinical Features**
In infants, suspect bowel obstruction if:
- No meconium is evacuated within the first 24 hours of birth.
- There is green or bilious vomiting.
- There is abdominal distension.

**In older children and adults, suspect bowel obstruction if there is :**

- Nausea and vomiting
- Abdominal pain
- Abdominal distension
- Altered bowel sounds
- Constipation
- Fever (if advanced obstruction is present)

**Note: If there is gross abdominal distension with no pain, suspect sigmoid volvulus.**

**Investigations**

- Full blood count
- Urinalysis
- Urea and electrolytes
- Radiograph of abdomen (erect AP and dorsaldecubitus)
  - Multipleair-fluid levels, gaseous distension of gut, double bubble sign in children, among others
  - Volvulus

**Management**

- Initiate resuscitation with nasogastric suction, intravenous fluids and nil by oral.
- Monitor vital signs appropriately.
- Take radiographs (if available). If not able refer to facility with ability to manage condition.
- Perform definitive management be it surgery or conservative management. Correct fluid and electrolyte imbalance.
  - Group and cross match blood
  - Deflate the distended stomach with nasogastric suction. This is more effective for small bowel. For large bowel obstruction high enema may be effective for faecal impaction only.
  - Give prophylactic antibiotics appropriately.

**Note** that high enema may be effective for faecal impaction only.

  - Address the cause of the obstruction by surgery intervention or conservative treatment.
  - Obstruction due to adhesions from previous surgery may open under conservative treatment.
- Emergency large bowel surgical resection usually involves creation of ade-functioning colostomy rather than performing primary resection and anastomosis if strangulation has taken place (Hartmann'sprocedure).

## 50.1.3 PERITONITIS

This is inflammation of the peritoneum which can be classified as acute or chronic. This causes can be due to:

- Bacteria: pyogenic bacteria of the gut
- Non-pyogenic bacteria in the gut such as tuberculosis

- Chemical causes which lead to asceptic peritonitis such as leaked pancreatic juices, bile juices among others.

Appreciate that peritonitis could be due to tuberculosis and could also be aseptic. The aseptic type is usually due to chemical irritants like pancreatic juices, among others. Peritonitis usually ends up producing adhesions that may cause future bowel obstructions of varying degrees.

**Clinical Features**
- Acute tendera bdomen,
- Abdominal distension,
- Ridigity and guarding
- Rebound tenderness,
- Fever.

Complications of peritonitis include the following:
- Abscess formation.
- Surgical site infection
- Wound dehiscence.
- Enterocutaneous fistulae.
- Adhesions
- Organ failure

**Investigations**
- Full blood count, PCV.
- Urea and electrolytes.
- Abdominal radiograph( erect AP and dorsal decubitus)–may show air fluid levels or air under the diaphragm in case of perforated viscera.
- Abdominal ultrasound.

**General Management**
- Correct fluid and electrolyte imbalance. These are usually disturbed by the movement of fluid and electrolytes into the third space. The disturbance could arise or be made worse by vomiting and/ordiarrhoea.
- Consider nasogastric suction, which is usually necessary because of organ hypotonia and dilatation.
- Use antibiotics to cover a broad spectrum of bacteria approporiately.
- Alleviate pain appropriately.

**Specific Management**
- Exploratory laparotomy
  - This is a must in secondary peritonitis in order to repair or remove the diseased organ. It also facilitates peritoneal lavage of the necrotic debris and pus.
  - If the facility permits, laparoscopic exploration is recommended for this minimizes complications and hastens patient recovery.
- Send pus for culture and sensitivity.
- Give appropriate physiotherapy.

## 50.1.4 APPENDICITIS

**Clinical Features**

- Anorexia, nausea and vomiting
- Abdominal pain
  - Pain then settles in the right lower quadrant and is localized at McBurney's point.
  - Pain may be relieved briefly after perforation but is accentuated by ensuring diffuse peritonitis.
- Abdominal tenderness
  - Localised tenderness in the right lower quadrant
  - Rebound tenderness
  - Pelvic tenderness in the right iliac fossa on rectal examination
- Muscle guarding and ridigity
- Cutaneous hyperaesthesia
- Fever

**Note: Look out for Roving's sign, Psoas sign**

**Investigations**

- Full blood count with neutrophlia. Normal values do not rule out appendicitis.
- Urea and electrolytes
- Random blood sugar
- Abdominal ultrasound: May show oedema of the appendix and adjacent ileocolic region, fluid around the appendix, intraluminal feacolith impaction in the appendix.
- CT scan of the abdomen if necessary.

**Note: Radiological investigations should not delay management of the patient.**

**Management**

- Initiate appropriate resuscitation. Maintain airway, breathing and circulation appropriately.
- Once diagnosis is made, give analgesics and appropriate antibiotics while preparing preparing for surgery.
- Starve the patient.
- Appendectomy is the treatment of choice, once definitive diagnosis is made.
- Give premedication when there is time (atropine 0.6mg IM stat and morphine 10mg IM stat).

**Note: Appendicular mass should be managed conservatively with the appropriate antibiotics, analgesics as detailed in the Oshner Shellen regime. Interval appendicectomy can be performed after two weeks.**

## 50.1.5 INTESTINAL ATRESIA

In the process of growth for humans, the gastrointestinal tract first develops into a tube that later canalizes. Failure of this process during any stage may result in intestinal atresia, which can affect any section of the bowel and can have varying degrees of severity.

**Clinical Features**

For an upper GIT lesion, bilious vomiting will be the main form of presentation with abdominal distension secondary to gaseous distension. Failure to pass meconium may occur for lower level lesions.

**Investigation**

- Full blood count
- Urea and electrolytes
- Plain radiograph to confirm fluid levels
- A thorough check for other anomalies will be required.

**Management**

- Initiate resuscitation measures with intravenous lines, nasogastric suction and fluid charts. Correct any fluid and electrolyte imbalance present.
- Carry out radiological investigation is possible in the facility.
- Surgical intervention as indicated.
- Refer appropriately for further surgical management.

**Management at Level 6 Facility**

- Resuscitation and stabilization.
- Completion of investigations as needed.
- Surgical intervention.

## 50.1.6 CHILDHOOD HERNIAS

### INGUINAL HERNIA

Inguinal hernia is an extension of the processus vaginalis, which fails to close during foetal development. Through this opening, abdominal content can herniate to varying extents into the inguinal canal and scrotal sac. The communicating type is the most common form and extends down into the scrotum. The non-communicating inguinal hernia is less common.

**Clinical Features**

- A bulge presents at either the internal or the external rings, or scrotum for males and inguinolabial region for females, that increases in magnitude with straining.
- Pain and discomfort, or it may present as an acute abdomen.
- Examination may reveal a reducible or irreducible mass.
- Trans-illumination test may be positive.

**Investigations**
- Usually clinical
- Ultrasound may assist in differential diagnosis

**Management**
- Inguinal hernias do not heal and must be corrected by elective herniorraphy for uncomplicated cases, to avoid complications.
- Emergency surgery shoud be done if complications like obstruction have set in.

**ABDOMINAL HERNIA**

This is a protrusion through the abdominal wall due to one of the following:
- Umbilical hernia, which is a mild condition as a result of a defect in the linea alba. The herniated bowel has a covering of subcutaneous tissue and skin.
- Omphalocele, which is due to the failure of development of the anterior abdominal wall at the area of insertion of the umbilicus, with the abdominal contents herniated out with only a peritoneal covering. There may be other associated anomalies.
- Gastrochesis, which is a herniation of small bowel contents with no covering at all and is often paraumbilical. Unlike omphaloceles, this condition does not have many associated anomalies.
- Umbilical hernia, which is a mild condition as a result of a defect in the linea alba. The herniated bowel has a covering of subcutaneous tissue and skin.

**Clinical Features**

There is protrusion of bowel contents through the abdominal wall to varying extents with or without other organs. Covering of the hernia varies and strangulation is a possibility.

**Investigations**
- Full blood count
- Urea and electrolytes
- Ultrasound has a role in the antenatal period.

**Management**
- Conservative management for small umbilical hernias with expectant observation.
- Elective herniotomy is performed if conservative management fails.
- Emergency herniotomy for strangulations or other surgical complications arising from the hernia.
- Surgical management best at specilaised facility.

**Note: For omphalocoel and gastro chesison early surgical intervention is recommended.**

## 50.1.7 IMPERFORATE ANUS

This is failure of the anal opening to canalize and is the commonest cause of intestinal obstruction in newborns. It presents with a wide variation in anatomical anomalies.

**Clinical Features**

**T**here is failure to pass meconium,or may pass meconium per urethra or vagina.

**Investigation**

- Invertogram
- Check for other anomalies

**Management**

- Divided sigmoid colostomy to defunction the colon should be performed immediately.
- Refer appropriately.

**Note:**

- Surgical management best at specialised facility even for apparently simple malformations.
- Definitive surgical intervention, which may range from minor anuloplasty (dilatation, incision) to more complicated pull through procedures at the appropriate facility.
  - Continued dilatation at home
  - Sitz baths
  - Colostomy closure if needed
- Counselling and attending to associated conditions.

## 50.1.8 INTUSSUSCEPTION

This occurs when a piece of, usually small, bowel invaginates into itself. This invagination may cause strangulation that leads to gangrene formation in the affected portion of the bowel.

**Clinical Features**

- Onset of acute abdominal pain sometimes associated with red currant jelly stools.
- Clinical examination reveals a mass of the interssuceptus in the right hypochondrium.

**Investigation**

- Ultrasound gives better detection rates.
- Plain abdominal radiograph may show evidence of obstruction but missed out in early disease.

**Management**

- Stabilize the patient adequately.
- Initiate conservative or surgical management appropriately.

## 50.1.9 INGUINAL HERNIA (ADULT)

This is usually an acquired condition and is often linked with activity associated with increase of abdominal pressure.

**Complications**

Complications of this condition include obstruction (when a hollow viscus goes through a ring of variable size and cannot be reduced), and incarceration (when non-hollow organ for example omentum, goes through a ring of variable size and cannot be reduced).

Strangulation is a process in which blood flow into the obstructed viscus is compromised, and if not corrected culminates in ischaemiaof the viscus supplied by the involved blood vessels. Pain and tenderness over the hernial area are ominous signs. Sudden change from reducible to irreducible status especially if discolouration of tissues over the area is present is an ominous sign.

**Clinical Features**

- Protrusion in the groin region, initially on straining and later may be spontaneous.
- Irritable or painful sensation in the groin.

**Examination**

Observation of the bulge with the patient coughing while standing and when lying down, and with a finger invaginated into the external ring, repeating the same examinations. This examination is able to differentiate femoral from inguinal hernia. There is no great advantage of differentiating indirect from direct inguinal hernia, pre-operatively.

**Management**
**Admit for**

- Emergency hernioraphy for obstructed or incarcerated inguinal hernia.
- Elective hernioraphy for non complicated hernias.

**Note:**

- In strangulation, with obstruction of viscus, especially bowel the usual resuscitative measures are carried out/continued before and after surgery. See details as per obstruction above.
- Surgical repair is necessary for all inguinal hernias.
- Umbilical, incisional, and lumbar hernias require similar treatment as above.

### 50.1.10 LOWER GASTROINTESTINAL BLEED

This may be frank bleeding depending on the cause. Common causes are:

- Haemorrhoids
- Anal fistulae and fissures

- Tumours: Benign (leiomyoma, fibromas, polyps) ormalignant
- Trauma
- Angiodysplasia
- Bleeding disorders

**Investigations**

- Full haemogram
- Urea and electrolytes
- Stool for occult blood
- Proctoscopy/Biopsy and Colonoscopy
- Abdomonal ultrasound
- Barium enema (double contrast

**Management**

- Do blood group cross match and transfuse if necessary
- Resuscitate appropriately
- Identify and treat primary pathology

## 50.2 Anorectal Conditions

**Clinical Features**

There is pain usually on defecation that prevents proper sitting and causes immobility (commonly due to abscess, thrombosed haemorrhoids, or acute fissure-in-ano). Painless bleeding is commonly due to haemorrhoids but may be due to colorectal carcinoma. A patient with a perianal mass complains of feeling a mass (usually prolapsed haemorrhoidsor anal tags) or has anal discharge that is associated with itching and is commonly associated with tumours, proctitis, and helminthic infestations. Perineal discharge, on the other hand, is usually due to fistulae and is common in obese people.

### 50.2.1 ANAL INCONTINENCE

Feacal incontinence is the inability to control bowel movements. It ranges from an occasional leakage of stool with passing gas to a complete loss of bowel control.

**Note: A thorough examination of the patient with digital rectal examination are critical for identifying the cause of anal incontinence. The following have been associated with anal incontinence:**

**Causes**

**The following have been associated to cause anal incontinence**

- Congenital abnormalities.
- Trauma to the sphincters and anorectal ring, injuries (obstetric, operative, abuse and accidental).

- Neurological abnormalities (due to spinal cord disease).
- Anorectal disease (rectal prolapse, third degree haemorrhoids and anorectal cancer).

**Investigations**
- Protoscopy
- CT Scan
- M.R.I

**Management**
- Manage the primary cause
- Management as per the cause.
- Appropriate physiotherapy

## 50.2.2 RECTAL PROLAPSE

Rectal prolapse may be partial (mucosal) or complete (whole thickness of rectal wall). It is a common occurrence in children and the elderly (especially females, who form 85% of affected adults population) but may occur at any age.

**Degree of prolapse**
- Primary prolapse with spontaneous reduction.
- Secondary prolapse with manual reduction.
- Tertiary prolapse that is irreducible.

**Clinical Features**

Reducible prolapse, often occurs during defecation and is associated with discomfort, bleeding, and mucus discharge. Prolapse may also be caused by mild exertion (e.g., through cough or walking) and may also be associated with incontinence of flatus and faeces. When uterine prolapse compounds rectal prolapse, urinary incontinence may also be a feature.

Rectal prolapse is also associated with benign prostatic hypertrophy, constipation, malnutrition, old age, and homosexuality /anal intercourse.

**Note:** Anorectal carcinomas should always be suspected if there are also ulcers, indurations, or masses in this area. During clinical examination it is important to check for patulous anus and for poor sphincter tone (on digital examination).

**Management**
- May be conservative or operative, depending on the condition of the patient.
- Primary and secondary prolapse: conservative treatment with stool softeners, e.g., lactulose 15ml 12 hourly.
- Tertiary prolapse: Perform definitive surgery.
- Complications include irreducibility of the prolapse with ulceration, bleeding, gangrene, and possible rupture of the bowel.
- Appropriate physiotherapy.

Complications include irreducibility of the proplapse with ulceration, bleeding, gangrene, and possible rapture.

## 50.2.3 PRURITIS ANI

This is a common condition especially in males. Causal factors include

- Skin conditions (psoriasis, lichen planus, contact eczema),
- Infective conditions (candidiasis, threadworms),
- Anal-rectal conditions (piles, fissures, fistula, proctitis, polyps),
- Gastro intestinal conditions (irritable bowel syndrome, ulcerative colitis, etc.),
- Drugs (quinidine, colchicine),
- Obesity.

**Clinical Features**

- History taking pruritis
- Physical examination
- Digital rectal exam
- Protoscopy

**Investigations**

- Stool ova and cyst
- Colonoscopy

**Management**

- Treatment is that of the cause.
- Improved personal hygiene for those affected.

## 50.2.4 FISSURE IN ANO

This is an elongated longitudinal ulcer of the lower anal canal. The commonest site is the midline posteriorly, followed by midline anteriorly. This condition occurs in children, but is more common in females in their midlife. It is uncommon in the elderly.

**Clinical Features**

- Pain during defecation that is often intense, may last for an hour or more, but subsides only to come again during the next defecation.
- Stool is frequently streaked with fresh blood.
- Constipation: Patient is reluctant to open bowel because of pain.
- Slight discharge occurs in chronic cases.
- A sentinel tag is usually demonstrated, with a tightly closed puckered anus.

**Management**

- Acute pain (less than 14 days): Conservative management: medications (perectal analgesics, laxatives).
- Chronic pain (more than 14 days): Lateral sphincterotomy.

**Note: Avoid doing digital examionation for it causes a lot of discomfort to the patient. Perform examination under anaesthesia (EUA).**

## 50.2.5 HAEMORRHOIDS

These are varicosities of the haemorrhoidal plexus that is often complicated by inflammation, thrombosis, and bleeding. Haemorrhoids are not commonly associated with pregnancy. Appropriate assessment is digital examination and proctoscopy (use good light).

**Clinical Features**

- Painless rectal bleeding
- Prolapse or
- Sensation of a mass in the anal area (especially during defecation),
- Mucous anal discharge

**Complications include**

- Profuse bleeding
- Thrombosis
- Infection
- profuse bleeding,

  all of which require surgical intervention for appropriate management.

**Management**

- Advise a high residue diet or bulk laxative to prevent constipation.
- Specific treatment includes:
  - Rubber-band ligation for 2°–3°haemorrhoids
  - Injection sclerotherapy.
  - Haemorrhoidectomy (for 2°–3° piles) where other methods have failed.
  - Management of associated compilations.

**Note:**

- Digital rectal examination and proctoscopy are painful and can be performed at examination under anaesthesia.
- It is important to consider carcinoma of the anus, anal chancre, tuberculosis ulcer (whose edges are undermined), and proctalgia fugax as important differential diagnoses that must be ruled out.
- Some heal spontaneously.

**Management**

- Local Anaesthetic and anti-inflammatory ointments (suppositories) appropriately
- Stool softners
- Saline sitzbath
- Operative treatment is recommended for cases refractory to conservative treatment
- Nutritional diet (high fibre diet).

## 50.2.6 ANORECTAL ABSCESS

There are four types of abscesses: submucosal, subcutaneous (perianal), ischiorectel, and high intermuscular. Usually there is no apparent cause, but certain underlying diseases such as Crohn's disease, ulcerative colitis, rectal cancer, HIV disease, diabetes mellitus, and active tuberculosis may be present. Complications for anorectal abscess include, fistula formation, recurrence of the abscess, and sinus formation.

**Clinical Features**

- Presents as acute painful swelling with fluctuation not always obvious and there is pain on defecation.
- Blood-stained purulent anal discharge.

**Management**

- Start on appropriate antibiotics
- Analgesics/anti-inflammatory appropriately
- Incise and drain under general anaesthesia (de-roof by making a cruciate incision and excising the four triangles of skin).
- Take a pus swab for culture and sensitivity.
- Advise saline sitz bath and stool softeners.

## 50.2.7 RECTAL TRAUMA

Rectal trauma may be caused by assault, road accidents, birth trauma.

**Clinical Features**

Patients present with pain, bleeding, and purulent rectal discharge. Clinical findings include anal laceration, features of peritonitis, and fever with or without foreign bodies in the rectum.

**Management**

- Address the primary problem.
- For mild to moderate cases, manage conservatively, which includes:
  - Administration of antibiotics appropriately
  - Analgesics.
  - Saline sitz bath
- For severe cases, carry out surgical interventions (defunctioning colostomy).
- Provide counselling and other support services of the patient as needed.

## 50.2.8 DISTAL COLON AND RECTAL CARCINOMA

Distal colon and rectal carcinoma is especially found in elderly patients, presenting with rectal bleeding, change in bowel habits, and sometimes with abdominal or pelvic pain or even intestinal obstruction. It is important to rule out familial conditions in the family history. Clinical examination for patients suspected to have distal colon and rectal carcinoma should include rectal examination.

**Investigations**

- Proctoscopy
- Colonoscopy and biopsy
- Investigation for spread includes:
  - Intrarectal ultrasound
  - Abdominal pelvic CT scan
  - MRI appropriately
  - Where available laparascopy.

**Management**

- Management is multidisciplinary speciality approach.
- Carry out curative or palliative surgical intervention.

## 50.3 Abscesses

**Clinical Features**

An abscess formation is the culmination of an uncontrolled localized infection. There is tissue necrosis with liquefaction (pus formation).

**Management**

Can be carried out at all levels with referral to higher level for more complicated abscesses or those requiring general anaesthesia. Caution should be exercised for special abscesses like brain abscess, mastoid abscess, as simple incision and drainage of these can result in severe injury or in chronic simuses. Such sinuses should be referred to higher level for appropriate management.

**Treatment involves:**

- Incision and drainage. (under anaesthesia)

**Note:**

- See ENT Section 55.7 for management of mastoid abscesses.
- The wound(s) is/are allowed to heal by granulation.
- Hand and foot abscesses will require multiple incisions, with counter incisions in some areas and elevation of the limbs.
- tablespoons of salt to thewater.
- Antibiotics are indicated in hand abscesses as per sensitivity and
- Face abscesses. Require antibiotic cover.
- Always send specimen of pus (and where possible abscess wall) for culture and sensitivity and histological exam.

### 50.3.1 FISTULA IN ANO

This condition may complicate anorectal abscesses, Crohn's disease, ulcerative colitis, tuberculosis, colloid carcinoma of the rectum, and HIV infections.

**Clinical Features**

- Persistent seropurulent discharge
- Periodic pain
- Pouting openings in the perennial area of the anal verge

**Note:** Appropriate examination involves palpating the anal internal opening for a nodule on digital examination; confirmation is made at proctoscopy.

**Investigations**

- Fistulogram

**Management**

- Determine the primary pathology.
- Deal with the primary pathology as well as the fistula (fistulectomy

## 50.4 Breast Conditions

Breast disease presents in a variety of forms as lumps, breast pain, nipple discharge, breast ulcers, or eczema.

### 50.4.1 BREAST ABSCESS

This condition is common during lactation, especially the second week of puerperium, and during pregnancy. It rarely occurs at other times.

**Clinical Features**

- Painful breast swelling
- Fever

**Investigations**

- Pus aspirate fro microbiology culture and sensitivity
- Full blood count

**Management**

- Incision and drainage
- Analgesics
- Broad spectrum antibiotics

### 50.4.2 BREAST LUMPS

Breast lumps can be the result of a wide number of conditions, including the following:

- Cystic lesions that may be due to breast abscess, fibrocystic disease, cystosarcomaphylloides (serocysticdisease),galactocele,and hydatid cysts.
- Solid lesions that may due to developing breast abscess, antibioma, fibroadenoma, giantfibroadenoma, intraductal

papilloma, tuberculosis lymphoma, neurofibrom, or carcinoma of breast.

**Investigations**
- Full blood count
- Triple assessment
  - History and physical examination
  - Fine needle aspirate
  - Ultrasound/Mammography

**Management**
Identify primary pathology and treat.

## 50.5 Central Nervous System

Conditions affecting the central nervous system (CNS) that may require intervention may be classified as follows:
- Congenital disorders (hydrocephalus, microcephaly, encephaloceles,etc.)
- Degenerative disorders
- Vascular disorders
- Infections (e.g., brainabscesses)
- Neoplasms
- Trauma

**(See neurosurgical textbooks for greater detail.)**



### 50.5.1 HYDROCEPHALUS

**See Paediatrics Section 39.6 for additional information.**

**Management**
- Initiate drainage procedure with ventro peritonial (VP) shunts.
- Treat underlying pathology if cause of the hydrocephalus is known.
- Manage associated conditions.
- Rehabilitation as appropriate for the particular patient.

### 50.5.2 INCREASED INTRACRANIAL PRESSURE AND SPACE-OCCUPYING LESIONS

This is usually caused by increases in mass content (e.g.,tumour,haemorrhage, oedema, or CSF).

**Clinical Features**
**Principal symptoms are**
- Headache
- Anorexia may be present, especially with tumours.
- Vomiting
- Visual disturbance

- ♦ Papilloedema may be detected by use of fundoscopy
- ♦ Weight loss
- ♦ Bradycardia
- ♦ Mild hypertension
- ♦ Intellectual deterioration

**NOTE:** Diagnosis is made on the basis of clinical history, neurological examination (papilloedema), and radiograph examination (cranial ultrasound in children, and CT scan head).

**Management**

- ♦ Clear airway and use endotracheal intubation if patient is in a coma.
- ♦ Give minimum daily fluid requirement in form of isotonic solution (e.g., Ringer's solution or normal saline).

- ♦ Maintain blood pressure at normal or above normal range.
- ♦ Administer mannitol in acute intracranial pressure: 1g/kg (as 20% solution) intravenously, with frusemide 0.7mg/kg(15 minutes after mannitol).

**NOTE:**

- ♦ Do not give mannitol in heart or renal failure, to a hypotensive patient, or in acute intracranial bleeds.
- ♦ Consider steroids (e.g.,dexamethasonene 4mg 8 hourly intravenous), as these can reduce brain oedema in tumours – but avoid in acute head injury.
- ♦ Maintain an input and output chart.
- ♦ Carry out definitve management.

## 50.5.3 BRAIN TUMOURS

- ♦ About 50% of intracranial tumours are metastatic: from lung, breast, thyroid, kidney, and prostate.
- ♦ The remaining arise from:
  - Meninges (e.g., meningioma – of braintumours).
  - Skull (e.g., osteomas, histiocytosis, multiple myeloma,etc.).
  - Pituitary and parapituitary adenomas (chromophobe,eosinophilic.basophilic and prolactinomas), and craniopharvngiomas. These will present with headaches, disturbed vision and some form of endocrine change (e.g., Cushing's syndrome,galactorrhoea,diabetic insipidus,etc.).
  - Intracerebral tumours: Gliomas, e.g., astrocytomas and oligodendrogliomas.
  - Ependymomas medulloblastomas
  - Diagnosis is made on the basis of clinical history and examination findings, CT scanning, angiography, and tumour biopsy.

**Management**

- ♦ Definitive diagnosis through invasive and non-invasive investigations including histology.
- ♦ Definitive treatment as per the final diagnosi

### 50.5.4 INTRACRANIAL INFECTIONS

These include osteomyelitis of the skull commonly complicating penetrating injuries,post craniotomy infections, intracranial infections complicating otitis media, mastoiditis, paranasal sinusitis, and scalp infections.

Conditions that may arise from infections are skull osteomyelitis, extradural and subdural empyema, cerebral abscess, and meningitis.

**Clinical Features**

Clinical features will vary depending on the site and spread of infection but will include local tenderness, focal neurological signs, etc., disordered consciousness, epilepsy, or signs of meningitis.

Diagnosis is made on the basis of clinical history and physical and neurological examination. Plain radiographs of skull may show opaque air sinuses or air bubbles in brain. Angiography or CT scan is used to confirm the diagnosis.

**M◆aAndaeggpumateendtose** of appropiate antibiotics

- ♦ Drainage (multiple burr holes, craniotomy,etc.)
- ♦ Excision of infected bone
- ♦ Drainage of infected sinuses or mastoid air cell.
- ♦ Long-term anticonvulsant therapy.
- ♦ Take specimens for culture and sensitivity. Commence antibiotic treatment.
- ♦ Arrange and/or provide rehabilitation as needed

## 50.6 Chest Conditions

### 50.6.1 CONGENITAL HEART DISEASE

For detailed description of the different congenital heart diseases please see Section 37.3 in Part II, or refer to a suitable text book.

- ♦ Surgical intervention is often needed for some of the congenital defects. For these, carry out a diagnostic work up.

**Note:**

- ♦ The objectives of surgical intervention are to:
  - Restore anatomy to as near normal as possible.
  - Maintain unidirectional blood flow.
  - Restore deranged physiology to as near normal as possible.

These can be achieved through various shunts, patches, electrophysiological procedures, and other corrections.

## 50.6.2 EMPYEMA THORACIS

- In empyema thoracis there is pus in the pleural space. The condition may be classified as acute, sub-acute, or chronic, depending on the duration of the presence. Immunosuppression is commonly associated with chest diseases (investigate in suspicious cases).
- Complications include chronicity with lung destruction, fistula formation, and chronic sinuses through the chest wall.

**Clinical Features**

Symptoms of underlying condition may be present. There may in addition be

- Shortness of breath
- Fever
- Sweating
- Diasphoresis
- Tachypnoea

- Tachycardia
- Dullness to percussion with reduced air entry on the affected side
- Weight loss.

**Investigations**

- Chest radiograph shows fluid in the affected side or an air fluid level.
- Carry out thoracocentesis (pus should be taken for culture and sensitivity).

**Management Improve general condition of the patient, e.g., nutritional status.**

**Management – Specific**

- Appropriate intravenous antibiotics directed at the primary pathogen (take pus for culture and sensitivity and AAFB studies). Treatment choice depends on the sensitivity report.
- Acute empyema: Tube thoracostomy drainage (underwater seal drainage)
- Sub-acute empyema: Tube thoracostomy drainage
- Chronic empyema: Refer to cardiothoracic unit for thoracotomy and decortication
- Anti-TB therapy where indicated. Refer to the national guidelines for tuberculosis treatment.
- Admit for underwater seal drainage.
- Chest physiotherapy.

**Note: Remember iatrogenic causes of empyema lead to very severe morbidity. It is therefore imperative to observe strict sterility at all times while carrying out invasive procedures on or in the chest cavity.**

## 50.7.1 ACHALASIA CARDIA

Main symptom here is dysphagia due to failure of relaxation of the lower oesophageal sphincter. This results in dysphagia with differing degrees of food tasis and regurgitation of feeds.

**Clinical Manifestations**

- Long-standing dysphagia, more for solids than liquids, and commonly in young patients.
- Vomiting of feeds also occurs, sometimes of foods taken some days back.
- Weight loss if present is usually only slight.

**Investigations**

- Barium swallow, endoscopy, and biopsy
- Manometry

**Management**

- Balloon dilatation
- Heller's myotomy

## 50.6.3 TRACHEO ESOPHAGEAL FISTULA (CHILDREN)

This is the communication between the trachea and the oesophagus. The condition tends to have life threatening complications and needs urgent treatment soon after birth. Refer to Table 50.1 for a summary of the various types of this condition. This has been dealt with in Part II, Section 26.14.4.

**Table 50.1: Prevalence of the various forms of tracheosophageal fistula**

| Anatomical characteristics | Percentage of cases |
|---|---|
| Oesophageal atresia with distal TEF | 87 |
| Isolated oesophageal atresia without TEF | 8 |
| Isolated TEF | 4 |
| Oesophageal atresia with proximal TEF | 1 |
| Oesophageal atresia with proximal and distal TEF | 1 |

**Note:**

**Surgical correction should be carried out as soon as patient is stabilized for surgery. Usually surgery is recommended within a few days of birth. Repair may be performed as a primary procedure or a staged procedure at higher level of health care. If at level 4, initiate resuscitation measures as for acute abdomen above (suction, antibiotics, fluids).**

**Management**

- Resuscitate and prepare for surgery.
- Correct any fluid and electrolyte imbalance. Look out for possible infection.
- Carry out necessary radiological investigations.
- Carry out urgent surgical correction. This may be performed as a primary procedure or a staged procedure.
  - Primary procedure in principle for mature children with no comorbidities or significant infective complications.
  - Secondary repair for premature if infection present.

## 50.7 Malignant Dysphagia

This presents as a difficulty in swallowing on attempted initiation of swallowing and can occur at any stage of the swallowing process. This particular symptom is more common than appreciated and can be due to many causes. Carcinoma of the oesophagus/ cardia, for example, is the most common cause in the older age group for adults.

**Clinical Presentation**

- There is progressive dysphagia
- Weight loss.
- Regurgitation suggests a cardia lesion.
- Patients tend to be wasted in the late stages.

- Dehydration. Up to 60% of these patients in Kenya will present with underweight (BMI less than 18kg/$m^2$). Most patients present late

**Investigation**

- Barium swallow
- Oesophagoscopy and biopsy.
- Staging with abdominal ultrasound, CTscan and other endoscopic techniques.

**Management**

- Conduct curative surgery for early disease and palliative measures (including palliative surgery) for later disease.
- Initiate intubation or stenting.
- Carry out surgical resection.
- Administer radiotherapy or chemotherapy.
- Counsel the patient and relatives; it is important that they understand the prognosis of the disease from the onset.

## 50.8 Lung Neoplasm

More cases are being seen in Kenya and the association with smoking is high. Squamous cell carcinoma is the commonest histological subtype.

**Clinical Features**

The clinical features of this condition include the following:

- Chronic cough
- Haemoptysis
- Wheezing or stridor
- Lung infection or other sequels of bronchialobstruction
- Features of spread – nodes, malignant effusions, fistulas, etc.
- Systemic symptoms like appetite loss

**Investigations**
- Chest radiograph
- CT chest

**Management**
- Evaluate for extent of disease.
- Carry out curative resection for early disease.
- Provide palliative care for late disease.
- In general, adopt a multidisciplinary approach to care.

## 50.9 Genitourinary System

Infections of the urogenital system are characterized by the following symptoms:
- Dysurea
- Urgency in micturition
- Colic pain in either flanks or the loins
- Pain on the lower abdomen due to inflammation of the urinary bladder(cystitis)
- Poor urinary stream
- Dribbling and hesitancy
- Nocturia
- Urinary incontinence
- Urinary retention
- Haematuria
- Renal failure

These symptoms overlap many specific conditions, hence a thorough examination is required to facilitate an accurate diagnosis. The following need to be done in this regard:
- Ask and check for urethral discharge.
- Palpate the urethra for areas of in duration (stricture).
- Palpate the lower abdomen for tenderness, masses in the urinary bladder.
- Bimanually palpate the kidney for masses or tenderness.
- Perform a rectal or vaginal examination:
  - Manually palpate the urinary bladder for masses.
  - Feel for the prostate (size,consistency,nodularity,tenderness, fixation of rectal mucosa).

### 50.9.1 POSTERIOR URETHAL VALVES

As a developmental anomaly a membrane develops in the posterior urethra of male fetuses and results in bladder neck obstruction. The resulting increase in pressure is associated with developmental alterations from the normal.

**Clinical Presentation**

Symptoms range from mild symptoms of repeated urinary tract infection to obstructive uropathy. Symptoms may also include
- Distended bladder

- Dilated ureters
- Ultimately renal failure.

**Note:** Failure to pass urine in the newborn within 24hrs should be investigated. However, this does not necessarily suggest posterior urethral valves problem.

**Investigations**
- Voiding cystourethrogram

**Management**
- Evaluate the patient.
- Conduct endoscopic surgical resection of themembrane.
- Follow up.

## 50.9.2 CHILDHOOD HYDROCELE

This is fluid within the processus vaginalis within the scrotum.

**Clinical features**
- Swelling in the scrotal sac that may spread down from the inguinal canal, in the communicating type, or remain localized to the scrotum in the non-communicating type.
- Communicating types are associated with straining and may develop strangulation if bowel contents enter.
- In non-communication type, one can palpate and grasp the sac towards the scrotum and get above it.

**Investigations**
- Trans-illumination test is positive

**Management**
- Hydrocelectomy for communicating hydrocele
- Conservative management for non communicating hydrocele

**Note:**
- Communicating hydrocele (or inguinoscrotal hernia with no bowel content) will not close spontaneously. This type has a high risk of incarceration.
- Observe infants presenting with noncommunicating hydrocele, as often these will resolve on their own as the hydrocele fluid is slowly reabsorbed. The only indications for surgery are: failure to resolve by 2 years, cause discomfort, become infected, or show variations is size.

## 50.9.3 TESTICULAR TORSION

This is a surgical emergency. A high level of suspicion is needed to avoid unnecessary morbidity.

**Clinical Features**

- Sudden onset of scrotal pain in a male.

**Note:**

The diagnosisis mostly clinical. Testicular torsion must be differentiated from epididymochitis

**Investigation**

- Urinalysis
- Full blood count
- Colour doppler can be useful, but its absence should not be delay diagnosis.

**Management**

- Rapid exploration of affected side and orchidopexy for both testes.

## 50.9.4 CIRCUMCISION

This is excision of the prepuce (foreskin of penis). Indications include ritual (religious, traditional, personal), phimosis, paraphimosis, recurrent herpes genitalis restricted to the prepuce, recurrent balanitis (inflammation of prepuce), balanoposthitis (inflammation of prepuce and glans penis), tight frenulum, long and adherent prepuce.

**Method**

For circumcision, the following are necessary:

- Clean and drape the perineum
- Use local anaesthesia, lignocaine 0.5% without adrenaline.
- Dilate the prepucial meatus with artery forceps.
- Retract foreskin and clean with warm saline.
- Make circular incision on inner skin approximately 3cm from the corona, taking care not to injure the urethra and the glans penis.
- Pull foreskin over glands penis and make incision with surgical knife over the coronal sulcus
- Leave adequate penile skin.
- Complete circumcision with scissors.
- Control all bleeders with clamps and ligatures.
- Suture incision with 3/0 vicrylon cutting needle.

**Note:**

- Use of Plastibel in circumcision of neonates is not recommended due to frequent injuries and is best left for experienced surgeons.
- Methods for infants, adolescent and adults are as described above. It can be performed under local anaesthetic.
- Do not use adrenaline.

## 50.9.5 ADOLESCENT HAEMATURIA

This clinical condition can be macroscopic or microscopic blood in the urine. In children, possible causes include

- glomerulonephritis

- anaphylactoid purpura (Henoch-Schönlein purpura)
- fever
- strenuous exercise
- mechanical trauma (masturbation)
- foreign bodies
- urinary tract infection (bacterial or parasitic)
- hypercalciuria/urolithiasis

- sickle cell disease/trait
- coagulopathy,
- tumours
- drugs/ toxins (NSAIDs, anticoagulants, cyclophosphamide, ritonavir, indinavir
- anatomic abnormalities (hydronephrosis, polycystic kidney disease, vascular malformations, and hyperuricosuria.

**Investigations**
- Urinalysis
- Abdomianl ultrasound for kidney, ureter and bladder
- CT abdomen

**Note:**
- Confirm the presence of and the extent of haematuria, as well as the primary cause.
- Determine secondary problems.

**Management**
- Treat the primary cause.
- Manage any complications.

## 50.9.6 HAEMATURIA IN THE ADULT

This is a common condition that has mostly benign causes. The commonest of these causes is urinary tract infection, while the most feared causes are malignancies of the urinary tract. Other causes include bleeding diathesis, urinary tract calculi, urinary tract trauma and hypertension. Macroscopic haematuria is more likely than microscopic haematuriato be due to urinary tract pathology. Ageing is associated with a higher incidence of significant urinary tract pathology.

**Investigation**
Should include the following:
- Urinalysis for culture and sensitivity
- Urine for cytology
- Urinary tract ultrasound
- Kidney ureter and bladder (KUB), radiograph
- Cytoscopy (rigid, flexible)
- Intravenous urogram
- CT scan abdomen

**Management**

- Identify and treat the underlying cause. Treatment for bladder cancer needs special emphasis as delay in diagnosis has morbidity and mortality implications.
- Address any complications that may have arisen.

## 50.9.7 URINARY RETENTION

- This is the inability to pass urine when the urinary bladder is full. There is an urge to micturate and if not relieved, there is severe pain with straining. The causes vary with age and gender. The common causes are:
  - For children, meatal stenosis, phimosis or paraphimosis, posterior urethra valves, ruptured urethra after trauma, and constipation.
  - For adults aged 20–50 years, urethral stricture, calculi (bladder and urethral stones), bladder tumours, ruptured urethra (trauma), and post operative (any perineal operation) clot retention.
  - For male adults older than 50 years, prostatism (benign prostatic enlargement, carcinoma of the prostate, prostatitis, prostatic fibrosis), calculi, urethral strictures, bladder tumours, ruptured urethra(trauma), and postoperative clot retention.
  - For females, bladder tumours, calculi, pelvic tumours (cancer cervix), urethral stenosis, and post operative clot retention (severe haematuria).

**Note:** Spinal cord compression with paraplegia/quadriplegia results in urinary retention.

**General Management**

- Relieve acute retention by catheterization:
- If catheterization fails, use cystofix or suprapubic cystostomy and refer.

**Specific Management**

- Perform circumcision for phimosis or paraphimosis [see circumcision].
- Carry out prostatectomy and urethroplasty as indicated.
- Treat cancer as indicated in cases of malignancy.

## 50.9.8 URETHRAL STRICTURE

Causes of urethral stricture include congenital, traumatic (usually follows fracture of pelvis), inflammatory (follows gonorrhoea infection, usually earlier in life) and instrumentation that results from indwelling catheter following endoscopy or postoperatively following prostatectomy or after amputation of penis.

**Clinical Features**

**U**sually occurs in younger patient (below 50 years). Early symptoms include passage of flakes in urine with early morning urethral discharge while the later symptoms include difficulties in micturition (narrow prolonged stream, dribbling, straining). There is urine retention with a distended urinary bladder.

History of urethral discharge in the past, history of pelvic injury, and history of instrumentation are significant. The urethra should be palpated for induration, and a rectal examination performed on all patients.

**Investigations**
- Urinalysis and culture and sensitivity
- Urea and electrolytes
- Micturating cystourethrogram and ascending urethrogram

**Management**
- Carry out supra pubic cystostomy or insert cystofix if there is retention of urine.
- Urethroplasty.

## 50.9.9 URETHRAL INJURIES

This may result from urethral trauma (for example a fall astride a projecting object, cycling accident), fracture of pelvis in road traffic accident, penetrating wounds (bullet wounds, etc.), and iatrogenic injuries.

**Clinical Features**
- Difficulty or inability in passing urine.
- There may be blood at the external meatus.

**Management**
- Admit for
  - Resuscitation and suprapubic catheterization.
  - Subcutaneous extravasation of urine and urethra stricture.
- Start on appropriate antibiotic cover.

**NOTE**
- Do not catheterize the patient per urethra.
- Give analgesia: Morphine or pethidine.
- If bladder is full, empty through as suprapubic cystostotomy, but if the patient has passed urine “leave alone”.
- Start antibiotics
- Group and cross-match blood.
- Order a plain pelvic radiograph. An ascending and descending urethrogram should be ordered thereafter.

**Note:** Definitive treatment will depend on which part of the urethra is ruptured, anterior (bulbous) or posterior (membranous). This is specialized treatment for which the patient should be referred to a urologist.

## 50.9.10 RUPTURED BLADDER

This usually follows a blow, a kick, or a fall on a distended bladder, gunshot or stab wounds, passage of instruments, endoscopic resection of prostate or bladder tumour, diathermy coagulation of bladder tumour, and operative procedures in the pelvis (for example, tubal ligation and hysterectomy).

**Clinical Features**

- Injury may be intraperitoneally or extraperitoneally.
  - Intrapentoneal rupture results in sudden agonizing pain in the hypogastrium, severe shock, with a rigid abdomen that distends slowly. The patient passes no urine. Rectal examination reveals a bulge in the pouch of Douglas.
  - Extraperitoneal rupture displays similar symptoms as in rupture of posterior urethra described above.
- The patient experiences pain, blood-stained urine, and may show other features of the primary pathology.
- Severe peritonitis is an ominous complication that may develop if the patient is not attended to within 12 hours. In situations of delayed attention, it may have a mortality rate of 100%.

**Investigations**

- Plain erect radiograph of the abdomen may show "ground glass" appearance of fluid in the lower abdomen.
- Intravenous urography will demonstrate a leak from the bladder.
- Abdominal ultrasound

**Management**

- Initiate resuscitation measures.
- Conduct laparotomy after resuscitative measures are taken.
- Repair the rupture in the bladder in two layers.
- Leave a urethral catheter in situ for 10–14 days.

## 50.9.11 BENIGN PROSTATE ENLARGEMENT(BPE)

Benign prostate enlargement causes lower urinary tract symptoms. A big prostate is not always symptomatic or problematic. A large prostate can cause damage to the kidneys, ureter, or bladder with minimal symptoms. Benign prostate enlargement is age related but not in a linear fashion. Symptoms increase with size, but also not in a linear fashion, and the condition does not always require surgery. Symptom evaluation of BPE must include the international prostate symptom score (Table 50.2).

**Clinical Examination**

Digital rectal examination (DRE) wil lreveal enlarged prostate, with smooth surfaces.

**Investigations**

- Urine for culture and sensitivity
- Urea Electrolyte and Creatinine levels
- Prostatic ultrasound (intrarectal)

- Prostatic specific antigen (PSA).

**Management**
**This includes:**

- Watchful waiting is for those with mild symptoms without damage to kidneys and ureters.
- Medical treatment (alphareductase inhibitors,e.g., finasteride 5mg daily; review treatment after 6 months. Note: May require treatment for several months before benefit is obtained)
- Surgical treatment if necessary.

Surgery is reserved for those with complications, like retention that fails trial without a catheter. Note that retention without such a trial does not qualify as an absolute indication for surgery.

## Table 50.2: International prostate symptom score(IPSS)

Name:_____ Date:_____

| | Not at all | Less than 1 time in 5 | Less than half the time | About half the time | More than alf the time | Almost always | **Your score** |
|---|---|---|---|---|---|---|---|
| ***Incomplete emptying*** Over the past month, how often have you had a sensation of not emptying your bladder completely after you finish urinating? | 0 | 1 | 2 | 3 | 4 | 5 | |
| ***Frequency*** Over the past month, how often have you had to urinate again less than two hours after you finished urinating? | 0 | 1 | 2 | 3 | 4 | 5 | |
| ***Intermittency*** Over the past month, how often have you found you stopped and started again several times when you urinated? | 0 | 1 | 2 | 3 | 4 | 5 | |
| ***Urgency*** Over the last month, how difficult have you found it to postpone urination? | 0 | 1 | 2 | 3 | 4 | 5 | |
| ***Weak stream*** Over the past month, how often have you had a weak urinary stream? | 0 | 1 | 2 | 3 | 4 | 5 | |
| ***Straining*** Over the past month, how often have you had to push or strain to begin urination? | 0 | 1 | 2 | 3 | 4 | 5 | |

| | None | 1 time | 2 times | 3 times | 4 times | 5 times or more | **Your score** |
|---|---|---|---|---|---|---|---|
| ***Nocturia*** Over the past month, how many times did you most typically get up to urinate from the time you went to bed until the time you got up in the morning? | 0 | 1 | 2 | 3 | 4 | 5 | |

| | |
|---|---|
| Total IPSS score | |

| **Quality of life due to urinary symptoms** | Delighted | Pleased | Mostly atisfied | Mixed – about equally satisfied and dissatisfied | Mostly dissatisfied | Unhappy | Terrible |
|---|---|---|---|---|---|---|---|
| If you were to spend the rest of your life with your urinary condition the way it is now, how would you feel about that? | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

**Total score:** 0–7 Mildly symptomatic; 8–19 moderately symptomatic; 20–35 severely symptomatic.

**Other absolute indications for surgery include:**

- Bladder stone
- Bladder diverticulum
- Intractable bleeding
- Raised creatinine
- Dilated ureters and kidney.
- Evaluation of the patient
- Conservative or definitive surgical management.
- Surgery includes:
  - Transurethral resection of the prostate (TURP)
  - Open prostatectomy



### 50.9.12 PROSTATE CARCINOMA

**Clinical Features**

There is poor urinary stream, haematuria, back or leg pain, as well as urinary urgency. Other features of secondary spread may also be present. Digital rectal examination typically reveals an irregular, firm prostate or nodule.

**Investigation**

- Measurement of PSA levels either total or ratio of free to bound.
- Biopsy for histology; Gleason score suggestive of prognostic outcome.
- Ct scan abdominal pelvic for staging of the disease.

**Management**

- Catheterize those with acute retention. If this fails, revert to a suprapubic cystostomy.
- Administer antibiotics for infection according to culture reports. Start on nitrofurantoin 100mg 6 hourly and await cultures.
- Initiate other emergency treatment as needed.
- Initiate hormonal therapy for advanced disease.
- Orchidectomy
- Provide nutritional support.

## 50.10 Ulcers and Tumours of the Skin

**The causes of these include the following:**

- Infections:
  - Bacterial: Mainly tuberculosis, leprosy, syphilis and anthrax
  - Fungal: For example, histoplasmosis.
  - Parasitic:For example, leishmaniasis
- Tumours:
  - Squamous cell carcinoma
  - Basal cell carcinoma
  - Melanoma
  - Kaposi's sarcoma

- Vascular:
  - Ischaemic (arterial)
  - Venous, venousinsufficiency
  - Sickle celldisease
  - Diabetes,
  - Thromboangitis
- Trauma
- Tropical ulcers

**Clinical Features**

Ulcers are mainly found in the lower limbs but may occur on any part of the body. Examination should be thorough and systematic. The following are, with brief examples, the characteristics to note:

- ***Site:*** For example, 95% of rodent ulcers (basal cell carcinoma) occur on the upper part of the face; carcinoma typically to the lower lip, while syphilitic chancre affects the upper lip.
- ***Size:*** Carcinoma spreads more rapidly than inflammatory ulcer.
- ***Shape:*** Rodent ulcers are usually circular while straight edges are found in dermatitis
- ***Edge:*** Undermined occurs in tuberculosis, rolled edges in basal cell carcinoma, (rodent), evert edges in squamous cell carcinoma, vertically punched out edges in syphilis and slopping edges in venous and traumatic ulcers.
- ***Base:*** Is palpably indurated in squamous cell carcinoma.
- ***Floor:*** When examined appears granulomatous in tuberculosis.
- ***Discharge:*** Purulent discharge indicates active infection while greenish discharge is seen in pseudomonas infection.
- ***Lymph nodes:*** Are enlarged mainly in malignant tumours.
- ***Pain:*** Occurs generally in malignant, tuberculous, and anal ulcers, while tropical ulcers are painless.

**Investigations**

This depends on the causative factor and may include:

- Full blood count
- Pus for culture and sensitivity
- Blood sugar
- VDRL
- Arteriography
- Biopsy for histology
- Mantoux test
- HIV screen
- Relevant radiographs to rule out bone involvement and/or infections.

**Management**

**The following are important:**

- Give antibiotics for infected wounds appropriately.

- Conduct regular cleaning and dressing with antiseptic for 3 days.
- Give tetanus toxoid 0.5ml IM.
- Identify primary cause and if able to manage at this level, then manage.
- Carry out wound excision/skin graft if no healing of the wound observed.
- Order histology for chronic ulcers to rule out malignant conditions.
- If necessary, treat malignant and varicose ulcers by amputation and stripping of the varicose veins, respectively.

# 51. Dental and Oral Conditions

Oral health is an integral part of general health. It entails the health of the mouth (the oral cavity), the jaws, the teeth, and all contiguous structures. The kind of diseases, disorders, and conditions that may be diagnosed in this area of the body can be particularly diverse. Since the mouth constitutes the main gateway into the entire body, disease processes and disorders elsewhere in the entire body may also be reflected and diagnosed here. This chapter discusses the most common diseases, conditions, and disorders that health clinicians may encounter in their daily practice.

## 51.1 Bacterial Infections

The mouth is a favourite habitat of a myriad range of disease causing and commensal micro organisms. These include nearly the entire range of aerobes and anaerobes, as well as Gram-positive and Gram-negative microbes.

Commonly, sites and sources of bacterial infection in the orofacial area include:

- Carious (decayed) teeth
- Root remnants in the jaws
- Periodontal infection
- Pericoronal infection
- Pre-exisitng pathology such as bone cysts, bone dysplasia and neoplasms
- Trauma to tissues

Remarkably, bacterial infections in the oral cavity may take diverse clinical courses and presentation as outlined in the subsequent sections.

### 51.1.1 DENTAL CARIES AND PULPITIS

Dental caries is a microbial infection characterized by the demineralization of the **inorganic** component and destruction of the **organic** component of the teeth. It involves progressive damage of the enamel, dentine, and cementum initiated by microbial activity on any tooth surface in the oral cavity. It is also the most common cause of pulpal disease, which results from bacterial invasion of dentine and eventually the pulp. The spillage of microbial toxins into the tooth pulp through the caries lesion precipitates pulpitis.

#### DENTAL CARIES WITHOUT PULPITIS

**Clinical Features**

Clinically the tooth presents with a cavity and the patient complains of mild pain on either chewing or extremes of temperatures.

**Management**

- Dental radiographs: BBW/IOPA
- Oral hygiene instructions
- Diet counselling

- Fluoride therapy, especially for high caries risk
- Analgesics: Paracetamol 1gm orally 8 hourly or ibuprofen 400 mg orally 8 hourly. Adjust dose according to age.
- Restorative procedures for carious teeth. Either composite resin, amalgam, glass ionomer cement or compomer restoration.

### DENTAL CARIES WITH PULPITIS

**Clinical Features**

- Sharp severe pain especially at night.
- Extreme tenderness of the affected tooth, which may imply impending pus formation.
- The tooth may be tender to percussion.

**Management**

In the absence of allergy, amoxicillin 500 mg orally 8 hourly and metronidazole 400 mg orally 8 hourly remain the most useful drugs.

- Depending on severity, direct or indirect pulp capping with CaOH2 may be considered for permanent teeth. Deciduous teeth where indicated would require pulpotomy and stainless steel crowns.
- Endodontic treatment with restoration of the tooth with irreversible pulpitis. Thereafter, crown prosthesis. For deciduous teeth, pulpectomy followed by restoration and stainless steel crown.
- Incision and drainage in the case of dentoalveolar abscess.
- Tooth extraction for grossly carious teeth.
- Provision of dental prosthesis where necessary.

## 51.1.2 PERIAPICAL AND DENTOALVEOLAR ABSCESS

It occurs secondary to an infective process in the pulp.

**Clinical Features**

Clinically presents with pain and localized swelling adjacent to the carious tooth. This swelling can be purulent and can spread to the adjacent mucosa depending on severity.

**Treatment**

- Initiate analgesics treatment: Ibuprofen 400mg orally 8 hourly.
- In the presence of an abscess: Give amoxicillin 500 mg 8 hourly orally and metronidazole 400 mg 8. hourly.
- Incise and drain the abscess; swab for culture and sensitivity.
- Institute root canal treatment for the offending tooth.
- Wait 3 days to extract the tooth if it is grossly carious to allow the abscess to subside. Provide dental prosthesis thereafter.

## 51.1.3 BACTERIAL SIALEDENITIS

Bacterial infections can lead to the inflammation of the salivary glands. Bacterial sialedenitis commonly affects the parotid gland, submandibular glands are rarely affected.

### ACUTE BACTERIAL SIALEDENITIS

**Clinical Features**

This is characterized by a sudden onset of unilateral pain mostly located at the angle of the mandible. The affected gland is enlarged, tender, and very painful. There is purulent discharge from the Stensen's duct. Patient may be febrile with other signs of inflammation. The condition is common in debilitated and dehydrated patients predisposed to xerostomia.

**Management**

- Reverse the medical condition that may have contributed to formation
- Discontinue anti-sialogogues if possible
- Warm compresses give sialogogues (lemon drops)
- External salivary gland massage if tolerated
- Improve oral hygiene of the patient by debridement and irrigation
- Give amoxicillin 500mg 8 hourly
- Initiate analgesics treatment: Ibuprofen 400mg orally 8 hourly.
- Carry out surgically drainage if indicated using needle aspiration.

### CHRONIC BACTERIAL SIALEDENITIS

This is chronic or recurrent and may be idiopathic or associated with factors that cause ductal obstruction. The disease starts mostly as an unilateral swelling at the angle of the mandible. The recurrent type shows periods of remission.

**Management**

- Give amoxicillin 500mg 8 hourly orally.
- Initiate analgesics treatment: Ibuprofen 400mg orally 8 hourly.
- Excise the sialolith.
- In intractable cases, excise the salivary gland.

## 51.1.4 CELLULITIS AND ABSCESS FORMATION

Orofacial cellulitis may emanate from any of the sources and sites given earlier. The principal micro-organisms that precipitate cellulitis produce diverse toxins, enzymes, and cytokines that destroy tissue to facilitate infection, which spreads through the contiguous fascial planes. In this way there is always the danger of the spillage of the infection into the bloodstream (septicaemia) and any adjacent vital organs and structures. When an acute infection emanates from the mandibular structures or the floor of the mouth and rapidly spreads to involve the

bilateral fascial planes, it often culminates in a deadly condition referred to as Ludwig's Angina.

**Note: All clinicians must endeavor to recognize these conditions most promptly, since death can occur in a matter of hours.**

**Clinical Features**

There is massive bilateral upper neck swelling with board-like feel on palpation. Tongue is raised towards the roof of the mouth and the floor is heavily indurated, the tissues having a cauterized-like surface. The patient is severely distressed because of respiratory embarrassment, and *onset of stridor is ominous because it implies impending death.*

**Management**

**The following management should be carried out:**

- Admit the patient and institute specialist consultation promptly.
  - Ensure secure airway during referral and provide competent escort.
  - If Ludwig's angina is diagnosed, then clinicians consulted may consider surgical intervention including surgical decompression, and/or tracheostomy.
  - Where an abscess is diagnosed, incision and drainage must be performed promptly and antibiotics commenced after culture and sensitivity report.

**For most acute bacterial infections in the orofacial area, the following should be done:**

- Give amoxicillin 500mg orally 8 hourly for adults and amoxicillin suspension 125–250mg for children remain the most useful for empirical management. In case of allergy, erythromycin 500mg orally 8 orally.
- Consider metronidazole 400mg orally 8 hourly (for children metronidazole suspension100mg orally 8 hourly) for 5–7days, in addition to amoxicillin where anaerobic micro-organisms are suspected to play a major role.
  - In cases of severe infections, benzyl penicillin 2.4g IV 6 hourly + metronidazole 500mg IV 8 hourly+gentamicin 80mg IV 8 hourly.
  - Analgesics: Ibuprofen 400mg orally 8 hourly. For severe pain diclofenac 75mg IM 12 hourly.
- Rehydrate the patient with 5% dextrose alternating with normal saline.

**Clinicians must note that massive antimicrobial administration does not eliminate pus from tissues. Incision and drainage of the established pus is mandatory.**

## 51.1.5 CERVICOFACIAL NECROTIZING FASCITIS

This is a bacterial infection that often requires special attention since it is associated with extreme morbidity.

Cervicofacial necrotizing fascitis is a mixed bacterial infection whose pathogenesis principally involves extensive and rapid destruction of fascia, almost exclusively around the neck and craniofacial area. The exact pathophysiology of the exclusive fascial damage remains unknown, however. Paradoxically, no specific micro-organisms have been implicated in the pathology of this condition. Once fascia is destroyed, the covering skin remains without nutrients and support, thereby breaking down to expose the underlying structures. Since this condition may not be as uncommon as medical literature may imply, clinicians are prompted to recognize it. The hallmark of the condition is that it may present with little suppuration and yet there will be extensive fascial necrosis with consequent skin breakdown.

**Management**

- Admit the patient for management and intravenous medication with supportive rehydration
- Initiate antibiotics: Amoxicillin + clavulinic acid 1.2g IV 12 hourly + metronidazole 500mg IV 8 hourly+gentamicin 80mg IV 8 hourly and intramuscular diclofenac 75mg IM 12 hourly.
- Swab for culture and sensitivity.
- Conduct surgical consultation for appropriate intervention: Mop out necrotic tissue meticulously with copious antiseptic irrigation (hydrogen peroxide/ povidone iodine).
- Dress exposed tissues appropriately and allow for adequate healing before plastic surgery intervention.

## 51.1.6 PERIODONTAL (GUM) INFECTIONS

The periodontium is a functional unit whose main roles include the support of the teeth within the jawbones and the provision of sensory information relating to the function of chewing. The components of the periodontium, therefore, include the alveolar bone, cementum, the periodontal ligament, and the gingiva (gum). Acute and chronic periodontal disease is one of the most common ailments affecting mankind. Some evidence of deterioration of the periodontal tissues can be demonstrated in almost all dentate adults. The periodontal tissues, like other tissues, are subject to inflammatory, degenerative, dysplastic, and neoplastic pathological changes.

### GINGIVITIS

This is an inflammatory process that usually originates at the dentogingival junction and affects the functional gingival component of the periodontium. It is primarily a disease of the gingiva but may spread secondarily to the alveolar or oral mucosa. It presents with uneven red colour of the gums, thickened blunted margins, and swollen papillae. The gingiva is soft and boggy and may bleed on palpation. Gingivitis may lead to periodontitis which may result in sensitivity to both hot and cold temperature, sweet and acidic foods and drink.

**Management**

- Oral hygiene instructions.
- Chlorhexidine mouthwash 0.2% rinses or normal saline rinses.
- Dental prophylaxis.
- In severe gingival hypertrophy, gingivoplasty can be recommended.
- Scaling and polishing where applicable
- Provision of advice for management of sensitivity including suitable toothpaste and cleaning agents

## PERIODONTITIS

Inflammation of the supporting structures of the teeth associated with the loss of attachment and alveolar bone. Characterized by gingivitis, periodontal pocket, gingival recession, tooth mobility.

**Management**

- Oral hygiene instructions.
- Chlorehexidine mouthwash 0.2% rinses or normal saline rinses.
- Full mouth scaling.
- In severe cases root planingis required, periodontal splinting.
- In severe tooth mobility (>3) tooth extraction may be indicated.
- Comprehensive periodontal management is required for aggressive forms of periodontitis.

## PERICORONITIS

Is the inflammation of the gingiva covering a partially erupted or impacted tooth. Presents with deep pain, gingival swelling, pus production and gingivitis of the overlying gum.

**Management**

- Oral hygiene instructions.
- Chlorehexidine mouthwash 0.2% rinses or normal saline rinses
- In presence of an abscess initiate antibiotic therapy: Amoxicillin 500mg orally 8 hourly+ metronidazole 400mg orally 8 hourly. Ibuprofen 400mg orally 8 hourly.

**Surgical operculectomy is the modality of treatment. In the presence of an impacted tooth, surgical disimpactionis indicated.**

## ACUTE ULCERATIVE GINGIVITIS

This disease is reported to be highly prevalent in Africa where it affects children and in groups of persons with congenital disorders such as Down's syndrome. Significantly, nutritional deficiencies arising from the prevalent poor socio-economic status of many of our populations may predispose to the occurrence of most cases that present with acute ulcerative gingival conditions. Poor oral hygiene may be prevalent where economic empowerment is low.

**Management**

- Oral hygiene with antibiotics and mouthwash with povidone iodine1%8 hourly.
- Benzylpenicillin1.2g IV 6 hourly+metronidazole 500mg IV 8 hourly+ gentamicin 80mg IV 8 hourly.
- Try to address the primary cause.
- Definitive management – periodontal cleaning and debridement.

## GANGRENOUS STOMATITIS (CANCRUM ORIS, NOMA)

This is an infective condition of the orofacial tissues that may cause extensive tissue destruction with severe morbidity. The condition may initially manifest as an acute ulcerative necrotizing gingival infection that rapidly involves a block of the contiguous tissues culminating in their breakdown. Unfortunately, the clinical picture and changes associated with this condition may often be so rapid that even the keenest clinician may not notice the progression of the pathological events.

**Management**

- Admit the patient for empirical parenteral antimicrobial therapy (benzyl penicillin1.2g IV 6 hourly and metronidazole 500mg IV 8 hourly).
- Give diclofenac 75mg IM 12 hourly.
- Institute parenteral nutritional support.
- Improve oral hygiene accordingly.
- Initiate prompt specialist consultation where feasible. This will probably require a multidisciplinary approach.

## 51.1.7 BONE INFECTIONS

Infection in the jawbones may be localized or generalized. Generally, the localized forms of infection are the most common, with the focal osteitis/alveolitis (dry socket) occurring 1 to 7 days following a dental extraction. This probably is the most common bone infection following a dental extraction. Patients will complain of much more severe pain than a toothache. The pain is usually throbbing and deep seated. Analgesics often offer little help.

**Clinical Features**

Examination reveals a denuded, open tooth-socket with a scanty necrotic clot while the bone often appears literally exposed hence the term, dry socket. On the other hand, infection may involve a large part of the jawbone, most often the mandible. An infective source may be anywhere within the oral cavity.

Such infection would then be rightly designated as osteomyelitis. In its acute form, severe pain and fever are significant presentations and may eventually develop suppurative osteomyelitis that may lead to sequestration. In other situations the acute phase may progress into the chronic sclerosing type of osteomyelitis that is not associated with sequestration.

**Management of Focal Osteitis/Alveolitis**

- Investigate using appropriate radiographs. BBW/IOPA
- ***Under local anaesthesia***, perform measures to debride the sparse necrotic clot and provoke fresh clot formation. Perform surgical curettage and irrigate copiously with normal saline.

- Pack the socket with alvogyl.
- Give tabs ibuprofen 400mg orally 8 hourly.
- Administer metronidazole 400mg orally 8 hourly and amoxicillin 500mg orally 8 hourly as these may be of benefit where there is evidence of infection.

**Management of Jaw Osteomyelitis**
- Initiate ibuprofen 400mg orally 8 hourly to control pain.
- Acute forms will require parenteral administration of an appropriate antimicrobial agent, e.g., clindamycin 300mg IM 6 hourly
- Eliminate any focus of infection wherediagnosed.
- For chronic suppurative types, consider surgical intervention where sequestration has occurred.
- Investigate all patients to ascertain their immunological status.

## 51.2 Trauma of the Orofacial Tissues

Injury to the teeth and the supporting alveolar bone occurs quite frequently, especially among children. Other more severe injuries to the soft and skeletal tissues of the orofacial area commonly arise through road traffic accidents, sporting activities, and interpersonal violence. Such violence where guns and other missiles are used may lead to extensive tissue destruction with high morbidity. Management of injuries of the tissues in the maxillofacial area should follow the basic principles of resuscitation: secure the airway, maintain breathing, and ensure circulation as a priority.

### 51.2.1 OROFACIAL INJURIES

**Management of All Orofacial Injuries**
- Stabilize as appropriate and maintain an airway.
- Administer tetanus toxoid 0.5ml IM STAT.
- Give analgesics: Ibuprofen 400mg orally 8 hourly.
- If in level 4, refer to a higher levels for appropriate management.

Management of Jaw Fractures and Severe Soft Tissue Injuries
Mandibular fractures may present with swelling, pain and loss of function due to the derangement of occlusion: antimicrobial and analgesic cover is then mandatory**.**
- Give amoxicillin 500mg orally 8 hourly + metronidazole 400mg orally 8 hourly.
- For analgesia, give ibuprofen 400mg orally 8 hourly.
- Ensure that the fractured fragments are adequately reduced. Use of a crepe bandage around the jaw and over the head should minimize fragment movement.
- Order an orthopantomogram, as this is the most useful radiographic investigation and should reveal the nature and severity of the fracture.
- Refer the patient for specialist surgical management.

**Primary care for gunshot and missile-associated injuries entails the control of haemorrhage, surgical toilet, and suturing. Appropriate packing with**

**antiseptic dressings (povidone iodine 10%) may be indicated in deep cavitating injuries where there is severe tissue loss.**

- ***Do not be too aggressive at the primary surgical toilet procedure.*** Useful tissue maybe salvaged by employing multiple staged procedures. This facilitates easier reconstructive procedures afterwards.

- For all severe injuries of the mid and lower face, protect the cervica lspine. Hence choose any imaging investigation carefully. Where feasible and available, a CT scan of the full neck and cranium may be the most useful primary investigation.
- Avoid unnecessary plain radiographic views.



**Criteria for the Admission of a Patient with a Craniofacial Injury**

- Prolonged loss of consciousness reported.
- Clinician is notable to predict the consciousness status.
- There is evidence of severe blood loss necessitating replacement.
- There is persistent/recurrent headache.
- There is massive oedema in the facial region and especially in the floor of the mouth.
- Any condition that may adversely influence the stability of the airway.
- Evidence of general confusion of the patient.
- Clinician must use discretion to evaluate the minimum criteria that will necessitate the admission of an injured patient for appropriate management.
- Rhinitis due to the risk of cerebrospinal fluid leakage.

## 51.2.2 DENTAL INJURIES

**Often teeth are injured during trauma and are fractured, displaced, or completely avulsed.**

- Antimicrobial cover is then prescribed appropriately: amoxicillin 500mg orally 8 hourly+ metronidazole 400mg orally 8 hourly.
- Meticulous oral hygiene should be emphasized.
- Soft diet is advised.

### UNCOMPLICATED CROWN FRACTURE

**Clinical Findings**

- Fracture involves enamel or dentin and enamel.
- The pulp is not exposed.
- Pulp test may have a false negative initially.
- Observe pulp until a definitive pulpal diagnosis can bemade.

**Radiographic Findings**

**Fracture involves enamel and/or dentin. Pulp is not exposed.**

- 3 angulations radiographs should be taken to rule out displacement or fracture of the root.
- Radiograph of lip orcheek lacerations is recommended to search for tooth fragments or foreign material.

**Treatment**

- If tooth fragment is available, consider whether it can be bonded to the tooth.
- Restore the tooth with composite resin and consider composite bandage where applicable.
- In case of severe crown fracture, consider a fixed prosthesis (crown).
- Primary teeth: Smooth sharped ges. If possible the tooth can be restored with glass ionomer filling material or composite filling.

## COMPLICATED CROWN FRACTURE

**Clinical Features**

- Fracture involves enamel and dentin and the pulp is exposed.
- Pulp test may have a false negative nitially.
- Observe pulp until a definitive pulpal diagnosis can be made.

**Radiographic Findings**

**Fracture involves enamel and dentine and the pulp is exposed**

- 3 angulations radiographs should be taken to rule out displacement or fracture of the root.
- Radiograph of lip or cheek lacerations is recommended to search for tooth fragments or foreign material.

**Treatment**

- In immature teeth, pulp capping (exposure<1mm) or partial pulpotomy with CaOH2. This treatment is also the choice in young patients with completely formed teeth.
- In older patients, root canal treatment can be the treatment of choice, although pulp capping or partial pulpotomy be considered.
- If exposure is>24 hours between accident and treatment root canal treatment is indicated.
- In extensive crown fractures, fixed prosthesis can be considered (crown)
- Extraction may be the last option.
- Primary teeth: Pulpotomy or pulpectomy where indicated with subsequent restoration with a stainless-steel crown thereafter. In a case of poor prognosis, extraction is the choice of treatment with space maintenance.

**ROOT FRACTURE**

**Clinical Finding**

- The coronal segment may be mobile and may be displaced.
- The tooth may be tender to percussion.
- Pulp test may have a false negative initially.
- Observe pulp until a definitive pulpal diagnosis
- Transient crown discoloration (red or grey) may occur.

**Radiographic Findings**

- The fracture involves the root of the tooth and is in a horizontal or diagonal plane.

**Treatment**

- If displaced, reposition the coronal segment of the tooth as soon as possible.
- Check position radiographically.
- Stabilize the tooth with a flexible splint for 4 weeks. If the root fracture is near the cervical area of the tooth, stabilization is beneficial for a longer period of time (up to 4months).But this has poor prognosis.
- Monitor healing up to one year to determine pulpal status.
- If pulp necrosis develops, root canal treatment of the coronal tooth segment to the fracture line is indicated.
- In case of poor prognosis, extraction of the tooth is advised

**Primary teeth: If the coronal fragment is displaced, extract only that fragment. The apical fragment should be left to be resorbed.**



## 51.2.3 DENTAL-ALVEOLAR FRACTURE

**Clinical findings**

- The fracture involves the alveolar bone and may extend to adjacent bone.
- Segment mobility and dislocation are common findings.
- An occlusal change due to misalignment of the fractured alveolar segment is often noted.
- Sensibility testing may or may not be positive.

**Radiographic Findings**

- Fracture lines may be located at any level, from the marginal bone to the root apex.
- The panoramic technique is of great help in determining the course and position of fracture lines.

**Treatment**

- Reposition any displaced segment and then splint.
- Stabilize the segment for 4 weeks.
- Primary teeth: Same treatment as permanent. However, monitor permanent teeth in the fracture line.

## 51.2.4 CONCUSSION

**Clinical Findings**

- The tooth is tender to touch or tapping. No displacement, no mobility.
- Pulp tests are positive.
- Radiological findings are normal

**Treatment**

- No treatment is needed; same for primary teeth.
- Monitor pulpal condition for at least one year.

## 51.2.5 SUBLUXATION

**Clinical Findings**

- The tooth is tender to touch or tapping and has increased mobility but has not been significantly displaced.
- Bleeding from gingival crevice may be noted.
- May get a false negative pulp test.

**Radiological Findings**

Radiographic abnormalities are usually not found.

**Treatment**

- A flexible splint following repositioning with gentle finger pressure to stabilize the tooth for patient comfort can be used for upto 2 weeks
- Primary teeth: Same treatment as permanent.

## 51.2.6 INTRUSIVE LUXATION

**Clinical Findings**

The tooth is displaced axially into the alveolar bone. It is immobile and percussion may give a high, metallic (ankylotic) sound. Pulp test will give negative result. In not fully developed teeth, pulpal revascularization may occur.

**Radiographic Finding**

The periodontal ligament space may be absent from all or part of the root**.**

**Treatment**

- Teeth with incomplete root formation: Allow spontaneous repositioning to take place. If no movement is noted within 3 weeks, recommend rapid orthodontic repositioning.
- Teeth with complete root formation: Reposition the tooth either orthodontically or surgically as soon as possible. The pulp will likely be necrotic and root canal treatment using a temporary filling with calcium hydroxide is recommended to retain the tooth.
- Primary teeth :If the apex is displaced toward or through the labial bone plate, leave the tooth for spontaneous repositioning. If the apex is displaced in to the developing tooth germ, extract.

## 51.2.7 LATERAL LUXATION

**Clinical Findings**

The tooth is displaced, usually in a palatal/lingual or labial direction.

**Radiological Findings**

The widened periodontal ligament space is best seen on ecentricorocclusal exposures eg standard occlusal and bitewings.

**Treatment**

- Reposition the tooth with forceps to disengage it from its bony lock and gently reposition it into its original location.
- Stabilize the tooth for 4 weeks using a flexible splint.
- Monitor the pulpal condition. If the pulp becomes necrotic, root canal treatment is indicated.
- In developing teeth, confirm revascularization radiographically by evidence of continued root formation and possibly by positive sensibility testing.
- Primary teeth: If there is no occlusal interference, as is often the case in anterior open bite, allow the tooth to reposition spontaneously. When there is occlusal interference, with the use of local anaesthesia, gently reposition the tooth by combined labial and palatal pressure.
- In severe displacement, when the crown is dislocated in a labial direction, extract. If minor occlusal interference, slight occlusal reduction is indicated.



## 51.2.8 EXTRUSIVE LUXATION

**Clinical Findings**

The tooth appears elongated and is excessively mobile. Sensibility tests will likely give negative results.

**Radiological Findings**

Increased periodontal ligament space apically.

**Treatment**

- Reposition the tooth by gently reinserting it into the tooth socket.
- Stabilize the tooth for 2 weeks using a flexible splint.
- Primary teeth: Determine treatment on the basis of the degree of displacement, mobility, root formation, and the ability of the child to cope with the emergency situation. For minor extrusion (<3mm) in an immature developing tooth, careful repositioning or leaving the tooth for spontaneous alignment are acceptable treatment options. Extraction is the treatment of choice for severe extrusion in a fully formed primary tooth.

## 51.2.9 AVULSION

**Clinical Findings**

The tooth is completely out of the socket.

**Treatment**

- Reimplant immediately.
- Cleanse tooth with clean water. Storage/transport medium should include buccal succus, milk, normal saline, or saliva.
- Splint tooth for 4 weeks using a flexible splint.
- Primary teeth: DO NOT re implant.

## 51.3 Orofacial Congenital and Dysplastic Conditions

The most commonly encountered congenital malformations are clefts of the lip and palate. When they are particularly severe, these malformations may pose feeding problems for the affected babies from birth. Special methods of feeding the affected children have to be instituted to facilitate normal growth and weight gain while awaiting surgical intervention Fortunately severe forms of this condition that necessitate such drastic and innovative feeding methods are rare.

Dysplastic lesions may include those that lead to aberrant tissue growths such as congenital epulides and natal and neonatal teeth. Dysplastic lesions of bone may manifest much later in life and should be easy to recognize. Although rare, some bone dysplasias may manifest with endocrine disorders that could have generalized effects. In the presence of any tissue malformation, therefore, clinicians are advised to institute a full investigation of the affected patient.

Ankyloglossia is a common condition in newborns that interferes with breastfeeding and with speech at a later age.

**Management**

- For cases with severe clefts, ensure adequate feeding.
- Where facilities are available special feeding devices can be fabricated.
- Otherwise nasogastric feeding should be the most important.
- Natal and neonatal teeth do not generally cause any impairment. Refer for their removal to allay parent anxiety that they could be inhaled or swallowed.
- Most congenital epulides may be excised under local anaesthesia, e.g., lignocaine 2%+adrenaline1:80,000 local infiltration.They hardly recur.
- Bone dysplasias maybe monitored appropriately until criteria for surgical intervention are defined.
- Cases with clefts should be advised for immediate follow-up and management at an appropriate facility.
- Speech therapy is advisable in all cases of cleft palate.
- Frenotomy of ankyloglossia should be carried out to avoid speech disturbance.

## 51.4 Cysts and Benign Tumours of the Orofacial Region

Cysts are generally slow growing and painless occurring in soft tissue or facial bones. Eventually they cause swelling and disfigurement. As for the bony cysts, pain may manifest due to tissue tension and/or supervening infection.Similarly, benign tumours of the orofacial region may originate from either soft tissue or

bone. Those originating from bone are much more common and often manifest late when function and disfigurement prevail. Among these neoplasms, ameloblastoma is the most important since it is the most common and particularly locally infiltrative. Early identification of this condition is extremely important because of the capacity of this tumourto infiltrate the surrounding tissues. The ossifying/cementifying fibroma is the next most important benign tumour that should be diagnosed early since it can also cause severe disfigurement.

**Management**

- Institute appropriate radiographic imaging to define the nature of the lesion.
- Aspiration of soft tissue lesions for cytological analysis where feasible is useful.
- Incisional biopsy or Excision biopsy for all benign tumours is mandatory.
- The odontogenic keratocyst has now been classified as a benign infiltrative tumour of the jaw bones. A diagnostic incisional biopsy must, therefore, be performed to ascertain its existence before surgical excision is executed.
- Surgical management includes enucleation or marsupialization

## 51.5 Malignant Neoplasms of the Orofacial Region

- It must be recognized at the outset that the mouth, jaws, and facial region constitute an area of the body that manifests the highest diversity of neoplastic pathology. All clinicians ought to be particularly vigilant to this reality.
- Embryologically and developmentally, the oral cavity and the jaws consist of tissues and organs originating from all the three embryonic stem tissues: the ectoderm, mesoderm and endoderm. Basically, the classification of malignant neoplastic pathology any where in the body essentially follows this premise.
- These neoplasms maybe broadly classified as those of epithelial, mesenchymal, and vasorformative in origin.
- Owing to its prevalence, oral squamous cell carcinoma (OSCC) constitutes the most important malignant neoplasm of epithelial origin. The aetiological factors associated with this neoplasm include tobacco use and sustained alcohol consumption. Apparently, immunosuppressive conditions may precipitate the prevalence of OSCC. Malignant neoplasms whose cells of origin are mesenchymal in nature are broadly classified as sarcomas. As a group almost all these lesions have hardly any identified definite aetiological associations.
- Cells of the mononuclear-macrophage system (the reticulo-endothelial systems) may also give rise to malignant neoplasms manifesting in the orofacial region.
- Among these, lymphomas are common, with Burkitt's lymphoma being the most common type.

**Management**

- Order appropriate radiographic imaging, as it may be of value.
- Refer the patient immediately for a diagnostic biopsy procedure.
- Identify a centre that can deal effectively with specific neoplastic lesions and advise the patient accordingly.
- Where necessary, give analgesia for effective pain management.

## 51.6 Neuropathies of the Orofacial Region

### 51.6.1 PAROXYSMAL TRIGEMINAL NEURALGIA

This condition carries very high morbidity because of the severe often intractable pain associated with it. It is common among middle-aged and elderly persons. Patients may report sequential symmetrical tooth extraction with no relief of pain. There is no known aetiological factor. The pain will be reported as severe and lancinating, lasting only a few seconds at particular sites (trigger zones) known to the patient. Often, sleep may not be disturbed at night. During the day there are usually multiple attacks of pain.

**Management**

- Listen to the history of the pain carefully.
- Establish that there are no other lesions that may precipitate similar pain.
- Give analgesia–Diclofenac100mg orally once daily for 3days. After 3 days reassess.
- Always examine the patient while you have a syringe loaded with local anaesthetic with lignocaine 2% (preferably a dental syringe).
- In the event that an attack occurs, quickly infiltrate the anaesthetic directly at the trigger zone. The patient will report immediate pain relief. This is diagnostic of the condition.
- Institute treatment accordingly: Carbamazepine 100–200mg orally nocte ***OR*** 100mg orally 12 hourly constitute the mainstay of treatment. Always start with the lowest recommended dosage of either formulation. Monitor the condition for at least 1 week and adjust the dosages appropriately. Get a physician's review. Since this treatment is often open-ended, review the patients regularly and evaluate haematological indexes accordingly. An assay of the drug in the serum may also be necessary.
- Alternatively, absolute alcohol injections, electrocoagulation of gasserian ganglion & nerve resection.
- For patients who may have suffered for lengthy periods without treatment, emotional instability will be clinically apparent. Therefore, provide backup treatment with a tricyclic antidepressant e.g., amitriptyline 25mg orally 8 hourlyfor a week, then 50mg nocte asmaintenance. Note that suicidal tendencies among patients whose pain is poorly managed are remarkable.

### 51.6.2 FACIAL PALSY

Facial palsy may manifest as a result of a variety of factors, including trauma, deep seated craniofacial neoplastic lesions, and non-specific viral infections. More commonly, the idiopathic type of facial palsy (Bell's palsy) is seen. The history of the condition is often short and there may be no clear-cut associated aetiological events.

**Management**

- Take a clear history to try to determine the type of facial palsy.
- Order craniofacial radiographic imaging and/or magnetic resonance imaging where indicated.
- Use an eye pad to protect the eye on the affected side.
- Institute steroid treatment over a 10 day period. Tabs prednisolone 15mg 8 hourly for 2 days then10mg 8 hourly for the next 2 days then 5mg 8 hourly for the next 3 days, then 5mg 12 hourly for the remaining 3 days.
- Refer for any long-term definitive management.

### 51.6.3 HERPETIC INFECTIONS

The herpes group of viruses and especially Herpes zoster constitutes one of the most common causes of vesiculo-bullous lesions in the orofacial region. The lesions are usually of acute onset manifesting with irritating pain. Where there is underlying immunosuppression due to HIV infection, fulminating Herpes zoster infection may cause extensive damage of the periodontium leading to spontaneous tooth exfoliation from the affected jaw segments. After the acute phase of the herpetic infections, the cutaneous lesions heal with scar formation accompanied by hyperaesthesia over the affected area. This post-herpetic facial neuralgia is often difficult to manage effectively.

**Investigations**

**Investigate for HIV, carry out Mantoux and examine the sputum.**

**Management**

- Diagnosis of the acute lesions is often made clinically as the crops of vesicles are typical.

**Do not touch these lesions without gloved hands.**

- In the acute phase, administration of tabs aciclovir 200mg orally 5 times daily for 7 days is the mainstay of treatment. In immunocompromised, patients give 500mg orally 8 hourly for 10 days.
- Apply lignocaine 1% cream PRN 5–7 days to manage hyperaesthesia.
- Diclofenac 100mg orally once daily may be chosen where pain is persistent (post-herpetic neuralgia). Add carbamazepine 200mg orally 12 hourly if pain persists.

## 51.7 Temperomandibular Joint (TMJ)Disorders

### 51.7.1 TEMPOROMANDIBULAR JOINT DYSFUNCTION

Temporomandibular joint pain and dysfunction remain enigmatic in terms of aetiology and pathogenesis. The condition may be intertwined with stressful life events that are often difficult to elucidate clinically. The condition has become particularly common in persons in their 2$^{nd}$ decade of life and above.

Generally, TMJ pain can be most variable in quality, may be nonspecific and without any clear-cut associated local events. However, it is often possible to

correlate the manifestation of TMJ pain with painful conditions in other areas such as the spine, recurrent headaches, and even abdominal cramps. No radiographic or other imaging modality may demonstrate a tangible biologic basis for the dysfunction and pain. Currently, professional consensus worldwide indicates that this group of conditions should be referred to as temporomandibular joint disorders (TMDS).

**Management of TMDS**

- Essentially, manage emerging symptoms eg bruxism by provision of mouth guard and management with ibuprofen 400mg orally 8 hourly.
- Tricyclic antidepressants: amitriptylline 25mg orally 8 hourly for 7 days, remains useful.

**Investigate selectively to rule out any "organic" changes in TMJs.**

- Note that, overall, expensive and extensive high techinvestigations may yield little value in the management of the individual.
- Evaluate the patient and collectively settle on a management modality that the patient feels offers relief.
- Avoid active invasive surgical intervention unless there is firm evidence that surgery would offer help.
- Consult for alternative opinion – maxillofacial surgeons.

### 51.7.2 TMJ DISLOCATION

This is the excursion of the mandibular condyle beyond the normal range, where it is displaced out of the glenoid fossa, much anteriorly beyond the articular emminence, but still remaining within the TMJ capsule.

**Management**

- Analgesia paracetamol 1,000mg orally hourly or Ibuprofen 400mg 8 hourly.
- In acute dislocation, manipulation for reduction with or without anaesthesia. Local or general anaesthesia maybe required. Muscle relaxants or sedatives may also be indicated.
- In chronic recurrent dislocation:
  - Mandibilar manipulation for reduction with intermaxillary fixation to limit the mouth opening. (crepe bandage may be used as a substitute).
  - Surgical intervention,e.g.,eminectomy, capsule tightening, or creation of a mechanical block may be necessary.

## 51.8 Oroantral Communication and Fistula

This is an unnatural communication between the oral cavity and the maxillary sinus. Commonly occurs after the extraction of the upper posterior teeth.

**Clinical Features**

- Escape of fluids from the oral to the nasa lcavity
- Epistaxis
- Escape of air from the mouth to the nose
- Pain
- Persistent purulent or mucopurulent nasal discharge

**Management**

- Amoxicillin 500mg 8 hourly orally.
- Ibuprofen 400mg 8 hourly orally
- 0.9% normal saline nasal drops
- Surgical intervention where necessary

## 51.9 Edentulism

Teeth are missing as a result of trauma or disease, or congenitally missing.

**Management**

- Removable prosthesis: Partial dentures, complete dentures, oroverdentures.
- Fixed prosthesis: Crown and, bridge, or implants.
- Indeciduous dentition, space management should be considered to prevent loss of archlength.

## 51.10 Dental Fluorosis

This is disturbance of the tooth structure caused by excessive intake of fluoride during the tooth development stage. It is characterized by hypomineralization of the inorganic component, which will present with tooth discoloration, pitting of the teeth and in severe cases brown discoloration of the teeth with destruction of the surface. In some cases there is extreme sensitivity to temperature extremes.

**Management**

- Topical fluoride therapy where there is sensitivity.
- Microabrasion or bleaching may be attempted.
- Restorative techniques to restore aesthetics: Composite masking, porcelain veneers, porcelain crowns.

## 51.11 Orthodontics

This is defined as the aspect /speciality of dentistry that is concerned with growth and development of the head, neck and dentition. It also involves prevention and correction of occlusal anomalies. Deviation from the ideal occlusion may result from skeletal or dental discrepancy.

Early detection of anomalies in growth and development enhances chances of an ideal treatment outcome.

The diagnosis of the malocclusion can be classified into with appropriate sub-categorisation.

1. Class I
2. Class II
3. Class III

The severity and individual patient's need for orthodontic treatment can be categorised using the Index of Orthodontic Treatment Need ( IOTN ) a universally recognised index with Clinical and Aesthetic Components. The lowest index demonstrating little or no need for orthodontic treatment is Index 1 The highest need for treatment is Index 5. Each Index is sub-divided into sub-categories indicating specific clinical features. Ideally IOTN 1-3 can be treated in primary and secondary dental care settings. IOTN 4-5 ideally could be treated by secondary to tertiary dental care settings

**Management**

- Ensure the patient has established a stable oral health base prior to commencing orthodontic treatment to avoid adverse effects of untreated basic dental disease
- Record the single worst clinical feature of dental health from the following aspects
    - **M**issing teeth
    - **O**verjet
    - **C**rossbite
    - **D**isplacement
    - **O**verbite
- Clear statement of patient complaint / concern
- Full Clinical Assessment
    - Extra-oral
    - Intra-oral
- Clinical photographs
- Study casts
- Radiographic investigations as appropriate
    - Orthopantomogram
    - Lateral Cephalogram (as indicated)

**Treatment Outcomes**

These can be evaluated according to treatment goals.
Peer Assesment Rating (PAR) scoring of standardised study casts can provide a measurable assessment of achievement of treatment goals.

## 51.12 Forensic Odontology

Forensic Odontology deals with the professional handling, examination of dental evidence, expert interpretation and documentation of findings made in the interests of justice. It is often done in cases of medico-legal proceedings where the dentist is an expert witness.

The aim is to examine and evaluate injuries to the dentition and assess bite marks, maintain and interpret dental records in dental jurisprudence cases such as malpractice or dental fraud, identify unknown persons, dead or alive.

## 51.12.1 IDENTIFICATION

The **primary** means of identification, using the comparative dental identification method (only possible with excellent dental record systems in place).

**Management**

*The expert should compare the **antemortem** and **postmortem** dental characteristics using written notes, study casts and radiographs, photographs, that would reveal traditions, fillings etc.

## 51.12.2 DETERMINATION OF AGE

These are based on tooth development and eruption, with better accuracy if root length
and degree of mineralization are used.

**Management**

In patients under 20 years of age:

- Radiological methods :Radiographs are interpreted using standard charts relating age to degree of development e.g. Schour & Massler and Gustafson & Koch.H to give estimates
- Histological methods to assess formation and mineralization of enamel and dentine, neonatal lines using the dentition are useful.
- Conclusions of dental age analyses are usually accurate to approximately ±1.5 years (with ±4 year's variation for 3rd molars).
- In middle-aged and older adults assess age using periodontal disease progression, attrition and tooth loss. Note the accuracy using these highly-variable markers is in the range of ±10-12 years.

## 51.12.3 DETERMINATION OF GENDER

- Anatomical techniques for patients that are past puberty since a lot of similarities occur pre-puberty.
- Laboratory based techniques: include DNA testing, Identification of the Barr body, and the F body.

## 51.12.4 BITE MARK ANALYSIS

This is in regard to trauma caused by dentition whether on inanimate objects as evidence or bite marks on humans. Often they point to close combat situations, child abuse and sexual assault in relation to interpersonal violence.

**Management**

Ruling out self-inflicted bites, they should be consistent with a contributory history such as falls on the face. Determine the size and shape and nature of the dentition to determine whether it is of a child or adult. Use the Pretty's Index for Bitemark Severity & Significance.

# 51.13 Maxillofacial Injury

This injury can present with an apparently frightening clinical picture.

Do not panic! Traumatic injuries to the facial structures may be classified as:
- ♦ Soft Tissue Injuries±tissue loss
- ♦ Hard Tissue Injuries±bone loss
- ♦ Combined soft and hard tissue injuries

**Management**
The management principles of maxillofacial injuries are:

- ♦ Advanced trauma life support (ATLS) principles (ABCDE)
- ♦ Restore Occlusion
- ♦ Restore function
- ♦ Restore Aesthetics
- ♦ A thorough history and examination is paramount to the management of maxillofacial injuries.

— Patients with maxillofacial injury require immediate referral to higher levels for appropriate management.

## 51.13.1 ADVANCED TRAUMA LIFE SUPPORT

**Primary Survey**
Airway + cervical spine control: Note that maxillofacial injuries both soft tissue and hard tissue may compromise the airway.
- ♦ If the palate is collapsed on the roof of the mouth,scoop
- ♦ with your finger and try to elevate.
- ♦ If the tongue is pushed back in the direction of the pharynx, pull forward with forceps.
- ♦ Apply suture to hold in place if need be. Lay patient on the side.
- ♦ With severe nose injury, suck to clear the blood and insert nasopharyngeal tube if need be.
- ♦ Take precautions as above for possible neck injuries.
- ♦ If needed in very severe injury, perform tracheostomy with cuffed tube.
- ♦ Apply local pressure or nasal packs soaked in liquid paraffin.
- ♦ Perform direct suture of spurting bleeders.

**Breathing**
- ♦ Rule out other injuries such as head injury or chest injury that may impair breathing;relevant radiographs such as chest radiograph and CT scan of the head should be taken.
- ♦ If the patient is not breathing or oxygen saturations are low,intubate and ventilate.
- ♦ For chest injury management,refer to Section 49.2, on chest injuries

**Circulation**

Monitor vitals such as BP and pulse, which are pointers to impending or established shock;also monitor urine (insert urinary catheter if the patient is unconscious).

- Give benzylpenicillin 2.4gIM 8hourly+metronidazole 500mg IV 8hourly until the situation is managed.
- Administer fluids to maintain haemodynamic stability.
- Monitor fluid management asabove.
- Give tetanus toxoid 0.5ml IM stat.

**Disability**

Check for consciousness and other neurological deficits (Glasgow Coma Scale–GCS–and examination of all cranial nerves). Refer to Table 51.1.

**Table 51.1: Glasgow Coma Scale**

| SerialNo. | Category | Specific function | Score |
|---|---|---|---|
| l | **Eye opening (E)** | Spontaneous | 4 |
| | To voice | 3 | |
| | To pain | 2 | |
| | Nil | 1 | |
| 2 | **Best verbal response (V)** | Oriented, converses | 5 |
| | Conversesbutconfused | 4 | |
| | Inappropriate words | 3 | |
| | Incomprehensible words | 2 | |
| | Nil | 1 | |
| 3 | **Best motor response (M)** | Obeys | 5 |
| | Localizes pain | 4 | |
| | Flexion withdrawal | 3 | |
| | Flexion abnormal | 3 | |
| | Extension | 2 | |
| | Nil | 1 | |

**Resuscitation**

- Arrange transport with adequate resuscitation equip,ent if at Level 4.
- Ensure communication with the receiving facility has been made.

## 51.13.2 SOFT TISSUE INJURIES

As above plus:

- Tetanus toxoid 0.5ml IM STAT.
- Rabies vaccine in case of animal bites

(referto Section1.4.2,on rabies management).

- Antibiotic therapy (refer to Section 51.2,on management

of orofacial fractures in dental and orofacial conditions).

- Thorough debridement of necrotic tissues and

surgical toilet; all vital structures that are injured such as the parotid duct, facial nerve,and naso- lacrimal duct should be repaired.

♦ Primary closure if there is adequate tissue for approximation; plan for wound cover with skin graft or flaps if there is tissue loss.

— Always rule out underlying bone injury by taking appropriate radiographs.

## 51.13.3 HARD TISSUE INJURIES

♦ These may be classified as:

- Dentoalveolar
- Mandibular fractures
- Midface fractures (Le Forte I, II andIII)
- Panfacial fractures
- The bones of the mid face tend to stick out and are thus prone to being injured. The nose, zygoma, and mandible are the most prone to injury,with maxillary bone injuries being relatively less common and more complicated.

## 51.13.4 DENTOALVEOLAR FRACTURES

This is more common in children but can occur in adults
Check for missing teeth/fragments/fillings to
Rule out inhalation or swallowing (take chest x- ray, abdominal x-ray).
For mobile teeth, rule out fractures of the root using radiographs such as intra-oral periapical (IOPA), upper or lower standard occlusal or an orthopantomograms (OPG). Then reposition and splint. Teeth that have very poor support or are infected should be extracted
For alveolar fractures, reduce and splint with composite resin,dental wires (figure of 8), archbar,or acrylic resin splints.

**Management**

♦ Fixation should be maintained for 4–6 weeks in adults
and 2–3 weeks in children. (stainless-steel wire 0.5mm.
♦ Put the patient on a soft diet.
♦ Full blood count.
♦ Clean and repair associated soft tissue injuries of the gingivae and lips.
♦ Give antibiotic cover (refer to Section 51.2, on management of orofacial fractures indental and orofacial conditions),analgesics,and oral mouth wash.

## 51.13.5 MANDIBULAR FRACTURES

These may involve any part of the mandible i.e. the symphysis, parasymphysis, body, angle, ramus, condyle, and coronoid.
They may also be displaced or undisplaced, depending on the pull of the muscles attached to the mandible.
Plain radiographs demonstrate these fractures well–OPG, PA mandible(to assess linguo-buccal displacement), lower standard occlusal, lateralviews.

**Management**

- Closed reduction – Indications (these are not absolute indications) Undisplaced fractures involving the dentate mandible, children ind eveloping dentition and severely atrophic edentulous mandible.
- Maxillo-mandibular fixation (MMF) for 6weeks;10–14 days for children. This is done using archbars, eyelets, or Ivy loops(stainless-steelwire 0.5mm)
- Lingual-labial occlusal splints
- Circum-mandibular wiring
- Gunnings plints
- Antibiotic cover syrup amoxicillin 500mg 8 hourly orally and metronidazole 400mg 8 hourly orally.
- Normal saline rinse or chlorhexidine 0.2% mouth wash.
- Open reduction and internal fixation (ORIF)

**Indications:**

- Displaced unstable fractures segments.
- Associated midface fractures.
- When MMF is contraindicated such as in epileptics, mentally handicapped.

**Treatment**

- Semi-rigid fixation with trans-osseous wires(osteosynthesis)
- Lag screw
- Plates and screws; load sharing plates or load bearing plates(for edentulous atrophic mandible, comminuted and defect fractures).

## 51.13.6 MIDFACE FRACTURES

Investigations include plain radiographs: Occipito-mentalview(OMV),PA skull, OPG), CTscan

**Treatment**

- MMF + suspension wires
- ORIF – -semi-rigid fixation with trans-osseous wires – rigid plating with mini plates (1.5 and 2.0mmplates)

## 51.13.7 ZYGOMATIC COMPLEX FRACTURES

Investigations include OMV, submental vertex (for zygomatic arch fractures), CT scan (axial, coronal cuts + 3Dreconstruction)

**Treatment**

- Limited access treatment (reduction without fixation)for fractures without comminution
  - Gilles technique (through temporal region)
  - Keen technique (intraoral approach)
  - Dingman technique (lateral eyebrow approach)

- ORIF for laterally displaced fractures and those with comminution
  - Semi-rigid fixation with trans-osseous wires
  - Rigid fixation with miniplates (1.5 and 2.0mm plates

- Orbital Fractures
- Eye examination is mandatory.
  - If no ophthalmoplegia and fracture is minimally displaced, no intervention needed.
  - If there is entrapment of orbital contents or muscles, ORIF is done.
  - Miniplates are used for the orbital rims.
  - Consult an ophthalmic surgeon.

# 52. Ophthalmology

## 52.1 Clinical Guidelines For Eye Care

Eye diseases in Kenya are ranked eighth among the top 10 causes of morbidity. Blindness prevalence is estimated at 0.7%. At the current population this translates to about 224,000 people being blind, with close to 672,000 suffering from low vision. Eighty per cent of the causes of blindness are either curable or preventable through primary eye care.

### 52.1.1 WHAT IS IMPORTANT TO KNOW

- Always check the vision for all patients using the Snellen'schart.
- Take good eye history.
- Do eye examination using a torch.

### 52.1.2 WHAT TO BE CAUTIOUS ABOUT

- Never use steroid containing medicines on the eye without a prescription from an eye specialist.
- Never put any medicines on any eye that may have been perforated.
- Never use atropine drops or ointment without a prescription from an eye specialist.
- Never use traditional eye medicines in the eye.

## 52.2 Ophthalmia Neonatorum (Conjunctivitis of the Newborn)

**Clinical Features**
There is bilateral copious pus discharge in the first month of life.

- If signs of ophthalmia neonatorum develop, refer to higher level to attend eye clinic immediately.
- Apply tetracycline eye ointment 8 hourly.
- Apply gentamycin eye drops both eyes 2 hourly ***OR***
- Give IM gentamycin 5mg/kg single dose or Kanamycin 25mg single dose.
- Give doxycycline eye ointment to all newborns at birth.
- Manage complications like corneal ulcer when they are observed.

## 52.3 Senile Cataract

It is estimated that 43% of blindness in Kenya is due to cataract. The senile form is a slow lens thickening secondary to degeneration. The condition is highly amenable to correction.

**Clinical Features**
There is slowly progressive painless visual loss or blurring affecting one or both eyes with increasing glare, showing a white pupil.

**Management :** Lens extraction is the definitive management

## 52.4 Childhood Blindness

Approximately 10,000 cases of childhood blindness occur annually. The causes include congenital cataract, corneal diseases, measles disease, congenital glaucoma. and retinoblastoma.

**Clinical Features**

The features are dependent on underlying condition but may include:

- Poor vision (olderchild)
- Squint (lazyeye)
- White pupil
- Growth in the eye
- Protruding eyeball

**Management**

Refer to eye specialist for appropriate management.

## 52.5 Retinoblastoma

This condition usually occurs among under-5's and is diagnosed on average at about 24 months of age. It may present as a unilateral or bilateral lesion. Retinoblastoma is associated with increased risk of developing pineal tumour. Up to 40% of this condition is hereditary.

**Clinical Features**

- Leukocoria – White pupillary reflex
- Crossed eye or strabismus
- Red painful eye
- Poor vision

**Investigations**

- Indirect opthalmoscopy
- Examination under anaesthesia
- Skull and other radiographs +CT
- Metastatic screening

**Management**

- At level 4
  - For advanced disease, perform enucleation.
- At level 5 and 6
  - Laser andcryotherapy
  - Radiation techniques used but external beam associated with spread of disease through radiation induce tumours.

## 52.6 Trachoma

- Trachoma is the leading cause of preventable blindness in Kenya and accounts for 19% of blindness.
- Eighteen districts are trachoma endemic:Baringo,Kajiado,Narok,West Pokot, Turkana, Marsabit, Samburu, Koibatek, Meru North, Laikipia, Murang'a, Mbeere, Isiolo, Mwingi, Transmara, Kitui, Makueni, Moyale.

**Clinical Features**

There is mucopurulant discharge associated with conjunctiva and corneal scarring and inward turning of eye lids and lashes, causing pain and ulceration. There is loss of vision.

**Management**

- Give tetracycline eye ointment 3 times daily for 6 weeks ***OR***
- Give tabs azithromycin 1g annually for 3 years as masstreatment.
- Promote regular face washing.
- Improve environmental sanitation and disseminate health education.
- Correct entropion/trichiasis surgically.

## 52.7 Glaucoma

Glaucoma is associated with approximately 25,000 blind cases in Kenya.

**Clinical Features**

- There is unexplained gradual decrease in central or peripheral vision.
- In children, the cornea is bigger and hazy.

**Management**

- Treat at level 4 and above.
- Topical beta-blockers, e.g., timolol or betaxolol, 1 drop BD.
- Surgery is definitive.

## 52.8 Refractive Errors

**Clinical Features**

These include the following:

- Decreased vision
- Frontal headaches
- Squinting
- Inappropriate viewing distance
- Eye strain

**Management**

Refer to eye specialist for management of refractive errors.

## 52.9 Vitamin A Deficiency

**Clinical Features**

These include the following;

- Dry eye
- Foreign body sensation

- Eye pain
- Night blindness
- Severe loss of vision
- In most cases features are of gradual onset
- Complications include:
  - Corneal ulcers

**Management**
- Give vitamin A supplement, 200,000 IU as capsules once in 6 months, starting at 6 months to age 5years.
- Immunize against measles.
- Manage complications.

## 52.10 Herpes Zoster Ophthalmicus(HZO)

**Clinical Features**
The following features occur:
- Acute vesicular skin rash that follows the 5th cranial nerve dermatome
- Blurred vision
- Eye pain
- Red eye
- Fever
- Malaise

**Management**
- For pain relief–Diclofenac 50mg orally 8 hourly for 3 days or carbamazepine 200mg 12 hourly orally for 7 days
- Tetracycline eye ointment TDS.
- Pad the eye.
- Refer to eye clinic immediately.

## 52.11 Chalazion

This is an inflammation of the meibomian glands of the eyelid that typically forms a granulomatous inflammatory mass.

**Clinical Features**
The affected patient complains of eye discomfort. Typically there is a hard, painless eyelid swelling away from the lid margin.

**Management**
- Incision and drainage.

## 52.12 Painful Red Eye

A condition that should not be underestimated and one that signifies some underlying inflammatory process. A good history and physical examination may aid in identifying the primary cause. It is important to rule out emergency ophthalmic conditions and refer these immediately.

**Management**

- Analgesics – Paracetamol 1g TDS.
- Refer to eye clinic especially if there is visual loss, significant trauma, and tearing.

## 52.13 Unexplained Loss of Vision

This frightening condition can have many causes, some of which are associated with poor prognosis. Obvious causes like space occupying lesions, metabolic disorders, blood disorders, and HIV/AIDS should be looked for.

**Management**

Refer to eye specialist for appropriate management.

## 52.14 Allergic Conjunctivitis

This is an immune mediated conjunctivitis that may present seasonally or without a specific pattern.

**Clinical Features**

- Itching, which may be bilateral
- Watery discharge
- Redness
- Photophobia

**Management**

**Management of this condition includes the following:**

- Application of cold compress.
- Antihistamines – sodium chromoglycate eye drops TDS.
- Add steroid eye drops to the treatment.

## 52.15 Viral and Purulent Conjunctivitis

**Clinical Features**

- Watery eye or pus in the eye
- Redness of the eye

**Management**

**Management of this condition includes the following:**

- Tetracycline eye ointment 1% 8 hourly for 7 days ***OR***
- Gentamicin eye drops 0.3% 6 hourly for 7days.

- ♦ Prevention is by good eye hygiene.
- ♦ Refer if no improvement.

## 52.16 Asthenopia (Eye Strain)

**Clinical Features**
There is normal vision but pain while reading

**Management**
- ♦ Reassurance
- ♦ If pain persists refer to an eye specialist for appropriate management.

## 52.17 Corneal Ulcers

These commonly occur and involve loss of epithelium and usually heal spontaneously. Some form of trauma is associated with these ulcers in most cases.

**Clinical Features**
- ♦ Red eye
- ♦ Photophobia (intolerance of light)
- ♦ Sensation of foreign body in eye
- ♦ Tearing
- ♦ Pain

**Management**
- ♦ Tetracycline eye ointment 4 times daily, then refer to eye clinic immediately.
- ♦ Gentamicin eye drops 2 hourly as alternative.
- ♦ Eyepad.
- ♦ Manage complications.

## 52.18 Stye

This is an infection of the follicles or tarsal glands and is localized to the eyelids.

**Clinical Features**
There is an acute painful swelling localized on the lid margin that may cause swelling of the entire eyelid. On examination ensure the underside of the eyelid is examined**.**

**Management**
- ♦ Warm water compresses.
- ♦ Tetracycline eye ointment 1% 8 hourly for 1 week.
- ♦ If no improvement within a week refer to eye specialist.
- ♦ At specialized centres – surgical drainage.

# 52.19 Eye Trauma

The eye is a delicate external organ and it is easy for it to be injured. Eye injuries are generally classified as penetrating and non penetratingand include corneal and conjunctiva foreign bodies and abrasions, burns (dry heat and chemical burns), blunt trauma (contusion), penetrating injuries to the eye ball (perforations),injuries to eyelids, orbital injuries, and cranial nerve injuries.

**A good evaluation of eye injury includes the following:**

- Checking vision for all patients.
- Good lighting and magnifying lens make eye examination easier.
- Eye examination to be carried out should be thorough, noting that. A small entry wound does not always equate to minimal injury.

**Management**

- Corneal and conjunctival abrasions:
  - Pad the eye with doxycycline eye ointment 1% for 24 hours.
  - Stain with fluorescein and then manage accordingly.

- Foreign bodies:
  - Use moist cotton swabs.
  - Remove under local anaesthesia (by a trained person) then pad the eye.
  - Apply doxycycline eye ointment 1% 3 times a day.
  - Refer to higher level if at 4 or 5 and unable to remove or if the instruments are lacking.
- Blunt trauma:

• Give analgesics.
  - Rest the eye.
  - Caution as this may be a ruptured eyeball.
  - Deal with or refer those with poor vision and/or blood in the eye (hyphaema) immediately to next level if not able to manage.
- Chemical burn:
  - Urgently irrigate the eye with plenty of water or normal saline for 30 minutes.
  - Note that washing the face is not enough.
  - Use local anaesthetic ophthalmic drugs, e.g., lignocaine 4% eyedrops.

  - Pad with tetracycline eye ointment1%.
  - Refer immediately to higher centre, preferably with an eye specialist.
  - Deal with complications or refer to eye specialist very urgently. Complications depend on the concentration of the chemical and the duration it stayed in the eye.
- Penetrating eye injuries
  - Give an injection of tetanus toxoid (IM) STAT.
  - DO NOT apply topical medications to the eye.
  - Protect the eye with a clean pad or shield.
  - Refer without delay to a resident eye specialist. Communicated directly with specialist prior to transfer.
- Lid injuries

- Dress wound.
- Give tetanus toxoid.
- Lids have very good blood supply and so healing is good.
- Stitch minor cut not involving lid margin if you are a trained person.
- Avoid distorting the lid margin.
- Refer if tissue loss and all patients injured lacrimal drainage system (nasal angle of the eye).
- Septic lacerations should be cleaned and covered with systemic antibiotics benzylpenicillin1.2 g IV 6 hourly+gentamicin 80mg IM 8 hourly for 7 days.
- Note: Refer all patients with injuries involving the lid margin.

♦ Orbital injuries
- Proptosis (protruding eye) or diplopia (double vision) suggest serious eye injury for which specialist assessment and treatment are required.
- Tetanus toxoid injection STAT should be given if there is an open wound.
- Take orbital x-ray of patients with suspected fractures of the orbit.
- Give systemic antibiotics (penicillins: amoxicillin 500mg 8 hourly or amoxicillin+clavulanic acid 625mg 12 hourly), and analgesics (paracetamol 1g orally 8 hourly and for children refer to appropriate appendix).
- Refer for specialized treatment.

## 52.20 Orbital Cellulitis

**This is a deadly ophthalmic emergency.** It present with severe periorbital pain and swelling, proptosis, spiking temperatures and restricted eye movements. **May complicate with cerebral involvement.**

**Management**

♦ Aggressive as for meningitis.
- Ceftriaxone 1g BD injection wit metronidazole 500mg IV TDS for 10days.

♦ Strict monitoring of vital signs.

## 52.21 HIV and the Eye

**Examination**

♦ Painless blurring or loss of vision.
♦ Flash lights and floating spots.
♦ Retinal haemorrhages and cotton wool spots.

**Treatment**

♦ Anti-retroviral medications (see Section 2.1.6).
♦ Intra vitreous foscanate (pellet).

# 53. Orthopaedics and Fractures

## 53.1 Fractures

- Definition – Discontinuity of bone
- Classification
  - Closed fractures
  - Open (compound) fractures

Most fractures are secondary to trauma, although pathological fractures secondary to tumours, infections, osteoporosis, and congenital deformities also occur. Fractured bone segments may communicate with wound while the skin over it is intact (closed fractures) or with the skin broken and therefore exposed to the outside (open or compound). Compound fractures are always contaminated.

### 53.1.1 CLOSED FRACTURES

The bone fragments do not communicate through the skin.

**Clinical Features**

- Pain
- Swelling
- Loss of function
- Abnormal movements/deformity/crepitus
- Signs of blood loss and neurovascular complications,e.g.,pulselessness, cold extremity, and bleeding. Always look for compartment syndromes.

**Investigations**

- Full blood count
- Group and cross match blood for fractures of major bones
- AP and Lateral radiographs of the affected bones. Some fractures may need special views, e.g., hip fractures.

**Note: For skull fractures a CT scan head is indicated to rule out intracranial injuries**

**Management**

- Give appropriate analgesic.
- Splint the fracture; this prevents soft tissue damage and also reduces pain. Familiarize yourself with the Thomas splint and how to apply it appropriately.
- Reduce fracture under sedation or general anaesthesia.
- Immobilize with plaster of paris (POP) traction, or splints, e.g., Thomas or Braun splint. (Refer to table 53.1 for the period of immobilization in plaster.)

♦ Fixation: This operative procedure can be internal or external fixation. Internal fixation is recommended for femoral fractures.
**Note:** Must check the peripheral circulation and innervation within 24 hours of plaster application. Check radiograph before removing the splint

**Table 53.1: Period of immobilization in plaster**

| | Adults | Children |
|---|---|---|
| Upper limbs | 6–8 weeks | 3–4 weeks |
| Lower limbs | | |
| Femur only for children below 6 years | | 6 weeks |
| Tibia | 8–12 weeks | 4–5 weeks |

**For all fractures it is essential to check for neurovascular complications pre and post cast application. If present, immediately split the plaster or decompress the affected compartment.**
**Hazards of POP consist of the following:**
♦ Compartment syndrome
♦ Gangrene and even loss of limb
♦ Stiffness of joint
♦ Contractures
♦ Skin reactions
♦ When POP harbours insects

## 53.1.2 OPEN / COMPOUND FRACTURE

**The treatment is as for closed fractures except that these are contaminated and the following should be done first:**
- Thorough surgical debridement (in theatre)
- Give tetanus toxoid and antibiotics
- External fixation is preferred for these fractures
- Occupational therapy and orthotic fitting
- Rehabilitation

♦ Note that delayed healing may occur duet o:
- Poor immobilization
- Poor reduction
- Poor blood supply

- Infections
- Soft tissue interposition and
- Systemic diseases
- Poor nutritional status
- Smoking and use of alcohol
- Immunosuppression

♦ Complications of compound fractures include fat embolism, neurovascular injuries, infections, joint stiffness, nonunion, mal-union, and delayed union, non-union, compartment syndrome.

## 53.2 Joint and Tendon Injuries

These injuries are usually due to sports injuries, road accidents, assault and occupational hazards. They should be handled as emergencies. They can be classified as:

- Dislocations
- Fracture dislocations
- Haemarthrosis,which may occur as a complication of any of the above injuries or may occur spontaneously as in haemophilia.
- Ligamentous injuries may occur following twisting, traction or bending forces

**Usual sites of joint and tendon injuries include:**

- **The knee:** Commonly affected are the medial and lateral, collateral, and the cruciate ligaments, occasionally the menisci.
- **The ankle joint:** This is a major weight-bearing joint and its stability depends on the surrounding ligaments. Proper diagnosis and accurate reduction is important if congruency of the joint is to be maintained.
- **The elbow:** Dislocations here occur in the posterior direction resulting from a fall on an outstretched hand. Spasm of the triceps muscle then locks the elbow in the dislocated position.

**Clinical Features**

In general joint injuries present with the following:

- Pain
- Swelling
- Loss of function
- Deformity
- Crepitus (if there is an associated fracture)
- Neurovascular complications

**Diagnosis**

This is made after clinical examination and radiology.

**Management**

Treatment of dislocation should be urgent because of possible damage to neurovascular structures.

- Analgesics and anti-inflammatory (relieve pain and inflammation)
- Splint of the dislocation/fracture
- Urgently reduce and immobilize.
- Check radiograph. If not adequately reduced repeat the procedure or perform an open reduction.
- Reduce dislocation under general anaesthesia if need be.
- Stabilize reduced joint.
- Initiate physiotherapy and occupational therapy.
- Immobilize for 2 to 3 weeks.

## 53.3 Club Foot (Typical Talipes Equinovarus)

- Description
  - Heel inverted
  - Forefoot and mid foot inverted and adducted (Varus)
  - Ankle in equinius (the foot is planterflexed with toes at a lower level than heel)
- Incidence: Club foot is one of more common congenital deformities of the foot, as indicated in Table 53.2.
  - It is bilateral in 50% of cases, and heredity plays a role:
    - Monozygotic twins: 32.5%
    - Dizygotic twins: 2.9%
  - There is rapid decrease in incidence from first to second to third degree relatives (2.9% of siblings,0.6% of aunts and uncles, and 0.2% of cousins).
  - It is thought that intra-uterine mechanical factors may also play a role.

**Table 53.2: Prevalence of club foot**

| | |
|---|---|
| Sex ratio Male: Female ratio 2:1 | |
| Race incidence: | |
| Caucasian | 1.12 cases per thousand births |
| Japanese | 0.53 cases per thousand births |
| Chinese | 0.39 cases per thousand births |
| South African Black | 3.50 cases per thousand births |
| Polynesian | 6.81 cases per thousand births |

**Management**

- Early serial splinting is important. An above the knee cast is applied with the knee in 90° of flexion. Cast is changed once to twice weekly.
- Ponseti technique of casting has a greater chance of succeeding in foot correction.
- Where available, a foot abduction splint is used for several weeks after achieving foot correction.
- Complications
  - Pressure sores due to tight casts.
  - Rocker bottom deformity of the foot.
  - Failure to achieve correction.
- Where conservative treatment fails, carryout a surgical correction.

## 53.4 Acute Osteomyelitis

This is caused by haematogenous spread of bacteria from a primary source, which may or may not be obvious. The commonest causative agent is Staphylococcus aureus. Other organisms that may be responsible include Streptococcus pyogenes, Pneumococus pneumoniae, Staphylococci albus, and sometimes Salmonella typhi in sickle cell disease.

**Clinical Features**

- Pain is the major presenting symptom. The severity increases with time.
- Fever and the patient becomes toxic.
- Localised tenderness, loss of function of the limb, and swelling. Commonly involved bones are proximal tibia, distal femur, and distal humeri.

  **Note:**
- The clinician needs to have a high index of suspicion for this condition, especially in children following a minor fall.

**Investigations**

- Haemogram: A leucocytosis will be demonstrated.
- Radiograph of affected limb may not show any changes in the early stages; peritosteal elevation is a late feature (2–3weeks).
- Blood cultures and sensitivity.
- Sickling test in suspected cases of sickle cell disease.
- Pus culture and sensitivity.

**Management**

- Analgesia to relieve pain.
- Elevate and rest the limb.
- Administer appropriate parenteral antibiotic therapy for 3weeks:
  - Flucloxacillin: 50–100mg/kg per day IV 6 hourly ***OR***
  - For MRSA (methicillin resistant Staph.aureus): Parenteral vancomycin 12 hourly.
- If there is any indication that the situation is not changing or is deteriorating within about 24 to 48 hours.
- Perform surgical drainage if fever and tenderness persist after 24 hours of appropriate antibiotic therapy and pus is present. Always submit pus for cultures.
- Where fractures occur, manage appropriately.
- Address issues related to primary cause if possible.

## 53.5 Chronic Osteomyelitis

This follows inadequate management of acute osteomyelitis, infected compound fractures, spread from infected tissue including prosthesis, and bone surgery.

**Clinical Features**
Infection may remain quiescent, with acute or sub-acute exacerbations that manifest as discharging sinuses.

**Investigations**
As for acute osteomyelitis.

**Management**
- Antibiotic therapy, as per culture/sensitivityresults for a minimum period of 6 weeks.
- Surgical drainage, sequestrectomy, and irrigation as indicated.
- Any complications manage appropriately

## 53.6 Septic Arthritis

**This is an acute infection of the joint space.**

**Aetiology**
- Haematogenous spread from a primary focus elsewhere in the body.
- Direct penetrating injuries into the joint.
- Extension of infection from a compound fracture of the neighbouring bone.
- The commonest causative organisms are Staphylococcus aureus, Streptococcus pyogenes, and Haemophilias influenza, and to a lesser extent Salmonella typhimurium or typhi.
- Large joints such as shoulder, knee, ankle, and hip are more often affected.
- Septic arthritis is most common in children under 3 years of age.

**Clinical Features**
- Fever, chills and irritability
- Swollen, warm, very tender joint(s)
- Pseudoparalysis of the joint
- Multiple joints may be affected

**Investigations**
- Full haemogram – Anaemia and leukocytosis present
- Pus for culture and sensitivity
- Radiograph of the affected joint shows increased joint space, synovial thickening, and later rarefaction of the adjacent bone surfaces.

**Management**
- Admit the patient
- Start on broad spectrum antibiotics.
- Give appropriate analgesics and antibiotics

- Splint the joint and initiate physiotherapy.
- Aspirate the joint and send speicime for culture and sensitivity. If there is frank pus then perform urgent arthrotomy.
- Review daily until discharge.
- Review monthly after discharge.
- Rehabilitate as necessary

**Note:**

- Watch for features of a worsening condition, which include the following:
  - The fever persists for more than 7 days of full treatment.
  - The joint swelling does not subside within 3 weeks.
  - New joints get involved while on treatment.
  - As much as possible, refer the patient to an appropriate facility before the following complications have developed, or refer immediately if they present with any of these complications:
  - The affected joint starts to discharge pus spontaneously.
  - Shortening of the limb occurs.
  - There is persistent deformity of the joint.
  - There is loss of function related to the infection.



## 53.7 Osteogenic sarcoma

This is a highly malignant bone tumour of late childhood and early adulthood. Commonly involves long bones, i.e., distal femur and proximal humerus. This tumour presents with pain, noticeable swelling, tenderness, or pathological fractures.

**Investigations**

**X-ray affected limb:**

- Radiological findings show periosteal elevation with new bone formation (Codmann's triangle), sunray appearance; chest radiograph may show metastatic lesions.
- Ct scan chest in metastatic disease.

**Management**

Multidisplinary team approach

## 53.8 Lower Back Pain

**Aetiological factors**

- Trauma
- Inflammatory, e.g., rheumatoid arthritis, ankylosing spondylitis,etc.
- Degenerative: Spondylosis (degenerative disease), prolapsed intervertebral disc,spondylolisthesis
- Neoplastic: Usually secondary tumours
- Infection: Pyogenic, non-pyogenic (Tuberculosis – Pott'sdisease)
- Spinal stenosis: Congenital, degenerative
- Others: Kyphoscoliosis

**Clinical Features**

- Pain
  - Sharp and localized, chronic and diffuse
  - Referred pain(sciatica): Pain radiates into the lower limb, may be aggravated by coughing, straining,etc.
  - Stiffness
  - Deformity, e.g., TB spine

- Numbness or parasthesiain the lowerl imb
- Urinary retention or incontinence (can be due to pressure on cauda equina)
- There may be history of trauma, heavy lifting, neoplasm, connective tissue disorder like rheumatoid arthritis.
- Physical findings at presentation are demonstrable by:
  - Inspection
  - Skin–may show scars, pigmentation, abnormal hair.
  - Shape and posture maybe abnormal and suggestive.
  - Palpation
  - Feeling for tenderness is likely to elicit it.
  - Motion – May be impaired.
  - Sensation – May be diminished if nerves are involved.
  - Reflexes – May be diminished if nerves are involved.
  - Straight leg raising test - Discloses lumbosacral root tension.
  - Examining the other systems.

**Investigations**

- Plain radiographs: Anteroposterior, lateral, and oblique views of spine may show:
  - Osteophytes and disc degeneration in spondylosis
  - Loss of lumber lordosis, which signifies muscle spasm due to pain
  - Anterior shifts of an upper segment on a lower segment which indicates spondylolisthesis.
  - Bone destruction with sparing of intervertebral discs is noted in tumours
  - Sclerotic metastases are seen in cancer of the prostate
  - Bone destruction in infective conditions,e.g.,TB.There may be a gibbus (sharp angulation) deformity.
  - Fracture in traumatic cases.
- CT scan: May pick up structural bone changes,e.g.,fracture, tumour, and intervertebral discs prolapse.
- Magnetic resonance imaging (MRI): Discs, nerves, and other soft tissues are clearly seen.
- Other investigations include:
  - Those based on the likely working diagnosis,e.g., abdominal ultrasound in suspected tumours.
  - Erythrocyte sedimentation rate (ESR) in suspected tumour, TB, connective tissue disease.

**Management**

Most cases of disc prolapse will improve on conservative management.

- Give analgesics to control pain.
- In suspected tuberculosis, diagnostic test are readily available to confirm a disganosis. Treat with anti-tuberculosis drugs.
- Initiate physiotherapy for spondylosis.
- Spondylolisthesis give complete bed rest for 2 weeks and physiotherapy.

- Stable fractures will heal conservatively on bed rest (orthopaedic bed). A hard lumbosacral corset may be fitted after 6–8 weeks and used for a further 4–6 weeks or until the pain is bearable.
- For unstable fractures do decompression and stabilization.
- In suspected tumours involve multidisplinary team approach.

# 54. Ear, Nose, and Throat Conditions

## 54.1 Epistaxis

This is where there is bleeding through the nose due to nose picking, trauma (fall in games, assault, etc.), nasal and paranasal neoplasms. It could also be triggered by a nasal infection, systemic derangements, e.g., acute fevers, hypertension, renal disease with uraemia, abnormalities of blood clotting, and foreign bodies in the nose.

**Management**

- Immediate: Sit the patient up (to avoid aspiration).;
- Pinch the nose for 10–20 minutes. This is usually sufficient to stop bleeding.
- Apply ice or cold packs on the bridge of the nose.
  - To pack the nose, remove clots as aspirate. Apply lignocaine nasal spray 4%, then pack (preferably using Tiley's forceps) with ribbon gauze or narrow strip of gauze impregnated with liquid paraffin. Start packing from the floor of the nose towards the roof. The pack should fit lightly to be effective. Do not use adrenaline.
- Remove paraffin pack within 24-48 hours.
- Put a patient with a nasal pack on:
  - Broad spectrum antimicrobial, e.g., cotrimoxazole or amoxicillin for 7days.
  - Analgesic, e.g., paracetamol 500mg 8 hourly for 5 days (children 40mg/kg/day QDS).
- Attend to primary cause. Patient may require in patient treatment of the underlying causative factor. Treat the underlying cause and provide additional treatment with cautery or endoscopic therapies.
- Admit the patient if fluid replacement or blood transfusion is required.
- In an adult, the cause should be identified as epistaxis is a more sinister sign in an adult. Rule out malignancy.

## 54.2 Foreign Bodies in the Ears

The types of foreign bodies include metallic pieces (hair clips, smooth pellets, needles,etc.),wooden items(e.g.,matchsticks),vegetable matter like seeds, and insects.

**Clinical Features**

- Obvious history of foreign body insertion into the ear, conductive deafness, pain or discomfort in ear. Discharging ear, disturbing noise (insects), and bleeding (traumatic insertion especially by a child).
- Danger signs: Foreign bodies in the ear with bleeding from the ear and external evidence of trauma suggest foreign body entry into the middle ear.

**Management**

- Analgesia: Paracetamol 7.5mg/kg body weight orally 4–6hourly if painful.

- Most foreign bodies can be removed fairly easily with crocodile forceps, a hook, an ear probe or by suction and gentle syringing with warm, clean water.
- Rounded objects may rupture the ear drum if pushed further into th eear. Refer these cases.
- If a complication such as perforation of the eardrum or a foreign body in the middle ear is suspected,refer.
- Removal, especially if general anaesthesia needed as in some children.
- Examine patient for possible other pathologies.
- If you lack the instruments for extraction of foreign bodies, please refer.
- Complications:
  - Conductive deafness.
  - Vegetable matter is hygroscopic, thus it is advisable not to syringe as this can lead to inflammatory reaction in the canal walls resulting in otitis externa.
- See Section 27.6.1 for more details.

## 54.3 Foreign Bodies in the Nose

This is covered under Section 27.6.2 in these guidelines.

## 54.4 Foreign Bodies in the Oesophagus

The commonest objects are fish bones or meat in adults. The commonest objects encountered in children are coins. All other forms of foreign bodies can be found in psychiatric patients.

**Clinical Features**

There is pain in retrosternal area and/or in the back, dysphagia, pooling of saliva in the mouth, or regurgitation of food. The affected patient may present with dyspnoea and hoarseness if there is laryngeal oedema from compression by the foreign body and localized tenderness in the lower part of the neck. As a number of children are not able to communicate their problem, child may present later with complaints relating to the presence of a foreign body.

**Investigations**

Plain radiographs, antero-posterior and lateral views, may show opaque objects. Radiolucent objects are not seen on radiographs. However, an increase in the prevertebral soft tissue exceeding 1/3 of the antero-posterior distance of the patient's vertebral body is highly suggestive of the presence of a foreign body.

**Management**

- Removal.
- For sharp sided foreign bodies, refer immediately if not equipped to deal with them.
- Oesophagoscopy and removal of the foreign body.
- Other surgical procedure(s) should this fail.

## 54.5 Foreign Bodies in the Laryngotracheobronchial Tree

More common in children. The common objects are vegetable seeds (animate) and beads (inanimate).

**Clinical Features**

- Child has a sudden attack of cough, choking, and wheezing (a high index of suspicion is needed.)
- The child may present with stridor and/or dyspnoea.
- Auscultation may reveal reduced air entry on one side of the chest.

**Investigation and Management**

- Chest radiograph.
- Bronchoscopy for diagnosis and removal of foreign body.
- For long-standing cases, refer forsurgery.

## 54.6 Hearing Impairment

A high index of suspicion and proper history is important, especially among children born prematurely, those born with low birth-weight, those born after difficult delivery, those who develop yellowness of eye (neonatal jaundice), those whose mothers had febrile illness during pregnancy, and those treated for meningitis. Do not ignore parents' complaint of a child not hearing or a child who is slow to develop speech. Use different voice intensity to assess grossly the state of hearing. If hearing loss is suspected, refer at whatever age to higher level for appropriate management. A child with a hearing impairment can be helped at any age, but the earlier the better.

Refer to an institution specializing in dealing with hearing impairment with facilities for audiometry, tympanometry, and rehabilitation.

## 54.7 Mastoiditis

This is an infection of the mastoid air cells and mastoid bone occurring as a complication of acute otitis media or chronic suppurative otitis media.

**Clinical Features**

There is a painful swelling above the ear in children under 2 years of age. There is tenderness, oedema, and possible flatulence behind the ear in other children, often with preceding otitis media and mastoid tenderness. There is fever and sagging of the posterosuperior meatal wall. Complications include squint or facial nerve palsy on the same side as the mastoiditis.

**Management**

- Admit.
- Give antibiotics as for otitis media. Intravenous co-amoxyclavulin 1.2g IV BD or cefuroxime sodium 750mg TDS.

- If the swelling points and/or bursts to discharge pus, refer to higher centre as this condition requires a formal mastoidetomy to adequately clear all the pus and infected material. Inadequate incision and drainage will result in a chronic sinus.
- Manage appropriately if child develops signs of meningitis (seeSection13.4, meningitis) or brain abscess or other complications.

## 54.8 Laryngeal Trauma

These can be blunt or penetrating injuries. The priority is to secure and maintain an airway, then refer urgently to an ENT specialist for endoscopy and repair.

## 54.9 Allergic Rhinitis

Immunoglobulin IgE-mediated rhinitis is characterized by seasonal or perennial sneezing, nasal congestion, pruritus, and often conjunctivitis and pharyngitis. Symptoms vary in severity from day today or hour to hour.

**Management**

- Avoid/remove the allergen (precipitating factor).
- Give antihistamines: Chlorphenamine 4mg 6 hourly adults and 0.35mg/kg in children in 4 divided doses.
- Give topical steroids, as these are safe and effective: Budesonide nasal spray two puffs per day.
- Refer to a higher level if the following present:
  - Gross nasal obstruction (hypertrophied inferior turbinates).
  - Polyps.
  - Chronic sinusitis.
  - Deviated nasal septum.
- Give systemic steroids in severe cases for 7 days, then taper off. Dose: prednisone or prednisolone 10 mg TDS to start and taper off gradually with time.
- Carry out any corrective procedure as may be necessary, e.g., turbinectomy.

## 54.10 Parotid Mass

These may be true parotid swellings (e.g., parotitis, parotid abscess, cysts, dialecticism, tumours, etc.), or pseudoparotomegaly due to swellings in nearby structure (e.g., hypertrophy of the masseter, jaw swellings, parapharyngeal masses, lymph enlargement, facial nerve tumours, etc.). Parotid swellings may also occur in other systemic conditions(e.g.,malnutrition,diabetesmellitus,HIV/AIDS, Sjogren'ssyndrome).

Infective masses may be associated with other features of infection like fever and pain. Further, there may be local inflammation or discharge from the opening of the parotid duct. Most parotid swellings are painless unless infected or malignant. Presence of facial nerve palsy is highly suggestive of malignant process.

**Investigations**

- Haematological tests,e.g.,white blood counts, erythrocyte sedimentation rate, serum protein, HIVantibodies.
- Fine needle aspirate (FNA) for cytology.
- Open biopsy is contraindicated because of:
  - Risk of seeding of tumour in neoplastic conditions.
  - Risk of injury to the facial nerve or its branches.
- Should FNA report not be conclusive, then superficial or total parotidectomy (depending on suspected condition) is needed. Always look out for neurovascular complication.
- Radiology:Plain radiographs may show radio-opaque stones in the ductor gland, but these are rare in the parotid gland. They are commonest in the submandibular gland.
- Sialography may be done to confirm sialectasis.
- CTscan will show the extent of the mass and its relation to other structures, and is an essential pre operative investigation.

**Management**

- Viral parotitis may not require more than analgesics and bed rest. In the presence of bacterial infection, clindamycin is the antibiotic of choice.Give 3–6mg/kg 6 hourly in children and 150–300mg 6 hourly in adults for 10 days or 450mg QID in severe cases.
- Where an underlying systemic disease is the causative factor for parotomegaly, manage the condition as appropriate.
- Surgical intervention as may be required.

## 54.11 Acute Otitis Media

This is covered in Paediatrics, Section 27.1, of these guidelines.

## 54.12 Chronic Suppurative Otitis Media (CSOM)

There are 2 types of CSOM: Tubo-tympanic and attico-antral.

### 54.12.1 TUBO-TYMPANIC TYPE

There is discharge of pus from one or both ears for more than 2 weeks following untreated or unresolved acute otitis media with a central perforation. There is recurrent ear discharge usually after URTI. Secondary infection may be present with Gram-negative organisms, yeast, and fungi.

**Clinical Features**

A purulent discharge from the ear for more than 2 weeks, usually not foul smelling. There is impaired hearing with a central perforation in the ear drum.

**Management**

- Admission is NOT necessary.
- If no antibiotics were administered recently, treat with antibiotics as in acute otitis media.
- Dry the ear by wicking, and show the mother how to do this:
  - Roll a piece of clean absorbent cloth or cotton wool into a wick on an applicator and insert it in the child's ear gently,
  - Roll the wick in the ear, then remove it and replace it with a clean wick.
  - Watch the mother repeat this until the wick is dry when it comes out.
- Instil local antibiotic ear drops, e.g., ciprofloxacin ear drops.
- Tell the mother to continue to dry the ear b ywicking at home atleast 4 times a day until the wick stays dry. Tell her to observe strict ear hygiene and use cotton wool balls when washing.
- Reassess the child weekly. If the mother needs assistance in keeping. The ear dry, reassess more frequently.
- Refer if:
  - The patient develops mastoiditis (see Section 6.1.mastoiditis).
  - There is no improvement after 4 weeks.
  - The patient will benefit from tympanoplasty surgery.
  - The patient has attico-antral type of CSOM.
  - Patient complains of headache, earache, vertigo, or facial paralysis, which indicate complications.

### 54.12.2 ATTICOANTRAL

**Clinical Features**

There is foul smelling discharge and hearing impairment with attic or marginal perforation with cholesteatoma.

**Management**

Do not syringe such ears. Refer to ENT for management.

## 54.13 Ear, Nose and Throat Manifestations of HIV/ AIDS

**An estimated 40% of AIDS patients present with otolaryngological symptoms. These include:**

- Infections: These can be viral, bacterial, or fungal, e.g., rhinitis, sinusitis, pharyngitis, glossitis, tonsillitis, laryngitis, parotitis, deep neck space cellulitis and abscesses, otitis externa, otitis media, and labyrinthitis.
- Tumours: There is an increase in head and neck cancers associated with HIV/ AIDS, especially Kaposi's sarcoma, lymphomas, squamous cell carcinoma and salivary gland tumours.
- Other features: Adenoid hypertrophy, oropharyngeal and oral ulcers, atrophic rhinitis, lymphadenopathy, parotid cysts, otitis media with effusion, vertigo, deafness, tinnitus, and cranial nerve palsies.

**Management**

Is directed at the presenting lesion.

## 54.14 Tracheostomy

This is an artificial opening into the trachea through the neck in order to by-pass an obstruction of the airway and/or to provide access to the lower airway to facilitate ventilatory support. This procedure should only be performed at level 4 and above.

**Indications for this procedure include:**

- Emergency tracheostomy: Foreign bodies (in the upper airway), maxillofacial trauma (patient cannot breath and endotracheal intubation impossible), inflammatory conditions such as epiglottis, Ludwig's angina, retropharyngeal and other oropharyngeal abscesses with respiratory obstruction, tumours of head and neck with acute obstruction to airway (due to oedema, bleeding, infection, etc.).
- Elective tracheostomy (ventilation likely to continue for more than 2 weeks): Surgery for tumours of head and neck, major reconstructive facial surgery, prolonged ventilatory support surgery, e.g., in flail chest, acute respiratory distress syndrome, pneumonia, Guillain-Barre syndrome.

**Methods**

- In case of complete acute upper respiratory tract obstruction:
  - Give oxygen through a big bore needle or a cannula inserted through crico-thyroid membrane(Cricothyrotomy).
  - Quickly extend the neck over a rolled up towel or pillow.
  - Feel for the cricoid prominence (Adam's apple) and the depression just distal to its membrane.
  - Insert a big bore needle or cannula to the trachea (with or without local anaesthetic depending on circumstances).
- Tracheostomy technique:
  - Ideally performed in theatre, with patient properly cleaned and draped.
  - Position patient supine with neck extended over a pillow and head stabilized in tracheostomy position.
- Anaesthesia:
  - General anaesthesia through a tracheal tube if possible.
  - Local anaesthesia (lignocaine 1% with adrenaline), in extreme circumstances.
- Incision and fixing of endotracheal tube:
  - Transverse incision, 2cm below the lower angle of cricoid cartilage. Incision made through the skin, subcutaneous fat, anddeep cervical fascia.
  - Blunt dissection, then expose the anterior jugular vein, infrahyoid muscles, and occasionally thyroid isthmus ( which should be ligated and divided).
  - A cruciate incision or a circular window is then made through the third and fourth tracheal rings.
  - A tracheostomy, endotracheal, or other tube is then inserted.
  - The skin incision is closed loosely around the tube.
  - Fix the tube securely with well tied tapes.

NB Use as short a time as possible through this simple procedure. Humidification of the gases/air and frequent suction through the tube must be done. When a clear passageway has been established and ventilation restored, refer the patient. For continued care of the tracheostomy, decannulation, etc., refer to a relevant textbook for detail.

## 54.15 Nasopharangeal Carcinoma

**Clinical Features**

It first commonly presents as neck mass. As a general rule of thumb, any mass in the angle of the mandible can be assumed to be nasopharangeal carcinoma until proved otherwise. Other non specific symptoms may include congestion, rhinorrhea, epistaxis, and ear pain, to mention a few.

**Investigations**

- Chest radiograph
- CTscan
- Neck ultrasound
- At level 6: Endoscopy +/- tracheostomy and biopsy

**Management**

- Radiotherapy alone
- Surgery – Laryngectomy with radiotherapy

## 54.16 Carcinoma of the Larynx

**Clinical Features**

- Commonly first presents as neck mass. As a general rule of thumb any mass in the angle of the mandible can be assumed to be nasopharangeal carcinoma until proved otherwise.
- Non-specific symptoms may include congestion, rhinorrhea, epistaxis, ear pain, and others.

**Investigation**

- Endoscopy and biopsy
- CT and MRI to evaluate spread of disease
- Ultrasound

**Management**

Management at level 4–5:

- Refer all cases to a level 6 for further evaluation and management.
- Receive all cases referred back from level 6 for followup care as per prescribed schedule.

**Management at level 6:**

- Radiotherapy mainstay of treatment.
- Prognosis particularly poor when evidence of extensive spread present like cervical nodes of basal skull invasion.

# 55. Referral Systems for the Surgical Patient (Hospitals)

An efficient, smoothly operating pyramidal referral system is essential for the effective management of surgery patients. This is especially important in an emergency situation so as to provide rapid and effective surgical treatment to the patients

Referral systems can be 2-way; upward referral and downward referrals. Upward referral seek specialists and sub-specialist referral services or in a few cases referral out of the country. Downward referral is made to the local health facility nearest to the patient's home environment and best able to cope with the patient's needs. An efficient referral system ensures that the mix of patients admitted in health facilities countrywide is appropriate for the different health facilities. Beside referral between facilities, there are also referrals within institutions that are equally important to patient's wellbeing.

All referrals must be directed to the correct facility while maintaining the normal pyramidal referral system of flow within the health system as much as possible.

For surgical conditions presenting at level 2 and 3 that are their capacity, referral to the county hospital level with the surgeon should be initiated. Hospitals should take on cases they are able to handle. Where they cannot manage, they must refer to the next appropriate facility. All referrals must be carefully evaluated and the risks and benefits assessed critically before the decision to refer is made. On completion of treatment at the higher centre, there will be a need to refer the patient back to the initial facility or to rehabilitation. The basic guidelines for upward referral are as follows and will vary a little depending on the level in question.

## 55.1 Procedure for Upward Referral

1. Make the decision to refer on the basis of critical evaluation:
   a. Individual doctor decision (team leader)
   b. Team decision
   c. Ministry or other body making decision
2. Prepare documentation to accompany the patient:
   a. Admission details
   b. Diagnostic details and investigations carried out
   c. Medications and treatments initiated
   d. Reason for the transfer
3. Communicate with receiving unit, casualty, or clinic
4. Communicate with relatives
5. Prepare appropriate transportation
   a. Efficient and reliable for the job
   b. Exclusively allocated for the job
6. Appoint an appropriately qualified escort
7. Check on resuscitation equipment to accompany patient

## 55.2 Procedure for Downward Referral

1. Make the decision to refer.
2. Prepare documentation detailing:
   a. Admission identification details
   b. Final diagnosis
   c. Procedures carried out
   d. Medications
   e. Follow-up details, any rehabilitation requirement,etc.
3. Ensure that there are 3 legible copies of the referral note, 1 for the patient, another for the receiving unit, and third copy for the file.
4. In case of terminal disease, involve the hospice in the case.
5. Communicate with the receiving unit as appropriate and provide feed back as appropriate. This as a valuable impact on improvement of services.
6. Communicate with relatives.
7. Prepare appropriate transportation.
8. Appoint appropriately qualified escort .Usually the relatives will suffice.
9. Book patent for review in SOPC or ensure follow up in receiving unit.

## 55.3 Procedure for Internal Referral

An institutional referral system needs to be clear and functional. Such a system is just as important as patient care. Each facility should have a system for both the upward and downward flow of patients to mirror the national level. A simple guide for institutional referral system would include:

- Conduct casualty department review: Unit referrals and admission decisions are made here.
- Make a correct diagnosis.
- Place appropriate unit on call.
- Ensure patient is reviewed & handed over to the unit on-call doctor
- Ensure documentation accurate.
- Decide whether to treat as outpatient or to admit.
- If admission, admit and ensure handed over to the admitting ward doctor.
- If for outpatient treatment, ensure correct referral is made.
- NB:In event of incorrect clinic referral, the doctor should be responsible for correcting this error, not the patient.
- Refer to specialized clinics if not admitted/National Referral Centre (Team leader decision)

## 55.4 Constraints to an Effective Referral System

All team members at all levels need to be conscious of the dangers that face a coordinated referral system. Effort needs to be made to avoid the following dangers to the referral system

1. Lack of confidence in the facility by the community and tendency to by pass facility to the nearest suitable
   a. Poor community relationships
   b. Poor manpower utilization
2. Infrastructure that is non-functional

a. Strengthening of the middle level facilities
b. Ensuring communication related infrastructure in place
c. Treating patients at inappropriate level
d. At higher level a disease costs more to treat so no money for supplies,etc.

3. Poor communication within and with the outside of the facility
   a. Ensure management practices are improved within facility
   b. Involve the community in services
4. Lack of or poor utilization of human resources
   a. Brain drain
   b. Poor distribution of staff
   c. Frustration in the work place
   d. Need for better working relationships
5. Lack of drugs and other equipment
   a. Issues of finance andplanning
6. Accurate diagnosis and treatment plans
   a. Training of personnel

**Referrals should be respected.**

# 56. Disaster Management

A major disaster is a situation where the number, type and severity of injuries require extra-ordinary arrangement by the hospital to cope with. These include road accidents, train accidents, airline, boat and ferry accidents, factory fires and bomb blasts.

## 56.1 Requirements for a Disaster Plan

Every hospital should have in place (and periodically test) a plan for handling major emergencies. The plan should make provisions for:

- Immediate mobilization of a designated disaster team headed by a Team Leader
- Arrangements for emergency equipment and drugs
- Transport
- Communication equipment

The plan is carried out on multiple levels, including pre-hospital organization (e.g., at the scene), hospital organization (ensuring all systems are geared up to cope with an influx of injured), and other aspects of disaster management.

### 56.1.1 PRE-HOSPITAL ORGANIZATION

Important activities:

- Crowd control.
- Security and safety for the team and victims.
- Preliminary assessment of the casualties – Triage starts here.
- Transport to various medical facilities depends on the number of casualties and availability of facilities.
- The triage sieve.

### 56.1.2 HOSPITAL ORGANIZATION

The key to success of the management of a major disaster is command and control. Each facility needs to establish an effective control centre staffed by senior medical, nursing, and administrative coordinators with appropriate support staff.

### 56.1.3 THE TRIAGE SIEVE

This a flow chart that will assist you to identity the priority patients and respond appropriately in a disaster situation. Action steps are itemized in Section 56.2.

### 56.1.4 WHAT TO CONSIDER WHEN CHOOSING THE TRANSPORT

Transport to various medical facilities depends on the number of casualties and availability of facilities. Considerations include:

- Capacity: A bus may be more suitable for a large number of “delayed” priority casualties.

- Availability: Save the ambulances for the seriously injured.
- Suitability: Do you need a wheeled or a tracked vehicle? Is a helicopter more suitable?

### 56.1.5 WHEN YOU HAVE LOADED A PATIENT

Move to an appropriate hospital (are you going straight to a specialist centre?)
Observe in transit. What equipment do you need?
Verify the treatment before departure (do you have enough oxygen, fluids or analgesics.
Escort if necessary – Doctor, nurse or paramedic.



## 56.2 Triage Sort

**Actions on receiving notification of a "Major Incident Declared – Activate Plan" Coordinators meet and establish the control centre, if there is no prior warning.**

**The medical-incident officer is dispatched to the scene to:.**

- Liaise with the ambulance service about the details and status of the incident.
- Establish whether mobile medical teams are required.

- Disaster protocol is made available to all hospital personnel.
- The necessary supplies for emergency response are made available including the interagency emergency kit.

**The Team Leader and coordinators:**

- Collect the teams, ensure the members are properly clothed and equipped, and dispatch them to the scene.
- Establish a triage point.
- Clear the accident and emergency department of existing casualties and prepare for the reception of casualties.
- Warn theatres, the intensive care unit, pharmacy, laboratory service, x-ray service, and outpatient department about the possible disruption of activities; ask the intensive care unit to clear beds if possible.
- Establish an accurate bed state.
- Designate a ward for reception of admitted casualties and start emptying it of existing patients.
- Call all off duty staff.
- Organize staff as they arrive.
- Make the disaster protocol available to all hospital personnel.
- Make the necessary supplies for emergency response should be made available, including the interagency emergency kit.

**Each member of the disaster team–no matter how insignificant the involvement must be crystal clear about their role during the execution of the disaster plan.**

## 56.3 Triage Activities

The most surgically experienced person should triage (grade) the casualties:

### 56.3.1 TRIAGE I

Patients who have life threatening injuries such as penetrating chest or abdominal wounds, head injuries, or hypovolaemic shock. These are patients who can be saved by way of urgent surgery.

### 56.3.2 TRIAGE II

Patients who have such severe injuries that they are likely to die anyway.

### 56.3.3 TRIAGE III

Patients who have only minor injuries and will probably recover even if treatment is delayed. Operate this group last.

**The decision as to what to do with each patient is made by the triage officer. This is a continuing process and patients are reassessed regularly.**

# PART IV: Obstetrics and Gynaecology and Related Disciplines

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# 57. Gynaecology

This section mainly involves the cohorts of pregnant women and the newborn, adult women of reproductive age (WRA), postmenopausal women, and infants and children in relation to sexual assault.



## 57.1 Abortion (Miscarriage)

The old working clinical definition of abortion denotes the termination of pregnancy before the 28th week of gestation. With advancement in modern neonatology the technical definition denotes termination of pregnancy to a foetus weighing less than 500g. There are several types and clinical stages of abortion, as summarized in Table 58.1.

### 57.1.1 THERAPEUTIC ABORTION

This is where the health of the mother and/or foetus is at risk, therapeutic abortion may be performed if recommended by two senior and experienced doctors as per the Penal Code section 240 and the Medical Practitioners and Dentists Council Code of Ethics and Professional Conduct 2003. These are excerpted in the box below.

— The punishment for unlawful termination of pregnancy is provided for in Penal Code sections 158, 159, and 160.

### 57.1.2 UNSAFE ABORTION

WHO defines unsafe abortion as a procedure for terminating an unwanted pregnancy either by persons lacking the necessary skills or in an environment lacking the minimal medical standards or both. Illegally induced unsafe abortion by mainly unqualified people is associated with incompleteness, sepsis, genital and visceral injuries, and death. This is usually an obstetric emergency (Table 58.2). Investigations and management are as for septic abortion (Section 58.1.6, below).Repair of genital and visceral injuries is mandatory.

**Box 58.1: Laws and Guidelines Regarding Induced Abortion in Kenya**

**The Law and Guidelines Regarding Induced Abortion in Kenya**

***Penal Code Section 240***
"A person is not criminally responsible for performing in good faith and with reasonable care and skill a surgical operation upon any person for his benefit, or upon an unborn child for the preservation of the mother's life, if the performance of the operation is reasonable, having regard to the patient's state at the time and to all the circumstances of the case".

***Medical Practitioners and Dentists Board Code of Ethics and Professional Conduct 2003***
"The Laws of Kenya do not allow for termination of pregnancy 'on demand' and severe penalties are meted out to those found guilty of procuring or attempting to procure an abortion or miscarriage. There is room, however, for carrying out termination when in the opinion of the attending doctors it is necessary in the interest of the health of the mother or baby. In these circumstances, it is strongly advised that the practitioner consults with at least two senior and experienced colleagues, obtains their opinion in writing, and performs the operation openly in hospital if he considers himself competent to do so in the absence of a gynaecologist. In all cases of illegal termination of pregnancies, the sentences shall be suspension or erasure".

## 57.1.3 THREATENED ABORTION

**Clinical Features**
As shown in Table 57.1.

**Investigations**
- Haemogram, blood group and cross matching
- Blood slide for malaria parasites in endemic malarious areas
- Urinalysis and microscopy
- Ultrasound examination to exclude "Blighted Ovum" or hydatidiform mole, and is reassuring if normal intrauterine pregnancy is seen
- VDRL

**Management**
- Order bed rest at home or in facility.
- For pain, offer hyoscine butylbromide 20mg TDS and/orparacetamol 1g TDS for 5days.
- Sedate with phenobarbitone 30mg TDS for 5 days ***OR*** diazepam 5mg TDS for 5 days, to help allay anxiety and enforce bed rest.
- Evacuate uterus if more bleeding and signs of progression to incomplete abortion occur.

**Patient Education**
- If on bed rest at home, return to health facility if features of progression to incomplete abortion intensify, e.g., more bleeding.
- Abstain from sexual intercourse for atleast 2 weeks to prevent progression to incomplete abortion and risk of infection.

**Table 58.1: Diagnosis and management of various types and stages of abortion**

| Types of abortion | Diagnosis | Management |
|---|---|---|
| Threatened abortion | Mild abdominal pain and mild PV bleeding<br>Cervix closed | Bed rest<br>Mild sedation Follow up<br>Treat any underlying cause |
| Inevitable abortion | Abdominal pains PV Bleeding<br>Cervix open<br>All POCs still in uterus | Expedite expulsion by oxytocin 20IU in 500ml normal saline drip to be run over 4 hours ***OR***<br>Misoprostal 600µg per vaginum if greater than 14 weeks (include on EML) Evacuate if less or some POCs retained after expulsion<br>Give antibiotics as appropriate |
| Incomplete abortion | Abdominal pains<br>PV bleeding Cervix open<br>Some POCs retained | Evacuate uterus by MVA under paracervical block (2.5ml of 1% Lignocaine HclInj at 2, 4, 8, and 10 o'clock positions).Ensure it is not intra- vascular *OR* misoprostrol 600µg orally<br>Antibiotics: Doxycycline 100mg BD and metronidazole 400mg TDS for 7 days.<br>Analgesia: Ibuprofen 400mg TDS for 5 days |
| Complete abortion | Little or no bleeding or pain Uterus contracted<br>Cervix closed | Observe<br>Reassure<br>Discharge |
| Missed abortion | History of amenorrhoea Symptoms of pregnancy regress, uterine size smaller than dates<br>Mild PV bleeding | Induce if more than 12 weeks Evacuate if less than 12 weeks (Ultra sound scan if available)<br>*OR* Misoprostrol 800µg orally for less than 12 weeks |
| Molar abortion | Presents as threatened or incomplete, uterine size larger, grape like vesicles Ultrasound if available | Evacuate or induce as in missed abortion.<br>X-match and drip for evacuation as excess bleeding is a risk. Strict follow up for possible choriocarcinoma.<br>Manage as per details in Sections 57.1.11 and 57.1.12. |
| Septic abortion | Any of the above with symptoms and signs of infection | Parenteral broad spectrum antibiotics Evacuate with MVA in severe cases without delay<br>Or in mild cases misoprostrol 600µg orally.<br>Manage as per details in Section 57.1.6. |
| Habitual abortion | Three or more consecutive spontaneous abortions | Treat emergency. Management depends on underlying cause; refer to Section 57.1.8. |
| Therapeutic abortion | Life threatening conditions in woman/foetus, compliance with law and MPDC guideline | Manage as per details in Section 57.1.1. |

**Table 58.2: Recommended emergency abortion care activities by level of health care facility and staff**

| Level | Staff may include | Abortion care provided |
|---|---|---|
| First referral (Level 4: county, sub-county, mission hospital, nursing home) | Nurses, trained midwives, general practitioners, specialists with training in obstetrics and gynaecology | All activities as in Table 54.1 plus: Emergency uterine evacuation through the second trimester treatment of most abortion complications, blood cross match and transfusion; local and general anaesthesia; counselling; laparotomy and indicated surgery are available |
| Diagnosis & referral for severe complications,e.g.,septicaemia,peritonitis,renal failure | | |
| Secondary & tertiary referral (Levels 5 & 6) | Nurses, trained midwives, general practitioners, obstetrics and gynaecology specialists. | All activities above plus: Uterine evacuation as indicated for all emergency abortion treatment of severe complications (including bowel injury, tetanus, renal failure, gas gangrene, severe sepsis); treatment of coagulopathy and counselling |

Source:Adapted from *Clinical Management of Abortion Complications: A Practical Guide* (WHO, 1994).

## 57.1.4 COMPLETE ABORTION

**Clinical Features**

As shown in Table 58.1.

**Investigations**

As for threatened abortion.

**Management**

- Resuscitate firt with IV fluids (normal saline and dextrose) if the patient is in shock, consider blood transfusion if necessary. Free running till the state of rehydration achieved.
- Administer antibiotics: amoxicillin-clavulanate 625g BD ***OR*** doxycycline 100mg QDS for 10 days and metronidazole 400mg TDS for 7 days.
- Give ferrous sulphate 200mg TDS and folic acid 5mg OD in standard dosage for 3 months. Ferrous sulphate should be given ***after*** completing the course of doxycycline.

**Patient Education**

- If further pregnancy is desired, investigate further as under habitual abortion (Section 57.1.8).
- If further pregnancy is not desired, discuss and offer appropriate contraception.

## 57.1.5 INCOMPLETE ABORTION

**Clinical Features**

As shown in Table 57.1 as for threatened abortion.

**Management**

- Resuscitated with fluids (normal saline and dextrose). If the patient is in shock, transfer to higher level for appropriate management.
- Give oxytocin 10 IU IM or ergometrine 0.5mg IM STAT.
- Remove POC from cervical os digitally or with ovum forceps.
- Evacuate the uterus, preferably with manual vacuum aspiration (MVA) as soon as possible under para-cervical nerve block (10ml of 2% lignocaine HCL: 2.5ml injected at 2, 4, 8, and 10 o'clock positions of the cervix). NB: The uterus can be evacuated with either MVA or medication.
  - Misoprostrol 600µg orally in a single dose will achieve completion in over 90% of the cases.
  - For pain, IM diclofenac 75mg STAT
- Give antibiotics: Doxycycline 100mg BD +metronidazole 400 mg TDS for 7 days.

**Patient Education**

As for complete abortion.

## 57.1.6 SEPTIC ABORTION

**Clinical Features**

As shown in Table 57.1.

**Investigations**

- As for threatened abortion
- Blood cultures for patients in endotoxic shock

**Management**

- Admit:
  - All cases having evidence of septic abortion
  - All patients in endotoxic shock
  - Where laparotomy is indicated.
  - Where pelvic abscess develops
- Resuscitate as in incomplete abortion.
- If presentation is late or the sepsis is severe: Give IV crystalline penicillin 3 mega units 6 hourly and IV gentamicin 80mg 8 hourly + IV metronidazole 500mg 8 hourly for 3–5 days, also, IM diclofenac 75mg 12 hourly.
- If presentation is early and sepsis is mild: Give PO doxycycline 100mg 12 hourly ***OR*** PO amoxicillin/clavulanate 625 mg 12 hourly.
- Plus PO metronidazole 500mg 8 hourly for 7 days plus PO ibuprofen 400mg 8 hourly for 5 days.
- In severe cases, evacuate the uterus with MVA soon after initial antibiotic doses. In mild cases give misoprostrol 600µg orally to achieve expulsion of the POCs.
- Once stable, then may discharge on the above oral antibiotics and a painkiller.

**Patient Education**
As in complete abortion.

## 57.1.7 MISSED ABORTION

**Clinical Features**
As shown in Table 58.1.

**Investigations**
- As for threatened abortion
- Ultrasound, where available, will confirm foetal death
- Bleeding and clotting time in case disseminated intravascular coagulopathy (DIC) has developed.

**Management**
- Admit the patient for definitive treatment.
- If more than 12 weeks, induce with the prostaglandin tabs misoprostrol 400µg per vagina. Observe for spontaneous onset of abortion process, then examine for complete abortion; if incomplete do MVA.
- Then if less than 12 weeks, evacuate the uterus with MVA or misoprostrol 800µg orally. Start on antibiotics PO doxycycline 100mg 12 hourly,PO metronidazole 400mg 8 hourly for 5 days, and PO ibuprofen 400mg 8 hourly for 3 days on discharge.
- If complicated with DIC, fresh blood transfusion or fresh frozen plasma is life- saving.

**Patient Education**
- As for complete abortion (Section 58.1.4)

## 57.1.8 HABITUAL ABORTION

**All cases of habitual abortion should be reviewed by a gynaecologist.**

**Clinical Features**
As shown in Table 58.1.

**Investigations**
- As in threatened abortion,and
- Bloodsugar
- Urine C&S
- Blood grouping
- Brucella titres
- Widal test
- Blood urea
- Pelvic U/S
- VDRL/RPR
- HIV screening

**Management**

Management depends on the cause of the habitual abortion.

- Correct any anaemia and ensure positive general health.
- If VDRL serology is positive, confirm syphilis infection with TPHA test, treat patient plus spouse with benzathine penicillin 2.4 mega units IM weekly for 3 doses. More often a single injection will suffice. In penicillin sensitivity, use erythromycin 500mg QDS for 15 days.
- Control blood pressure to normal pre-pregnant levels.
- Ensure diabetes is controlled.
- For cases of recurrent urinary tract infections, order repeated urine cultures and appropriate chemotherapy.
- For brucellosis positive cases, give doxycycline 500mg QDS for 3 weeks + streptomycin1g IM daily for 3weeks. If pregnant, substitute cotrimoxazole for doxycycline.
- Offer cervical cerclage in next pregnancy in cases of cervical incompetence.
- For cases with poor luteal function, give a progesterone early in pregnancy,e.g., hydroxyprogesterone 500mg weekly until gestational age is 14weeks.Then continue with oral gestanon 5mg TDS up to the 6$^{th}$ month.

## 57.1.9 TERMINATION OF PREGNANCY

Therapeutic abortion is termination of pregnancy for medical indications (refer also to Section 58.1.1, above).

**Method of Therapeutic Abortion**

May be surgical or medical

**Surgical:**

- After 12 weeks: MVA or EVA
- In 13$^{th}$ –18$^{th}$ week, dilatation and evacuation (D&E) after cervical priming with misoprostrol 400µg for 3 hours.
- D&E should only be performed by skilled and experienced doctors.

**Medical:**

In 13–22 weeks:

- Give mifepristone 200mg orally, followed after 36–48 hours by misoprostrol 400µg orally every 3 hours for 5 doses.
- Ormisoprostrol 400µg orally every 3 hours for 5 doses. Refer also to Table 58.3.

**Table 58.3: Medication for therapeutic abortion**

| Option | Gestational age | Mifepristone Day 1 | Misoprostol | | | Efficacy |
|---|---|---|---|---|---|---|
| | | | Dose | Route | Timing | |
| Recommended option | Up to 9 weeks | 200mg orally (one 200mg tablet) | 800µg (four 200µg tablets) | Bucally or sub lingually | Day 3 | 95–98% |
| Other options | Up to 9 weeks | 200mg orally (one 200mg tablet) | 800µg (four 200µg tablets) | Vaginally | Day 2 or 3 | 93–97% |
| | | 200mg orally (one 200mg tablet) | 800µg (four 200µg tablets) Repeat after ±7 days if not aborted | Vaginally | 6–24 hours | 95–98% |
| | Up to 7 weeks | 200mg orally (one 200mg tablet) | 400µg (two 200µg tablets) | Orally | Day 2 or 3 | 89–93% |

Sources: Ashok, 2002; Britton, 2007; Creinin, 2004; Middleton, 2005; Shannon, 2006; Tang, 2003; von Hertzen, 2003; WHO, 2000.



## 57.1.10 POST ABORTION CARE(PAC)

Unsafe abortion is common in Kenya. It is often associated with serious medical and psychosocial complications/problems. All women should have access to comprehensive quality services for the management of post-abortion complications. PAC services include resuscitation, evacuation of uterus by MVA, post-abortion counselling, education, and linkages to other reproductive health and support services. Fertility may return soon (11 days) after an abortion. It also includes community participation. Family planning services help reduce repeat unsafe abortions. Midlevel providers (nurses and clinical officers) can be trained to provide PAC.

## 57.1.11 MOLAR ABORTION (HYDATIDIFORM MOLE)

Hydatidiform mole should be managed in levels 4,5, and 6 because of its potential to progress to choriocarcinoma.

**Clinical Features**

A hydatidiform mole usually presents as a threatened or incomplete abortion. In the threatened stage, before the cervix opens, the diagnosis of hydatidiform mole is suspected if bleeding does not settle within a week of bed rest. The uterine size is larger than gestational age and foetal parts are not palpable.

Foetal movements are not felt at gestation 18–20 weeks and beyond. Features of hyperemesis gravidarum, nausea, vomiting, and ptyalism are still present and severe after 3 months. When the cervix opens, passage of typical grape-like vesicles confirms the diagnosis. Bleeding may be very heavy when a mole aborts spontaneously.

**Investigations**

- Positive pregnancy test in dilutions after 12 weeks gestation.
- Confirmation is by ultrasound.

**Management**

- Treat shock with IV fluids or blood as necessary.
- Put up oxytocin drip ( 20 IU in 500mllitre of normal saline or 5% dextrose at 20 drops per minute) for 4 hours or until drip is over and give IV antibiotics crystalline penicillin 3 mega units 6 hourly, gentamycin 80mg 8 hourly, and PO ibuprofen 400mg TDS.
- Evacuate the mole with suction curettage; after evacuation continue oxytocin drip once the patient has stabilized. discharge home on oral antibiotics (doxycycline 100mg 12 hourly and PO metronidazole 400mg 8 hourly for 5 days) and ibuprofen 400mg 8 hourly for 5 days, and advise patient to return for admission for sharp curettage after 2 weeks.
- Repeat sharp curettage to make sure all remains of the mole have been evacuated and send tissues for histology.
- Provide reliable contraception for 1 year: combined pill, e.g., levonorgestrel 150µg ethinylestradiol,3 0µg(microgynon or nordette) once daily for 3 weeks with breaks of 1 week in between is the best choice. Follow up monthly for pelvic examination and repeat pregnancy tests.

## 57.1.12 CHORIOCARCINOMA

Choriocarcinoma is confirmed while following the protocol of management of hydatidiform mole. The condition needs to be reviewed by a gynaecologist. Treatment depends on risk classification. Criteria for high risk(poor prognosis) is indicated by the following:

- Duration of antecedent pregnancy event >4months.
- Beta HCG levels >40,000IU/ML.
- Metastases to brain, liver, orGIT.
- Failed chemotherapy (recurrence).
- Following term pregnancy.

## 57.2 Ectopic Pregnancy

Ectopic pregnancy is defined as a pregnancy outside the uterine cavity. Most of such pregnancies occur in the fallopian tube. It is usually due to partial tube blockage and therefore the patient is often subfertile. There are two types: acute ectopic pregnancy and chronic (slow leak) ectopic pregnancy. Differential diagnoses for this condition include pelvic inflammatory disease (PID), appendicitis, abortion, and ruptured ovarian cyst.

**Clinical Features**

For acute rupture ectopic pregnancy:

- Amenorrhoea 6–9 weeks.
- Abdominal pain of sudden onset.
- Shock and anaemia.
- Abdominal distension and tenderness.
- Shoulder tip pain due to haemoperitoneal diaphragmatic irritation.
- Cervical excitation tenderness present.

**For chronic (slow-leak) ectopic pregnancy:**

- Abdominal pain.
- Irregular PV bleeding, usually dark blood (amenorrhoea may be present).
- Anaemia, fainting attacks.
- Low grade fever.
- Low abdominal and pelvic tenderness and possibly a mass.
- Cervical excitation present.

**Investigations**

- Paracentesis of non-clotting blood is diagnostic in acute and some chronic cases.
- Culdocentesis in experienced hands is positive with dark blood,especially in chronic cases.
- Group and cross-match blood. Haematocrit and/or haemoglobin estimation.

**Management**

—**Admit to comprehensive emergency obstetric care facility all patients suspected to have ectopic pregnancy.**

- Start IV line with saline and plasma expanders after obtaining specimen for grouping and cross-matching to treat shock.
- Perform emergency laparotomy.
- Perform routine salpingectomy of damaged tube. Make a note of the condition of the other tube and ovary in the record and discharge summary.
- Where experienced gynaecologist is available, initiate conservative management of affected tube.
- Transfuse if necessary.
- Discharge on haematinics.
- Review in out patient gynaecology clinic to offer contraceptives or further evaluate sub-fertility status.

## 57.3 Infertility

Infertility is usually defined as the failure to conceive after 1 year of sexual intercourse without contraception. It is divided into 2 categories:

- Primary: The woman has never conceived inspite of having unprotected sexual intercourse for at least 12months
- Secondary: The woman has previously conceived but is subsequently unable to conceive for 12 months despite unprotected sexual intercourse.

**Causes of infertility include the following:**

- Tubal factor: There is bilateral occlusion of fallopian tubes as a result of PID.
- Male factor: The sperm ducts are damaged as a result of previous STIs leading to abnormalities of sperm function.
- Endocrine disorders affecting the woman.
- Tropical diseases in male and female, including leprosy, filariasis, schistosomiasis, or tuberculosis.
- Cervical mucus abnormalities.
- Congenital disorders.

Any couple desiring children who do not achieve a pregnancy within 1 year of adequate exposure should have a systematic evaluation of their reproductive function. Most patients will require a detailed work-up, thus patients should be referred to a gynaecologist after a good history and examination rule out immediately treatable causes. Since infertility results from female *OR* male problems, both partners should be prepared to undergo evaluation. Diagnosis depends on:

- History from couple and individually.
- Physical examination of both partners.

**Investigations**

- Basal body temperature
- Semen alysis
- HSG for tubal patency
- Hormone assays where indicated
- Dye laparoscopy.

**Management**

- Definitive treatment depends on the cause as per the investigations above and may include:
- Counselling on sexual technique and fertility awareness
- Ovulation induction: Clomiphene citrate 50mg OD for 5 days starting from day 2–5 of menstrual cycle
- Tubal surgery
- Vas surgery
- Assisted reproduction
- Adoption

## 57.4 Pelvic Masses

Do simple screening by history and physical examination for any lower abdominal swellings, but refer to higher level for appropriate management, which may include further investigations. The differential diagnoses for pelvic masses include normal pregnancy, distended urinary bladder, uterine fibroids, pelvic abscess, tubal-ovarian mass, and ovarian cyst.



### 57.4.1 NORMAL PREGNANCY

This is easy to diagnose from menstrual history, clinical signs, and ultrasound if available

### 57.4.2 DISTENDED URINARY BLADDER

Acute retention of urine is the commonest. It is commonly associated with acute urinary tract infection in young girls and may be associated with other pelvic tumours in older women. Catheterization, urine examination (urinalysis, microscopy, culture, and sensitivity), and appropriate antibiotic based on culture will suffice in urinary tract infection (UTI).

### 57.4.3 UTERINE FIBROIDS

**Clinical Features**

Benign uterine growths may be sub-serous, interstitial, or submucous. They occur commonly in age group about 30 years and above and are associated with nulliparity, low parity, sub-fertility, and infertility. The condition presents with features of mass in the lower abdomen or dysmenorrhea or heavy periods. Vaginal examination reveals a mass that is firm, nodular, and non-tender, and moves with the cervix. Diagnosis is essentially clinical.

**Investigations**

- Haemoglobin, VDRL, blood group, blood urea,urinalysis
- IVU in selected cases
- Hysterosalpingography in subfertile and infertility cases
- Ultrasound where facilities exist.

**Management**

- Treat associated pelvic inflammatory disease: Antibiotics – amoxicillin/ clavulanate 625mg BD for 7 days ***OR*** doxycycline 100mg BD for 7 days + metronidazole 400mg TDS for 7 days. NSAIDs–ibuprofen 400mg TDS for 3 days.
- Correct any anaemia associated with menorrhagia by haematinics (ferrous sulphate 200mg TDS and folic acid 5mg OD for 1 month) or blood transfusion.
- Where fertility is desired, plan myomectomy and where obstetric career is complete, plan hysterectomy with conservation of 1 ovary in women under 45 years of age.

## 57.4.4 PELVIC ABSCESS AND TUBO-OVARIAN MASS

**Clinical Features**

Essential features for diagnosis of this condition include the following:

- History of STI or pelvic infection
- Lower abdominal and pelvic pain
- Nausea and vomiting
- Tender adnexal mass
- Fever and tachycardia
- Rebound tenderness

**Investigations**

- Haemogram, ESR
- Urinalysis
- Urea and electrolytes
- Blood sugar
- Group and cross-match
- Ultrasound
- Culdocentesis

**Management**

- Give parenteral broad-spectrum antibiotics ceftriaxone 1g BD IV +gentamycin 80mg TDS IV, metronidazole 500mg TDS IV, for 3–7 days. Then change to oral medications.
- Carry out appropriate surgery: Laparotomy and drainage/excision.
- Initiate physiotherapy.

## 57.4.5 OVARIAN CYSTS

**Clinical Features**

- These are usually benign and may occur in women of any age group. Menses are usually normal in simple cysts. Abnormal menses including amenorrhoea occur in functional cysts. Ovarian cysts may undergo torsion to cause acute pain.
- A cystic mass in one or other side of pelvis is essential for diagnosis.

**Investigations**

- Haemogram, urinalysis
- Plain abdominal x-ray may be useful in calcified tumours and some dermoid cysts
- Ultrasound

**Management**

- Cysts greater than 4cm need laparotomy.
- Cystectomy or salpingo-oophorectomy and histology.

**Patient Education**

- Annual pelvic examination and ultrasound.

## 57.5 Menstrual Disturbances

Most women suffer some form of menstrual disturbance in their lifetime. The common types are mentioned here.

### 57.5.1 AMENORRHOEA

Amenorrhoea means the absence of menstruation for 2 cycles or more. It is a symptom and not a disease. Primary amenorrhoea refers to a patient who at any age has never menstruated. Secondary amenorrhoea refers to cessation of the periods after menstruation has been established. There are 2 varieties of amenorrhoea: cryptomenorrhoea (hidden periods) and true amenorrhoea (primary and secondary).

#### CRYPTOMENORRHOEA

**Clinical Features**

The menstrual fluid is retained in the genital tract. The commonest variety seen is imperforate hymen occurring after menarche (12–14 years) with cyclic abdominal pains. Vulval inspection will reveal bluish bulging hymen. There may or may not be lower abdominal mass.

Management

- Admit to hospital for cruciate incision, which is a cure for imperforate hymen.
- Give antibiotics and analgesics (PO amoxicillin/clavulanate 625mg 12 hourly and ibuprofen 400mg 8 hourly for 5 days)
- Ascertain whether healing; if so, follow up is not necessary.

#### TRUE AMENORRHOEA

True amenorrhoea can be physiological as the period before puberty, during pregnancy, during lactation, and after the menopause. It may also be pathological.

**Clinical Features**

The clinical features depend on age of presentation in physiological type and on the level of disturbance in the pathological type of amenorrhoea.

**Investigations**

- In the physiological type of amenorrhoea, a good menstrual history and physical examination are usually sufficient to confirm physiological amenorrhoea. A pregnancy test and/or ultrasound usually confirm early pregnancy.
- In the pathological type, the causes may be uterine lesions, ovarian lesions, pituitary disorders, other endocrine disorders, psychiatric illness or emotional stress,and severe general illness. Primary amenorrhoea is investigated after age 18 and secondary amenorrhoea at any age when 2 or more cycles are missed. Refer to a gynaecologist.

**Management**

- In physiological amenorrhoea, reassurance is sufficient.
- In the pathological type, management depends on the cause.

## 57.5.2 DYSFUNCTIONAL UTERINE BLEEDING (DUB)

A normal menstrual period lasts 2–7 days, average lasts 3–5 days, and a normal cycle lasts between 21 and 35 days. Menorrhagia is excessive bleeding at the menstrual periods. Polymenorrhoea refers to frequent cycles shorter than 21 days. Epimenorrhoea refers to frequent and heavy periods. Metrorrhagia refers to irregular uterine bleeding independent of or in between regular periods.

Dysfunctional uterine bleeding refers to those cases in which the bleeding is due neither to some obvious local disorder, such as pelvic infection or new growth, nor to some complication of pregnancy. This denotes some form of hormonal imbalance to be confirmed or excluded on MVA histology and hormonal assays.

Metropathia haemorrhagica describes periods of amenorrhea of 6–12 weeks followed by prolonged spotting 2–4 weeks. On curettage and histology there is cystic glandular hyperplasia.

**Clinical Features**

Irregular periods associated with lack of ovulation, which are commonest at puberty and during perimenopausal period and at times during the reproductive years (14–44 years). As a consequence, there may be anaemia and poor health.

At puberty it may be associated with changes in climate and environment, school examinations, stress, intercurrent illness, and pregnancy. It is important to exclude abortion, ectopic pregnancy, and fibroids during the reproductive years, while pregnancy and uterine and cervical cancers should be excluded during perimenopausal years.

**Investigations**

- Haemoglobin estimation
- Pregnancy test
- Curettage and histology (avoid in young girls)
- HSG and semen analysis in those with associated infertility

**Management**

- At puberty, reassurance may suffice
- Women whose irregular periods are with associated anovulation need hormonal therapy at any age. Those with associated infertility can be given ovulation inducers such as clomiphene after HSG and semen analysis.Those not desiring children can have cyclicity of periods re-established using contraceptive pills for 3 cycles.

**Follow up is not necessary after healing is ascertained.**

- Manage cases of fibroids and genital cancer as appropriate.
- Those with pregnancy complications can be similarly managed, as appropriate.
- Those with anaemia require transfusion or haematinics with iron and folate in standard doses.
- Sometimes and more often, curettage is curative but it maybe so in patients amenable to spontaneous cure. Follow up is as appropriate.



## 57.5.3 DYSMENORRHOEA

Dysmenorrhoea is pain before or during period, sufficient to interfere with the woman's normal occupation. It may be associated with nausea, vomiting and disturbance of bowel function. There are 2 types of dysmenorrhoea, primary and secondary.

### PRIMARY DYSMENORRHOEA

**Clinical Features**

Primary dysmenorrhea is the more common type, occurring in girls or young women less than 20 years of age. The pain is spasmodic or colicky in nature. It starts on the first day, and may last a few hours or throughout the period. It may be associated with nausea, vomiting, and/or diarrhoea or constipation. It may be incapacitating and interfere with normal daily activity. Good history and examination are necessary to rule out co-existing disease.

**Investigations**

- Haemoglobin estimation in cases of anaemia

**Management**

- Reassure.
- Counsel on stress and treat as appropriate.
- Analgesics: paracetamol 1g TDS or ibuprofen 200mg TDS.
- Suppression of ovulation by use of contraceptive pill for 3 cycles, for example microgynon.
- MVA or D&C are not recommended as a remedy in young girls.
- Note that in a majority of cases, pain may cease after first delivery. Follow up as appropriate.

### SECONDARY DYSMENORRHOEA

**Clinical Features**

This is secondary to organic disease, for example PID, fibroids, and associated infertility. Features of underlying cause may be evident. Often the pain precedes the onset of a period by a week to 10 days.

**Investigations**

- In line with underlying cause

**Management**

- Administer paracetamol 1g TDS or ibuprofen 200mg TDS as in primary dysmenorrhoea
- Treat underlying cause.

### 57.5.4 PREMENSTRUAL TENSION SYNDROME

**Clinical Features**

This manifests as premenstrual discomfort in lower abdomen and back 7–10 days preceding menses. It gives a sensation of distension or pelvic engorgement. There is relief after flow begins. It is accompanied by nervous irritability, depression, headache, listlessness, and discomfort in breasts.

Occasionally there is fluid retention. A good history and physical examination are important for accurate diagnosis.

**Management**

- Reassurance
- Mild tranquillizers: Phenobarbitone 30mg nocte ***OR*** diazepam 5mg nocte.
- Norethisterone (progestin) 5mg BD orally days 19–26 of cycle for 3 cycles.

## 57.6 Neoplasms (Potentially Malignant Conditions)

Health service providers should sensitize community members on symptoms of gynaecological cancer and advise them to seek help from health facilities.

Women should also be encouraged to have routine annual gynaecological checkups by qualified health personnel. Health service providers should use simple cancer screening technologies such as visual inspection with acetic acid (VIA), visual inspection with Lugol's Iodine (VILI), and breast examination. They should refer suspicious cases to higher levels for appropriate management.

Neoplasms may present as pelvic masses. Refer to Section 58.4 on pelvic masses.

### 57.6.1 OVARIAN CANCER

**Clinical Features**

Usually occurs in women aged 40 years and above. Usually presents late with mass in lower abdomen. Pain and irregular vaginal bleeding are late features. Ascites and wasting are further late features. In late cases the mass is usually irregular and fixed. Diagnosis is essentially clinical but confirmed with biopsy.

**Investigations**

- Haemoglobin, blood group, urinalysis, blood urea
- Ultrasound
- Intravenous urogram (IVU)
- Ascitic tap for cytology
- Fine needle aspiration and cytology (FNAC)
- Laparotomy for biopsy and histology and staging

**Management**

Surgery is the mainstay treatment. To prepare:

- Improve general health with high protein diet and transfusion where necessary.
- Carry out palliative surgery in inoperable cases and staging.

- Perform total abdominal hysterectomy and bilateral salpingo-oophorectomy in operable cases.
- Administer chemotherapy in addition to surgery; available drugs include vincristine, vinblastine, alkeran, cyclophosphamide, and cisplatinum, as directed by the oncologist.
- Admit level 4–6 for
  - Surgery and/o rchemotherapy.
  - Confirmation of diagnosis.



**Prevention**

Annual pelvic examination and pelvic ultrasound are recommended as preventive measures for early detection and management.

## 57.6.2 CANCER OF THE CERVIX

This is the most common gynaecological cancer. The risk factors for this condition are early age of first coitus, multiple sexual partners, having spouses with multiple sexual partners, high parity, infection with human papilloma virus (HPV), and infection with Herpes simplex type II.

**Clinical Features**

- Commonest in age group 30 and above.
- There is post-coital bleeding.
- There is post-menopausal bleeding.
- There is foul smelling vaginal discharge.
- There is intermenstrual PV bleeding.
- Many patients present late with advanced disease.
- Pain, anaemia, cachexia are late presenting features.
- Diagnosis is confirmed by histology.

**Investigations**

- Speculum examination shows easily bleeding lesion on the cervix
- Haemoglobin
- Biopsy

**— A high index of suspicion is essential as early detection is important**

**Management**

- Provide general supportive care, e.g., correction of anaemia.
- Undertake examination under anaesthesia for staging and biopsy of the lesion, for confirmation by histology.
- Provide supportive treatment, surgery, and/or radiotherapy.
- Refer to a specialist as appropriate.
- If histology confirms malignancy, admit for investigations.

**Prevention**

- Avoid risk factors listed above.
- Pap smear every 3 years for early detection.
- Visual inspection (of cervix) with acetic acid (VIA) or Lugol's iodine (VILI) are simple screening methods that can be used for all women from sexual debut.
- HPV vaccine before sexual debut and for those HPV negative.

## 57.6.3 CARCINOMA OF THE ENDOMETRIUM

This is probably the third commonest cancer in women in Kenya after cervix and breast. Main age is peri and post menopausal. Associated with low parity, obesity, diabetes, and hypertension and may be preceded by endometrial hyperplasia due to unopposed oestrogen stimulation of endometrium. Presents with abnormal uterine bleeding at the perimenopausal or post-menopausal period.
Clinical findings may be unremarkable in early disease but enlarged uterus and evidence of metastasis may be evident in late cases. Diagnosis is confirmed by histology of endometrial biopsy obtained through MVA, fractional curettage, or Novak or Kevorkian curets. Treatment is by extended total abdominal hysteretomy (TAH) but adjuvant chemotherapy and/or radiation may be needed in advanced cases. High doses of progesterone are especially useful in advanced disease.

**Management**

Management is by a gynaecologist in conjunction with an oncologist.

## 57.6.4 CARCINOMA OF THE VULVA

This accounts for 3–4% of all gynaecological cancers.

**Clinical Features**

- Majority of patients present after menopause.
- It may be preceded by pruritic conditions of the vulva.
- Presents as an ulcer on the vulva.
- May have inguinal lymphadenopathy.
- Diagnosis is by clinical features and confirmed by biopsy and histology.
- Differential diagnoses include granuloma inguinale, lymphogranuloma venereum, syphilitic chancre or gummata, and chancroid.

**Management**

- Suspicious lesions should be referred to a gynaecologist.
- Treatment is by surgery (radical vulvectomy).
- Extent of surgery will depend on the primary tumour.
- Radiotherapy and chemotherapy and surgery for advanced disease.

### 57.6.5 CARCINOMA OF THE VAGINA

♦ Accounts for 1% of gynaecologic malignancies. Peak. Incidence is from age 45 to 65.

**Clinical Features**

There is post-coital bleeding, dyspareunia, watery discharge, urinary frequency or urgency, and painful defecation. Cancers are commonly found in the upper part of the vagina on posterior wall. Speculum and digital examination reveals growth in the vaginal wall.

**Investigations**

- ♦ Pap smear: Reveals carcinomatous cells
- ♦ Schiller'stest
- ♦ Biopsy

**Management**

- ♦ Depends on location and extent of the disease
- ♦ A tumour localized in the upper 1/3 of the vagina is treated either by radical hysterectomy with upper vaginectomy and pelvic lymph node dissection or with radium and external radiotherapy
- ♦ Treatment of secondary carcinomas and 1°carcinoma is usually combined and may be either radiotherapy or radical surgery. The 5-year survival rate without recurrence is about 30%.



## 57.7 Pelvic Inflammatory Disease (PID)

Pelvic inflammatory disease is the inflammation of pelvic structures above the cervical os. It is essentially a consequence of STI (gonorrhoea and Chlamydia trachomatis), but can follow puerperal sepsis or abortion. Gonorrhoea and chlamydia trachomatis principally result in endosalpingitis, whereas puerperal and post-abortion sepsis result in exosalpingitis. PID may be acute, subacute, acute on chronic, or chronic. Tuberculosis is another important cause of PID.

**Clinical Features**

Acute PID is diagnosed by:

- ♦ Lower abdominal pain usually starting soon after a menstrual period
- ♦ Fever
- ♦ Signs of pelvic peritonitis in lower abdomen
- ♦ Bilateral adnexal tenderness and positive cervical excitation on n vaginal examination
- ♦ The patient may be toxic with vomiting

Chronic PID is diagnosed by:

- ♦ Chronic or recurrent lower abdominal pains
- ♦ Dyspareunia
- ♦ Infertility
- ♦ Mucopurulent cervical discharge

- Bilateral adnexal tenderness
- Adnexal induration and/or masses (tubo-ovarian)

**Diagnosis is mainly clinical.**

— **Tuberculosis is diagnosed by biopsy: endometrial or pelvic.**

**Investigations**
- Urethral and cervical smears may be helpful in acute cases for gram-stain and culture
- Haemoglobin
- Blood slide for malaria parasites
- Urinalysis
- VDRL

**Management**
- Acute PID: Mild to moderate where the patient is not toxic and there are no features of peritonitis:
  - PO amoxicillin/clavulanate 625mg 12 hourly for 7 days ***OR*** doxycycline 100mg BD for 7 days 12 hourly with PO metronidazole 400mg 8 hourly for 7days; avoid alcohol. Add PO ibuprofen 400mg 8 hourly and hyoscine butylbromide 20mg PO 8 hourly for 5 days.
  - STI related PID:
  - Amoxicillin 3g STAT + amoxicillin-clavulanate 625mg STAT + probenecid 1g **+ doxycycline 500mg QDS for 10 days. In pregnancy use erythromycin 500mg QDS for 10 days + metronidazole 400mg TDS for 10 days.**
- Acute PID – Severe cases with toxicity and features of peritonitis:
  - Start IV fluids.
  - Parenteral or oral analgesic,e.g.,morphine 10mg IM PRN(3 doses),then change to PO ibuprofen 400mg TDS for 7 days.
- IV crystalline penicillin 3 mega units 6 hourly ***OR*** ceftriaxone 1gm BD + IV gentamicin 80mg 8 hourly+metronidazole 500mg IV 8 hourly for 3–5 days. Then give PO metronidazole 400mg 8 hourly and doxycycline 100mg 12 hourly for 10 days and PO ibuprofen 400mg 8 hourly for 5 days.
- If fever persists after 48–72 hrs of antibiotic cover:
  - Perform bimanual pelvic examination. Confirm with pelvic ultrasound.

**If there is pelvic collection (bulge in pouch of Douglas) and/or adnexal masses, pelvic abscess is suspected and laparotomy for drainage done.**
- At laparotomy, do drainage and peritoneal toilet with warm saline; leave drain in situ for about 3 days and continue parenteral antibiotics postoperatively.
- Chronic PID
  - Antibiotics as for mild to moderate acute PID.
  - Spouse or sexual partner is also investigated and treated for STI.
  - Physiotherapy for chronic pelvic pain.

- Admit level 4 or above in presence of:
  - Severe PID, which is indicated by
  – Dehydration

– Suspicion of abscess
– Febrile patient
– Suspicion of induced abortion
• Acute PID if
– There is vomiting
– Follow up cannot be guaranteed

**Patient Education**
In case of partner(s), trace and treat contacts and advise on condom use to avoid re-infection.

## 57.8 Abscesses and Fistulas



### 57.8.1 BARTHOLIN'S ABSCESS

Bartholin's glands are located bilaterally in the vulva, adjacent to the vaginal orifice. Cysts arise when the glands' ducts become occluded. Bartholin's abscesses occur when the gland becomes secondarily infected with one of many common bacterial pathogens.

**Clinical Features**
Patient may complain of any combination of symptoms including local pain, low-grade fever, perineal discomfort, labial swelling, dyspareunia, purulent PV discharge, and difficulty in sitting. Physical examination may reveal tender, fluctuant abscess lateral to and near the posterior fourchette, local swelling, erythema, labial oedema, and painful inguinal adenopathy. Most abscesses develop over 2–3 days and spontaneous rupture often occurs within 72 hours.

**Management**
♦ Treatment of acute phase includes bed rest, analgesics,e.g.,PO ibuprofen 400mg TDS for 5 days, hot wet compresses.
♦ Metronidazole 400mgs TDS for 7 days
♦ PO doxycycline, 100 mg BD for 7 days, then re-evaluate.
♦ When abscess formation is obvious, incision and drainage as follows:
  • Apply local anaesthesia lignocaine1%.
  • Incise distended mucosa as close to hymenal ring as possible or through skin if point of abscess is obvious.
  • Marsupialize to prevent recurrence.
  • Pack cavity with gauze impregnated with liquid paraffin for 24 hours.
♦ Continue with warm sitz (saline) baths till the wound is healed.

### 57.8.2 GENITAL FISTULAS

**This is communication between the genital tract and the urinary or alimentary tracts and may occur singly or in combination. It is due to:**

- Obstetrical injury: Obstructed labour usually leads to pressure necrosis of the bladder and vaginal wall and the rectum. Necrotic tissue sloughs off, leading to vesicovaginal fistula (VVF) and recto-vesical fistula (RVF).
- Instrumental delivery may cause perforation of the vagina and rectum.
- Operative injury: A fistula maybe caused during total abdominal hysterectomy and caesarean section.
- Extension of disease: Malignancy of the bowel or any pelvic abscess may perforate into the rectum and posterior vaginal wall.
- Radiotherapy: Heavy radiation of the pelvis causes ischaemic necrosis of the bladder wall and bowel, causing urinary or faecal fistula.

**Clinical Features**

The patient complains of urinary or faecal incontinence or both. Secondary amenorrhoea is common.

**Management**

- Confirm diagnosis using Sims's peculum.
- Examination under anaesthesia is always mandatory for the diagnosis and definition of fistula. In case of recently formed VVF, continuous bladder drainage for 2 weeks is useful because a small fistula may close or a large fistula may reduce in size.
- Vulval excoriation is treated by water repellent substances,e.g.,KY jelly, to be applied before repair is done. If a VVF co-exists with RVF, the VVF is repaired first.
- Admit for
  - Confirmation of diagnosis and definition to plan treatment.
  - Physiotherapy for sphincter strengthening and for lower limb weakness.
- Refer to higher level if
  - Diagnosis is confirmed after examination.
  - Reconstructive surgery is deferred 3 months after the initial injury or after a previous attempt at repair to allow all tissue reaction to subside.

## 57.9 Sexual Assault

(See also National Guidelines for Medical Management of Rape and Sexual Assault – DRH/MOH)

Sexual assault (rape) is a violent crime directed predominantly against women. Under Kenyan laws rape is defined as carnal knowledge of a woman without her consent or by use of force, duress, or pretence. A girl below 18 years of age is not legally deemed to be able to give consent (Children Act). Neither are mentally retarded or psychiatric women.

**Clinical Features**

These will range from none or mild to very severe injuries that may be life threatening. The medical personnel must approach the rape victim with great understanding, respect, and concern for her well being. The patient may appear deceptively calm and is usually withdrawn and detached. Careful history and medical record are important because this information will be required in court. If the patient has eaten, drunk, bathed, or douched, this may affect the outcome of laboratory tests. History must be taken to evaluate the risk of sexually transmitted disease and pregnancy.

During physical examination, it is important to document location, nature, and extent of any external trauma to face, neck, breast, trunk, limbs, the genitalia, and vagina; in addition, cervical trauma must be documented. Clothes and attire are retained as exhibits. Psychological trauma is evaluated and managed.

**Investigations**

- Swabs for microscopy and culture from:
  - Vagina
  - Throat
  - Rectum
  - Urethra
- Swab the cervix and vagina for sperm microscopy. Pap smear may preserve sperms for later identification.
- Pubic hair combings and clippings.
- Scraping of fingernails for DNA studies for purposes of identification of the assailant
- Blood for baseline RPR and HIV serology; repeated after 3 months.
- Urine for baseline pregnancy test; repeated after 4 weeks.

**Management**

- Encourage patients and relatives to report all cases to the police. Disco urage private deals by perpetrators to evade the law.
- Treat physical injuries, noting that some tears or cuts may require surgical repair.
- Administer tetanus toxoid for soiled lacerations,
- Give prophylactic treatment to prevent pregnancy after ruling out already existing pregnancy. This is ethynyloestradiol 50µg+levonorgestrel150µg2 tabs STAT and 2 tabs 12 hours later, ***OR*** ethynyl oestradiol 30µg + levonorgestrel 150µg 4 tablets STAT followed by 4 after 12 hours, ***OR*** levonorgestrel 750µg/L STAT and 1 after 12 hours.
- Give prophylaxis against sexually transmitted disease.
- Give HIV post exposure prophylaxis (see Section 2.1.8).
- The patient may be put on tranquillisers,e.g.,PO diazepam 5–10mg 8 hourly for about 10 days.

- ♦ If the perpetrator is available for examination, document clinical evidence that may connect him/her with the victim/survivor (hair, blood, semen, scratch or teeth marks) and take specimens accordingly.
- ♦ Ensure psychological and psychiatric review; this is essential.
  - Long-term psychological and psychiatric care may be required.
- ♦ Initiate major or reconstructive surgery as required.

# 58. Obstetrics

In this section, attention turns to the care and treatment of the woman during and before pregnancy, during and after the birth of the child, as well as on the welfare of the child. This focus is consistent with the first cohort defined by the Kenya Essential Package for Health (KEPH) – pregnancy and the newborn (up to 2 weeks).

## 58.1 Antenatal Care and Complications

Uncomplicated antenatal care can be provided at all levels of the health care system, while complicated antenatal care should be carried out only at levels 4 to 6.

### 58.1.1 ANTENATAL CARE

Antenatal care is organized to achieve several main objectives:

- Prevention and treatment of pregnancy complications.
- Provision of nutritional, social, emotional, or physical support.
- Detection and treatment of disorders or diseases.
- Provision of patient education.
- Planning for labour and delivery.

**CONDUCT OF ANTENATAL CARE**

Antenatal care should start as early as possible. The first visit should be in the first trimester. During this visit a detailed history is taken. It should include age, marital status, occupation, education, ethnic origin, area of residence, drinking, smoking and any substance abuse habits, as well as past obstetric and gynaecological history. Records of each pregnancy in chronological order should include date, place, maturity, labour, delivery, weight, sex and fate of the infant, and any puerperal morbidity**.**

The patient's past medical and surgical history is recorded, as is any family history of diabetes, hypertension, TB, hereditary diseases, and multiple pregnancy. The history of the current pregnancy is enquired into: last menstrual period (LMP), estimated delivery date (EDD), maturity at present, any problems encountered so far, e.g., bleeding. LMP is the first day of LMP; gestation is calculated in weeks from LMP; EDD is calculated by adding 7 days to LMP and 9 to the month, e.g., LMP 1/1/93, EDD 8/10/93.

**Physical exam is then done, to include:**

- BP, weight,urinalysis
- General physical exam
- Abdominal exam: Fundal height, lie, presentation, foetal heart sounds, presence of multiple gestation, sizes of liver and spleen, and presence of other masses.
- Vaginal exam: This is indicated as follows:
- At early pregnancy to confirm and date pregnancy.

- In late pregnancy at 36 weeks to assess pelvic adequacy.
- In labour to confirm diagnosis and monitor progress.
- Other times to evaluate symptoms and complaints from patient.

**Investigations**

Should include a minimum of:

- Blood group: ABO + Rhesus, VDRL, haemoglobin, HIV screening
- Other tests as appropriate for individual patient.



## 58.1.2 SCREENING FOR NEW WHO MODEL OF FOCUSED ANTENATAL CARE

This is a classification mechanism, presented in Figures 58.1 and 58.2.Those who check NO for all questions follow the 4 visits model, while those with problems may require extra visits. The 4 visits are 1st by 16 weeks, 2nd at 24– 28, 3rd at 32 weeks, and 4th at 36 weeks.

**Table 59.1: Common complaints in pregnancy**

| Complaint | What to do | What to avoid |
|---|---|---|
| Abdominal pain, backache | Exclude UTI and local lesion; if none reassure. Physiotherapy | Avoid unnecessary medication |
| Morning sickness (nausea & vomiting) | Reassure up to 3 months. If severe with dehydration admit for hydration. Exclude UTI, malaria, and typhoid | Avoid anti-emetics |
| Indigestion (flatulence, heartburn & constipation) | High roughage diet. If severe give mild laxative and antacid, e.g., Bisacodyl 5mg in the morning 2 at bedtime x 5 days. Magnesium trisilicate 10ml TDS x 5 days | Avoid strong laxatives or enema |
| Ptyalism (Excessive salivation) | Reassurance | Avoid anti-cholinergic drugs |
| Food fads; pica (Craving for unusual foods and substances) | Advise on balanced diet. Eat according to desire. Give haematinic supplements as for prophylaxis | Discourage harmful and contaminated materials, e.g.,soil |
| Generalized pruritus | Reassurance: Mild anti-pruritic (chlopheniramine 4mg TDS) 5 days; Exclude skin and systemic diseases | Avoid steroids |
| Pruritus vulvae | See under vaginal discharge | Avoid douching |
| Muscle cramps | Calcium lactate 300mg daily Physiotherapy | Avoid NSAIDs |
| Fatigue | Reassurance; bed rest 3–7 days Advise on balanced diet | Avoid drugs |
| Breast tenderness | Reassure; advise on breast support | Avoid NSAIDs and breast massaging |
| Bleeding gums | Oral hygiene, massage gums, vitamins ABC Refer to dentist if necessary | Do notexcise hypertrophied gums(epulis) |

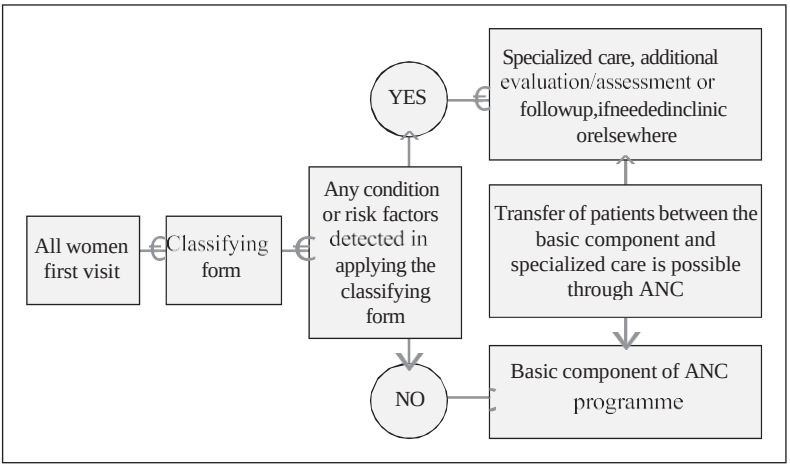
**At each return visit antenatal care should include:**

- Interval history of symptomatology and/or problems. Date of first foetal movements.
- Weight: amount and pattern of weight change. Blood pressure, check for oedema.
- Urinalysis for glucose, proteins, ketones. Obstetric examination,vaginal examination/speculum as indicated.
- Repeat laboratory tests, if necessary,e.g.,
  - PCV at 28–36 weeks
  - Serology for syphilis and HIV at 36 weeks
  - If Rh –ve, indirect Coomb's test every 4 weeks.

**Special laboratory tests as indicated for individual patients to assess maternal/ foetal wellbeing:**

- Examination of amniotic fluid.
- Foetal-heart movements monitoring and evaluation.

**Figure 59.1: The new WHO antenatal care model**

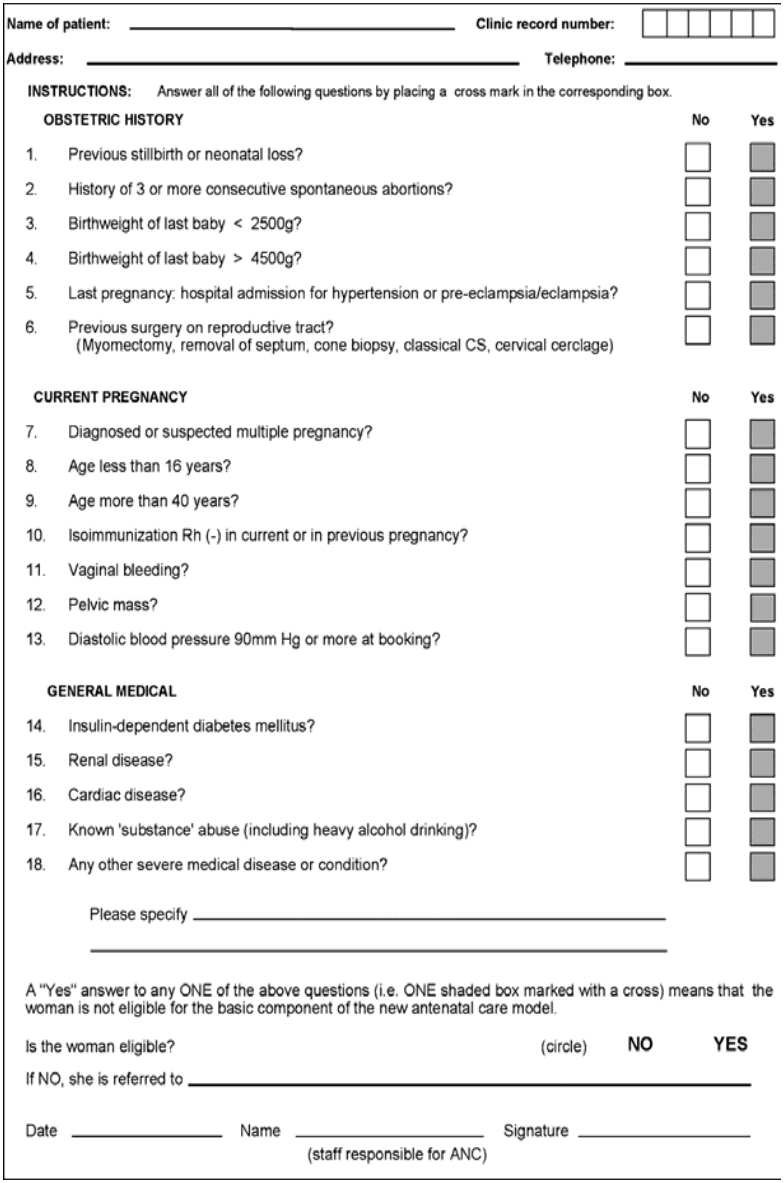



**Other issues include the following:**

- Decisions on place and expected mode of delivery should be made and agreed with the patient not later than 36 weeks of gestation.
- Counselling should be provided for family planning in general and for postpartum voluntary surgical contraception (VSC). Duly signed informed consent forms should be available at admission.
- Patients should be advised to report to the health facility promptly if they have PV bleeding, draining of amniotic fluid, blurred vision, or labour pains.

**Figure 59.2: Criteria for classifying women in the basic component of the new**

natal care model



Name of patient: _____ Clinic record number: ☐☐☐☐☐☐

Address: _____ Telephone: _____

**INSTRUCTIONS:** Answer all of the following questions by placing a cross mark in the corresponding box.

| | **OBSTETRIC HISTORY** | **No** | **Yes** |
|---|---|---|---|
| 1. | Previous stillbirth or neonatal loss? | ☐ | ■ |
| 2. | History of 3 or more consecutive spontaneous abortions? | ☐ | ■ |
| 3. | Birthweight of last baby < 2500g? | ☐ | ■ |
| 4. | Birthweight of last baby > 4500g? | ☐ | ■ |
| 5. | Last pregnancy: hospital admission for hypertension or pre-eclampsia/eclampsia? | ☐ | ■ |
| 6. | Previous surgery on reproductive tract? (Myomectomy, removal of septum, cone biopsy, classical CS, cervical cerclage) | ☐ | ■ |

| | **CURRENT PREGNANCY** | **No** | **Yes** |
|---|---|---|---|
| 7. | Diagnosed or suspected multiple pregnancy? | ☐ | ■ |
| 8. | Age less than 16 years? | ☐ | ■ |
| 9. | Age more than 40 years? | ☐ | ■ |
| 10. | Isoimmunization Rh (-) in current or in previous pregnancy? | ☐ | ■ |
| 11. | Vaginal bleeding? | ☐ | ■ |
| 12. | Pelvic mass? | ☐ | ■ |
| 13. | Diastolic blood pressure 90mm Hg or more at booking? | ☐ | ■ |

| | **GENERAL MEDICAL** | **No** | **Yes** |
|---|---|---|---|
| 14. | Insulin-dependent diabetes mellitus? | ☐ | ■ |
| 15. | Renal disease? | ☐ | ■ |
| 16. | Cardiac disease? | ☐ | ■ |
| 17. | Known 'substance' abuse (including heavy alcohol drinking)? | ☐ | ■ |
| 18. | Any other severe medical disease or condition? | ☐ | ■ |

Please specify _____

_____

A "Yes" answer to any ONE of the above questions (i.e. ONE shaded box marked with a cross) means that the woman is not eligible for the basic component of the new antenatal care model.

Is the woman eligible? (circle) **NO** **YES**

If NO, she is referred to _____

Date _____ Name _____ Signature _____

(staff responsible for ANC)

### 58.1.3 MANAGEMENT OF HIGH-RISK PREGNANCIES

Every pregnancy faces risks. However, it is necessary to detect current problems or complications and manage them. Notably, it is not possible to predict future complications and prevent them. All pregnant women need to be assisted to recognize danger signs and to report for management of complications at all time.



**High risk patients should be managed at levels 4 to 6.**

**In the past the following have been considered high risk criteria (history or current):**

- Extremes of reproductive age: Below 18 and above 35.
- Primigravida: Especially too young, too short, too old.
- High parity 5+, short birth interval.
- Large infants: 4,000g or above.
- Prematurity: LBW below 2,500g.
- Obstructed and difficult labours.
- Still births, neonatal deaths, abortions, caesarean section.
- Genetic or familial diseases.
- Medical diseases: Diabetes, cardiac, renal, hypertension, Rhesus, anaemia,HIV infection.
- Antepartum haemorrhage, postpartum haemorrhage, DVT, IUGR, PROM, postdates, CPD, and multiple pregnancy.

**Principles of management include:**

- Prenatal investigations and counselling in appropriate cases
- Early start of antenatal care
- Close medical supervision during pregnancy
- Special tests and examinations to evaluate foetal development and wellbeing as well as maternal wellbeing
- Timely intervention for therapy and delivery.

## 58.2 Anaemia in Pregnancy

Anaemia in pregnancy is a major obstetric problem in Kenya. Locally, anaemia is generally accepted as Hb <10g%. Mild anaemia Hb 8–10g, moderate Hb 6–7g, severe Hb 4–5g, very severe below Hb 4g. In severe anaemia the pregnancy is in danger of abortion, premature labour, or IUFD, while in very severe anaemia the mother's life is also in danger.

Most cases are due to iron deficiency as a result of dietary deficiency or blood loss from hookworm infestations. Anaemia is also due to haemolysisdue to malaria, sickle cell disease, and folate deficiency due to inadequate intake especially in urban areas. Iron deficiency and folic acid deficiency often occur together causing "Dimorphic Anaemia".

**Clinical Features**

There is general weakness, dizziness, pallor, and oedema; in addition, in haemolytic anaemia there is jaundice and hepatosplenomegaly.

**Investigations**

- Full haemogram
- Stool for hookworm ova and schistosomalova, where applicable
- Urine urobilinogen and schistosomal ova, where applicable
- Blood slide for malaria parasites
- Sickling test

**Prevention**

- Balanced diet
- Prophylaxis iron (ferrous sulphate 200mg TDS+folate 5mg OD), throughout pregnancy
- Antimalaria prophylaxis (ITP)
- Early detection
- Routine antenatal Hb screening at first visit and near term

**Management**

**As summarized in Table 58.2, principles of management include:**

- Raise Hb (oral or parenteral haematinics, transfusion – refer if needed).
- Remove cause-dietary deficiency,treat malaria, treat hookworms,give haematinics if dietary deficiency exists.
- Prevent recurrence.

**— Whereas mild anaemia can be cared for at all levels of health care, moderate to severe anaemia needs to be taken care of at 4 to 6 level facilities.**

**Table 58.1: : Management of anaemia in pregnancy**

| Severity | Hb(g/dl) | Management |
|---|---|---|
| Mild | 8–10 | Treat cause. Oral haematinics, ferrous sulphate 200mg TDS and folic acid 5mg for 1 month. Improved diet. |
| Moderate | 6–7 | Give ferrous sulphate 200mg TDS and folic acid 5mg OD for 3 months. Improved diet. |
| Severe | 4–5 | Transfusion of blood and give ferrous sulphate 200mg TDS and folic acid 5mg OD for 3 months. Improved diet. |
| Very severe | Below 4 | Resuscitation and treatment as for severe cases. |

### USE OF BLOOD TRANSFUSION IN PREGNANCY

For severe and very severe anaemia, especially where cardiac failure or labour is imminent (see Section 58.3, antepartum haemorrhage, and 58.14.3, postpartum haemorrhage), do the following:

- Transfuse slowly: 500ml whole blood in 4–6 hours with branula G18.
- Give frusemide 80mg IV STAT.
- Give packed cells, if available; cover with malaria PO artemether lumefantrine fullcourse.

**In very severe anaemia, transfuse blood bearing in mind the HIV risk in blood transfusion: Use screened blood only and sparingly.**

### 58.2.1 COMPLICATIONS OF ANAEMIA IN PREGNANCY

**The complications of anaemia in pregnancy include the following:**

- Cardiac failure, which may lead to death.
- May worsen effects of minor postpartum haemorrhage (PPH) leading to death.
- May worsen effects of minor hypoxia during anaesthesia, causing death.
- Reduces resistance to infection.
- Causes late abortions, premature labours.
- Increases perinatal mortality and morbidity even in term babies.
- Results in babies becoming anaemic (iron deficiency) after 2–3 months of life.
- Administer prophylactic haematinics.

## 58.3 Antepartum Haemorrhage (APH)

Antepartum haemorrhage (APH) is defined as vaginal bleeding after the 20th week of pregnancy. APH is associated with increased foetal and maternal morbidity and mortality. The foetal and maternal status will depend on amount, duration, and cause of bleeding. The causes of APH are:

- Extraplacental bleeding*:* From sites other than the placental surface, including cervical lesions, e.g., trauma, cancer of the cervix, cervical polyps; vaginal lesions, e.g., tears/lacerations(rare), and infections; and vulvo perineal tears(rare).
- Placental causes:
  - Placental abruption (abruptio placentae): This is defined as occurring when a normally implanted placenta separates from the uterine wall (decidua basalis) after the 20th week and prior to the 3rd stage of labour. Bleeding may be absent, mild, moderate, or severe (this does not reflect extent of separation or severity). Bleeding may be concealed when little or no bleeding is seen PV or revealed when bleeding PV is evident.
  - Placenta praevia: This occurs when any part of the placenta implants in lower part/segment of the uterus. Further clinical classification is feasible depending on the relationship to internal cervical as:
  - Minor degree:
    - Type 1: Placenta in the lower uterine segment but not encroaching the internal os.
    - Type 2: Placenta partially encroaches internal osbut not during labour.

– Major degree:
  - Type3:Placenta partially encroaches the internal os and remains the same even during labour.
  - Type4: Placenta totally covers the internal os and this relationship does not change during labour.

♦ ***Vasapraevia:*** This is a rare cause of antepartum haemorrhage in which the umbilical cord is inserted into placental membranes with blood vessels traversing and presenting over the internal cervical os.

**Investigations**

♦ Haemoglobin
♦ Urinalysis: Haematuria,proteinuria
♦ Bedside clotting time
♦ Bleeding time
♦ Platelet count
♦ Others: Ultrasound, which offers a high degree of diagnostic accuracy in antepartum haemorrhage

**Management – General**

Always admit to hospital a patient with a history of antepartum haemorrhage even if bleeding is not apparent and the patient appears quite well.

**Take a careful history and note:**

- Amount and character of bleeding
- Any associated pain
- History of bleeding earlier in pregnancy
- History of trauma

**Do a thorough physical exam, including abdominal examination for:**

- Tenderness/guarding
- Contractions
- Foetal heart presence

**Carry out speculum examination:**

- Bleeding from uterus
- Other sites of bleeding
- Cervical dilatation

**In patients with antepartum haemorrhage:**

- Quickly evaluate the maternal and foetal status.
- Take blood for grouping and cross-matching.
- Start IV 5% dextrose or normal saline using a wide bore branula.
- Monitor vital signs; blood pressure, respiratory rate, pulse rate, temperature and insert an indwelling urethral catheter.

**If bleeding is severe or patient is in shock then:**

- Ensure open airway and breathing.
- Establish and maintain adequate circulation: may transfuse whole blood or packed cells.
- Monitor fluid input and output: insert an indwelling Foley's catheter.

**Management – Specific management depends generally on:**

- Gestational maturity
- Condition of foetus
- Continuous bleeding or not
- Onset of spontaneous labour

**Management – Specific**

Essentials of diagnosis:

- Abruptio placentae
  - Continuous abdominal and/or back pain
  - Irritable, tender and often hypertonic uterus
  - Visible or concealed haemorrhage
  - Board-like rigidity
  - Evidence of foetal distress
- Rupture of the uterus may be confused with abruptio placentae. The following features suggest rupture of the uterus:
  - Efforts at resuscitation of the mother unrewarding (e.g.,blood pressure remains low while the pulse remains rapid and thready).
  - Uterine contractions absent.
  - Difficulties in determining shape and outline of the uterus( due to peritoneal irritation and the empty uterus):***This is a very important sign.***
  - For mothers who have been in labour, recession of the foetal presenting part and disappearance of foetal heart sounds suggest rupture of the uterus.
- Once rupture of the uterus has been ruled out, then treatment for abruptio placentae should be instituted.

**Principles of treatment:**

- Rapid correction of hypovolaemia/shock or anaemia, as above
- Correction of coagulation defect:
  - Whole blood
  - Fresh frozen plasma
- Early uterine emptying
- Vaginal delivery whenever possible
- Prevention of postpartum haemorrhage
- Thorough physical examination, including abdominal examination for:
  - Tenderness/guarding
  - Contractions
  - Foetal heart presence
- Speculum examination: Bleeding from uterus, other sites of bleeding, cervical dilatation
- If above measures do not establish diagnosis, then do examinations under anaesthesia (EUA) in theatre; rule out placenta praevia then do:
  - Artificial rupture of membranes start oxytocin infusion(if no contraindications) 5 units in 500ml of 5% dextrose(10 drops per minute for 30 minutes and increase by 10 drops every half hourly to a maximum of 60 drops per minute or 3 contractions per 10 minutes, which ever is earlier). This is done when vaginal delivery is evaluated as imminent and feasible.

- Indications for abdominal delivery: Caesarean section,hysterotomy

- Intrauterine foetal death with severe uterine bleeding
- Severe degree of placental abruption with a viable foetus
- Haemorrhage severe enough that it jeopardizes life of mother
- Any incidental complication of labour

Postpartum: Continue oxytocin for about 2 hours.

**Placenta Praevia**

The management of placenta praevia depends on gestation, extent of bleeding, and clinical findings. Conservative management is done when: bleeding is minimal and a significant risk of prematurity exists. The decision follows evaluation based on complete examination of maternal and foetal status.

**Speculum examination is mandatory. The following must be done:**

- Hospitalization mandatory in a place with caesarean section facilities.
- Restriction of physical activities.
- Weekly haemoglobin.
- Avoid unnecessary physical examinations.
- Ultrasound monitor if possible.

**Patient may be discharged if placenta is normally situated and be re-admitted at 38 weeks (as below); then:**

- If no bleeding recurs by 37 weeks prepare patient for theatre under a DOUBLE SET-UP for EUA and for caesarean section.
- If a minor degree of placenta praevia is found, then do artificial rupture of membranes (ARM), start oxytocin, and DELIVER.
- If a major degree of placenta praevia is found, prepare the patient for theatre immediately for caesarean section.
- Do caesarean section if: Bleeding is severe and a threat to life, in doubt about degree of placenta praevia, and any contraindication for normal delivery.

**Level of care for antepartum haemorrhage is 4–6.**

## 58.4 Cardiac Disease in Pregnancy

In Kenya, cardiac disease in pregnancy is often of rheumatic heart disease origin.



**Clinical Features**

There may be history of rheumatic fever in childhood, or known rheumatic heart disease, dyspnoea, palpitations, body oedema, cough, easy fatigability, evidence of heart enlargement, murmurs, thrills, left parasternal heave, prominent neck veins, and tachycardia. There may also be hepatomegaly, ascites, and basal crepitations.

**Investigations**

- Shielded chest x-ray in early pregnancy
- Electrocardiogram
- Routine antenatal profile (haemoglobin, VDRL, blood group,urinalysis)
- Urine C&S, blood culture, urea and electrolytes

**Management**

- This depends on functional classification of the New York Heart Association:
  - Class I Asymptomatic
  - Class II Symptomatic with heavy work
  - Class III Symptomatic with light work or exercise
  - Class IV Symptomatic at rest
- Class I and II are managed as outpatients until 34–36 weeks when they are admitted for bed rest and observation in hospital level 4–6.
- Class III and IV are admitted on first visit at any gestation for entire duration of pregnancy, level 5 and 6.

**Management – Supportive**

- Bed rest
- Haematinic supplementation: Ferrous sulphate 200mg TDS+folic acid 5mg OD combination.
- Treat intercurrent infections: Dependent on organisms identified and site of infection.
- Avoid undue physical and emotional stress.
- Regular urine analysis and culture.
- Ensure dental hygiene.
- Regular urea and electrolyte determination.

**Management – Pharmacological**

- Digitalization is indicated in imminent and overt cardiac failure, if not previously on digoxin. Consult cardiologist on medication regimes.
- Rapid digitalization by mouth,1–1.5mg in divided doses over 24 hours, less urgent digitalization 250–500mcg daily (higher dose may be divided).
- Continue maintenance therapy with digoxin 0.25mg, frusemide 40–80mg.
- Continue prophylactic IM benzathine penicillin 2.4 mega units monthly.

**Labour and Delivery**

- Spontaneous labour and delivery are preferred.
- Prop up & have oxygen and emergency tray available
- Start antibiotics PO amoxicillin 2g+IV gentamicin 160mg STAT then PO amoxicillin1g 8 hourly and IV gentamicin 80mg 8 hourly for 2weeks.
- Adequate analgesia with morphine 10mg IM STAT at 4–6cm cervical dilatation.
- Avoid lithotomy position.
- Assisted vacuum delivery in second stage.
- Massage uterus after delivery of placenta to achieve uterine contraction.
- Give oxytocin 10 IU IM if needed to achieve uterine contraction or to control postpartum haemorrhage.
- Give frusemide 80mg IV STAT after 3rd stage of labour.
- Observe closely for evidence of cardiac failure.
- Keep in hospital for 2 weeks. Continue antibiotics for entire period. Discharge through the cardiac clinic.

**Patient Education**

- Advise on family planning. Cardiac patients should have small families of 1or 2 children or none. Suitable methods include minilaparotomy tubal ligation under local anaesthesia, vasectomy, barrier methods, progesterone-only agents pillsor implants. Oestrogen containing methods are contraindicated such patients.
- Levels of care for cardiac disease in pregnancy:
  - Classes I and II: Levels 4–6
  - Classes III and IV: Levels 5–6

## 58.5 Diabetes in Pregnancy

Diabetes mellitus is a metabolic disorder characterized by elevated glucose levels in blood. Covered in Section 11.3 in Part I.

**Clinical Features**

- ***Overt diabetes:*** If not already diagnosed the symptoms include polydipsia, polyuria, weightloss, blurred vision, and lethargy. Glycosuria is common but not diagnostic.
- ***Gestational diabetes: This will occur in 1–5% of pregnancies.*** Historical risk factors include previous gestational diabetes, family history of diabetes, previous macrosomic infant or unexplained stillbirth, polyhydramnios, obesity, and advanced maternal age. Glycosuria maybe present but is not diagnostic.
- ***Complications of diabetes:*** These include chronic hypertension and nephropathy,pregnancy induced hypertension, foetal macrosomia, intrauterine growth retardation, polyhydramnios, foetus distress, and hypoglycaemia.

**Investigations**

- Postprandial blood glucose level
- Glucose tolerance test (GTT) to confirm diabetes

**Management**

- Diabetes in pregnancy should be managed in hospital (levels 4-6).
- Regular daily physical activity should be maintained.
- Diet should be 30–35 calories/kg/day,i.e.,1,800–2,400 calories per day; carbohydrate 200g/day and protein 90g/day.
- Non-insulin requiring gestational diabetes can be managed by diet alone and monitored with serial blood sugar:
- If not controlled by diet the patient should start on soluble insulin under the supervision of the diabetic team during admission. Start with 10 units of soluble insulin TDS subcutaneous to maintain the sugar under 7–10mmol/L. To change the dosage as required. Once controlled to convert to insulin 70/30 give 2/3 of the daily dose of soluble in the morning and 1/3 in the evening. To prevent PET, start aspirin 60–75mg OD; start at 16 week and stop at 36 weeks to avoid excess bleeding.
- Delivery:
  - Non-insulin requiring gestational diabetic should be delivered at term.
  - Well controlled insulin-requiring diabetic should progress to 38 weeks before delivery.
  - Insulin dependent diabetic with hypertension, renal, retinal, or cardiac disease, or pre-eclamptic intrauterine growth retardation must be delivered by 37 weeks. When in labour give 1/2 of the daily dose as insulin soluble STAT subcutaneously and then put the other half of the daily dose as insulin soluble in an infusion of 1 litre 5% dextrose to be given over 8 hours.
  - Intrapartum blood glucose is monitored hourly and insulin doses adjusted accordingly in small doses (Discontinue usual insulin regime).
- Postpartum:
  - Insulin requirement can alter after delivery; serial glucose monitoring should be done allowing adjustment of insulin dose to achieve stable control.

**Patient Education**

This should involve the following:

- Pre-pregnancy counselling to facilitate achieving optimum glucose control before pregnancy to minimize foetal complications in diabetic pregnancy.
- Family planning: Advise on a small family.
- Recommended family planning methods include VSC, barrier methods, implants, IUD, and progesterone-only pill.
- Oestrogen containing methods are contraindicated.

## 58.6 Drugs in Pregnancy

Drugs taken by the mother during pregnancy can be harmful to the developing foetus in a variety of ways. Drugs taken just before delivery can also affect the baby. Table 58.3 provides guidelines on drugs that are considered safe or relatively safe in pregnancy. Even these drugs should be used with caution, however,and only when necessary. *Drugs that are contraindicated should be avoided*.

**Table 58.2: Drug use in pregnancy**

| Types of medication | Degree of safety for use in pregnancy | | |
|---|---|---|---|
| | **Safe or relatively safe** | **Some risk – Use with caution** | **Contraindicated in pregnancy** |
| Analgesics | Codeine, morphine, paracetamol, pethidine | Indomethacin, salicylates | |
| Anti-convulsants | Ethosuximide, phenobarbitone, primidone | Clonazepam, phenytoin | |
| Anti-microbials | Ampicillin, amoxycillin, cephalosporins, clindamycin, dicloxacillin, erythromycin, gentamicin, isoniazid, miconazole, oxacillin, penicillin | Chloramphenicol, metronidazole, nitrofurantoin, streptomycin, sulfonamides, trimethoprim, rifampicin, kanamycin | Tetracycline |
| Anticoagulants | Dipyridamole, heparin | Dicumarol, warfarin | |
| Antiemetics | Hydroxyzine, meclizine, prochlorperazine | Phenothiazines | |
| Antihypertensive | Hydralazine, methyldopa, propranolol | Diazoxide | Nitroprusside |
| Bronchodilators | Aminophylline, beclomethasone | Cromolyn sodium | |
| Cardiac drugs | Atropine, digoxin, lidocaine, procainamide, quinidine | Dispyramide, nifedipine | |
| Decongestants | Pseudoephedrine | | |
| Diuretics | Frusemide, Hydrochlorothiazide | | Acetazolamide |
| Gastrointestinal drugs | Antacids, cimetidine, ranitidine | | |
| Hypoglycemics | Insulin | | Chlorpropamide, tolbutamide |
| Sedative &psychiatrics | Barbiturates, flurazepam | Diazepam, chlordiazepoxide, haloperidol, lithium,phenothiazines, tricyclic antidepressants | |
| Thyroid preparations | L-thyroxine, propylthiouracil | | Iodide |
| Vaccines | Polio, tetanus, rabies | | Rubella, measles, smallpox |
| Other drugs | Ferrous sulphate, probenecid | | Antineoplastic drugs, oestrogens, DES |

# 58.7 Malaria in Pregnancy

Falciparum malaria is particularly dangerous in pregnant women. The clinical features of malaria in pregnancy depend to a large extent on the immune status of the woman, which in turn is determined by her previous exposure to malaria. (More details are given in Part I, Section 11.5, malaria in pregnancy.)

**Clinical Features**

- Non-immune (women from non-endemic area): High risk of maternal perinatal mortality. Acute febrile illness; severe haemolytic anaemia; hypoglycaemia; coma/convulsions; pulmonary oedema; abortion; intrauterine death; premature labour; intrauterine growth retardation.
- Semi-immune (women from endemic area): May be asymptomatic, despite placental infection. Causes severe anaemia and low birth weight. More common in primigravidae than multigravidae. One of the dangers of malaria in these settings is that it is not detected or suspected. Antimalarials should form part of the case management of all women with severe anaemia who are from endemic areas, irrespective of whether they have a fever or a positive blood slide (see Part I, Section 11.1, anaemia inpregnancy).



**Investigations**

- Haemogram
- Blood slide for peripheral blood film for identification of parasites. This may be negative in a woman from endemic areas, despite placental parasitization.

**Management**

This is consists of supportive and pharmacological portions of management.

**Supportive:**

- Correct dehydration.
- Evacuate if incomplete/inevitable abortion.
- Deliver if foetal death or established labour.

**Management – Pharmacological**

For clinical disease it is essential to use the most effective antimalaria drug available.

**Immediate treatment is essential.**

- **For uncomplicated disease the following is recommended:**
  PO Quinine hydrochloride 600mg 8 hourly for 7days. 1st trimester: quinine + clindamycin (10mg/kg bw twice a day) for 7 days (or quinine monotherapy if clindamycin is not available). An ACT or oral artesunate + clindamycin is an alternative if quinine + clindamycin is not available or fails.
- Give PO paracetamol l1g 8 hourly or PO ibuprofen 400mg 8 hourly for 3 days.
- ***OR*** PO artemether 20mg–lumefantrine 120mg 4tabs STAT, then after 8 hours, then 12hourly for 2 more days (4 STAT at 0, 8, 24 36,48 and 60 hours). After a meal.

**This treatment can be used in 2nd and 3rd trimester and even in the 1st trimester if quinine not available.**

**For severe or complicated disease the following is recommended:**

Severe Malaria is a medical emergency that puts both the life of the mother and foetus at high risk. Aggressive management is essential.

- Quinine hydrochloride parenterally IV 900mg over 4 hours, then 600mg in 5% dextrose solution to run for 4 hours, then 10% dextrose 500ml for the next 4 hours,to continue with the cycle IV 8 hourly, until able to take oral then change to oral quinine 600mg TDS. Quinine given for 7 days. Or change to full course of arthemether-lumefantrine once able to take orally. Dextrose use helps avoid quinine-induced maternal hypoglycaemia (encourage oral glucose)
- Give diclofenac 75mg BD for 1 day then change to PO paracetamol 1g 8 hourly or PO ibuprofen 400mg 8 hourly for 3 days.
- Other drugs that can be used for treatment in pregnancy areartemisinin derivatives in absence of quinine.

**Prevention**

- In endemic areas all women should receive 4 doses of sulfadoxine/pyrimethamine: 1 in the 2nd trimester (between 16 and 27 weeks) and 1 in the 3rd trimester (between 28 and 36 weeks). Doses should be given at last 4 weeksa part.
- Non-immune pregnant women should be advised not to visit a malarious area. If travel is not avoidable they should take special precautions in order to prevent being bitten, such as using mosquito repellents and an insecticide treated bed net.
- In addition, they should take chemoprophylaxis of either daily sulphadoxine + pyrimethamine 3 tablets STAT in the 2nd or 3rd trimesters. Check with the malaria guidelines.
- Drugs that are contraindicated in pregnancy are: tetracycline preparations (oxytetracycline, minocycline, doxycycline), and primaquine.

— Health care providers should refer to the latest edition of National Guidelines for Treatment and Control of Malaria as protocols may change from time to time.

## 58.8 Multiple Pregnancy

This is a situation where there is more than one foetus in-utero. In most situations it is a twin pregnancy but pregnancy involving more fetuses like triplets may be encountered. Multiple pregnancy may be associated with the use of fertility drugs and generally with higher risk for adverse outcomes (antenatal, intrapartum, and postpartum) than for a singleton.

**Clinical Features**

The uterus is larger than dates; there are multiple foetal parts or more than two foetal poles. There may be a family history of twins and on examination foetal heart rates can be identified at two different areas with a difference of 15 beats per minute. There is increased risk for having PET, polyhydramnios, anaemia, APH, PPH, malpresentation, congenital foetal anomalies, and premature labour.

**Investigations**

- Definitive diagnosis is made by ultrasound, but where it is lacking a plain abdominal radiograph can be taken between 34 and 36 weeks.
- Other investigations as for routine antenatalcare.

**Management – Antenatal Care**

- Preferably in a hospital "High Risk" clinic, levels 4–6.
- Monthly haemoglobin check
- Administration of:
  - Ferrous sulphate 200mg TDS
  - Folic acid 5mg OD
- Monitor for associated obstetric complications,e.g.,preeclamptic toxaemia, antepartum haemorrhage, anaemia, malpresentation.
- Ultrasound at 34–36 weeks gestation (or radiography if not available) to determine:
  - Presentation of 1st twin.
  - Detect anomalies, e.g., conjoined twins.
- Mode of delivery
  - Admission may be necessary to observe and manage for premature labour.
  - Bed rest while at home.

**Management – Intrapartum**

- Mode of delivery determined by presentation of 1st twin:
  - If cephalic allow vaginal delivery.
  - Any other presentation or anomaly, then caesarean section.
- Vaginal delivery:
  - Monitor as per normal labour (refer to normal labour and delivery).
  - After delivery of 1st twin the lie and presentation of the 2nd foetus is determined. Foetal heart also evaluated.
  - If longitudinal, cephalic and foetal heart are satisfactory, then perform (Amniotomy) ARM and await spontaneous delivery.

- If lie is not longitudinal, do external cephalic version (ECV). If ECV fails, then do internal podalic version and perform assisted breech delivery after bringing down a leg and stabilizing the head.
- If longitudinal lie and cephalic presentation with ruptured membranes but within adequate contractions and stable foetal heart rate, then oxytocin at 5 units per 500ml at 30 drops per minute and deliver normally.

**Management – Retained 2nd Twin**

Perform abdominal and vaginal examination and assess: membranes – if intact or ruptured; lie and presentation; whether cervix oedematous. Look for evidence of foetal and maternal distress and manage accordingly. If assessment is favourable, then oxytocin and delivery. Caesarean section if evaluation is poor.

**Management – 3rd Stage**

- Oxytocin 10 IU IM administered after delivery of 2nd twin.
- Look for and anticipate postpartum haemorrhage.

**Patient Education**

- Family planning
- Infant feeding
- Early antenatal visit at subsequent pregnancies.
- Level of care for multiple gestation is 4—6.

## 58.9 Pre-Eclampsia and Eclampsia



Pre-eclampsia (PET) and eclampsia are a continuum of the same syndrome. PET is defined as the onset of hypertension with either proteinuria, oedema, or both at a gestation of 20 weeks or more. Hypertension is here defined as a blood pressure of 140/90mmHg or higher on more than 2 occasions of about 6 hours apart. Eclampsia is the presence of convulsive fits in a patient with PET. Eclampsia carries a high foetal mortality and high maternal morbidity, and in cases of poor management a high maternal mortality as well. The aetiology of pre-eclampsia and eclampsia remains unknown, remaining as “a disease of theories”

**The risk factors associated with pre-eclampsia and eclampsia are:**

- Parity, mostly affecting primigravidae.
- Positive family history of PET.
- Associated with the following medical diseases:
  - Diabetes mellitus
  - Chronic hypertension
  - Renal disease; chronic pyelonephritis, acute glomerulonephritis, polycystic kidneys.
- Age extremes.
- Obstetric conditions:
  - Multiple pregnancy
  - Hydatidiform mole
  - Hydrops fetalis

**Clinical Features**

For management purposes the clinical features may be graded by the criteria given in Table 58.5.

**Table 58.3: PET grading**

| Category | Diastolic BP | Proteinuria (dipstix) | Oedema (variable) |
|---|---|---|---|
| Mild | Up to 100mmHg | - | + |
| Severe | >100mmHg | ++ | ++ |

**Imminent eclampsia manifests as severe PE with these features:**

- Headaches
- Nausea and vomiting
- Epigastric pain
- Visual disturbances, e.g., blurred vision, diplopia, blindness, ocular signs
- Restlessness
- Oliguria

**Investigations**

- Haemoglobin, PCV (packed cell volume)
- Urinalysis for protein (bedside)
  - Qualitative: Dipstix
  - Quantitative: Esbach's reagent
- Blood urea and electrolytes
- Liver function tests
- Coagulation tests (where available)
- Ultrasound may be done to evaluate foetal status

**Management –- General**

- Proper management of pre-eclamptic toxaemia is necessary to optimize the maternal and foetal outcome.
- The optimal time for delivery to be considered.
- Continuous assessment of maternal and foetal conditions.
- Bed rest.
- Drug therapy wherea ppropriate.
- Delivery options must be evaluated.
- Admit level 4–6 for:
  - PET at term for delivery.
  - Severe PET at any gestation.
  - Imminent eclampsia for management and delivery.
  - Eclampsia for management and delivery.
  - Complicating obstetric condition, e.g., antepartum haemorrhage (abruptio), premature labour.
  - Foetal conditions:
    - Intrauterine foetal death.
    - Intrauterine growth retardation.
- Mild pre-eclamptic toxaemia can be managed at level 2–6 as out patient with weekly:
  - Blood pressure record

- Body weight
- Urinalysis by dipstix
- Foetal heart rate
- Foetal/uterine size

♦ Advise on bed rest at home on sedation with phenobarbitone 30mg TDS and to report to hospital level 4–6 if:
- Onset of features suggesting severity (see above).
- Decrease/change in foetal movements.

♦ Admit level 4–6 at 38 weeks for delivery:
- Surfactant test.
- Bishop's score.

**Management –- Severe Pre-Eclamptic**

Admit and manage in hospital level 4–6 as follows

♦ General
- Absolute bed rest
- BP 4 hourly
- Daily urinalysis by dipstixif more than ++, then do quantitative:
  – Daily foetal heart rates
  – Foetal kick count chart
- Weekly blood urea and electrolytes
- Weekly haemoglobin
- Input/output chart (if necessary)

♦ Pharmacological
- Tabs phenobarbitone 30mg TDS
- Tabs methyldopa 250mg TDS then build upto 500mg QDS (depending on response)

♦ If this regimen does not work, deliver immediately.

**The definitive treatment of severe preeclamptic toxaemia is delivery.**

♦ Delivery
- Admit preferably in a quiet room with 24-hour nursing coverage (a pre-eclamptic toxaemia room)
- Put an indwelling Foley's catheter for monitoring output of urine.
- Keep an input/output chart.
- Prevent convulsion.
- Give magnesium sulphate 50% 5g IV over 5 min and then 50% 5g in each buttock deep IM. If MgSO4 not available:
  – Put anI IV line and put in 40mg of IV diazepam. This to titrate against evel of consciousness to keep them well sedated but arousable. The diazepam to be put in 500ml of 5% dextrose
- Control blood pressure:
  – Through another IV linemix 40mg hydralazine in 500ml of 5% dextrose and titrate against the blood pressure level to maintain a diastolic blood pressure of 90–100mmHg. (Where patient is allergic to hydralazine, use sublingual nifedipine 10mg BD.)
- Do a vaginal examination and decide on the mode of delivery, either:
  – Abdominal delivery (caesarean section, hysterotomy)

- Vaginal delivery (artificial rupture of membrane and oxytocin drip IV); if vaginal delivery is not recommended then do abdominal delivery (caesarean section,hysterotomy)

♦ Intrapartum management:
- Maintain the above guidelines.
- If foetus is alive, monitor the foetal heart rate ½ hourly to detect signs of foetal distress.
- Maintain partogram (see Section 58.13.1, on normal labour).
- Do vacuum extraction with episiotomy if required at 2nd stage.
- Continue MgSO4 or diazepam and hydralazine as above for 24–48 hours.

**Management – Eclampsia**

♦ Admit in the acute/pre-eclampsia room.

♦ Management – General ("ABC" = airway, breathing,circulation)
- Assess the level of consciousness of the mother.
- Clear the airway: Suction excess secretions.
- Nurse on the lateral position.
- Introduce a mouth gag, plastic airway, or spatula.
- Administer oxygen through a nasal catheter.
- Introduce an indwelling Foley's catheter to monitor urine output and check for proteinuria

♦ Management –Pharmacological
- Control the convulsions:
  - Magnesium sulphate 50% 5g IV over 5 min and then 50% 5g in each buttock deep IM. Monitor its side effects through the knee reflex; absence signifies toxicity.
  - If MgSO4 is not available, use diazepam 20mg IV immediately.
  - Then put an IV line of 500ml 5% dextrose with 40mg diazepam to keep patient deeply sedated but arousable.
- Control the blood pressure: If the diastolic blood pressure is 110mmHg or more then administer IV hydralazine 20mg STAT; then 40mg in 500ml of 5% dextrose, titrate according to BP.
- Carry out emergency investigations:
  - Haemogram
  - Urea and electrolytes
  - Liver enzymes/and bilirubin levels
  - Urine analysis

♦ Ensure a urine output of 30ml/hr;if less, delay the delivery or refer for ICU. Determine the mode and expedite delivery immediately.

♦ NOTE: Imminent eclampsia is managed as eclampsia; prophylactic antibiotics ceftriaxone 1g BD ***OR*** IV flucloxacillin 500mg 6 hourly to be given. IV frusemide 40mg STAT to be administered if there is pulmonary oedema.

♦ Initiate obstetric physiotherapy.

## 58.10 Chronic Hypertension

Chronic hypertension is managed along the same lines as pre-eclamptic toxaemia. To note:

- Involve the physician.
- Provide contraception with caution.

## 58.11 Rhesus (Rh) Incompatibility

Rhesus isoimmunization occurs in pregnancy where a Rhesus-negative mother is pregnant with a Rhesus-positive foetus. Other ways of isoimmunization include transfusion with Rhesus incompatible blood, ectopic pregnancy, hydatidiform mole, and abortion.

**Clinical Features**

Usually none, but severe isoimmunization can lead to spontaneous abortion, intrauterine foetal death (hydrops foetalis), and neonatal death. Severely affected neonates who require exchange transfusion need to be referred to higher level for appropriate management to avoid hyperbilirubinaemia.

**Investigations**

- Blood groups and Rhesus factor in all pregnant women.
- Rhesus status of husbands of women who are Rh-negative. If he is Rh-negative, then the foetus should be Rh-negative and hence no risk of isoimmunization in the mother. Do remember, however, that extramarital pregnancies do occur.
- Rhesus antibody screening in those who are Rhesus-negative (i.e.,indirect Coombs'test) as soon as possible and every month starting at 20 weeks.
- If Rhesus antibody titreis above 1:8 then do amniocentesis for bilirubin spectrophotometry. The results of this are read on the Liley's graph and the pregnancy managed accordingly.

**Management –** Level 4–6 with Obstetrician and Paediatrician

Pregnancies that are severely affected while the foetus is premature can benefit from intrauterine transfusion. Rhesus disease should be managed by an obstetrician.

**Prevention**

- A Rh-negative woman who delivers a Rh-positive baby must have anti D 500mcg IM within 72 hours of delivery if they are not already isoimmunized (i.e., Rh antibody negative or negative indirect Coombs test, or rhesus- negative baby)
- The same applies for un-isoimmunized Rh-negative mothers who have an abortion, ectopic pregnancy, hydatidiform mole, or obstetric amniocentesis.

## 58.12 Urinary Tract Infection (UTI) in Pregnancy

This is an infection of the urethra, bladder, ureter, and kidney. It is more common in pregnancy because of the physiological changes that cause dilatation of the urinary system and relative stasis of urine. Glycosuria and amminoaciduria in pregnancy also encourage bacterial growth. UTI can lead to abortion, premature labour, low birth weight, and intrauterine growth retardation.

### 58.12.1 ASYMPTOMATIC BACTERIURIA

**Clinical Features**

This condition occurs when there are 100,000 or more bacteria per milliliter of urine without any symptoms. It occurs in 2–10% of all pregnant women. If left untreated, pyelonephritis will develop in2 5–30%.

**Investigations**

- Urinalysis
- Urine culture and sensitivity

**Management**

- Oral antibiotic therapy, oral amoxicillin/clavulanate 625mg 12 hourly ***OR*** PO nitrofurantoin 100mg 8 hourly ***OR*** erythromycin 500mg 8 hourly. All for 10 days.
- Can be managed at all levels of health care provided culture and sensitivity results are provided.

### 58.12.2 URETHRITIS AND CYSTITIS

**Clinical Features**

There is dysuria, frequency, urgency, hesitancy, suprapubic pain, and false labour.

**Investigations**

Urine specimen for microscopy, culture and sensitivity

**Management**

- Advice on adequate hydration
- Oral antibiotic therapy as above
- Pain relief using hyoscinebutylbromide 20mg TDS ***OR*** paracetamol 1g TDS for 5 days.

### 58.12.3 PYELONEPHRITIS

**Clinical Features**

There is fever, vomiting, renal angle tenderness, particularly on the right, and rarely premature labour.

**Investigations**

- Urine culture will usually grow E. coli or K.enterobacteria.

**Management**

- Admit immediately
- Hydration using intravenous fluids normal saline 500ml to run for 8 hours.

- Antibiotic therapy as aboveu ntil the patient responds. Then continue orally for 10 days. If patient is vomiting, ampicillin 500mg IM QDS then change to oral therapy for 10 days.
- Recurrence cases are high and may indicate resistant organism, urologic abnormalities (e.g., polycystic kidneys),renal calculi, ureteric obstruction,or perinephric abscess. Ultrasound if available may be helpful. However, x-ray examinations may be done after the puerperium.

# 58.13 Intrapartum Care and Complications

## 58.13.1 NORMAL LABOUR AND DELIVERY

Normal labour and delivery can be managed at all levels of health care. It should be managed by a skilled provider linked to emergency obstetric care (EmOC) facilities through an effective referral system. Normal labour is characterized by onset of regular uterine contractions at term, accompanied by progressive cervical dilatation and expulsion of the foetus.



### STAGES OF LABOUR

**Labour is divided into 3 stages:**

- 1st Stage: From onset to full dilatation of the cervix.
- 2nd Stage: From full dilatation to expulsion of the foetus.
- 3rd Stage: From delivery of the baby to delivery of placenta.

### MANAGEMENT OF LABOUR

**Proper management of labour reduces maternal and perinatal mortality and morbidity. It includes:**

- Provision of a rapid counselling and testing for HIV for those who missed during prenatal period.
- Making correct diagnosis of labour, with cervical effacement and dilatation 3– 4cm and regular uterine contractions.
- Regular assessment consisting of maternal BP TPR 1 hourly, foetal heartrate half hourly, VE 4 hourly
- Use of partogram, which is a simple but essential tool in labour management. It is a graphic display of labour record to show progress of labour in terms of cervical dilatation, descent of the head, foetal condition, and maternal condition. An “alert line” and an “action line” should be noted. Parameters are charted against time. The partogramis especially useful where there is shortage of staff, and where majority of labours and deliveries are managed by midwives, clinical officers, or medical officers, or if patients have to be transferred to other facilities for operative deliveries (e.g.,caesarean section).
- The expected rate of cervical dilatation is at least 1cm/hour: Avoid artificial rupture of membranes unless there is a cleari ndication.
- Vaginal examination is done at least 4 hourly to assess cervical dilatation, moulding, caput, position. Descent is assessed by abdominal palpation, noting the number of fifths of the head felt above the pelvic brim.
- Foetal condition is monitored by the foetal heart sounds and the colour of the liquor.

- Maternal condition is monitored by BP, temperature, pulse, and urinalysis. Most normal labours are completed by 12hours. The few(approximately 20%) that go beyond 12 hours should be critically evaluated to rule out cephalopelvic disproportion (CPD), inadequate uterine contraction, malpresentation, or malposition.

**Management – Supportive**

**Proper management of the 1st stage ensures the woman reaches 2nd stage strong enough for safe delivery. Patients in labour require:**

- Psychological support.
- Appropriate analgesia if desired by patient,e.g.,morphine 10mg IM STAT at 4–6cm cervical dilation.
- Hydration and nourishment.

**Management –Pharmacological**

- Oxytocin drip indicated for inadequate or in coordinate uterine action in absence of cephalopelvic disproportion or foetal distress:
  - Dose is 2.5–5 IU in 500ml of 5% dextrose starting at 10 drops per minute (DPM) increasing by 10 DPM every half hour to maximum of 60 DPM or when 3 contractions in 10minutes, lasting over 20 seconds, are achieved.
  - Contraindicated in Para 5 and above and in patients with a previous scar, who should be referred to operative delivery.

- Dextrose drip (5% or 10%):
  - Indicated in mild foetal distress (light meconium staining of liquor with normal foetal heart rate) and maternal dehydration.
  - Flush with normal saline, then give at 30 drops per minute or 20 cc of 50% dextrose bolus.

## NORMAL DELIVERY

**Clinical Features**

Second stage (full dilatation) is recognized to have been achieved when contractions become strong and frequent, patient grunts and bears down and develops the urge to push, the head further descends, the perineum bulges and the overlying skin becomes tense and glistening, and the anus may “gape”.

**Management**

- Full dilatation should be confirmed by digital vaginal examination (VE).
- Mother should be encouraged to bear down with contractions and relaxin between.
- At crowning, perineum should be supported with the fingers to prevent perineal tear.
- If necessary episiotomy should be done at this time under local anaesthesia.
- When head is born, it is allowed to rest, the cord round neck is checked and loosened if present.
- Anterior shoulder is delivered followed by the posterior.
- Oxytocin 10 IU IM is given after delivery of shoulders (hypertension,cardiac disease, delivery of first twin).
- Cord is clamped and cut, leaving adequate length for administration of drugs if needed.

- Application of tetracycline 1% eye ointment is recommended as prophylaxis against ophthalmia neonatorum.
- APGAR (A=appearance,P=pulse,G=grimace,A=activity,R=respiration) scoring is done.
- Identification tag applied; baby is wrapped in warm towels and given to the mother to introduce breastfeeding.
- Baby is given a full physical examination when stable.
- Following delivery of the baby, the mother is observed for signs of placental separation indicated by uterus becoming harder and more globular, occurrence of sudden gush of blood per vagina, rising higher of the uterus in the abdomen, and lengthening of the cord outside the vagina.. When this happens:
  - The placenta should be delivered by controlled cord traction.
  - The uterus should be gently massaged.
  - The placenta and membranes should be examined for completeness, infarcts, retroplacental clot, and any other abnormalities.
  - The placenta should be weighed.
- The perineum, vagina, and cervix are then examined for tears. The episiotomy and any tears discovered are repaired immediately. Patients are then observed closely for 1–2 hours before being transferred to the postnatal ward. This period of observation after delivery of the placenta is called 4th Stage of Labour and involves monitoring of blood pressure (BP), temperature (T), and pulse rate hourly, together with uterine palpation, vulva inspection, and estimation of degree of blood loss.



## 58.13.2 COMPLICATED LABOUR AND DELIVERY

Complications of labour may affect the mother, the baby, or both. Most complications are associated with obstructed labour. Cephalopelvic disproportion (CPD) is the major cause of obstructed labour and ruptured uterus.

**Maternal complications of labour include:**

- Genital tract infection
- Fistula formation
- Laceration of the genital tract
- Peripheral nerve palsies
- Foot drop

**Foetal/infant complications of labour include:**

- Foetal distress
- Meconium aspiration
- Hypoxia/Asphyxia
- Injuries
- Foetal death

## CEPHALOPELVIC DISPROPORTION (CPD)

This occurs when the baby is too big for the pelvis or the pelvis is too small for the baby. CPD may be due to faults in the pelvis or faults in the foetus or a combination of both.

- ♦ The faults in pelvis maybe:
  - Contracted pelvis
  - Deformed pelvis
- ♦ The faults in the foetus maybe:
  - Too large baby
  - Hydrocephalus
  - Foetal monsters
  - Locked twins (rare)

CPD is the most important cause of obstructed labour. Other causes of obstructed labour are malpresentations or malpositions of the foetus, and soft tissue abnormalities of the genital tract. Obstructed labour is the commonest cause of ruptured uterus and a major cause of maternal mortality. Obstructed labour and ruptured uterus can be prevented by appropriately timed caesarean section.

## OBSTRUCTED LABOUR

**The requirements for a diagnosis of obstructed labour are:**

- ♦ The cervix fails to dilate despite good uterine contractions.
- ♦ There is oedema of the cervix and vulva.
- ♦ The head fails to descend.
- ♦ The degree of moulding increases.
- ♦ Bandl's ring occurs.
- ♦ There is urinary retention, blood stained urine on catheterization.
- ♦ There is foetal distress.
- ♦ There is maternal distress, manifested by:
  - Dehydration
  - Fever
  - Tachycardia

**Management**

- ♦ Give supportive management.
  - Resuscitation:
    - – Rehydration (IV fluids),
    - – Parenteral antibiotics: Ceftriaxone 1g BD, metronidazole 500mg TDS, and gentamicin 80mg TDS.
    - – Bladder care (empty bladder and continuous bladder drainage for atleast 7–14 days).
  - Relief of obstruction:
    - – Caesarean section or
    - – Destructive operation if the foetus is dead.
- ♦ Do laparotomy: if there is rupture of the uterus:
  - Repair or
  - Subtotal hysterectomy.
- ♦ Initiate physiotherapy.

## RUPTURED UTERUS

**Ruptured uterus is an obstetric catastrophe and should be prevented. Major causes are:**

- Obstructed labour
- Previous operations on uterus (C/S,myomectomy)
- Ecbolic herbs and improper use of oxytocin
- Grand multiparity
- Perforations during evacuation of uterus or D&C are a type of ruptured uterus

**Clinical Features**

- Clinical features may be insidious ("quiet") or obvious ("classical"). In classical cases the patient who was in labour complains of severe abdominal pains, has PV bleeding and goes into shock. Examination shows hypovolaemic shock with signs of intraperitoneal haemorrhage.
- Impending rupture of the uterus can be diagnosed by:
  - Observing rise in maternal pulse (more than 100 beats per minute).
  - Localized abdominal pains.
  - Foetal distress (irregular foetal heart, meconium stain).
  - PV bleeding.

**Management**

- Quick resuscitation with drip,blood.
- Cross-match adequate blood.
- Arrange for laparotomy as soon as possible or refer.
- Decision to repair the tear or remove uterus (hysterectomy) depends on extent and number of tears. Whichever is best to achieve haemostasis quickly is done.

## CAESAREAN SECTION

- When properly applied, caesarean section is an important operation in reducing maternal and perinatal mortality and morbidity.
- The major indications for caesarean section are:
  - Cephalopelvic disproportions (CPD)
  - Foetal distress
  - Previous caesarean section: 2 or more caesarean sections or 1 caesarean section with CPD
  - Malpresentations: Breech, transverse lie
  - Cord prolapse or presentation
  - Antepartum haemorrhage
  - Placenta praevia (major types), placental abruptions (sometimes)
  - Hypertensive disease: Where induction is unlikely to succeed or is contraindicated
- Types of caesarean section operation:
  - Lower uterine segment transverse incision – Routinely done nowadays because of its low morbidity and safety during subsequent pregnancies.

- Classical caesarean section–Vertical incision in upper uterine segment; done very rarely for:
  - Inaccessible lower segment because of tumours or adhesions
  - Avoiding dissemination in cancer of cervix
  - Impacted shoulder presentation

♦ Preparation for caesarean section and procedure:
- Catheterization of the bladder inserted in the theatre.
- Empty the stomach (if not fasted).
- Premedicate with atropine IM 0.6 mg half an hour before operation and start antibiotics at a high dose crystalline penicillin 5 mega units for a clean operation and ceftriaxone 1g STAT if infection is suspected.
- Cross-match1–2 units of blood, fix drip normal saline 500 ml over 8 hours (15 drops perminute).
- Anaesthesia may be general or regional; requires special skills to avoid foetal respiratory depression and maternal gastric acid aspiration.
- Preparation of operation field done when mother is awake to shorten induction delivery interval to 10 minutes or less.
- Incision through the abdomen and uterus done quickly (but carefully) to avoid foetal respiratory depression.

♦ Post operative care after caesarean section:
- Patient requires:
  - IV fluids normal saline 500ml alternate with 5% dextrose 500ml every 6 hours for 24hours,
  - Analgesia morphine IM 10mg every 4 hourly if required and give antibiotics if indicated ceftriatoxe 1g OD 3 days.
  - **Close observation, vital signs, BP, temp, pulse, respiration half hourly for the first hour or until awake and then monitor every 4 hours.**
- Early postoperative ambulation is encouraged.

♦ Chest and leg exercises are also given to prevent hypostatic pneumonia and deep venous thrombosis (DVT).
♦ Patient can be discharged from 4 to 7 days.
♦ Alternate stitches are removed on the 6$^{th}$ day and all stitches on the 7$^{th}$ day.

## INDUCTION OF LABOUR

♦ This is artificial initiation of the process of labour; the indications are:
- Intrauterine foetal death from any cause
- Prolonged gestation (postdates, 41 weeks and above)
- Diabetes mellitus
- Pre-eclampsia and eclampsia
- Rhesus isoimmunization

♦ Technique for induction o flabour:
- Generally, induction is achieved by ARM and oxytocin drip as described above in active management of labour according to Bishops score:
- If 7 and above, warm bath and then ARM and oxytocin 5 IU in 500ml of 5% dextrose (10 drops per minute for 30 minutes and increase by 10 drops

every half hour to a maximum of 60 drops per minute or 3 contractions per 10 minutes, whichever is earlier).

- If less than 7, cervical ripening is indicated. The following option is available:
  - Foley's catheter (can only be used when the membranes are intact) inflated maximally and left for 8–12 hours will normally achieve ripening.
  - Misoprostol 25mcg tablet inserted per vaginum.

### OPERATIVE VAGINAL DELIVERY

Level 3 with specially trained, competent and experienced provider may perform vacuum delivery (ventouse). Indications and case selection must be appropriate to avoid maternal and/or foetal injuries. These include:

- Poor maternal effort.
- Delayed second stage (within 30 minutes from full dilatation) in the absence of CPD.
- Cord prolapse in 2nd stage.

**Requirements for vacuum delivery are:**

- Cephalic presentation
- Full cervical dilation
- Low head (good descent)
- Empty bladder
- Episiotomy

**Contraindications for vacuum delivery are:**

- CPD
- Previous caesarean or myomectomy scar
- Malpresentation (breech, transverse lie, oblique, etc.)
- Malpositions (brow and face malpositions)

## 58.14 Postpartum Care and Complications

Post-natal care is the care of the woman in the immediate postpartum period and within 6 weeks of delivery. This is the time the woman is returning to her normal pre-pregnant status. Postnatal care can be given at all levels by a skilled provider appropriately supported. Targeted postnatal care has a minimum of 3 checkups. The emphasis is starting early in the postpartum period, with the 1st review 24 to 48 hours after delivery, the 2nd review within 2 weeks after delivery, and the 3rd review between 4 and 6 weeks after delivery.

The aim of post natal care is to protect and promote maternal and infant health, support breastfeeding, and provide family planning counselling and services.

### 58.14.1 IMMEDIATE POSTPARTUM CARE

**This includes the following:**

- Repairing the episiotomy as soon as possible.
- Observing and monitoring maternal BP, pulse and temperature closely for 1–2 hours.

- Ensuring that the uterus is well contracted, lochia loss is normal, and urine has been passed.
- Encouraging the mother to establish bonding and initiate breastfeeding.
- Giving paracetamol 2 tabs TDS for after pains and episiotomy pain and providing rapid counselling and testing for HIV for those whose status is unknown and also giving the prophylactic ARVs to the baby (within 72 hours) if mother is positive.
- Transferring the mother to postnatal ward.
- Continuing the above observations at least twice daily.
- Encouraging rooming-in (or "bedding-in") of mother and baby.
- Continuing to give paracetamol 2 tabs TDS.
- Advising on nutritious diet, and generous fluid intake for successful lactation.
- Giving the baby first immunizations (BCG and first polio).
- Documenting and notifying the birth to the civil registrar.
- If no problem, discharging after 24–48 hrs to avoid ward congestion. Women who deliver at home should come for checkup with their babies within 24–48 hours.

## 58.14.2 FOLLOW UP VISITS AND REVIEW

A follow up is carried out at 1–2 weeks to check and treat for secondary PPH, sub-involution of the uterus, puerperal infection, and whether baby is well and breastfeeding. For those not breastfeeding, the visit and review should be at 1 month for family planning.

**Otherwise 3rd visit is at 4–6 weeks to check:**

- For any problems in mother or baby
- Whether periods and/or intercourse has resumed and to provide counselling on family planning, baby care, breastfeeding and immunizations.

**At 6 weeks provide family planning service if required. Suitable methods for lactating mothers include:**

- Progesterone-only pill (e.g.,microlut)
- Intrauterine device("coil")
- Depo-provera or noristerat("injection")
- Voluntary surgical contraception (VSC): Tuballigation
- Norplant/jadelle

## 58.14.3 COMPLICATIONS OF PUERPERIUM

The puerperium is defined as the period 6 weeks following parturition. This is a time when complex adaptations of physiology and behaviour occur in women. Although usually a low risk period, life threatening emergencies or serious complications may occur that must be recognized and managed efficiently. For the majority, however, a minimum of interference is warranted. Those caring for women postpartum should be sensitive to the initiation of family bonding, a special process not to be disturbed unless maternal or neonatal complications arise.

Some of the maternal complications include postpartum haemorrhage, puerperal sepsis, deep vein thrombosis, psychosis, breast engorgement, mastitis, or breast abscess.

## POSTPARTUM HAEMORRHAGE (PPH)

Postpartum haemorrhage is a condition that can sometimes be preventable by proper management of all stages of labour. An understanding of the factors that predispose to postpartum haemorrhage will lead to the practice of precautionary measures that minimize its occurrence. All levels managing labour and delivery (1–6) should be able to diagnose this condition. The skilled health provider should be supported through an effective referral system. Level 1–3 should refer to 4–6 after first aid and should send donors.

Postpartum haemorrhage is defined as bleeding from the genital tract after delivery. It is further defined as primary or secondary postpartum haemorrhage.

- In primary postpartum haemorrhage: Bleeding of more than 500ml within the first 24 hours postpartum.
- In secondary postpartum haemorrhage: Abnormal bleeding occurring after 24 hours and up to 6 weeks postpartum.

Clinical experience and empiric estimates of blood loss are important for diagnosis of postpartum haemorrhage to be made.

Patients at high risk of developing postpartum haemorrhage include the following:

- Prolonged or obstructed labour
- Grand multiparity
- Past history of PPH
- Past history of retained placenta
- Multiple pregnancy
- Polyhydramnios
- Antepartum haemorrhage either placental abruptio or placenta praevia.

**The commonest causes of PPH are:**

- Uterine atony
- Failure of adequate contraction and retraction of uterus after delivery associated with:
- Prolonged labour
- Precipitate labour
- Over-distension of the uterus by,e.g., multiple pregnancy and/or polyhydramnios
- Grand multiparity
- Fibroids
- Halothane use in general anaesthesia
- Concealed haemorrhage in placenta abruptios leading to intramyometrial haemorrhage and manifested as Couvelaire uterus
- Uterine sub-involution.
- Retained placental fragments or membranes. This is a common complication in which there is delay in completion of the 3$^{rd}$ stage of labour due to adherent

placenta. Adherent placenta manifests usually as actual placental invasion of the myometrial wall in the following forms:
  - Placenta accreta: Which is superficial myometrial invasion.
  - Placenta increta: Which is deep myometrial invasion.
  - Placenta percreta: Which is uterine perforation by placenta.
- Lacerations or tears of the birth canal: This can be cervical,vaginal,or vulvoperineal.
- Other causes include disseminated intravascular coagulation (DIC), which is usually secondary to other causes like intrauterine foetal death, amniotic fluid embolism, abruptio placentae, and pre-eclampsia/eclampsia.
- Rupture of the uterus where there is previous scar, oxytocin hyper-stimulation, obstructed labour in multigravidae, and use of ecbolic herbs.
- Uterine inversion and when there isexcessive cord traction, adherent placentae, manual removal of placenta, and poort echnique of placental delivery.

**Investigations**
- Hb or PCV, most important
- Bleeding time
- Clotting time
- Coagulation factors

**Management**
- General measures include:
  - Put up an IV line
  - Take blood for group and cross-match
  - Put in a self-retaining catheter,Foley
  - Determine cause
- Specific measures
  - These depend on the cause

## UTERINE ATONY
- Do a bimanual uterine massage and express any clots; this may also provoke contractions.
- Put up an oxytocin drip 20 units in 500ml dextrose or normal saline to run at 20 drops per minute for about 2 hours.
- Give prostaglandins when and where available, as these are also useful:
  - Misoprostrol 600mcg orally or per rectum.
- Surgery:
  - Subtotal hysterectomy if above measures do not achieve haemostasis.

Retained placenta also causes uterine atony. The following is recommended:

- Apply general measures as above.
- For manual removal of the placenta in lithotomy position on the delivery couch, administer:
  - Morphine 10mg IM STAT
  - 10mg diazepam IV,then
  - Try manual removal of placenta using the ulnar surface of the righth and with the left hand supporting the uterus. If this is not possible, see below.

## ADHERENT PLACENTA

- This will require management in the major theatre in some cases of placenta accreta for manual removal and limited instrument use, e.g., ovum forceps, blunt curette under general anaesthesia.
- Other types will require surgery, i.e., subtotal hysterectomy.

### LACERATIONS/TEARS OF GENITAL TRACT

## CERVICAL TEAR

**The following is important for cervical tear:**

- Review in lithotomy position and in good light.
- Secure a good exposure of cervix by two Sims' speculums.
- Carry out a careful evaluation of the extent of the tear.
- Repair cervix with No. 1 chromic catgut under local anaesthesia (lignocaine HCL 1%) and achieve haemostasis. Then give antibiotics (PO amoxicillin/ clavulanate 625mg BD for 5 days) and PO paracetamol 1g 8 hourly for 3days.

- NB: General anaesthesia maybe required if upper limit of tear is not defined or laparotomy is further required.

## VAGINAL TEAR

**The following are important for vaginal tear:**

- Examine in lithotomy position.
- Carry ligation of bleeders and repair of tears and laceration with No.1 chromic catgut under local anaesthesia (lignocaine HCL 1%).
- Carry out evacuation of haematomata. Then give antibiotics (PO amoxicillin/ clavulanate 625mg BD for 5 days) and PO paracetamol 1g 8hourly for 3days.

## VULVOPERINEAL TEAR

Proper management of episiotomy:

- Define upperend.
- Stitch vaginal epithelium with continuous Chromic catgut No.1 suture under local anaesthesia (lignocaine HCL1%):
  - Stitch muscle layer with the same interrupted stitch.
  - Stitch skin with interrupted catgut.
- Repair all other tears.
- Then give antibiotics (PO amoxicillin/clavulanate 625mg BD for 5 days) and PO paracetamol 1g 8 hourly for 3 days.

**If disseminated intravascular coagulopathy (DIC) develops:**

- Administer fresh blood.
- Administer fresh frozen plasma.
- Carry out surgery as appropriate.

### RUPTURED UTERUS

- Carry out laparotomy and then:
  - Repair of the tear,or
  - Hysterectomy.
- Give broad spectrum antibiotics ceftriaxone 1g OD for 3 days and analgesics morphine 10mg IM 4 hourly for 24 hours.

### UTERINE INVERSION

**Perform manual replacement:**

- If inversion recognized before corpus is trapped,
  - Carry out manual compression an dinsertion
  - Initiate oxytocin drip 20IU in 500ml 5% dextrose 30 drops per minute until the uterus is well contracted and haemorrhage well controlled.
  - The inserting fist should remain until uterine cavity is well contracted.
- If above is not possible then:
  - Give general anaesthesia using halothane to relax uterus.
  - Replace and compress uterus.
  - Use oxytocin as above.
  - Leave fist during the G/A till uterus is well contracted.
- If replacement is not successful with the above measures, then hysterectomy and appropriate treatment are recommended.

## 58.15 Puerperal Infections

These are any postpartum infections of the genital tract complicating labour or delivery. An important contributor is wound sepsis after caesarean section. Extragenital causes of puerperal fever must be considered and looked for. These include upper and lower urinary tract infections, deep vein thrombosis, respiratory tract infections, and mastitis with associated breast engorgement.

**Clinical Features**

There is fever of greater than 38°C during the first 6 weeks after delivery. Other features include lethargy, general malaise, toxicity, dehydration, lower abdominal tenderness, foul-smelling lochia, parametrial pain and thickening and retained membranes**.**

### 58.15.1 PUERPERAL SEPSIS

This is usually a polymicrobic infection presenting as a combination of endometritis, endomyometritis, and endoparametritis. Associated risk factors are: prolonged labour, prolonged rupture of membranes, low socio-economic status, caesarean section, and underlying chronic debilitating disease. Anaerobic organisms are encountered in most infections associated with puerperal sepsis.

**Investigations**

- Haemoglobin, PCV
- Total white cell count (TBC) and differential
- Culture of lochia cervical specimen
- Blood cultures
- Urinalysis and culture
- Sputum: Gram-stain,culture
- Chest x-ray

**Management – General**

**General measures/non-pharmacological therapy on admission:**

- Rehydration: Start an IV line of 500ml normal saline to run over 8 hours.
- At the same time:
  - Take blood for urgent group and cross-match, haemoglobin,whitecell count, blood cultures.
  - Give blood transfusion if necessary.
- Keep patient warm.
- Arrange for infant care in nursery or by relatives.
- Evacuate uterus for any remaining placental tissue or membranes.

**Management – Pharmacological**

- Oral therapy:
  - Amoxicillin capsules 500mg TDS for 5days + metronidazole tablets 200mg TDS for 5 days + paracetamol tablets 2TDS for 5 days.
- Parenteral therapy:
  - Ceftriaxone injection 1g IV or IM BD + gentamicin 80mg IV or IM TDS+ metronidazole 500mg IV TDS, all for 3 days then oral treatment.

**Management – Surgical**

- Laparotomy to be done if any complicating sequalae occur, the most common one being pelvic abscess. Others are abdominal abscess and diffuse peritonitis.
- Wound sepsis following caesarean section may require surgical wound debridement to remove haematomata, necrotic material.
- Admit if
  - Patient toxic
  - Patient febrile >39°C
  - Patient dehydrated
  - Patient not able to take oral drugs
  - Pelvic abscess suspected

## 58.15.2 SEPTIC PELVIC THROMBOPHLEBITIS

This condition occurs with development of ovarian vein thrombophlebitis in a patient with preceding pelvic soft tissue infection. Presenting as a definite mass extending caudally, this is a rare condition that is diagnosed mainly by exclusion and has poor response to therapy.

**Treatment**

- Give ceftriaxone injection 1g IV 12 hourly + gentamicin 80mg IV 8 hourly + metronidazole 500mg IV 8 hourly and IM diclofenac 75mg 12 hourly, all for 3–5 days, then oral treatment for 5–7 days as above including heparin10,000 units 6 hourly subcutaneously until symptoms (pain, swelling and warmth of the involved limb) abate. Then taper heparin dosage within a week (heparin 10,000 units for 2 days then heparin 5,000IU for 2 days and then finally heparin 2,500 IU for 3 days, then stop the heparin).
- Start warfarin 2mg OD for 2 days with the heparin at 5,000 IU, then warfarin 4mg OD for 3 days with heparin 2,500 IU ) and then stop the heparin and change to oral warfarin 5mg OD a day for 3 months. Monitor heparin with KCCT(Kaolin Cephalin Clotting Time) and warfarin with Prothrombin Time Index (PTI).
- Ensure availability of antidotes (protamine sulphate for heparin overdosage with IV heparin: Give protamine sulphate IV;1mg neutralizes 80–100 units of heparin when given within 15 minutes of heparin. If longer than 15 minutes less protamine is required, since heparin is rapidly excreted. Max of 50mg of protamine sulphate) and (Vitamin K dosage to be added for warfarin).
- Note that surgery may be indicated.

## 58.16 Extra-Genital Differential Diagnoses

These include urinary tract infections, deep vein thrombosis, and respiratory tract infections. Respiratory complications are an infrequent cause of puerperal morbidity. Lobar pneumonia is the most serious infection and may be complicated by atelectasis. Patients who have delivered through caesarean section are most susceptible to developing this condition.



### 58.16.1 BREAST CONDITIONS

**These involve the following conditions:**

- Breast engorgement: This is accompanied by inflammation of breast and fever. Adequate breastfeeding and paracetamol 1g TDS for 5 days are usually sufficient.
- Mastitis: This is infection of the parenchyma of the mammary glands. It may occur any time postpartum but usually 2–3 weeks after. Predisposing factors include:
  - Breastfeeding perse.
  - Fissures in nipple.
  - Recent weaning.

Diagnosis of mastitis is usually made on basis of the pain on the same side, localized cellulitis, and axillary lymph nodes that may be palpable and tender. The most common causative organism is Staphylococcus aureus.

**Management**

- Expressing milk on affected side
- Applying ice packs
- Supporting affected breast.
- Using antibiotics: PO flucloxacillin 500mg 6 hourly for 7 days.

- For pain and inflammation, adding PO ibuprofen 400mg 8 hourly for 3 days.

— *Breast abscess may be a sequelae of mastitis.* In addition to the measures above, incision and drainage will be necessary, as well as stoppage of breastfeeding when there is a purulent discharge. If abscess does not respond to this, refer to a specialist on lactation management.

## 58.16.2 DEEP VEIN THROMBOSIS (DVT)

- Antibiotics: PO amoxicillin/clavulanate 625mg 12 hourly ***OR*** PO flucloxacillin 500mg 6 hourly for 7 days.
- PO ibuprofen 400mg Diclofenac 50mg 8 hourly for 3 days.

**Clinical features, investigations, and general management are described under Section 3.1 in Part I.**

**Management**

- Give heparin 10,000 units 6 hourly subcutaneously until symptoms (pain, swelling, warmth of the involved limb) abate, then taper heparin dosage within a week (heparin10,000 units for 2 days, then heparin 5,000 IU for 2 days, and finally heparin 2,500 IU for 3 days, then stop the heparin).

- Start warfarin 2mg OD for 2 days with the heparin at 5,000IU, then warfarin 4mg OD for 3 days with heparin 2,500 IU, and then stop the heparin and change to oral warfarin 5mg OD a days for 3 months.
- Monitor heparin with KCCT and warfarin with PTI. Ensure availability of antidotes. Use protamine sulphate for overdosage with IV heparin: Give protamine sulphate IV; 1mg neutralizes 80–100 units of heparin when given within 15 minutes of heparin. If longer than 15 minutes less protamine is required since heparin is rapidly excreted. Max of 50mg of protamin sulphate) and vitamin K (major bleeding – stop warfarin, give vitamin K 5–10mg by slow IV if INR>8.0; no bleeding or minor bleeding – stop warfarin and give vitamin K 500µg by slow I : INR 6.0–8.0; no bleeding or minor bleeding stop warfarin restart when INR<5.0). Monitor as above for INR to be in the range of 2.0–2.5 times the normal.

**Patient Education**

- Avoid oestrogen containing contraceptives.
- Non hormonal progesterone only contraceptives are appropriate.
- Avoid prolonged bed rest. Exercise legs even during bedrest.

## 58.16.3 PUERPERAL PSYCHOSIS

**The following are risk factors of puerperal psychosis:**

- Family history of major psychological illness of close relative, e.g.,mother.
- Major emotional complications during and after a previous pregnancy.
- “Reaction” of current pregnancy.
- “Fear” of labour from a previous experience.
- Traumatic childhood.
- Deprivation of emotional support during adult life, e.g., single mother.

- Severe prolonged or multiple somatic symptoms with no apparent organic cause during current/or prior pregnancy.
- Major sustained mood changes or repeated rapid mood swings or abnormal sleep patterns.
- Refer to Mental Illness chapter for clinical features and management.

# 59. Family Planning

Family planning (FP) means that "everyone should plan their family so that all children are born when wanted, expected, and welcome". The health benefits of family planning play a major role in protecting the lives of infants, children, women, and the family as a whole. (See also Family Planning Guidelines for Service Providers, MOH/DRH 2005).



## 59.1 Family Planning Methods

There are many available types of family planning methods, and many categories of people can be involved in the provision of FP advice, information, and services. This is as long as such people have received the necessary training and instruction. Similarly, FP can be provided in varied settings (from levels 1 to 6) and within facilities operated by various providers (public, mission, private), provided they conform to the basic requirement for the provision of the particular FP method. (FP Guidelines for Service Providers MOH/DRH 2005).

Refer to Table 60.1 for a summary of the different types of methods and their suitability for different types of clients. Table 60.2 provides a guide to the various methods in terms of their effectiveness, ease of use, compatibility with breastfeeding, return to fertility after stopping, and other pertinent issues.

## 59.2 Hormonal Contraceptives

Methods in this category work by affecting the body's hormonal system in various ways. They are contraceptives only, and do not provide protection against STIs and HIV.

**Table 59. 1 Family planning methods and their suitability for various types of users**

| Method recommended for the group | Not recommended for the group |
|---|---|
| *Combinedpill* | With suspected pregnancy |
| Women under 40 years, of any parity<br>Women who want highly effective contraception<br>Breastfeeding mothers after 6 months postpartum<br>Younger women/adolescents who are sexually active and have been adequately counselled | Who are over 35 years and smoke<br>With history of blood clotting disorders or heart disease<br>With lump in either breast, liver disease With unexplained abnormal vaginal bleeding. With BP over 140/90mm/Hg confirmed on revisit<br><br>With suspected pregnancy |
| *Progestin-only pill* | With history of blood clotting disorders or heart |
| Women of reproductive age, of any parity<br>Breastfeeding mothers after 4–6 weeks postpartum | disease<br>With lump in either breast, liver disease<br>Unexplained abnormal vaginal bleeding<br><br>With suspected pregnancy |
| *Injectable methods* | With history of blood clotting disorders or heart |
| Women of proven fertility Breastfeeding mothers after 6 weeks postpartum<br>Women who want long-term contraception<br>Women who want atleast 2y ears between pregnancies | disease<br>With lump in either breast, liver disease With unexplained abnormal vagina lbleeding |
| *Implants* | With suspected pregnancy |
| Women needing long-term protection<br>Breastfeeding mothers after 6 weeks postpartum<br>(Long term highly effective contraception)<br>Women who have their desired family size but do not want permanent surgical contraception | With history of blood clotting disorders or heart disease<br>With lump in either breast, liver disease With unexplained abnormal vaginal bleeding<br>With suspected pregnancy, history of PID or Ectopic pregan |
| *Intrauterine devices* | With anaemia or heavy menstrual bleeding |
| Women who have delivered 1 or more times<br>Breastfeeding mothers<br>Women who want long-term contraception<br>Women in a stable monogamous sexual relationship<br>Women after 6 weeks postpartum; before 6 weeks if provider has specialized IUD insertion training | Having no menses after 6 weeks postpartum<br>With history of heart disease<br>With abnormalities or cancer of pelvic organs<br>Having unexplained vaginal bleeding or severe menstrual pains<br>At risk of exposure to STDs<br><br>Who desire or require highly effective protection against pregnancy<br>Who are allergic to latex |
| ***Male and female condom*** | |
| Men who desire to take contraceptive initiative<br>Couples needing an immediately effective method<br>Couples waiting to rule out a suspected pregnancy<br>Couples at risk of exposure to HIV, STDs | *Continued* |

**Table 59.1, *continued***

| Method recommended for the group | Not recommended for the group |
|---|---|
| Natural family planning | |
| Couples willing to learn about the woman's cycle and to practise abstinence 1–2 weeks each cycle<br>Couples who, for religious or any other reasons, desire to practise periodic abstinence | Who need/want more effective contraception<br>With irregular menstrual cycle<br>Who are breastfeeding<br>Who must not become pregnant for health or any other reasons<br>Who are unwilling to abstain during fertile period |
| Tubal ligation or vasectomy | |
| Couples or individuals who have been fully counselled,understand and have voluntarily signed consent form<br>Couples with desired family size<br>Women for whom age or health problems might cause an unsafe pregnancy<br>Couples who are certain they want no more children regardless of accidental death of a child or children | Who do not fully understand VSC(Voluntary surgical contraception) or are unwilling to agree to items on the consent form<br><br>Note: Men or women whose spouses oppose VSC should be considered on a case by case basis for the procedure. |

**Table 59.2:Guide to family planning methods**

| Method | Pregnancy rate? | Used at inter-course? | Effect on STD risk? | Compatible with breast-feeding? | Return to fertility after stopping? |
|---|---|---|---|---|---|
| Male sterilization | 0.15 (0.1) | No | None | Yes | Permanent method |
| Female sterilization | 0.4 (0.2) | No | None | Yes | Permanent method |
| Implants | 0.2 (0.04) | No | Probably none | Yes, but not preferred method. Wait 6 weeks postpartum | Immediate on removal |
| Combined oral contra-ceptives | 1–8 (0.1–3) | No | May protect against some forms of PID, but increase risk of infection with some STDs | After 6 months postpartum, but not preferred method if breastfeeding | Immediate to short delay (average 2–3 months) |
| Progestin-only minipill | 3–10 (0.5–3) | No | None | Yes, but not preferred method. Wait 6 weeks postpartum | Immediate to short delay |
| Injectables | 0.3–0.4 | No | Unknown | Yes, but not preferred method. Wait 6 weeks postpartum | Delayed 4–12 months |

*Continued*

**Table 59.2,** ***continued***

| Method | Pregnancy rate? | Usedat inter-course? | Effect on STD risk? | Compatible with breast-feeding? | Return to fertility after stopping? |
|---|---|---|---|---|---|
| Intrauterine devices (IUCD) | 3 (0.3–2) | No | Increase risk of PID in women at risk of STDs | Yes | Immediate after removal by trained provider |
| Condoms | 12 (2) | Yes | Protective (70% against AIDS) | Yes | Immediate |
| Natural family planning | 20 (1–9) | No | None | No, method not reliable | Immediate |

## 59.2.1 COMBINED ORAL CONTRACEPTIVE PILL

This pill contains a combination of progestogen and oestrogenin proportion and quantity that vary across the various preparations. The pill acts by inhibiting ovulation and thickening cervical mucus, thus providing a physical barrier to spermatozoa and making the endometrium too thin for implantation.

**Client Education**

**This should contain the following information with regard to the pill:**

- It is highly protective against pregnancy.
- Pregnancy rate increases if the pill is not taken regularly.
- It may be associated with minor complaints, such as nausea, headache, weight gain, and gastrointestinal upsets.
- It is unsuitable for breastfeeding mothers because of its suppressive effect on milkoutput.
- If you forget to take a pill, take it as soon as you remember. Take the next pill at the regular time even if this means taking 2 pills on the same day.
- Return to the clinic in case of the following:
  - Suspected pregnancy
  - Swelling or pain in legs
  - Yellowing of skin or eyes
  - Pain in abdomen,chest,or arms or shortness of breath
  - Severe headaches, depression, or vision difficulties
- Side effects: Although many side effects of oral contraceptives use have been eliminated with low dose pills, some women still experience irregular menstrual bleeding, nausea, weight gain, headaches, skin colour changes, and other side effects. These may go away after several months or continue as long as oral contraceptives are taken.
- Complications:
  - There is increased risk of cardiovascular disease in women over 35 years of age who smoke.
  - There is increased risk of hypertension.
  - Users exposed to STIs maybe at risk of serious diseases, including PID and possibly cervical cancer.

- Non-contraceptive benefits:
  - Reduces menstrual flow (lighter, shorter periods).
  - Decreases dysmenorrhoea.
  - Protects against ovarian and endometrial cancer.
  - Decreases benign breast disease.
  - Gives some protection against ectopic pregnancy.

## 59.2.2 PROGESTOGEN-ONLY PILL

This is a pill that is taken daily and contains only a progestogen. It acts by altering cervical mucus, making it thicker/denser, thus preventing sperm transport. It also suppresses ovulation and inhibits implantation of fertilized ovum.

**Client Education**

**This should include the following:**

- Used in breastfeeding mothers because it does not interfere with lactation.
- Has a high level of pregnancy protection.
- There is need for compliance on a daily regimen.
- Unrelated to sexual intercourse.
- May cause menstrual irregularities.
- If client forgets to take 1 pill, take it as soon as they remember (see combined pills)
- Clients should return to the clinic immediately for a pregnancy check if 45 days have passed since the last menstrual period.
- Side effects: Users may experience irregular bleeding patterns.
- Complications: Studies to date have shown no long-term complications.
- Non-contraceptive benefits:
  - Does not affect lactation.
  - Lighter shorter periods.
  - Decreased breast tenderness.
  - Does not increase blood clotting.
  - Decreases dysmenorrhoea.
  - Protects against endometrial cancer.

## 59.2.3 EMERGENCY CONTRACEPTIVES

**Emergency contraceptives reduce the occurrence of pregnancy in unprotected intercourse from 8% to 2% (75% protection). Indications are the following:**

- Unprotected intercourse
- Rape
- Condom leakage
- Condom breakage/slippage.

### COMBINED ORAL EMERGENCY CONTRACEPTIVES

- Ethynyl oestradiol 50mcg + levonorgestrel 150µg 2tabs orally STAT and 2 tabs 12 hours later.
- Ethynyloestradiol 30mcg+levonorgestrel 150µg 4 tablets STAT followed by 4 tabs after 12 hours.

### PROGESTERONE ONLY EMERGENCY CONTRACEPTIVE

Levonorgestrel 750µg 1 tab STAT and 1 tab after 12 hours.

## 59.2.4 INJECTABLE CONTRACEPTIVES

These are either progesterone only or combined progesterone plus oestrogen. They comprise long-acting progestogen usually administered as deep intramuscular injections. They act by suppressing ovulation, inducing a thin atrophic endometrium, and producing a thick cervical mucus that is difficult for sperm to penetrate. They are available in these forms:

- Depot Medroxy progesterone Acetate (Dmpa): 150mg per vial and given as a deep (depot) intramuscular injection every 3 months
- Norethisterone Enanthate (Net En): 200mg vials given at 2-month intervals

**Client Education**

- They may be associated with heavy menses, amenorrhoea, or spotting.
- Regular administration is required.
- It is necessary to return to the clinic as scheduled to continue using this method.
- Return to the clinic if one suspects pregnancy, or experiences dizziness or heavyb leeding.
- Side effects: Users may experience menstrual irregularity (amenorrhoea, spotting, and, rarely, heavy bleeding).
- Complications: Studies to date have shown no long-term complications.
- Advantages: They contain natural oestrogens and hence have a protective effect on CVS and CNS and give better cycle control.

## 59.2.5 SUB-DERMAL IMPLANTS

Implants consist of 2 rods of levonogestrel 75mg that are inserted under the skin of the arm. These rods slowly release progestogen for up to 5 years. They act by thickening cervical mucus, suppressing ovulation, and causing atrophic changes in the endometrium that make it unsuitable for zygote implantation. Etonogestrel 68mg is a single rod of progesterone-only contraceptive implant that gives protection for years

**Client Education**

The following is important:

- May be associated with prolonged menses, spotting, or amenorrhoea.
- Requires a minor surgical procedure for insertion and removal.
- If possible, return to the same clinic if you desire implant or removal.

- Return for removal anytime you desire, but it can be kept in place for 5 years.
- Return to the clinic if you:
  - Suspect pregnancy
  - Experience pain, swelling or pus at the implant site
  - Experience dizziness or headache.
  - Experience heavy bleeding
- Benefits include the following:
  - Highly effective
  - Immediate return to fertility
  - Continuous, long-term protection
  - Reduced menstrual flow
  - Protection against endometrial cancer and ectopic pregnancy
  - Does not affect lactation
- Side effects: Users may experience infection at the insertion site, irregular menstrual bleeding (longer bleeding episodes, amenorrhoea, or spotting).
- Complications: Studies to date have shown no serious long-term complications.



## 59.3 Intrauterine Contraceptive Devices(IUCD)

IUCDs form a highly effective, long-term family planning method that is in widespread use around the world. The modern IUCD is a plastic device usually bound with copper wire that is placed in the uterus through the cervix. Lippes's loop has no copper. The IUCDs act by preventing implantation of fertilized ovum, inhibiting sperm mobility, and inhibiting fertilization. Copper T 380 A is effective for 12 years.

**Client Education**

The following is important:

- It is important to check regularly to ensure IUCD is in place
- May cause dysmenorrhea and menorrhagia.
- Return to the clinic if you have:
  - Signs of pregnancy, heavy bleeding or spotting.
  - Abnormal sexual pain or vaginal discharge.
  - Chills or fever.
  - Dysmenorrhoea and menorrhagia.
  - Desire for removal.
- Benefits include the following:
  - Highly and immediately effective.
  - Long-term protection with immediate return to fertility upon removal.
  - Does not interfere with intercourse.
  - Can be used by women who are breastfeeding.
- Side effects: Users may experience pain on insertion and increased menstrual bleeding and abdominal cramps for the first 1–2 periods.

- Complications: Increased risk of anaemia if heavy bleeding occurs, perforation (rare) and increased risk of PID and associated infertility, especially within 4 months of insertion and in women at risk of STDs.
- Displaced IUCDs: When threads are not visible at cervix and pregnancy is ruled out, then
  - Attempt removal with an alligator or simple artery forceps.
  - If this fails, then localization by ultrasound, plain x-ray with tracer IUCD and removal.
- If one conceives with an IUCD remove it if possible; otherwise leave alone until delivery (ultrasound if possible) and counsel client accordingly.

## 59.4 Barrier Methods

### 59.4.1 THE MALE CONDOM

Condoms present a physical barrier to sperm deposition into the vagina. Condoms also offer some protection against STIs, including HIV/AIDS, HBV, and carcinoma of the cervix.

**Client Education**

The following is important:

- Before every intercourse, place condom on erect penis, leaving tip empty to collect semen.
- Withdraw the penis from the vagina after each ejaculation while the penis is still erect.
- Remove condom after use.
- Do not re-use condoms.
- Discard used condom immediately in toilet or pitl atrine.
- Using spermicides with condoms increases the effectiveness.
- Complications may include local irritation if allergic to latex/lubricants.
- May interfere with sexual pleasure for some people.
- Benefits:
  - Fairly effective if used properly.
  - Immediately effective.
  - Highly effective protection against STIs/HIV.
  - May prevent premature ejaculation
- Side effects: Some users experience sensitivity to rubber or lubricants.

### 59.4.2 THE FEMALE CONDOM

The female condom is a thin (0.05mm) polyurethane sheath, 7.8cm in diameter and 17cm long. It is soft, loose fitting, and has 2 flexible rings. One ring is inserted into the vagina and acts as an internal anchor. The other ring forms the open edge of the device and remains outside the vagina after insertion.

The female condom provides protection for one act of intercourse. It can be inserted (up to 8 hours) before intercourse but must be removed immediately after. There are no complications associated with it. Unlike the male condom, it can be washed and reused.

## 59.5 Surgical Contraception

Many factors have contributed to improved safety of voluntary surgical contraceptive in the last 30 years. These include improved anaesthetic methods, better surgical techniques, asepsis, improved training of personnel, and better selection and monitoring of clients.

### 59.5.1 TUBAL LIGATION

This is a voluntary irreversible procedure for fallopian tubal occlusion that can be done under general or local anaesthesia by minilaparotomy or laparoscopy.

**Client Education**

- The procedure is more or less irreversible(permanent).
- Failure is very rare when done by trained professional.
- Counselling is absolutely necessary.
- There is no loss of libido or vigour or health.
- It is necessary to return to the clinic if the client experiences:
  - Postoperative fever, pus, or pain at the surgical site.
  - Weakness or rapid pulse.
  - Vomiting or persistent abdominal pain.
- Benefits:
  - Permanent; highly and immediately effective.
  - No change in sexual function.
  - Good for client if pregnancy would be a serious health risk.
  - Does not affect lactation.
- Side effects: Some users experience minor pain, bleeding, and wound infection following procedure.
- Complications: Injury to other organs (e.g., gut, bladder) and – rarely – death. Risk of complications is increased if general anaesthesia is used.

### 59.5.2 VASECTOMY

This is a voluntary surgical procedure, done under local anaesthesia, to cut and ligate the vas deferens so that spermatozoa cannot be ejaculated. It is gradually becoming accepted in Kenya.

**Client Education**

Counselling is necessary, as this procedure is permanent and irreversible.

- It is necessary to use condoms for at least 15 ejaculations or 3 months to ensure azoospermia.
- Return to the clinic in case of:
  - Postoperative fever.
  - Excessive swelling, pus, or pain at the surgical site.
- Side effects: Some users experience minor swelling, pain, infection, and bruising following procedure. For pain give ibuprofen 400mg TDS for 5days and for infection flucloxacillin 500mg QDS for 7 days.
- Complications: Risk of serious complications or death is extremely low.

## 59.6 Periodic Abstinence (Natural Family Planning)

In this method, the couple avoids sexual intercourse during ovulation and for a safety margin before and after ovulation. Various techniques may be used to determine the fertile period: cervical mucus, basal body temperature, rhythm. The benefits include:

- No physical side effects and it is cheap.
- No need for prescriptions by medical personnel.
- Improved knowledge of reproductive system and possible closer relationship between couples.

**Client Education**

- Requires high motivation
- Has a high failure rate
- Assumes a regular, perfect menstrual cycle
- Requires proper record-keeping
- Has no health risks, except for pregnancy
- Side effects:None.
- Complications:None.

# PART V: The Referral Framework

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# 60. The Referral Framework

The Government of Kenya is actively promoting comprehensive care for all people at the community level. The elements of comprehensive care include clinical, nursing, psychological, and social support. Maintaining the continuum of care is essential and requires a strong linkage between the community and the health system. This occurs through an effective and efficient referral system.

A referral system is a network of service providers and facilities that link to provide a continuum of care for acute and chronic illnesses. It includes individuals and organizations working to provide care and support to those who need it. There are typically four levels to a referral network in the health system: the community, primary, secondary, and tertiary.

As defined by the Kenya Essential Package for Health (KEPH), these 4 levels of the referral network incorporate the community level (level 1), with its households, community health workers (CHWs), traditional birth attendants (TBAs), traditional herbalists, and community health extension workers (CHEWs). At the primary care level, dispensaries and health centres (KEPH levels 2 and 3) are the first points of linkage between the community and the formal health system. The CHEWs and health management committees strengthen them. The sub-county hospitals (level 4), the county hospitals (level 5), and the national referral hospital (level 6) provide levels of increasing specialization of care to support the community level. Providers at all of these levels should be able to recognize complications, gauge their severity, provide prompt treatment based on their capacity as defined by the norms and standards for each level of care, and refer any clients they are unable to treat to a facility where they know adequate treatment is available.

A referral system aims to improve clients' access to services, reduce the time it takes to receive critical care, and avoid unnecessary delays. Meeting clients' needs entails a collective effort of many providers, both formal and informal. Legal referral arrangements, proper communication, and standard tools must be in place to strengthen access to existing services and enhance linkages between and among the providers.

The service provider initiating a referral at any level of the referral system is responsible for documenting the referral activity and following up with clients to ensure they receive the necessary care. An adequate system provides continuity and high-quality care to patients, enhancing the utilization of available resources and encouraging clients to participate actively in decisions that directly affect their lives.

Coordinated service delivery and strong communication among health care providers is necessary to ensure that access to required services is as quick as possible, Referrals can be easily traced and followed up, referral outcomes can be documented, feedback from clients on the services they received can be noted, gaps in the system can be identified, and steps taken to

improve service provi- sion. For this, effective communication and transport arrangements are crucial.

The following elements are essential:

- *Availability, accessibility, and affordability:* Services must be based on prevailing local health problems, and provided in a way that local needs can be addressed.
- *Coordination, coordination, coordination:* Referral activities within and between different service providers with different resources and different mandates demand focused attention. This is best facilitated by having a team or specific individuals designated to coordinate these referral activities.
- *Relationships:* Higher level health facility providers should take the lead in establishing and maintaining referrals by supporting lower level providers,with both the clients and the providers working as partners.
- *Effective communication and transport arrangements :* Identification of the most cost-effective means of transport should be done. One way is to choose a member of the community with a vehicle to assist other community members with transport during referrals in such a manner that the costs incurred can be covered and taken care of within such an arrangement.
- *Feedback:* echanisms should be established to help with the tracking of referrals from the point of initiation to the point of delivery. This will provide evidence that the client completed the referral process.
- *Monitoring and quality control:* Monitoring and evaluation mechanisms for the continuous assessment and improvement of the referral process and outcomes are crucial and need to be initiated and maintained.

## 61. General Guidelines

An efficient and effective pyramidal referral system is essential for the effective management of surgical patients. It is especially important in emergency situation to provide rapid and effective treatment to the patients. A referral system can function either upwards or downwards concerning the levels of health care. Upward referral seeks specific medical care from specialists and subspecialists found at the higher levels of health care or even outside the country. Downward referral engages the local facility nearest a patient's home environment because they no longer need the more specialized health care at the higher level. Instead, they require ongoing medical care that is best able to cope with the patient's needs and this often happens to be at locations nearest to the patient's home.

An efficient referral system ensures an appropriate mix of patients with different needs, admitted in different health facilities countrywide. This means that all referrals must be directed at the correct facility while maintaining the normal pyramidal referral system of flow within the health system as much as possible. Besides referrals between facilities, there are also referrals within institutions. Such referrals are necessary and essential for patient's well-being. Hospitals should only manage cases they can handle, and in situations where they cannot

adequately take care of them they must refer them to the next appropriate facility. All referrals must be carefully evaluated and the risks and benefits critically assessed before the decision to refer is made. The basic guidelines for upward referral are shown below and will vary a little depending on the level in question.

## 61.1 Procedure for Upward Referral

**The upward referral consists of the following components:**

1. Critical evaluation and decision to refer is made by:
a) Individual doctor or health care provider.
b)Management team taking care of the patient.
c) Ministry or other administrative body in charge of the welfare of the patient.

2. Documentation is prepared that includes the following:
a)Admission details.
b)Diagnostic details and investigations carried out.
c)Medications and treatments given to the patient.
d) The reason for the transfer of the patient.

**— This documentation MUST accompany the patient being referred.**

3. Appropriate communication with respect to the referral is made:
a) With the receiving unit or health facility.
b) With the relatives

4. Preparation of appropriate transportation is made:
a) Efficient and reliable means of transport to effect the referral is secured.
b) The means of transport secured is exclusively allocated for transportation of the referred patient.
5. An appropriately qualified escort is appointed.
6. A systematic check to ensure that the resuscitation equipment to accompany the patient is available and functioning well.

## 61.2 Procedure for Downward Referral

On completion of treatment at the higher centre, there will be a need to refer the patient back to the initial facility. This is done for purposes of feedback to the facility and for ongoing rehabilitation or palliative care of the patient.

The downward referral is a mirror of the upward referral, except that in this situation the patient has already received specialized care. Being sent back to the lower health facilities is for continuing care, for feedback and for rehabilitation or palliative care.

**The downward referral consequently consists of the following components:**

1. Decision to refer the patient downwards.
2. Documentation detailing the following aspects with respect to the patient being referred:

a) Admission/identification details.
b) The final diagnosis for the patient.
c) Procedures carried out during hospitalization in the referring facility or unit.
d) Medications provided while the patient was hospitalized in the referring health facility.
e) Follow-up details and any rehabilitation requirements.
f) In case of terminal disease the hospice needs to be involved in the referral process and the follow up of the patient.

**There must be legible copies of the referral note or letter: one for the patient, another for the unit receiving the patient, and a third for the file.**

3. Communication is made with receiving unit or facility as appropriate and feedback obtained as appropriate. Such communication enhances the efficiency of the referral system.

4. Communication is made with the relatives with regard to the planned downward referral and the need for the referral for this patient.

5. Preparation is made for the appropriate transportation for the intended referral.

6. An appropriately qualified escort is appointed, although in many situations the relatives would be sufficient to provide such an escort.

7. Booking is made for the patient to be reviewed in the outpatient clinics in the referring facility, unless arrangements are made for the patient to be reviewed in the receiving unit or health facility.

## 61.3 Guidelines for an Institutional Referral System

An institutional referral system needs to be clear and functional. Such a system is just as important as patient care. Each facility needs to have a system for both the upward and downward flow of patients to mirror that at the national level.

A simple institutional referral system should have the following features:

1. Casualty department review

a) Make a correct diagnosis.
b) Call appropriate unit.
c) Ensure patient is reviewed.
d) Ensure patient is handed over to the unit on-call doctor.
e) Ensure documentation is accurate.

2. Making unit referrals and admission decisions:

a) Decision on whether treatment should be provided to the patient on an outpatient basis or after admission as an inpatient.

b) If patient is admitted, ensure the patient is handed over to the admitting ward doctor.

c) If the decision is made to provide care to the patient on an out patient basis, then correct referral should be made.

d) In the event of incorrect clinic referral, the doctor rather than the patient should be responsible for correcting this error.

If the patient referred is not admitted, they should be referred to specialized clinics at the facility. Referrals should be made to the National Referral Centre

## 61.4 Dangers and Barriers to a Coordinated Referral System

All team members at all levels need to be conscious of the dangers that face a coordinated referral system. Effort needs to be made to avoid these dangers to the referral system. These dangers and barriers involve the following:

- Lack of confidence in the facility by the community and the tendency by the community to bypass the facility and go to the next nearest facility they consider more suitable. Such a situation could be due to:
  - Poor community relationships.
  - Poor manpower utilization.

- An infrastructure that is non functional:
  - Broken down, understaffed and under supplied middle level facilities.
  - Inadequate or inappropriate communication infrastructure at the various levels of care. This is exacerbated by poor management practices at the health facilities and failure to involve the community in the process.
  - Treating patients with specified conditions at inappropriate levels.
  - Inadequate funding and supplies for higher levels of health care, so that they are unable to provide expected services.

- Lack of or poor utilization of human resources, due to the following:
  - Brain drain because of perceived low wages, inadequate infrastructure, and non conducive working environments in the public health services, to NGOs or private health facilities with better remuneration, or even out of the country.
  - Poor distribution of staff.
  - Frustration in the workplace.
  - Poor workingrelationships.

- Lack of drugs and other equipment, which is to an extent related to issues of poor planning and inadequate financing.

- Inaccurate diagnosis and treatment plans for facilities because of inadequate training for personnel at the facilities.
- Lack of working quality control and M&E measures.

# PART VI: Principles of Oxygen Use

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# 62. Introduction

The aim of this section is to describe the indications and procedure for use of oxygen therapy and its mode of delivery.
Oxygen is regarded as a drug that must be prescribed when used as a form of medical treatment. Clinicians must bear in mind that supplemental oxygen is given to improve oxygenation, but does not treat the underlying causes of hypoxaemia which must be urgently diagnosed and treated.

## Definitions

**Oxygen therapy**-is a lifesaving treatment that provides the patient with supplemental oxygen through a device.
**Hypoxaemia**-is the medical term for a low blood oxygen level, and should always be monitored and treated by a skilled health worker.
**Hypoxia**-is low oxygen in the tissues such as can occur in acute illness due to failure in any of the systems that deliver and circulate oxygen. Hypoxaemia is the most common cause of hypoxia.

## 62.1 Principles of Oxygen Use

### 62.1.1 SIGNS AND SYMPTOMS OF HYPOXEMIA

These include the following

- Shortness of breath
- Fast breathing
- Increased heart rate and blood pressure, which may progress to low heart rate and low blood pressure if not treated
- Tiredness
- Anxiety or agitation
- Paleness, which may progress to Cyanosis if not treated.
- Headache, which may progress to confusion,
- Blurred vision,
- Loss of muscle coordination and eventually coma if not treate
- Tissue damage due to hypoxia

### 62.1.2 HOW TO DETERMINE WHEN OXYGEN THERAPY IS NEEDED

Apart from using the symptoms, blood oxygen levels can be determined through the following;

i) **Pulse oximetry**: *Pulse oximetry is a test in which a tiny clip like electronic device (the pulse oximeter) is used to find out blood oxygen saturation. For healthy individuals, oxygen will usually fall between 95% and 100%.*

When the blood oxygen levels registers between 90 and 92%, a patient may need supplemental oxygen, and readings below 90% indicate immediate medical attention and intervention is required.

ii) **Arterial blood gas saturation**: *This is a blood gas test that gives a more accurate measure of how much oxygen and carbon dioxide is present in blood. It is a measure of how efficiently the lungs are exchanging gases*.

## 62.1.3 INDICATIONS FOR OXYGEN THERAPY

Some conditions where oxygen therapy can be prescribed include, but are not limited to the following:

- Chronic obstructive pulmonary disease, Pulmonary fibrosis, Pneumonia, Severe asthmatic attack, Cystic fibrosis, pleural effusion, pulmonary embolism, pneumothorax, sleep apnoea.
- Cardiac arrest or resuscitation, acute heart failure, severe anaemia, post - operative breathlessness.
- Hypoxia from excessive bleeding, shock, sepsis, major trauma, drowning, anaphylaxis, major pulmonary haemorrhage, status epilepticus and carbon monoxide poisoning

***Note: Refer to the specific section dealing with these conditions for the detailed information on their management and oxygen therapy in those conditions***.

## 62.1.4 ADMINISTRATION OF OXYGEN/STARTING THERAPY

Knowing when to start patients on oxygen therapy can save many lives. However ongoing assessment and evaluation should be carried out to ensure the therapy is safe and effective. Oxygen should be prescribed to achieve a target saturation of 94-98% for most acutely ill patients or 88-92% or patient specific target range for those at risk of hypercapnic respiratory failure. The specific ranges are provided in the relevant sections within these guidelines.

Some key points before the administration of oxygen include the following:

- Red cell transfusion is recommended for patients whose haemoglobin is less than 5g/dl. Stable patients at even this level of haemoglobin may not need blood transfusion.
- Hypoxia is an indication that oxygen therapy should be started.
- Oxygen does not treat breathlessness in the absence of hypoxaemia.
- A target oxygen saturation range should be prescribed to guide therapy.
- A lower target saturation range should be prescribed for patients who are at risk of hypercapnia.

- The amount of oxygen received by the patient is dependent on the delivery device used, thus health workers should ensure appropriate and correct selection and use of the devices.

When starting oxygen therapy, the health worker should be keen on the following;

- Document the baseline observations including saturations, respiratory rate, blood pressure and pulse rate.
- Ensure pulse oximetry is available to monitor response to oxygen therapy.
- Note the patients respiratory effort, colour and level of consciousness.
- Confirm the prescription of oxygen with a stated target saturation range ( except in dire emergencies such as cardiac arrest where documentation may be done post procedure).
- Ensure delivery device is connected via tubing to oxygen supply and turned on to the appropriate rate.
- Have explained to the patient and get appropriate consent where possible.
- Reassure the patient if they are very breathless.
- Monitor response to oxygen therapy-recheck saturations, vital signs, colour and level of consciousness.
- Document all adjustments, with saturations recorded.
- Weaning with reduction of oxygen therapy should be considered upon satisfactory oxygen saturation in the patient. Oxygen should be discontinued once the patient can maintain saturations within or above the target range breathing air.



### 62.1.5 HUMIDIFICATION OF OXYGEN

When oxygen is delivered at a flow rate of 1-4L/min by mask or nasal prongs, the oropharynx or nasopharynx provides adequate humidification. Humidification is necessary at higher flow rates or when oxygen is delivered directly to the trachea. The type of humidification device selected will depend on the oxygen delivery system that is in use, and the patients requirements.

## 62.2 Methods of delivering oxygen

Oxygen is delivered via variable performance or fixed performance devices.

**Variable performance devices**, also known as uncontrolled oxygen systems or low flow oxygen delivery devices include non-re-breathing masks, nasal cannulae and simple face masks. The amount of oxygen delivered through these devices is dependent on the oxygen flow rate, the patient's inspiratory volume, respiratory rate and proportion of room air added during breathing.
Non rebreather masks (or Reservoir masks) are recommended for short term use in patients who are critically ill. Oxygen at 10-15L/Min via a reservoir mask delivers oxygen at concentrations of 60-85%.

The simple face mask is usually intended for short term use, such as post-operative recovery period, with oxygen being delivered at 2-10L/min and supplemented with air drawn into the mask during breathing
Nasal cannulae are used for patients who are stable to provide supplementary oxygen therapy. They are more comfortable and well tolerated by patients. They do not need to be removed when the patient is talking or eating. Oxygen is inhaled even when breathing through the mouth.

**Fixed performance devices**, also known as controlled oxygen or high flow delivery systems deliver a fixed proportion of air and oxygen via a venture valve, thus ensuring accurate concentration of oxygen is delivered. Fixed performance devices are used in acute illness patients who are at risk of carbon dioxide retention and include venturi mask, trans-tracheal catheters, continuous positive airway pressure (CPAP) therapy and ventilators.

## 62.3 Risks of oxygen therapy

Like any drug, there should be clear indications for treatment with oxygen and appropriate methods of its delivery. Inappropriate dose and failure to monitor treatment can have serious consequences including hyperoxaemia, carbon dioxide narcosis, depressed ventilation and lung collapse. Vigilant monitoring to detect and correct adverse effects is essential. To ensure safe and effective treatment prescriptions should cover the flow rate, delivery system, duration, and monitoring of treatment.

# PART VII: Management of Blood and Blood Products in Hospitals

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# 63. Introduction

The Ministry of Health prepared this section about the management of blood and blood products. It is meant to assist physicians and other health care providers in the correct selection of patients for transfusion, and the safe administration of blood and blood products.

Severe anaemia is a major health problem in Kenya and is frequently treated with blood transfusion. Transfusion of blood and products saves lives, but it is not without risks or costs. Some of the possible complications include the transmission of infectious diseases such as HIV, Hepatitis B, Hepatitis C, syphilis, malaria, as well as haemolytic and non-haemolytic transfusion reactions, immunosuppression, and alloimmunization. Safe blood is a scarce and valuable resource that is expensive to collect, process, and administer. Limiting transfusion to patients whose chance of survival or quality of life is improved with blood will help to lower the high demand for blood and will reduce unnecessary exposure of patients to the risks of transfusion.



For effective implementation of blood transfusion, hospitals should establish a Hospital Transfusion Committee. This committee serves to ensure that the quality of blood transfusion services and practices is maintained at a high level.The committee will oversee all policies and procedures relating to blood utilization for the hospital, namely selection of patients for transfusion; ordering, distribution, handling, and administration of appropriate blood and blood components; and the monitoring of the effects of blood on patients, including the investigation of blood transfusion reactions (refer to heamovigilance guidelines).

The following are recommended for appropriate transfusion practice:

- Blood should be transfused only when required to save a life. The decision to transfuse should be based on an estimate of the patient's risk for developing complications of inadequate tissue-oxygen delivery and should be based on both the haematological and the clinical status of the patient.
- Red cell transfusion is recommended for patients whose haemoglobin is less than 5g/dl. Stable patients at even this level of haemoglobin may not need blood transfusion.
- Effort should be made to stabilize patients through use of intravenous therapy with crystalloids and colloid solutions and oxygen therapy before blood is available.
- A patient should be re-evaluated by clinical and nursing staff immediately prior to blood transfusion to ensure that the transfusion is still required. The patient may have stabilized with supportive measures and may no longerneed transfusion. The patient should not be transfused purely because compatible blodd is available.
- Effective transfusion requires a minimum of 1 unit of blood for an adult or 20ml whole blood (10–15 ml packed cells) per kilogram body weight for a child.

- Before transfusing a second unit, the post transfusion haemoglobin level should be compared wih the pre-transfusion value to assess the efficzcy of the transfusion.
- The underlying cause of the need for the blood transfusion needs to be identified and appropriately managed.

# 64. Use of Red Blood Cell Products

The following general information is important when using red cell blood products:

- A red blood cell (RBC) transfusion is intended to increase the delivery of oxygen to the tissues. Red blood cells can be transfused as either whole blood or packed red blood cells (PRBCs).
- A unit of whole blood measures approximately 400–500 ml and has a hematocrit of 38-54%. A unit of parked red blood cells (PRBCs) consists of the red blood cells concentrated from a unit of whole blood. Each unit of PRBCs contains approximately 230-330 ml of RBCs and 50–70 ml of plasma. The hematocrit of PRBCs is 60 to 80%. Each unit of blood contains approximately 60g of haemoglobin and 250 mg of iron, predominantly in the form of haemoglobin. Both whole blood and PRBCs contain a small amount of citrate anticoagulant and additional preservative solutions. Blood units that are collected in CPDA-1 anticoagulant can be stored for up to 35 days and 42 days for PRBCS with additive solution (saline adenine glucose, mannitol).
- PRBCs should not be used to treat long-standing anaemia that can be corrected with non-transfusion therapy such as iron to increase blood volume, oncotic pressure, coagulation factors, or platelets.
- Red blood cells must be compatible with the ABO antibodies present in the recipient patient's serum, and must be cross-matched in order to confirm compatibility. Unless the patient is bleeding or haemolysing, the post-transfusion haemoglobin can usually be accurately predicted. One unit of blood (or the equivalent volume in a child) usually increases the patient's haemoglobin by 1g/dl. In acute haemorrhage, blood transfusion should be initiated as soon as possible to offset the deficit;however,too rapid infusion of large volumes of cold blood with excess extracellular potassium, reduced pH, and excess citrate can sometimes have undesired effects on cardiac rhythm.
- The risk of mortality increases significantly in otherwise stable patients when the haemoglobin level falls to approximately 3.5–4 g/dl. In ischaemic heart disease,the risk of mortality significantly increases when the haemoglobin falls between 6 and 7.5g/dl. Perioperative RBC transfusion experience suggests that patients usually require transfusion when their haemoglobin level is less than 6g/dl, and only rarely when their haemoglobin level is above 10g/dl. For levels between 6 and 7g/dl, the transfusion needs depend on the amount of blood loss, underlying coronary/cardiac disease, and overall patient status.

## 64.1 Acute Blood Loss, Chronic Anaemia and Surgery Transfusion

### 64.1.1 ACUTE BLOOD LOSS

In a patient with acute blood loss, an early haemoglobin level will not accurately reflect the severity of blood loss until there has been adequate plasma volume replacement. Serial haemoglobin levels are required to determine the need for red cell transfusion as well as evaluation of the clinical status of the patient.

The following is anticipated with varying degrees of blood loss:

- As a general rule,a loss of blood volume of less than 15% results in minimal symptoms; 15–30% results in tachycardia; 30–40% in signs of shock; and greater than 40% in signs of severe shock.
- Some patients with underlying diseases may require transfusion at 30–40% blood loss.All patients with losses greater than this require transfusion.
- The first treatment for hypotension, shock, and acute blood loss is volume expansion with normal saline (without dextrose), infused in a volume at least three times the volume lost. Normal saline up to 50ml/kg is recommended for initial volume replacement. This should be followed by colloid solution, e.g. 6% dextran or 6% hydroxy-ethylstarch, given in equal volume the blood volume lost. The 6% dextran should not exceed 50ml/kg body weight, and the 6% hydroxy-ethyl starch 20ml/kg body weight in 24 hours.
- The decision to transfuse should be made on the basis of parameters such as heart rate, blood pressure, haemoglobin, and the presence of active bleeding.

In order to restore blood volume and oxygen-carrying capacity in patients with massive haemorrhage (blood loss greater than 40%), blood may be required. In massive transfusion (more than four units within 1 hour in an adult, or the replacement of the equivalent of the patient's blood volume within 24 hours), platelets or fresh frozen plasma should be given according to the results of the patient's platelet count and coagulation profile, if possible. Consider giving ABO compatible fresh frozen plasma (FFP) in a dose of 15ml/kg if the prothrombin time (PT) is prolonged , and platelet concentrate (4–6 donor units for an adult) when the platelet count falls below 20,000/$mm^3$. If the platelet count or coagulation profile is not available, consider giving 2 units of FFP and 6 donor units of platelet concentrate for every 6 units of blood transfused within a period of 24 hours.

**Table 64.1: Massive Transfusion Protocol**

| | PRBCs | FFPs | Platelets | Cryoprecipitate |
|---|---|---|---|---|
| Round 1 | 6U | 6U | 6U | |
| Round 2 | 6U | 6U | 6U | 10U |
| Round 3 | Tranexamic Acid 1 g over 10 min | | | |
| Round 4 | 6U | 6U | 6U | 10U |

**Adapted from the Guidelines of Appropriate Use of Blood and Blood Products**

### 64.1.2 PERIOPERATIVE TRANSFUSION

It is important to know the following with regard to perioperative transfusion:

- In the perioperative patient, transfusion decisions should be based not only on haemoglobin level but also on clinical signs, symptoms and prior medical history. In anaesthetized patients, vital signs alone do not reflect the patient's real situation. During the intraoperative period, the patient's cardiopulmonary reserve, the amount of anticipated blood loss, oxygen consumption and the presence of atherosclerotic heart disease affect the decision for transfusion.
- Prior to elective surgery,all efforts should be made to correct anaemia without the use of blood. Patients with a Hb level less than 5g/dl may need transfusion prior to surgery if anaemia cannot be corrected by other means.
- Blood should be cross-matched and made available for immediate use during surgery for patients with a high likelihood of needing a transfusion.
  Transfusion may be necessary during surgery for patients with a Hb level less than 8g/dl or who lose more than 1 litre of blood during surgery.
  In the case of post operative or postpartum haemorrhage, the source of bleeding must be identified and stopped. Transfusion is not indicated as treatment of anaemia in postoperative or postpartum patients if no active bleeding exists.

### 64.1.3 CHRONIC ANAEMIA

With respect to chronic anaemia,the following is recommended:
Blood should be used only to relieve clinical signs of cardiac and respiratory distress in severely anaemic patients, in order to achieve haemodynamic stability. Blood should not be used to correct chronic anaemia. Most patients with chronic anaemia have nutritional and/or mild blood loss anaemia that responds rapidly and effectively to specific therapies. In case of transfusion, it should be done preferably using PRBCs and should be done slowly with careful monitoring of the patient. This is because these patients have normal blood

volumes and the transfusion of whole blood may cause circulatory overload, with harmful effects.

- Do not transfuse above 7g/dl Hb unless the patient is symptomatic. ·
- Treat nutritional and mild blood loss anemia with specific therapeutic agents as indicated (iron, folic acid, VitaminB12).
- Use specific strategies for congenital anaemias including sickle cell disease.

### 64.1.4 RED BLOOD CELL TRANSFUSION GUIDELINES

The following guidelines are recommended for red blood cell transfusion for acute, pre-operative blood loss and also for chronic anaemia.

**Acute and Peri operative Blood Loss**

1) Evaluate patient for risk of ischaemia
2) Estimate blood loss:
   - If >30-40% of rapid blood loss: transfuse RBCs and use volume expanders
   - <30-40% of rapid blood loss: RBCs not usually needed in otherwise healthy person
3) Monitor vital signs:
   - Tachycardia and hypotension not corrected with volume expanders: RBCs needed
4) Measure haemoglobin:
   - If Hb >10g/dl: RBCs rarely needed
   - Hb <5g/dl: RBCs needed
   - Hb 5–10g/dl: RBCs may be needed,determined by additional clinical conditions

**Chronic Anaemia**

- Transufuse only to decrease symptoms and tom minimize risk (generally at Hb of lesss than 5g/dl). Do not transfuse above 5g/dl Hb unless the paient is symptomatic.
- Treat nutritional and mild blood loss anaemia with specific therapeutic agents as indicated (iron, folic acid, B12).
- Use specific strategies for sickle cell disease and thalassaemia.

## 64.2 Blood Transfusion in Pregnancy

Anemia in pregnancy is defined as first trimester hemoglobin (Hb) of less than 11.0 g/dl, second/third trimester Hb of less than 10.5 g/dl, and postpartum Hb of less than 10.0 g/dl. For normocytic or microcytic anemia, a trial of oral iron should be considered as the first step and further tests should be undertaken if there is no demonstrable rise in Hb at 2 weeks and compliance has been checked. Pregnant women should be offered screening for anemia at first antenatal visit and at 28 weeks. Women with multiple pregnancies should have an additional full blood count done at 20–24 weeks.

The following information is important with regard to blood transfusion in pregnancy:

- In pregnancy, maternal plasma volume increases by 40%, and red cell mass by 25%. Blood loss is usually well tolerated during pregnancy. The mean blood loss during vaginal delivery is 500 ml, while 1,000 ml is lost during caesarean delivery. Indications for transfusion in the pregnant and postpartum patient are similar to those for the non-pregnant patient.
- In addition to the clinical assessment of pallor, all women should have their haemoglobin measured at the first antenatal visit, and subsequently once

during every trimester. Clinical evaluation of mucous membranes (conjunctiva and tongue) or palmar pallor may not detect mild or moderate anaemia that may lead to adverse effects later in pregnancy or at the time of delivery.

- All women should have ABO blood grouping and Rhesus (Rh) factor typing performed at the first antenatal visit. Where facilities exist, a screen for unexpected antibodies should be done. All Rh-negative women, with no evidence of immunization, delivering a Rh-positive foetus (or who have an abortion) should be given Rh immunoglobulin (RhoGAM) in adose of 300mg IM within 72 hours of delivery or abortion.
- Nutritional education must be an integral part of routine antenatal care, including recommendations for protein and dark green leafy vegetables in the diet.
- Women with Hb of less than 10 g/dl should receive ferrous sulphate 200 mg (60mg elemental iron) 3 times a day throughout pregnancy.Clinically stable pregnant women with severe anaemia (<7 g/dl) should be evaluated for the cause of their anaemia and treated appropriately. These women should be monitored every 2 to 4 weeks,including measurement of the Hb level. It may be necessary to admit or refer women with a Hb level persistently lower than 7g/dl for closer clinical monitoring and treatment.
- Blood transfusion should be considered for pregnant women with Hb level less than 5g/dl who become symptomatic with dyspnoea, shock, or orthostatic hypotension.
- Blood should be ordered and made available in the delivery room for immediate transfusion incase of haemorrhage at the time of delivery for pregnant women with a Hb level less than 7g/dl. Pregnant women with a Hb less than 7g/dl should be referred for delivery at facilities where blood transfusion is available.
- Blood transfusion is not indicated in anaemic women who are clinically stable after delivery.
- In the case of postpartum haemorrhage,the source of bleeding must be identified and corrected. The first therapy of acute blood loss is volume replacement.

**Treatment and Management of Anemia in pregnancy**

- In the case of postpartum haemorrhage,the source of bleeding must be identified and corrected. The first therapy of acute blood loss is volume replacement.
- Oral iron should be the preferred first-line treatment for iron deficiency.

- Parenteral iron is indicated when oral iron is not tolerated or absorbed or patient compliance is in doubt or if the woman is approaching term and there is insufficient time for oral supplementation to be effective.
- Women should receive information on improvement of dietary iron intake and factors affecting absorption of dietary iron.
- The role of recombinant human erythropoietin (rHuEPO) for non-end-stage renal anemia is still to be established and it should only be used in the context of a controlled clinical trial or on the expert advice of the hematologist.

- Active management of the third stage of labor is recommended to minimize blood loss.
- Women at high risk of hemorrhage should be advised to deliver in hospital.
- Optimisation of hemoglobin in the antenatal period reduces the risk.
- Selected sickle cell pregnancy complications such as recurrent foetal loss.

## 64.3 Paediatric and Neonatal Transfusions

The following information is important for paediatric and neonatal blood transfusions:

**Paediatric Transfusion**

- If Hb is < 4g/dl,transfuse.
- If Hb is >4g/dl and <5g/dl, transfuse when signs of respiratory distress or cardiac failure are present. If patient is clinically stable, monitor closely and treat the cause of the anaemia.
- If Hb is>5g/dl, transfusion is usually not necessary unless in cases of shock or severe burns. Otherwise, treat the cause of the underlying anaemia.
- Transfuse with 10–15ml/kg of PRBCs or 20ml/kg of whole blood. In the presence of profound anaemia or very high malaria parasitaemia, a higher amount may be needed.

**Management**

***Transfusion volume = bodyweight (kg) x Hb deficit (g/dl) x 3 (packed RBC) or 5 (whole blood)***

**Neonate Tranfusion**

The total blood volume of neonates is small, although the volume is higher per kg of body weight than that of older children or adults (85ml/kg for full-term and 100–105ml/kg for pre-term). Transfusions are generally given in very small increments, increasing the risk of infectious disease transmission through multiple donor exposures.

Blood transfusion in pre-term infants is often given for the anaemia of prematurity, associated with delayed renal production of erythropoietin due to decreased sensitivity to lower haematocrit levels. This commonly develops in

neonates after 2 weeks of life. Neonates, especially pre-term, may require multiple transfusions.

In neonates, a dose of 15ml/kg of packed red blood cells will increase the haemoglobin by approximately 3g/dl.

Transfuse with 10–15ml/kg PRBCs for:

- Acute blood loss of >10% of blood volume.

- Haemoglobin <7g/dl.
- Haemoglobin<8g/dl in a newborn with apnoea, bradycardia, tachycardia, tachypnoea, or decreased vigour.
- Haemoglobin of <12g/dl with moderate to severe respiratory distress or severe congenital heart disease and absence of weight gain for 7 days with no other explanation.

note: **Avoid using blood donated by blood relatives to transfuse neonates.**

## 64.4 Congenital Anaemias

Children with congenital anaemias such as sickle cell diseases (Hb S/S, Hb S/C, Hb S/-thalassaemia) like all other children, should only be transfused when they develop cardio-respiratory symptoms from severe anaemia, or the indications listed below.

- Indications for Red Blood Cell Transfusion in Sickle Cell Disease
  - Symptomatic anaemia due to:
    - Aplastic crisis
    - Splenic sequestration
    - Accelerated haemolysis (due to haemolytic anaemia or sickle cell crisis)
    - Preoperative preparation for most types of surgery
  - Chronic transfusion:
    - Prevention of recurrent occlusive stroke (< 30% HbS)
    - Selected sickle cell pregnancy complications such as recurrent fetal loss

## 64.5 Plasma Transfusions

The following is recommended for plasma transfusions:
- Correction of coagulation abnormalities with bleeding e.g Hemophilias and coagulation factor deficiencies
- Massive transfusion
- Bleeding due to warfarin therapy refractory to vitamin K
- The dose is 10-20ml/kg of group specific plasma transfused over 2-4hrs.
- The amount of FFP to be given should be pegged on normalization of PT and a PTT.

## 64.6 Autologous Transfusions

The following is important for autologous transfusions:

- For elective surgery in patients with Hb level of 10g/dl or greater, 2–4 units of blood may be collected from the patient prior to surgery for the patient's own use during surgery (autologous transfusion). Collections should be at least 7 days apart, and the last donation should be at least 2-3 days before surgery.
- There is no indication for a single-unit autologous transfusion in an adult.

**Unused autologous units can be released into the general donor pool, provided the patient meets all criteria for blood donation and the units are fully screened and tested.**

- Preoperative isovolaemic haemodilution maybe performed prior to surgery. This can be accomplished by removal of 2 or more units of blood and replacement with an equal volume of crystalloid. This technique improves tissue perfusion during surgery and makes the units of blood available for autologous transfusion during and after surgery.

**Contra indications**

- Pre-existing anemia
- Heart disease
- Uncontrolled hypertension
- Extremes of age

# 65. Transfusion Reactions

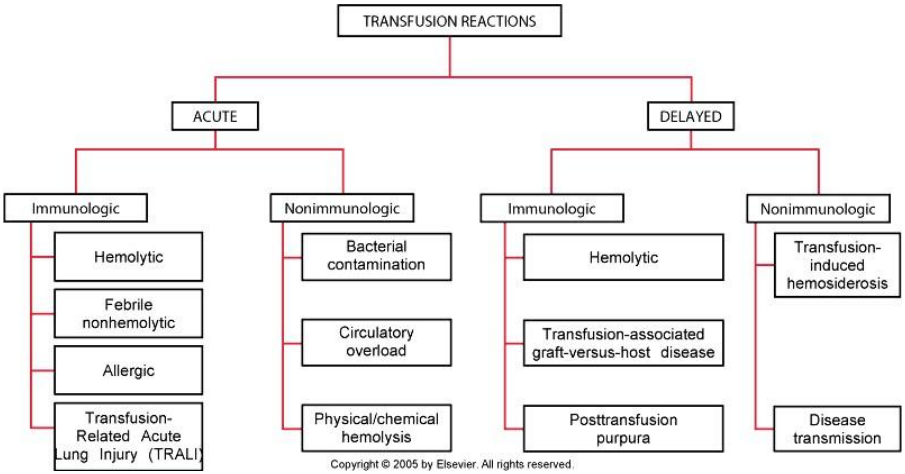
- Although transfusion can be a life saving therapy,it can result in many adverse effects. Approximately 1% of all transfusions lead to some type of adverse reaction. Many measures have been taken to reduce transfusion related risks, including donor risk screening and laboratory testing of blood products, but it is not possible to provide a blood supply with zero risk. Therefore, physicians must carefully weigh the benefits of transfusion against the risks.
- Transfusion reactions can be caused by immunological or non-immunological mechanisms, and may be immediate or delayed for some time after the transfusion. The majority of immediate serious reactions are immunological and are caused by clerical errors, including incorrect recording of blood type,
- cross-match results, or patient name resulting in transfusion of the wrong unit or the wrong patient. The importance of proper patient identification and specimen labelling cannot be overemphasized. Other common serious complications of blood transfusion are related to infectious disease transmission. The most serious of the transmitted agents are HIV and Hepatitis B and C.
- All transfusions should be given under the supervision of a clinician. The patient should be monitored closely for the first 15 minutes of the transfusion since it is during this period that serious haemolytic transfusion reactions can first be detected. The transfusion should be regulated to infuse for a maximum

  of 4 hours, with monitoring of the vital signs by the nursing staff every 30 minutes. Any change in vital signs (temperature, pulse, respiratory rate, blood pressure) or level of consciousness may be an indication of a transfusion reaction. The symptoms and signs of a transfusion reaction include pruritus, palpitations, lumbar pain, pain along the entry vein, fever, hypotension, tachypnoea, tachycardia, and altered level of consciousness.
- Blood should be setup for transfusion within 30 minutes of leaving the laboratory. Unused blood from the theatre or wards should be returned immediately (within 30 minutes) to the laboratory.

## 65.1 Types of Transfusion Reactions

The following flow chart lists common types of transfusion reactions:

**Figure 65.1: Common types of transfusion reactions**




The majority of transfusion reactions are febrile reactions. These reactions are usually characterized by a mild temperature elevation without other clinical signs or symptoms. These can be managed with antipyretics, without having to stop the transfusion. The most common cause of serious haemolytic transfusion reaction is the administration of ABO incompatible blood. If serious transfusion reaction is suspected, the transfusion should be stopped immediately. The patient should have an IV line kept open with saline and vital signs should be monitored. The laboratory should be notified of the suspected transfusion reaction, and a transfusion reaction work-up immediately initiated. The laboratory should report all suspected transfusion reactions to the Hospital Transfusion Committe**e.**

**Table 65.1: Signs and symptoms of acute hemolytic transfusion reactions**

| General | Cardiac/respiratory | Renal | Haematological |
|---|---|---|---|
| -Fever, chills, flushing<br>-Nausea, vomiting<br>-Headache<br>-Pain at infusion site<br>-Back or loin pain<br>-Pruritis<br>-Altered levels of consciousness | -Chest pain<br>-Dyspnoea and tachypnea<br>-Hypotension<br>-Tachcardia | -<br>Haemoglobinuria<br>-Oliguria<br>-Anuria | -Anemia<br>-Unexplained bleeding (disseminated intravascular coagulation)<br>-hrombocytopaenia |

**Management of transfusion reaction**

- Stop the transfusion but keep the IV line open with normal saline.
- Monitor the vital signs of the patient.
- Inform the laboratory about a possible transfusion reaction.
- Check the clerical information to ensure that the patient is receiving the correct blood.
- Take the following blood samples from the patient (from the opposite arm):
  - 10 ml of blood into a plain tube. Check the colour of the plasma for haemolysis.
  - 2ml of blood into an EDTA tube.
- Collect a sample of the first voided urine.

**Send to the laboratory:**

- All samples correctly labelled.
- The blood that reacted, together with the attached transfusion set.
- All empty blood bags of already transfused units.
- Laboratory request form filled in.
- Report all investigations to the Hospital Transfusion Committee.

# Part VIII: Forensic Medicine

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# 66. Fundamental Principles of Forensic Medicine

Forensic medicine is the application of medical knowledge to investigate crime particularly in establishing the causes of injury or death. Forensic medicine includes examining and managing the living, injured as well as persons dying under unnatural circumstances. It requires accurate documentation and preservation of forensic medical evidence. Standardisation of care, reports, improving consistency and quality of opinions prepared by experts will facilitate better delivery of justice.



## 66.1 Legal frameworks

Criminal law deals with relationships between the state and the individual and it is the area where forensic medical expertise is most commonly required. Criminal trials involve crimes 'against public interest'; including offences against the person (e.g. murder, assault, grievous bodily harm, rape), property (e.g. burglary, theft, robbery), and public safety and security of the state (terrorism). On the other hand, civil law is concerned with resolving disputes between individuals.

## 66.2 Medical practitioner and the law

Medical practitioners may encounter the law like any other citizen or in their day-to-day professional practice particularly when they may manage patients whose medical condition is of forensic interest. A medical practitioner is expected to conduct a full medical-forensic examination on the patient (and prescribe the appropriate medical treatment); collect and preserve the necessary medical forensic samples and inform and forward to the investigating officer or their representative, the samples collected, while maintaining a record of the chain of custody. The should then Initiate appropriate referral to the relevant areas for the necessary subsequent care.

The medical practitioner may then be required to give evidence at a court of law regarding the patient, in which case she/he may be a professional (gives an account of the events) or expert witness (gives an opinion based on medical facts).

When required to give evidence at a court of law, the medical practitioner is expected to prepare a statement in advance. This statement will take the form of a medical report and will be based on the notes made at the time of encounter with the patient, which may be many months or many years later.

**Preparation of medical reports**

***General considerations***

- The diversity of uses of a report is reflected in the individuals or groups that may request one: a report may be requested by the police, prosecutors, coroners, judges, medical administrators, government departments or regulatory bodies.
- The most important question that medical practitioners must ask themselves before agreeing to write a report is whether they: (1) have the expertise to write such a report and (2) have the authority to write such a report.
- Generally, when medical records need to be reviewed, written permission to access and use those records has to been given, either by the individual themselves, or by an individual or body with the power to give that consent. If consent has not been sought, advice should be sought from the relevant court or body for permission to proceed.
- An order from a court, if valid, should be obeyed.
- For medical confidentiality, the consent of a living patient is required and, if at all possible, this should be given in writing to the medical practitioner. Exceptions may exist, particularly where serious crime is involved in which case the medical practitioners have a public duty to assist the law-enforcement agencies.
- If no consent was provided, this should be stated in the report, as should the basis on which the report was written.
- It is also important to remember that consent to disclose the effects of an alleged assault does not imply consent to disclose all the medical details of the victim, and a medical practitioner must limit her/his report to relevant details only.
- Issues that relate to terrorism, child abuse, use of a weapon and other violent crime must be reported.
- The basis of most reports and statements lies in the contemporaneous notes made at the time of an examination and it is essential to remember that copies of these notes may be required in court as part of evidence.
- The medical practitioner should ensure clarity and simplicity of expression to make the whole process simpler. A clear, concise and complete report or statement may prevent the need for court attendance at all, and if you do have to give evidence, it is much easier to do so from a report that is legible.
- Medical reports can be constructed along the same lines as the clinical notes – they should structured, detailed (but not over-elaborate) and accurate. A good report will give the relevant facts clearly, concisely and completely, and in a way that an intelligent person without medical training can understand.
- Medical abbreviations should be used with care and highly technical terms, especially those relating to complex pieces of equipment or techniques, should be explained in simple, but not condescending, terms.
- Abbreviations in common usage such as ECG can generally be used without explanation although occasionally further explanation is required.
- It is preferable not to submit handwritten or proforma type statements unless absolutely unavoidable.
- A simple professional witness statement (one that simply reports facts found at examination) may be headed by specific legal wording.

***Autopsy reports***

- Are a specialist report and may be commissioned by the Coroner, the police or any other legally competent person or body, there may be standardized protocols or proforma.
- The authority to perform the examination will replace the consent given by a live patient, and is equally important.
- The history and background to the death will be obtained by the police or the Coroner's officer, but the doctor should seek any additional details that appear to be relevant, including speaking to any clinicians involved in the care of the deceased and reviewing the hospital notes.
- A visit to the scene of death in non-suspicious deaths, especially if there are any unusual or unexplained aspects, is to be encouraged.
- An autopsy report is confidential and should only be disclosed to the legal authority who commissioned the examination.
- Disclosure to others, who must be interested parties, may only be made with the specific permission of the commissioning authority and, in general terms, it would be sensible to allow that authority to deal with any requests for copies of the report.

***Structure of a statement or report***

When instructed to prepare an expert report always clarify whether or not a specific structure is required and if so, follow it assiduously. An example is a P3 form or a police autopsy request form. But generally:

- The name, address and contacts of the health facility should be captured in the letter head. This could be that of the medical practitioner in the event of a private practice.
- The medical practioner's professional address and qualifications should follow.
- Indicate who requested the statement, and when.
- The date of the report is essential.
- A summary of the medical history (as given by the patient or any other relevant persons) touching only on the relevant details should be provided.
- The time(s), date(s) and place(s) of any examination(s) should be listed, as should the details of any other person who was present during the examination(s).
- The medical practitioner should confirm their understanding of their role at the time (e.g. 'I was called by the police to examine an alleged victim of assault to document his injuries').
- Confirm that the patient has given consent for the release of the medical information (if no consent is available it must be sought).
- By referral to contemporaneous notes outline the history that you were aware of (... 'Mr X told me that...').
- In simple terms summarize medical findings. If information other than observation during a physical examination (e.g. medical records, laboratory or imaging) formed part of the basis of management of the patient, these too must be recorded.
- If opinions of consultants and any other persons were sought, these too will need to be documented.
- The treatments offered to the patient (including surgical procedures) should be provided in summary form.

- Any significant complications should also be documented, as well as a summary of the overall clinical course and outcome of the disease/injury process.
- Conclusions and recommendations relevant to the particular case may be added.
- The medical practitioner should sign off the report with their unique mark and forward it to the requesting authority via the chain of custody.

## 66.3 Ethics of forensic medical practice

The laws governing the practice of medicine vary from country to country, but the broad principles of medical ethics are universal and include;

- Compassion - understanding and concern for another person's distress.
- Informed consent - The right of patients to make decisions about their healthcare with adequate information provided by the medical team.
- Confidentiality - ability of a medical practitioner to keep secret information obtained from a patient in the course of a professional relationship.
- Competence - Skills required to carry out a task successfully.
- Autonomy - self determination and the right to decline or choose treatment.
- Non-maleficence – do no harm.
- Beneficence – acting in the patient's best interests.
- Dignity - the state or quality of being worthy of honour or respect.
- Honesty – providing informed consent
- Justice – honor or standards to support fair treatment and due reward.

# 67. Forensic Medical Evidence

## 67.1 Definition

Forensic evidence is any item or information gathered at the scene of a crime, or at related locations, which is found to be relevant to an investigation is analyzed using scientific methods to aid in solving a crime or administration of justice. Maintaining evidence is paramount and strict procedures must be observed by all involved in the investigation when it comes to collecting, labeling, and analyzing it. Above all, every effort must be made to ensure that evidence is not lost, damaged, or contaminated.

## 67.2 Types of Forensic Evidence

Evidence can be classified into:

a) Direct and indirect (circumstantial) evidence
b) Physical or biological evidence
c) Reconstructive evidence
d) Associative evidence (class or individual)
e) Trace evidence



## 67.3 Collection, Packaging, Transport and Analysis of Forensic Evidence

### 67.3.1 COLLECTION

The first items to be collected are those which are fragile and could easily be damaged such as fingerprints, shoe prints, fibers, and hair. A systematic approach must be taken to ensure that the collection of one item of evidence will not destroy another for use back in the laboratory to distinguish relevant from irrelevant evidence. Control samples are used in the laboratory to distinguish relevant from irrelevant evidence.

### 67.3.2 PACKAGING

Each item of evidence is packaged separately to avoid contamination and damage.

### 67.3.3 TRANSPORT

The evidence is handled through a strict chain of custody. Every time an item is transferred from one person to another, it is signed and accounted for. Forensic medical specimens (and reports) should be handed over to the investigating/escorting police officer upon collection for onward transmission to the forensic laboratory. Break in the chain of custody may render the evidence inadmissible in a court of law.

## 67.3.4 ANALYSIS

Analysis of forensic evidence is typically carried out by the forensic laboratory (government analyst) for all specimens except histology which is handled by the cancer diagnostic laboratory. In some cases specimens analyzed in clinical/pathology laboratories may too constitute forensic medical reports, these include infectious disease diagnostics, biochemistry, hematology, immunological, histopathological and other relevant tests required. The aim of analysis of forensic evidence is to ascertain identity of the forensic evidence and carry out comparison studies with the control samples with the aim to to establish links through evidence.

## 67.3.5 CHAIN OF CUSTODY

Chain of custody refers to the chronological documentation of the processes through which forensic evidence is taken including any persons who handle it. It is an important process for the following reasons:

- Ensures admissibility of evidence in court
- Ensures evidence is not lost
- Ensures the integrity of the evidence
- Ensures traceability of the evidence

- Ensures evidence is not tampered with or switched
- Ensures availability of the evidence for look back purposes when needed
- Reduces the number of people who handle the evidence
- Ensures the evidence presented to court is actually the evidence that was collected at the scene

## 67.3.6 DOCUMENTATION OF FORENSIC EVIDENCE

Documentation creates a permanent record of events, lesions, processes and activities. It is particularly important in ensuring important facts are available for future reference. This is more so in forensic medical practice where a medical practitioner may be called upon to give evidence in a court of law or a board many months years after the examination when some of the facts have been forgotten.

## 67.3.7 METHODS OF DOCUMENTATION OF FORENSIC MEDICAL EVIDENCE

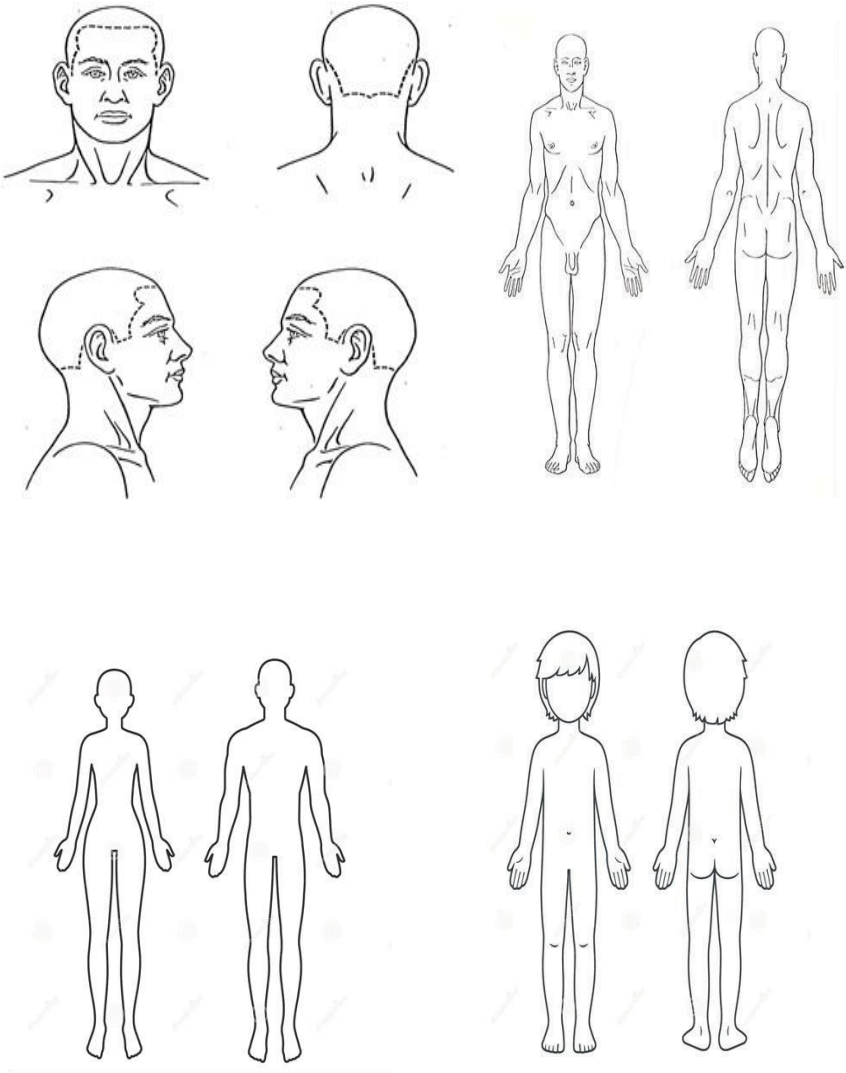
### MEDICAL/CLINICAL NOTES

Clinical notes must be thorough and include history, findings in general examination, systemic examination, tests ordered (laboratory and imaging) and their findings, treatment plans (including medical and surgical procedures), opinions of any consultations and follow up review of the patient including any significant complication(s).

## SKETCHES AND CHARTS

They give a visual representation of location of lesions and are particularly important in documentation of injuries.

**Figure 64.1: Body Charts**



## STILL PHOTOGRAPHS

Still photographs are particularly important in documentation of complex situations, lesions or injuries where narrative description may fail to capture some details such as differences in colour or complex relationships. Colour photographs are particularly helpful. Medical practitioners should avoid using personal cameras for forensic evidence documentation. Centres offering forensic medical services should provide still cameras.

## VIDEO

Video recording is quite helpful in documenting procedures and processes and is able to capture a variety of activities and information at the same time. This service is mainly provided by the police.

## MEDICAL IMAGING

This is required for specific types of forensic cases such as gunshot injuries, child abuse, sexal offences,etc where available.

# 68. Clinical Forensic Medicine

Clinical forensic medicine includes a wide range of procedures from examination of victims of injuries, sexual assault, victim/ accused examination, custodial torture victims to much more. Forensic medical aspects of clinical practice traverse all levels of practice, from the very basics, to community level, to sophisticated care in level six facilities. It is worth noting that the medico-legal aspects of any case must always be secondary to life saving treatment of the patient.

## 68.1 Crowd Control Agents

Chemical restraints can be used for various reasons for control of a violent or agitated patient, to disperse crowds, or to limit access to some areas.
The agents include but are not limited to:

- Capsaicin oleum (OC) also known as pepper spray
- Chloracetothenon (CN) also known as mace
  Chlorobenzylidenemalononitrile (CS) also known as tear ga**s**

**Clinical Features**

- Dermal irritation with rashes and burns, contact dermatitis, contact dermatitis, lachrymation, ocular pain and irritation, conjunctivitis, transient impairment of vision, respiratory distress, blepharospasm.
- Nausea, vomiting, bad taste, headache from the tear gas.
- Following prolonged exposure or exposure to large amounts of irritant, pulmonary edema, periocular edema can occur.
- Long term effects include asthma, glaucoma, cataracts, corneal scarring.

**Management**

- Ensure you wear a mask and gloves before touching the patient.
- Remove and dispose contaminated clothes.
- Decontaminate exposed skin with soap and water.
- Topical antibiotics for corneal abrasion
- Analgesics for pain
- Systemic antihistamines for dermatitis
- Systemic steroids for severe form of contact dermatitis

## 68.2 Forensic Aspects Relevant to Restraint

Most law enforcement agencies and government-sanctioned caregivers have policies that guide the use of force. However, during an encounter, use of various means of control may be escalated.

### 68.2.1 WRIST RESTRAINTS

These include handcuff or rope injuries. Watch out for the following signs and symptoms

- Wrist scarring at bony prominences. Are mostly circumferential or form part of the circumference.

- Skin lesions like bruising.
- Ulnar or median nerve neuropathy.
- Scaphoid or radial styloid fractures.

**Management**
- Analgesics
- Clean wounds with antiseptic
- Wrist radiographs to rule out fractures
- Nerve conduction tests to check for nerve damage
- Antibiotics to treat infected skin lesions
- Document history, examination findings and treatment given in official medical forms or P3 if provided.
- Photograph the injuries.

## 68.2.2 NECK HOLDS

This involves pressure on or around the neck which can be fatal. Such holds can be choke holds or carotid holds. Choke holds can cause airway collapse. Carotid holds can cause loss of consciousness.The elderly, people with Down's syndrome, pregnant women are at higher risk of severe injuries and even death.

**Clinical Features**
- Neck pains
- Asphyxiation
- Altered mental status
- Fractured hyoid
- Bruises in the neck region. Describe the bruises in terms of shape, size, location and colour.

**Management**
- Take a detailed hiistory and carry out a thorough medical examination.
- Stabilize the neck and provide oxygen if required.
- Refer for specialized care if needed.
    - Antibiotics to treat infected skin lesion
    - Dcoumnt history, examination findings and treatment given in official medical forms or P3 if provided.
    - Photograph the injuries

## 68.2.3 TASER

It is a hand held electrical weapon, powered by two 3V batteries, that induces neuromuscular incapacitation and pain by the application of a small electrical charge.

A detailed history should be documented which should include; the date, place and specific time of the incident, any available witnesses, what was the victim doing at time of the incident, any symptoms associated with the application of electric current and mode of delivery of the electric charge.

**Clinical Features**

- Localised superficial burns and erythema.
- Musculoskeletal injury from the intense muscle contraction.
- Ethmoid bone fracture.
- Spinal vertebral compression fractures.
- Bony injuries from falls (including non-fatal and fatal head injury).
- Triggering of epileptic seizures.
- Cardiac effects-affects cardiac rhythm (ventricular fibrillation)

**Management**

- Remove darts penetrating skin by stabilizing the skin surrounding the taser dart and while firmly grasping the probe, remove it with rapid traction.
- Clean the skin with antiseptic.
- A complete general and system-specific examination to document visible injury should be undertaken to identify any taser- associated complications.
- ECG, X-ray, ultrasound, CT or MRI scans may be indicated depending on examination findings.
- Refer to specialist if extensive injuries are found.

## 68.3 Sexual Offences

***(See classification of sexual offences as per The Sexual Offences Act).***

The services required include early identification of victims, screening, comprehensive history taking, examination, documentation, collection, preservation and linkage of evidential material. It also includes appropriate referral, presentation of evidence as expert witness and the maintenance of a chain of custody.

Patients seen at lower levels can receive the minimum package of care as listed. However, it is good to note that cases requiring referral should have warm referrals made to avoid retraumatization. This involves contacting the facility that the patients are being referred to and provision of a comprehensive referral form.

### 68.3.1 MANAGEMENT OF A SEXUAL VIOLENCE VICTIM (ADULT)

- Make sure that the consultation room has adequate lighting and it's private.
- Before a health worker sees a patient, they should ensure that the necessary furniture, equipment, medication and supplies are available.
- It is advisable for the health care provider to be of the gender that the patient is comfortable.
- The consultation room should be clean, private, disability-accessible, clean and with all the requirements needed e.g. Post rape care (PRC) forms etc.

## HISTORY TAKING IN SEXUAL OFFENCES

A comprehensive history taking is necessary.

- Ensure that you acquire informed consent and ensure confidentiality. The consent should be documented.
- Examination of a person without their consent is detrimental and the health practitioner (medical officer or specialized consultant) or designated person (nurse or clinical officer) can be liable for breach of patient autonomy.
- Consent for the unconscious and mentally ill survivors can be given by their caregiver or an authorized person.
- Ensure confidentiality and explain to the patient the instances where this can be breeched e.g. under court order, shared confidentiality.
- For victims with extensive injuries, first aid is prioritized before comprehensive history and examination can be done.
- The history is taken in three parts;
  - General history including history of the incident and any significant medical history
  - Gynecology history
  - Psychological history

**General history**

The history should include;

- Date, time, place, number of person involved.
- A detailed history of the incident.
- Is the perpetrator known or unknown?
- Details of physical injuries
- Has the victim canged clothes, urinated, defecated, or douched after the incident?
- Any history on use of alcohol, medication or drugs for both the perpetrator and the survivor.
- History on use of condoms, lubricants, foreign objects or any sex devices.
- Any history on use of restraints
- Any history on use of weapons
- Any symptoms after the incident- pain in urinating, defecation, urethral discharge, vaginal discharge, anal discharge, scrotal/anal discomfort
- Any significant medical condition.

**Gynecology history**

This history is important and should include;

- Menstrual history- age of menarche, details of menstrual cycle, last normal menstrual period.
- Marital history, history pertaining to pregnancy, vaginal delivery and lactation
- Current sexual partner/s
- Last consensual sexual intercourse
- Sexual history prior to the incident.

**Psychological history**

This assesses the mental health status of the survivor.

The psychological assessment includes: general appearance, rapport, behavior, mood, affect, speech, insight, thought content and process, perception, orientation, judgement, memory, attention, alertness.

**Physical examination**

- Ensure privacy and dignity as you examine the patient. Avoid unnecessary exposure.
- Be sure to explain to the patient the whole examination procedure before and during the process. Inform them that they have the right to stop the examination at any point.
- Before the examination begins, the survivor should stand and undress on a white paper or bed sheet. This is to ensure any evidential material on them is collected in the white sheet.
- Conduct a head to toe physical examination.
- General examination- appearance, height, weight, gait, demeanor, mental status, vital signs.
- Examine the face, ears, nose and mouth- check for periorbital edema, conjunctival hemorrhage, shadow bruising behind the ear, oral examination in case of physical injuries or suspected oral penetration.
- Examine the neck for any bruising which could be a sign of life threatening injuries like strangulation.

- Examine the trunk for any injuries, including the back.
- Check for any abrasions and bruises over arms indicating struggle (defensive injuries).
- With the victim lying down palpate the abdomen for any tenderness or masses like pregnancy.
- Check for bruises or any fluids e.g. semen on the inner thighs. While the patient is standing, examine the legs and buttocks for any abrasions, lacerations, bruising, tram line injuries and evidential material like dirt, semen, vaginal discharge etc.

**Genital examination**

Inspect the external genitalia for any visible discharge like vaginal bleeding, matted pubic hair, signs of healed tears, bruises or lacerations in the labias, clitoris, perineum, hymen or hymenal remnants. Take any swabs before attempting any digital or speculum examination. Document genital structure assessment using the clock face analogy.

**Speculum examination**

This can be done with or without sedation as per guidelines. Check for vaginal, cervical, fourchette tears as you collect any evidential material.

**Rectal examination:** in cases of suspected anal penetration for tears, bleeding.

**Age estimation**: when needed.

**Evidence collection, preservation and linkage**

The principles to follow while collecting evidence include:

- Wear gloves during the whole examination process and change gloves with every different specimen collected. This is to avoid contamination.

- Collect the evidence early, ideally within 72 hours. However, this does not negate need to examine the survivor if they present to the facility after 72 hours.
- Handle all the specimens appropriately. Store fluids in appropriate temperatures e.g. urine and blood between $2^0$ C to $8^0$ C for short term storage (frozen at -$20^0$ C for longer storage). Air dry the biological evidential material like stained clothes and swabs under a shade, avoiding direct sunlight.
- All samples should be labelled using the survivor's or suspect's name and date of birth; type of specimen; date and time of collection and health care provider's name.
- Ensure security by keeping the collected samples in places that guarantee safety e.g. locked rooms/cupboards with authorized entry. Sign across the label to make it tamper proof.
- Maintain chain of custody by ensuring that for every collected sample, any subsequent handling is recorded. This can be through maintenance of a chain of custody book and documentation of SGBV samples in a different sample documentation book in the laboratory.
- Ensure all the samples are collected in threes; two for the government chemist and one for the facility.

**Investigations**

- The health care provider collects evidential material for preservation as well as onward transmission to the testing laboratory (commonly the government chemist).
- Blood samples are required for toxicology and DNA (deoxyribonucleic acid) analysis.
- Clothes for detection of blood, semen, foreign material.
- DNA analysis done on the samples collected.
- Sanitary pads, used condoms, panty liners, tampons and diapers for detection of semen and spermatozoa, DNA analysis.
- Foreign material e.g. soil, grass for transfer evidence analysis.
- Swabs from buccal cavity, inner thighs, vulvar, vaginal walls, introitus, cervical, anal opening, anal canal, rectal area may be required depending in the type of case, for DNA analysis, pathological organisms, semen and spermatozoa detection.
- Blood samples should be collected for for HIV test, liver function test, Hepatitis B test, VDRL, toxicology and DNA analysis. Collect 10mls in two purple vacutainers and 10mls in two red vacutainers.
- Tool marks such as those caused by a knife, hot iron, fire arm among others will require photography for possible identification of the tools causing injuries and documentation of the injuries.
- Nail scrapings and clippings are required for DNA analysis.
- Urine samples are required for microscopy for detection of spermatozoa, culture and sensitivity for pathological organisms, pregnancy testing and toxicology. Collect about 100mls by asceptic techniques.
- Hair samples such as pubic hairs for DNA analysis.

**Note: All samples should be handled by following a strict chain of custody.**

**Treatment**

The basic management of sexual offences victims involves

- **General wound care,**
  - Clean the lacerations and abrasions with antiseptic.
  - Primary suturing is done for minor, clean wounds under local anaesthesia. Consider general anaesthesia in cases of extensisve wounds.
  - Delayed suturing or healing by secondary intention is done for dirty wounds.
- **Sexually transmitted infections**
  - Offer STI treatment to survivors of penetration.
- **PEP for HIV**
  - Provide PEP as per treatment guidelines. (Refer to the latest PEP guidelines) within 72 hours of exposure.
- **Pregnancy prevention**
  - For penetrative sexual violence, provide emergency contraception within 120 hours.
  - One 750 µcg Levonorgestrel pill to be taken as soon as possible within 120 hours. Repeat the same dose in 12 hours. Can also be given as a single dose given on first contact.
  - IUCD inserted within 120 hours.
  - Estrogen pills such as Microgynon given as four tablets to be taken as soon as possible within 120 hours. Repeat the same dose in 12 hours.
  - Estrogen pills such as Eugynon given as two tablets to be taken as soon as possible within 120 hours. Repeat the same dose in 12 hours.
  - Ulipristal Acetate (UPA) given as a 30mg single dose within 120 hours.
- **Hepatitis B Vaccination**
  - For patients who have never been vaccinated against Hepatitis B, give 1st dose at initial visit, second dose at 1-2 months after 1st dose and third dose 4-6 months after 1st dose.
  - For patients who have at least one dose, finish the vaccination schedule.
  - For fully vaccinated patients, do not vaccinate.
- **Psychosocial support**
  - The healthcare provider should offer first line support.
  - Refer to a psychologist or psychiatrist for trauma counselling.
- **Follow up visits**
  - Follow up visits are done as per the schedule.
  - A minimum of 5 visits are scheduled at 2 weeks, 4 weeks, 6 weeks and 3 months respectively.
    Refer to the annexed algorithm for services offered on each visit.

**Documentation**

According to Medical Regulation Act of 2012, the post rape care form has to be filled for the survivor of sexual violence. History and examination findings are documented in the PRC (Post Rape Care) forms. P3 Forms are police issued documents that are filled in the facility. Do not fill a P3 that does not have Part 1 filled.

Fill the P3 with clinical findings at the time of the examination. You can also add the findings during the first visit but stating the date of that initial examination and the document from which the information is acquired e.g. PRC. Ensure clarity and completeness while documenting findings.

**Examination of perpetrators**
The examination principles applied are the same as for the victims.

## 68.3.2 VIOLENCE AGAINST CHILDREN

Health care providers have a mandate to ensure privacy, confidentiality, safety, non-discrimination, non-maleficence and all rights to ongoing development are upheld. They should obtain informed consent or assent as below:

**Table 64.2:** Obtaining informed consent or assent for a child

| Age Group | Child | Caregiver | If No Caregiver or Not In Child's Best Interest | Means |
|---|---|---|---|---|
| 0-5 | _ | Informed Consent | Other trusted adult's or case worker's informed consent | Written consent |
| 6-11 | Informed Assent | Informed Consent | Other trusted adult's or caseworker's informed consent | Oral Assent, Written Consent |
| 12-14 | Informed Assent | Informed Consent | Other trusted adult's or child's informed assent. Sufficient level of maturity (of the child) can take due weight | Written Assent, Written Consent |
| 15-18 | Informed Consent | Obtain informed consent with child's permission | Child's informed consent and sufficient level of maturity takes due weight | Written consent |

Source: Kenya National Guidelines on Management of Sexual Violence,2014

- Ensure that the health care provider examining the child is trained. The safety and wellbeing of the child supersedes evidence collection. Manage life-threatening injuries first.
- Provide first aid and **refer if not adequately trained to examine the child**. (Provide comprehensive information in the referral note and notify the facility you refer to).
- Inform the children's services for the purposes of ensuring safety to the child. Please note that the guardian can also be the perpetrator so watch out for the signs which include witness reports, overprotective guardians, repeat unexplainable injuries or medical conditions from the medical history, fear in the child, or from history given by the child.

- The history taken is the same as the adult history. Additionally, take note of history of repeated violence and gynecological history for adolescents.
- Special considerations in history include age and disability. For children below 5 years, history is given by the non-offending caregiver or adult accompanying the child. For children between 5-10 years, history is obtained from the child and supplemental history is given by the adult. For older children, history is from the child.in this older age group it's advisable to exclude the guardian from the history taking process.
- Child-friendly techniques like use of toys, role play, drawing, drama etc. should be used for the younger children (below 10 years).

**Examination**

The examination principles applied are the same as for the adult. Special circumstances for the child include doing a tanner staging for developmental assessment. An accurate description of injuries is mandatory.

- While doing anogenital examination for cases of sexual violence, place the child in supine frog leg, supine knee chest, lateral or prone knee chest position. Labial traction and separation is done to visualize the introitus in girls. Anal examination is best done using supine or prone knee chest position.
- Determine any injuries and document.
- Of importance in female patient examination, while documenting the hymenal changes, describe the size, patency, shape and margins of the orifice; thickness of the hymen tissue. To visualize the hymen better, use a moistened cotton-tipped swab to sweep around the hymenal edge.
- A small (8Fr) sterile foleys catheter can also be used in case of any detected abnormality. The catheter is introduced through the hymenal opening into the vagina and the balloon is inflated using 2-3mls of water to a degree that is tolerated. Do not use the catheter if the hymen is intact.
- Avoid speculum, digital and bimanual examination in pre-pubertal girls unless medically indicated. Speculum can be used in post pubertal girls. Indications for speculum use include foreign body in the vagina, per vaginal bleeding and genital injury requiring surgical repair.
- Do not conduct virginity testing.
- In the male patient: check for injuries on the foreskin, penis, prostate and anus, urethral discharge, anal sphincter tone. In an older child, the foreskin should be gently pulled back to examine the penis.

**Psychological assessment** should be done by a qualified health care professional.

**Investigations**

- Collect samples as per the adult guidelines.
  Urine samples are relied on heavily for spermatozoa detection and DNA analysis in cases where cervical, high vaginal swabs are not available. However, the external genitalia samples e.g. vulvar swabs should still be collected.

**Treatment**

- Physical injuries are treated as per guidelines.
- The basic treatment for sexual violence child is employed.
- Medication is given based on the weight.
- In pregnancy prevention, emergency contraceptive pills (ECPs) can be offered to girls who have attained menarche and those in the beginning stages of puberty (Tanner stage 2 or 3).
- HPV vaccination should be given to girls over 9 years using the national vaccination guidelines.

## 68.4 Traffic Medicine

Traffic medicine embraces all disciplines, techniques, and methods aimed at reducing the harm traffic crashes inflict on human beings. A majority of harm results from road vehicles. However traffic medicine also includes injuries from all vehicles travelling over land, sea, air, under-water and in space. The health worker treating the victim of a crash, the people transporting the victim from the crash site to a health centre, those in the institutions dispatching help to the crash site, are all involved in traffic medicine.

### 68.4.1 EXAMINATION OF VICTIMS OF ROAD TRAFFIC ACCIDENT

**Important consideration in history taking**

- Should take place in the casualty department for the referred clients.
- All referral medical notes investigations and evidence should be safely and securely stored.
- Emergency care services should be provided as per the guidelines
- Linkage to specialized care centers like ICU should be considered.
- Accurate documentation of all medical and surgical procedures is required, photographs may be used to illustrate injuries.
- Documentation should include date, time, palce of accident and any other information relevant to the accident.
- Whenever possible, the healthcare worker should find out if the victim was a pedestrian and if so what kind of vehicle hit them, the direction from which they were hit, if the pedestrian was lifted from the ground or even if they were run over following a hit.
- For pillion riders, the healthcare worker should find out if the person injured was wearing a helmet and if they were thrown off the vehicle.
- Details of what structures or objects (including other vehicles) the survivor impacted after the crash will aid in evaluation of secondary injuries.
- The position of the survivor in the vehicle as well as details on use of seat belts should be elicited and documented.
- Presence and deployment of airbags is an important consideration as well aas the possibility of the survivor having been injected from the vehicle.
- First aid services offered at the crash site and details of the persons who took the survivor to hospital should be sought and documented.
- Any other medical/surgical conditions in the survivors should be sought.

- Survivors of road crashes should be assessed and tested for alcohol, medical or recreational drugs. This is especially important for drivers, pilots, captains or pillion riders
- The healthcare worker should find out if the crash was reported to the police and an OB number issued. The police station to which the matter was reported and the OB number should be recorded.

**Forensic medical traffic accident examination**
- General condition, vital signs
- Injuries- Describe the type, location, dimensions, pattern of distribution, nature of injuries, colour changes and age of injuries.
- Pattern injuries e.g. tire marking.
- Other systems for medical conditions e.g. vision.

**Forensic investigations**
- Radiography/ sonography as needed to assess severity of injury.
- Blood/ urine sample for alcohol/ drugs.
- Photographs of pattern injuries. (By use of a facility designated camera).

**Management of accident victims**
- Provide the emergency care services as needed.
- Refer for specialized care to the level four five and six facilities as needed.
- Health care provider should preserve the medical notes and any evidence for legal purposes.
- Filling of p3 form conclusively and clearly.
- Forensic clinician presentation of evidence before court of law.
- Forensic clinician to ensure chain of custody of all documents and evidence collected.

**Checklist**
- Procedure done
- History elicited
- Complete external examination
- Appropriate investigation
- Final opinion after investigation
- Referred to a higher centre when needed?
- Nature of injury
- Duration of injury
- Documentation
- Blood sample for alcohol and drugs
- Scene visited

## 68.5 Injury assessment, documentation and interpretation

The accurate description of injuries should fall within the capabilities of most health workers. Interpretation requires considerable skill and expertise and often is best left to a forensic physician or pathologist. Nevertheless, other health workers should offer some advice and comment, albeit of a general nature, on how a particular injury or group of injuries was caused. Indeed, in some circumstances it is the ordinary practitioner who draws attention to the possible medico legal significance of some injuries and initiates an investigation into their cause, e.g., pediatricians in cases of non-accidental injury in children.

### 68.5.1 DESCRIPTION OF FORENSIC INJURIES

When describing an injury, it is essential to comment on the nature of the wound, its size, shape, and its location. The age of the injury also needs to be considered. It is extremely useful to record injuries on body charts or anatomic line drawings, which can be used in court if the health worker is called to give evidence.

In cases of serious assault or when injuries have distinctive characteristics or patterning, it is essential that the wounds be photographed with a suitable metric scale alongside.

### 68.5.2 NATURE OF THE INJURY

In legal terms, a "wound" requires the integrity of the skin surface to be breached and theoretically at least would exclude bruising and indeed abrasions. It is therefore essential that for medico-legal purposes a standard nomenclature be adopted when describing injuries.
The following classification is adopted by forensic physicians and pathologists.

- **Bruises:** often called contusions due to the application of blunt force. The blow ruptures small blood vessels beneath the intact skin, and blood then escapes to infiltrate the surrounding subcutaneous tissues under the pumping action of the heart.
- **Abrasions:** also known as scratches superficial injury involving only the outer layers of the skin and not penetrating the full thickness of the epidermis. Some abrasions may be contaminated with foreign material such as dirt or glass, which have important medico-legal significance. Such material should be carefully preserved for subsequent forensic analysis.
- **Lacerations:** sometimes known as cuts and tears
- **Incisions:** colloquially called slashes
- **Stab wounds**—sometimes known as penetrating wounds.

**Note:** A variety of wound types may co-exist following trauma. Furthermore, a single wound may show features of different types.

### DESCRIBING SIZE AND SHAPE OF FORENSIC INJURIES

Ascertained using a ruler or a pair of calipers and recorded in centimeters or millimeters. The shape of the wound should also be noted; simple terms such as circular, triangular, V-shaped, or crescentic best express this characteristic, but if the wound shape is irregular or complex then it is possibly easier to record this feature on a body chart. Wounds also may have depth, but it is often not possible to determine this accurately in the living.

### DESCRIBING POSITION OF FORENSIC INJURIES

The best method of pinpointing the location of an injury is to use fixed anatomic landmarks. On the head, one can use the eyes, ears, nose etc. The advantages of using simple anatomical diagrams and body charts for locating the injury are self-evident.

### DOCUMENTING TRANSIENT LESIONS

Swelling, redness, and tenderness, although frequently caused by trauma, are not specific signs of injury but are rather an indication of the effects of the injury. It is however important to examine for and record these features. An example would be red marks outlining an injury such as the imprint of a hand on the slapped face or buttock of a child. These features should be photographed at the earliest opportunity as such changes may fade or change colour with time. The colour and appearance of these transient lesions is of value in dating the injury.

## 68.6 Examination of victims of assault

### 68.6.1 SHARP OBJECT TRAUMA

- Location of examination should be any place with privacy safe to the victim and with available necessary equipment.
- Preliminaries- name, age, gender, address
- Whether police were informed and a report prepared at the police station and an OB.NO ( Occurrence Book No) issued.
- Indicate whether the victim was assaulted by a single person or a group of persons. Inquire if It was anticipated and defensive or unanticipated. What kind of weapon was used and Who has accompanied the victim-relatives/police. If the suspect is a known/unknown person.
- Whether the victim was taken to any other hospital before being examined and intervention done/ report prepared anywhere else. It's important to note the handedness of the victim and suspect. Any consumption of alcohol or drugs should be documented.

### 68.6.2 OTHER FORENSIC INJURIES EXAMINATION

- Place of examination**.** Should be examined at a private place with all necessary equipment. Infection prevention protocols should be adhered to.
- Examination of clothes for cuts corresponding to injuries over the body.
- The Site, dimensions and depth (plane) of injuries, margins, edges, surrounding tissues, active bleeding present/absent, underlying neurovascular bundle damaged or not, underlying tissues protruding or not,

undermining/beveling if present should be well documented and possibly illustrated on the body map.

- Indicate whether single wound of entry (penetrating) or both entry and exit (perforating) are present in stab injuries- shape of injury, orientation with the axis of the body, distance from anatomical landmarks, distance from other injuries (in case of multiple injuries), distance from heel of injured. Look for Defense injuries in the periphery: incised wounds/stabs over forearm, arm, wrists, and hands.
- Diagrammatic representation in cases of forensic interest is fundamental.

## 68.6.3 INVESTIGATION FOR FORENSIC DOCUMENTATION

- The request forms must indicate that the investigations are for legal purposes and be correctly labeled indicating date, time and all other vital particulars. These may include the following
  - Radiography to rule out underlying fracture/dislocation, haemo/pneumothorax/ air under the diaphragm.
  - Computed tomography of brain to look for intracranial bleed/contusions.
  - Ultrasonography of abdomen and pelvis to look for collection of blood/fluid.
  - Doppler/ vascular studies to look for damage to vessels.
  - Expert opinion for vision/ hearing defects and other medical departmental findings including Blood, urine samples for alcohol/drugs.

## 68.6.4 MANAGEMENT INJURED VICTIMS

- Provide the emergency care services to the needy as per protocols.
- Refer for specialized care appropriately.
- Ensure all medical notes are complete, accurate and preserved for future reference.
- Handle all forensic evidence obtained through appropriate chain of custody.
- P3 forms should be filled accurately and conclusively.
- The health care provider should be available to present evidence at a court of law.

**Checklist**

**Its best practice to maintain an audit sheet to ensure all the necessary measures has been adhered to and documentation done with preservation of all physical evidence.**

- Correct procedure followed
- History elicited
- Complete external examination carried out
- Appropriate investigation ordered and done
- Final opinion after investigation offered
- Other expert opinion sought as needed
- Referral to a higher level of care done as required
- Nature of injury documented
- Type of weapon documented, if possible

- Age of injury assessed
- Tract of the injury assessed where relevant
- Opinion on injury compatibility with suspected weapon offered, where possible
- Blood samples collected for alcohol, drugs and any other tests as required
- Clothes examined and preserved
- Clothes forwarded to the police as part of evidence



## 68.7 Thermal Injuries

Thermal injuries may be broadly classified into two:

- Thermal injuries due to heat
- Thermal injuries due to cold

**Injuries due to heat**

Injuries due to heat may be caused by moist or dry heat.

Injuries due to moist heat are mainly scalds. Scalds are caused by contact with a hot liquid such as in immersion, pouring or splashing of the hot liquid on a person. The affected skin is soggy, blanched, and blistered. The shape of the injury is contoured. The depth of the burn is variable. Forensic examination of scalds involves determining an upper fluid level for immersion cases or a drip pattern in cases where fluid is splashed on a person.

- Injuries due to dry heat are caused by contact of the skin with open flames. Common patterns of injuries due to dry heat include:
  - **Contact burns:** direct contact with a hot object. Characteristically, the burn is shaped like the hot object with sharply defined edges and usually of uniform depth. The burn may blister.
  - **Flame burns**: caused by flames from fires such as matches and lighters that come into close or direct contact with the skin, causing charring and skin loss with singeing of hairs.
  - **Cigarette burns**: inflicted direct contact leaves a characteristically well-demarcated circular or oval mark with rolled edges and a cratered center, which may blister and tends to scar. Accidental contact with a cigarette tends to leave a more superficial, irregular area of erythema with a tail.
  - **Friction burns** dragging or rubbing injury causing superficial skin loss, with broken blisters, usually on bony prominences.

**Electrical Injuries**

- **Electrical burns**: small, deeply penetrating burns with an entry and exit wound with possible necrosis of underlying tissues.
- **Chemical burns**: the chemical in liquid form is drank, poured, or splashed onto the skin, or in solid form is rubbed on the skin. The skin may stain, may have the appearance of a scald, and may scar.
- **Radiant burns**: more extensive areas of erythema and blistering on exposed body parts.

## 68.7.1 DIFFERENTIAL DIAGNOSIS OF THERMAL INJURIES

- Infections such as Staphyloccal, streptococcal (impetigo, scalded skin syndrome).
- Allergy – urticaria, contact dermatitis.
- Insect bites.
- Bullous diseases such as Porphyria, Erythema Multiforme

## 68.7.2 MEDICAL EXAMINATION OF VICTIMS OF FLAME BURNS

**Factors to observe in history taking.**

- Find and document the date, place of incident, possible of exposure.
- Enquire about the possible aussative agent of the fire.
- Enquire whether the matter was reported and an OB number issued.
- Enquire and document about the type of clothing worn by the vioctim at the time of the incident.
- Find out any history of damage to structures, vehicles and other objects in the vicinity of the fire that may cause other injuries other than those due to fire.

**Factors to consider during examination of thermal injuries**

- Document vital signs
- Examine the location, depth of burns, percentage of burns (total burn surface area, TBSA).
- Sketch the area involved.
- Examine for facial burns that may indicate possibility of inhalational burns
- Examine for signs of infection
- Examine for associated injuries aused by damage to structures in the vicinity of the fire.

Associated injuries due to trapping in masonry. Respiratory disturbances due to CO inhalation.

**Investigation of thermal burns**

- Clothes, burnt hair and cuticles for detection of combustible substances.
- Photography of burns
- Scene of crime visit notes; illustrated diagrams and photographs by a clinical forensic physician.

**Checklist**

It is best practice to maintain an audit sheet to ensure all the necessary measures has been adhered to and documentation done with preservation of all physical evidence.

- Correct procedure followed
- Detailed history
- Surface area involved/ sketch
- Depth of burns
- Injuries over body
- Clothes/ hair/ skin preserved

**Examination of Victims of Scald Burns**

**Key findings on Forensic History**

- Day, time, place of incident.
- Substance causing burns: water/oil/other chemicals.
- Duration of contact with skin.
- Type of cloth worn by victim at the time of incident.

**Factors to consider on examination**

This must involve the mental status of the patient and vital signs. Further, clothing, area of distribution, depth of burns, percentage of burns, sketch of area involved and signs of dribbling should be examined. An examination of scalp, face, body hair Inflammation in old burns and signs of secondary infection should also be conducted.

**Investigation**

- Clothes and cuticle for detection of chemicals.
- Saline swab for detection of chemical substances.
- Photography of burns lesions.
- Scene of crime visit notes sketch maps and photographs

**Audit sheet**

- Correct procedure followed
- Detailed History
- Surface area involved/ sketch
- Depth of burns
- Clothes/ hair/ skin preserved
- Scene of crime documentation

## 68.7.3 EXAMINATION OF VICTIMS OF ELECTROCUTION

**History taking**

- Day, time, place of incident.
- Source of current: domestic/ occupational/ others
- Type, strength, tension, resistance of current.
- Was there a contact? If yes Duration of contact?
- Was the scene wet or dry?
- Type of cloth worn by victim at the time of incident

**Examination of electrocution victims**

- Mental status of the patient, vital signs.
- Entry wound, exit wound of electrocution (if any).
- Number, site, size, location, surrounding area.
- Associated flash/ flame burns.

**Investigation of victims of electrocution**

- Photography of burns
- Scene of crime visit notes and sketch maps

**Management burns victims**

- Provide the emergency care services to the needy as per protocols.
- Refer for specialized care.
- Forensic clinician maintain the forensic medical notes and preserve evidence for legal purposes.
- Filling of p3 form conclusively and clearly.
- Forensic clinician presentation of evidence before court of law.
- Forensic clinician to ensure chain of custody of all documents and evidence collected.

**Audit sheet**

Its best practice to maintain an audit sheet to ensure all the necessary measures has been adhered to and documentation done with preservation of all physical evidence

- Correct procedure followed
- History
- Surface area involved/ sketch
- Depth of burns

## 68.8 Care of Detainee and Custodial Medicine

A health worker will often be asked to assess the fitness of an adult or juvenile for detention in police custody. This is usually in connection with an arrest for an offense committed, or detained by immigration, or requiring a place of safety (children and the mentally ill), or remanded or sentenced (convicted) prisoners.

A person in police custody is referred to as a detainee and guidance may have to be given to the custodians regarding their care. If an individual detained in police custody appears to be suffering from a mental or physical illness and needs medical attention or has sustained any injuries whether at arrest or prior to arrest, such attention should be sought as soon as possible. Increasingly the police have to deal with individuals who misuse alcohol and drugs or are mentally disordered. When the detainee's behavior gives rise to concern, medical advice should be sought.

Custody staff should also seek medical advice if a detainee requests a doctor or requires medication or if the custody staff suspect that the detainee is suffering from an infectious disease and needs advice. In some areas, when a person under arrest is discharged from the hospital and taken to a police station, a doctor will be called to review the detainee and assess whether he or she is fit to be detained and fit for trial.



## 68.8.1 ADMINISTRATION OF MEDICATION

- Ensure clear and detailed instructions regarding any medication to be administered while the detainee is in police custody i.e. times of administration, and special instructions) are given to custodians with confirmation that these instructions are understood.
- Sufficient quantity of medication should be prescribed to cover the time in detention.
- The medication should be given to the police in appropriately labeled individual containers or sachets.
- Records should be kept showing that prescribed medication is given correctly and timely and that unused medicines are accounted for.
- Medication should be stored under lock.
- Police should ensure that when administering medication, they are accompanied by a witness and detainee to be observed taking medication to prevent hoarding.
- Detainees arrested with medications on their persons, medical advice should be sought as to whether they should be allowed to self-administer them.
- Medication brought with the prisoner or collected from home should be checked to ensure that it has the correct name and dosage and that the quantity left is consistent with date of issue.
- If the medicine is unlabeled, it is preferable to issue a new prescription.

## 68.8.2 CONDITIONS OF DETENTION

- Medical examiner should ensure that the conditions of detention are satisfactory with regard to:
  - The temperature and ventilation of the detention cells,
  - Cleanliness of the cell,
  - Bedding and personal hygiene,
  - Access to dietary needs, and fluids as appropriate,
  - Period of rest of 8 hours during each 24 hours.

### 68.8.3 INFECTIOUS DISEASES IN DETENTION AREAS

- The clinician may be called to advise the police/custodial homes regarding infectious diseases.
- These populations are indeed at high risk for blood-borne viruses such as Hepatitis and HIV/AIDS, all individuals should be considered a potential risk, and observation of good clinical practice relating to body fluids to avoid contamination risks is fundamental.
- Concerns regarding untreated acute infections such as open Tuberculosis should warrant transfer to the nearest Health units for assessment regarding treatment.
- Scabies may be treated in the custodial setting; however, bedding and cells should be professionally cleaned.

### 68.8.4 PERSONAL SAFETY ISSUES

Certain health care groups are at increased risk of violence in the work-place e.g. those working in clinical forensic medicine and accident and emergency services.

**Strategies for interviewing a difficult patient**

- Being fully aware of the person's history (be prepared!), and considering how the person sees you (as uninterested or hostile?)
- Being polite and respectful.
- Avoiding confrontation.
- Using appropriate eye contact.
- Keeping calm, and showing interest.
- Look for signs of tension and find out why tension may be increasing.
- Being ready to leave if necessary and consider the need to have a chaperone.

## 68.9 Drug Searches

Individuals unlawfully in possession of illicit drugs or involved in drug trafficking may ingest drugs or pack them into certain body cavities ("body packers" or "mules"). A person about to be arrested by the police may swallow drugs.

Forensic clinicians may be called by the police to carry out intimate searches of those arrested. A discussion of possible implications in the ingestion of drugs and obtaining fully informed consent is paramount before carrying out any search that may involve examination of the mouth, nostrils, ears, foreskin, rectum, or vagina.

### 68.9.1 HOW TO CARRY OUT DRUG SEARCHES

All drug searches should be carried out in premises where there are full facilities for resuscitation in case of significant quantities of drug leaks into the bloodstream. This can result in acute intoxication and even death from overdose. Medical problems such as bowel obstruction may also occur.

In a genuine emergency, when there is no possibility of obtaining consent, the doctor has a duty to carry out treatment to safeguard the life and health of a patient in accordance with what would be accepted as appropriate treatment in the best interest of the patient.

### 68.9.2 COLLECTION OF FORENSIC SAMPLES

Samples from a detainee such as dental impressions, blood, saliva, urine, hair, fingernail scrapings and cuttings, swabs (e.g., mouth, penile) may be requested by police authorities in connection with the investigation of an offense.

These samples should only be taken by a clinician or nurse for evidential purposes with the detainee's fully informed consent and should be packaged in accordance with local procedures to ensure the chain of evidence.

A police officer in the rank of inspector of police and above may give consent if the victim is unable. This must be signed in the presence of a health worker as a witness.



## 68.10 Identification of the living

The clinician should document the available biodata of each client if available including; name age sex physical address local chief etc. If the person is unknown, the following criteria should apply:

### 68.10.1 IDENTIFICATION CRITERIA

These could be Primary, Secondary, Tertiary

**Primary identification criteria**

- Fingerprints lifted by the police officers only at any safe and secure location with necessary equipment.
- DNA samples collected by clinician and packed accordingly ie. Blood. Hair strands for submission to the government chemists.
- Unique medical characteristics eg Use of prosthesis. should be documented.

**Examination of dental structures**

Forensic odontology techniques are widely used in the establishing or confirmation of identity when bodies are found, or after mass disaster. Pre-existing dental records and charts and radiographic images can be compared with examination of the teeth of the deceased. The odontologist may also be

asked to make dental charts of bodies whose identity remains unknown or unconfirmed despite a police investigation, so that, should dental information

become available at a later date, the two sets of records may then be compared. Neither a living individual nor a body can be identified simply by taking a dental chart – that chart has to be compared with, and found to match, a chart whose origins are known.

**Secondary criteria**
Features such as deformity, marks and scars, X-rays, personal effects and distinctive clothing.

**Tertiary criteria**
Features that provide some assistance in identification include clothing, photographs location.

**Additional techniques**
Techniques such as gait analysis from CCTV can be useful when other features cannot be used These must be sent to the police for analysis and documentation.

All unidentified person's cases must be reported to the police station for further investigation action and linkage to other necessary institutions.

## 68.11 Fitness for trial

The custodial interrogation of suspects is an essential component of all systems of criminal investigation. The confessions and other incriminating statements that are obtained during these interrogations play an important role in prosecutions. They are relied on as evidence of guilt in a substantial number of trials.

A forensic physician should hence perform a thorough examination to detainee to ascertain fitness of trial.

**A detained person may be unfit for interview when**:

- Conducting an interview could worsen any existing physical or mental illness to a significant degree.
- Anything said or done by the detained person at the time of detention may be considered unreliable in subsequent court proceedings because of the physical or mental state of the detained person."

### 68.11.1 QUANTIFYING RISK OF UNRELIABILITY

In making an assessment, the clinician should quantify the risk of unreliability into one of the following categories:

- Definite unlikely to be fit for interview at any stage (e.g., severe dementia, severe mental handicap).
- Major risk unfit for interview at present. Reassessment or further review is considered necessary to establish fitness at a later stage (e.g., drunkenness,

intoxication with drugs, severe drug withdrawal, severe physical illness, major mental disorders that may be amenable to treatment such as mania and acute confusion states).

- Some risk Precautions are advised, such as the presence of an appropriate adult or referral for other medical or psychiatric advice (e.g., mental illness such as hypomania, schizophrenia and depression, mild dementia or mental handicap, significant anxiety).
- No discernible risk Fit for interview, in so far as the interview can proceed without any special precautions.

## 68.11.2 SCHEME OF EXAMINATION

When assessing the fitness of a detainee for an interview, the traditional medical model of taking a history and then conducting an examination should be employed. As always, informed consent should be obtained and detailed and contemporaneous notes should be taken.

**History taking**

**Location**

- Private safe clean environment with an examination couch, good lighting and ventilation with basic equipment
  Much background information should be obtained and, when possible, an indication of how long any interview is likely to take.

**General medical history**

- Enquiry made about any significant illness and any prescribed medication.
- Ask about history of psychiatric illness, past or present,
- Enquire about alcohol and drug misuse.
- Questions about the person's educational background.
- Ensures the detainee has not been deprived of food or sleep
- Enquire about significant social distractions (for example, a single parent may make a false confession in order to obtain early release from custody.

Detainees should be asked whether they have been detained before and any unpleasant experiences in custody

**The Examination**

- Should include observations on the general appearance, physical examination as appropriate, and mental state examination.

- A functional assessment should be performed as to whether the detainee is aware of the reason for arrest, is aware of legal rights, and is capable of making a rational decision.

# 69. Forensic Pathology

Forensic pathology is a subspecialty in pathology that deals with forensic investigation of deaths for persons who die suddenly, unexpectedly or violently to determine cause and manner of death. It involves performing post mortem examinations, attending death scenes, carrying out forensic exhumations and testifying at a court of law.

## 69.1 Death in the community

This section applies to persons who die at the facility, persons brought in dead or persons who die on arrival to the facility.

- The medical officer takes a clinical history and examines the body to confirm the person has died then prepares clinical notes to that effect and fills out the verbal autopsy form. The medical officer must determine if the death is of forensic importance from history and examination.
- Confirmed deaths of no forensic interest are transferred to the mortuary for preservation. The medical officer issues a burial permit (D1) in preparation for disposal of the body. She/he also prepares and submits the register for death.
- Confirmed deaths of forensic importance are referred to the police for forensic death investigation.

## 69.2 General concepts in medical investigation of death

### 69.2.1 DEFINITIONS

- Clinically, death refers to the irreversible cessation of all vital functions of life especially as indicated by permanent stoppage of the heart, respiration, and brain activity. This is somatic death and means that the individual will never again communicate or deliberately interact with the environment. The individual is irreversibly unconscious and unaware of both the world and their own existence.
- Scientifically, death occurs at individual cells. Cellular death means the cessation of respiration (the utilization of oxygen) and the normal metabolic activity in the body tissues and cells.
- Understanding of the physiology of death impacts medico-legal aspects of resuscitation, the vegetative state, brain death and cadaveric organ donations.
- Death may have forensic and non-forensic interests:
  - Forensic deaths are those deaths that are sudden, unexpected or due to violence arising from unnatural causes such as accident, murder, suicide, among others.

- Non forensic deaths are those that arise from disease processes whose pathophysiological processes can be explained such as hypertension, tuberculosis, malignant diseases, among others.

## 69.3 Clinical confirmation of death

### 69.3.1 PROCEDURE IN DEATH CONFIRMATION

- Wash your hands and don PPE if appropriate.
- Confirm the identity of the patient by checking their wrist band.
- Inspect for obvious signs of life such as movement and respiratory effort.
- Assess the patient's response to verbal stimuli (e.g. "Hello, Mr Smith, can you hear me?").
- Assess the patient's response to pain using one of the following methods:
  - Apply pressure to the patient's fingernail.
  - Perform a trapezius squeeze.
  - Apply supraorbital pressure.
- Assess the patient's pupillary reflexes using a pen torch: after death, the pupils become fixed and dilated.
- Palpate the carotid artery for a pulse: after death, this will be absent.
- Perform auscultation in an attempt to identify any heart or respiratory sounds:
  - Listen for heart sounds for at least 2 minutes.
  - Listen for respiratory sounds for at least 3 minutes.
- The recommended amount of time to listen for heart and respiratory sounds can vary, but it is generally accepted that a minimum of five minutes of auscultation is required to establish that irreversible cardiorespiratory arrest has occurred.
- Wash your hands, dispose of PPE appropriately and exit the room, making sure the relevant doors and/or curtains are closed/drawn behind you.

### 69.3.2 APPEARANCE OF THE BODY AFTER DEATH

Changes occurring after death may be classified based on the time interval since death:

- Immediate changes after death:
  - Heart stops
  - Breathing ceases
  - Fall in blood pressure and cessation of circulation of the blood usually render the skin, conjunctivae and mucous membranes pale.
  - Nervous activity ceases and reflexes are lost - reflexes are lost and breathing stops. In the eye the corneal reflex ceases and the pupils stop reacting to light.
  - Retinal vessels, viewed with an ophthalmoscope, show the break-up or fragmentation of the columns of blood, which is called 'trucking or 'shunting'.
  - Eyes lose their intraocular tension.

- Muscles become flaccid (primary flaccidity), with complete loss of tone which may lead to voiding of urine, emission of semen and regurgitation of gastric contents.

- Intermidiate change:
  - Rigor mortis – stiffening of muscle due to lack of ATP.
  - 'Cadaveric rigidity' is said to be the stiffness of muscles that has its onset immediately at death, and the basis for this concept is the finding of items gripped firmly in the hand of the deceased before the onset of normal rigor. Most cases are said to be related to individuals who are at high levels of emotional or physical stress immediately before death.
  - Post-mortem hypostasis or post-mortem lividity describes the visual manifestation of simple fluid movement occurring within the blood vessels leading to filling of the dependent blood vessels.
  - Postmortem cooling is the loss of heat from the body to the environment till an equilibrium is achieved.
- Late postmortem changes:

  These changes are characterized by decomposition and putrefaction. Early features of body decomposition include:
  - Purging
  - Marbling
  - Skin slippage
  - Green discolouration
  - Bloating
  - Offensive smell

  Late features of decomposition include:
  - Mummification
  - Adipocere formation
  - Skeletalization

# 69.4 Postmortem examination

## 69.4.1 DEFINITION

Postmortem examination refers to the examination of a body after death.

## 69.4.2 TYPES OF POSTMORTEM EXAMINATION

- View and grant postmortem involves the external examination of a body without dissection.
- Autopsy (also referred to as necropsy) involves external and internal examination of a body and requires evisceration and dissection of internal organs. Autopsies may be classified as:
  - clinical (done to determine the extent of a disease for which a patient was being treated
  - forensic (for the investigation of sudden, suspicious, obscure, unnatural, litigious or criminal deaths).

### 69.4.3 PURPOSE OR OBJECTIVE OF POSTMORTEM EXAMINATION

- To make a positive identification of the body and to assess the size, physique and nourishment.
- To determine the cause of death or, in the newborn, whether live birth occurred.
- To determine the mode of dying and time of death, where necessary and possible.
- To demonstrate all external and internal abnormalities, malformations and diseases.
- To detect, describe and measure any external and internal injuries.
- To obtain samples for analysis, microbiological and histological examination, and any other necessary investigations.
- To retain relevant organs and tissues as evidence.
- To obtain photographs and video for evidential and teaching use.
- To provide a full written report of the autopsy findings.
- To offer an expert interpretation of those findings.
- To restore the body to the best possible cosmetic condition before release to the relatives.

### 69.4.4 PRELIMINARIES FOR AUTOPSY EXAMINATION

- Authorization and consent – authorization for forensic autopsies is required from the investigating authority and consent from family, guardian or relatives is required for clinical autopsies.
- Examination of the scene of death may offer important information for the autopsy including estimation of the postmortem interval.
- Identification of the deceased person. Methods of identification of deceased persons include:
  - Physical features such as facial appearance, eye color, skin pigmentation, hair colour and texture, tattoos, scars, statures, occupational stigmata and racial features. This is the commonest method used to identify fresh bodies.
  - Finger, palm, foot and lip prints – finger prints are the commonest used prints in identification of deceased persons. The finger prints are compared with previously obtained prints when the deceased was alive.
  - Molecular identification by DNA analysis is reliable but expensive method of identification and therefore not for routine use.
  - Dental identification involves examination of dental structures and comparing with previous dental records.
  - Identification of skeletalized remains may give information on age, sex stature
- History of the case is important to guide the autopsy process. The history of the case may be lacking for forensic autopsies.
- Infection prevention and control – Bodies of deceased persons and the environment in which they are found may pose risk of infection and adequate measures must be taken to mitigate these risks. These measures include use of appropriate personal protective equipment, proper medical waste management, engineering controls, among others.

## 69.4.5 PROCEDURES IN POSTMORTEM EXAMINATION

- External examination involves examining the outside appearance of the body for, postmortem changes, nutritional status, skin colour, congenital abnormalities, evidence of disease processes, injuries and presence of foreign bodies. The examination should include body orifices.
- Internal examination involves examination of eviscerated internal organs for injuries, disease processes, congenital abnormalities and foreign bodies.
- Ancillary laboratory investigations including toxicology, microbiology and histology may add useful information to the autopsy examination.
- Autopsy radiology should be undertaken where facilities are available but should be considered for ballistic injuries, air embolism, barotrauma and pneumothorax among others.
- Forensic photography is useful in creating a record of the findings at autopsy.
- The autopsy report is an integral part of the procedure and should receive as much attention as any physical procedure in the autopsy room. It is a permanent record of the findings at autopsy and is especially vital for medico-legal purposes where every effort must be made at the time to make it as comprehensive and useful as possible. It should include the following:
  - Full personal details of the deceased subject, unless unidentified.
    - This includes the name, gender, age, occupation and address.
    - The place, date and time of the autopsy.
    - The name, qualifications and status of the pathologist/medical officer.
    - Persons present at the examination.
    - The authority commissioning the autopsy.
    - A record of who identified the body.
    - The name and address of the deceased subject's regular (or last) medical attendant, where available.
    - The date and time of death, where known.
    - The history and circumstances of the death.
  - External examination - The 'external examination' should record all external findings including:
    The height, weight and apparent state of nutrition.
    - The presence of natural disease such as oedema, abdominal swelling, cutaneous disease, senile changes, etc.
    - Identifying features such as skin colour, tattoos, scars, congenital or acquired deformities, dentures, eye colour and hair colour.
    - The presence of rigor, hypostasis, decomposition and abnormal skin coloration.
    - The condition of the eyes, including petechiae, arcus senilis, pupil size, and the condition of iris and lens.
    - Condition of mouth and lips, including injuries, teeth and presence of foreign material.
    - Condition of external genitals and anus.
    - Listing and description of all external injuries, recent and old.
    - Evidence of medical intervention.
- Internal examination records all abnormalities, usually in a conventional sequence such as:

- Cardiovascular system: heart weight, any dilatation, ventricular preponderance, congenital defects, the pericardium, epicardium, endocardium, valves, arteries, myocardium, aorta, other great vessels and peripheral vessels.
- Respiratory system: external nares, glottis, larynx, trachea, bronchi, pleural cavities, pleura, lungs (including weight) and pulmonary arteries.
- Gastrointestinal system: mouth, pharynx, oesophagus, peritoneal cavity, omentum, stomach, duodenum, small and large intestine, liver (weight), pancreas, gall bladder and rectum.
- Endocrine system: pituitary, thyroid, thymus and adrenals.
- Reticuloendothelial system: spleen (weight) and lymph nodes.
- Genitourinary system: kidneys (weight), ureters, bladder, prostate, uterus, ovaries and testes.
- Musculoskeletal system: skull, spine, remaining skeleton and musculature where necessary.
- Central nervous system: scalp, skull, meninges, cerebral vessels, brain (weight), middle ears, venous sinuses and spinal cord (when examined).

- A list of specimens and samples retained for further examination. Those handed to other agencies, such as the forensic science laboratory, should be formally identified by means of serial numbers and the name of the person to whom they were handed.
- The results of further examinations such as histology, microbiology, toxicology and serology. When the main report is issued soon after the autopsy, these will not yet be available and a supplementary report will be necessary.
- A summary of the lesions displayed by the autopsy
- Discussion of the findings, if necessary in the light of known history.
- An opinion as to the definite or most likely sequence of events leading to the death.
- A formal cause of death, in the format recommended by the World Health Organization, suitable for the completion of a death certificate.
- The signature of the pathologist/medical officer.

Postmortem artifacts should be identified and documented. These may include those due to autolysis and decomposition, heat fractures of bones, animal scavenging.

Resuscitation artefacts and features of medical intervention should be identified and differentiated from trauma. These may include rib fractures, venipuncture marks, electric defibrillator pad marks, damage to the mouth, palate, pharynx and larynx from attempts to introduce a laryngoscope or airway, among others

## 69.4.6 POSTMORTEM EXAMINATION OF WOUNDS

Accurate assessment, classification, documentation and interpretation of wounds is of paramount importance in forensic pathology.

**Classification of wounds**
Wounds are classified according to appearance and causation into:

- Wounds due to application of physical force:
  - Wounds due to blunt force trauma are injuries caused by objects without a cutting edge. They include:
    - Abrasions - An abrasion is the most superficial of injuries and is one that does not penetrate the full thickness of the epidermis. Thus, typically, abrasions do not bleed, as blood vessels are confined to the dermis. Deeper abrasions, however, may penetrate the dermis and cause bleeding. Different types of abrasions have forensic importance and relate to the mode of causation such as brush abrasions, crushing abrasions, fingernail abrasions, patterned abrasions and postmortem abrasions.
    - Contusions - an abrasions lies beneath an intact epidermis and consists of an extravascular collection of blood that has leaked from blood vessels damaged by mechanical impact. Dating contusions is of forensic importance.
    - Lacerations – lacerations are wounds caused as a result of a blunt force compressing or stretching the skin; they may extend through the full thickness of the skin and can bleed profusely. They typically have irregular edges and bridging tissues.
  - Wounds due to sharp force trauma are caused by objects with a cutting edge and include:
    - Incisions – are generally longer that they are deep and typically have regular edges with cleanly divided tissues.
    - Stabs – are generally deeper that they are long with cleanly divided tissues and regular edges. Examination of stab wounds may give an indication of the weapon/object used.
    - Chop wounds are a type of wounds caused by heavy relatively blunt objects and are associated with contused edges.
    - Bite wounds - Bite marks are commonly associated with bruising and may be associated with lacerations if severe force is applied.

- Wounds caused by application of heat or cold:
  - Wounds due to application of heat – include burns (due to application of dry heat) and scalds (due to application of moist heat).
  - Ante-mortem burn wounds should be differentiated from postmortem burn wounds.
  - Death in fire injuries may also result from inhalation of fumes by thermal injury to the air passages and lungs by hot gases, or form carbon monoxide poisoning.
  - Wounds due to application of cold include frost erythema and frost bite.

- Chemical exposure to the body may cause chemical burns
  - Examination of burn wounds should determine the burn surface area and the depth (degree) of burns.
- Wounds caused by electrical injury include blister lesions and spark burns.

### 69.4.7 INJURY DOCUMENTATION

Documentation of injuries may take the form of medical/clinical notes, photographs, video and body charts.

### 69.4.8 INJURY INTERPRETATION

Examination of the site, orientation and pattern(s) of the wounds will often reveal useful indications about the causation of the wound. Documentation of injuries should include:

- Type of injury
- Size and shape of injury
- Site of injury
- Position of injury
- Age of injury
- Orientation of injury
- Pattern of injuries, where applicable

## 69.5 Ballistic injuries

Refers to injuries caused by the impact of a projectile in flight on the body. These Injuries include those due to gunshots and explosives.

### 69.5.1 GUNSHOT INJURIES

For purposes of forensic examination of ballistic injuries, firearms may be classified as smooth barreled (low velocity) or riffled (high velocity) firearms.

- Injuries caused by smooth-barreled firearms

These firearms discharge pellets that leave the muzzle in a compact mass and spread out as they travel away from the gun. The further away from the gun that the victim is situated, the larger the pellet spread, and the larger the area of potential damage. The appearance of the wound is therefore determined by the distance of the victim from the muzzle of the gun:

- Contact wounds are created when the gun muzzle abuts the skin and usually results in a circular entrance wound that approximates the size of the muzzle. The wound edge will be regular and often has a clean-cut appearance with no individual pellet marks apparent. There will commonly be smoke soiling of at least some of the margin of the wound. Radial or stellate splitting of the skin may occur where the entry is over a bony surface. The wound will also show a muzzle mark/impression.
- A close discharge, within a few centimetres of the surface, will also produce a wound with a similar appearance, but as there is now room for muzzle gases to escape, there will be no muzzle mark. More smoke soiling

can occur, and burning of skin, with singeing and clubbing of melted hairs, can be seen around the wound. There is also, very commonly, powder 'tattooing' of the skin around the entry wound.

- At intermediate ranges, between 20 cm and 1 m, there will be diminishing smoke soiling and burning of the skin, but powder tattooing may persist. The spread of shot will begin, first causing an irregular rim to the wound.
- At a range of over 1 m, smoke damage and tattooing generally do not occur and injuries caused at longer ranges will depend upon the spread of the shot. Satellite pellet holes begin to be seen around the main central wound at a range of about 2–3 m.
- At long ranges, such as 20–50 m, there is a uniform peppering of shot, and this is rarely fatal.
  - Shotguns rarely produce an exit wound when fired into the chest or abdomen, although single pellet exit wounds can occasionally be seen. Exit wounds can be seen when a shotgun is fired into the head, neck or mouth. The exit wound in these cases may be a huge ragged aperture.

♦ Injuries caused by rifled weapons

- Entry wounds:
  - Contact wounds from a rifled weapon are generally circular, unless over a bony area such as the head, where splitting caused by the propellant gas is common. There may be a muzzle mark on the skin surface if the gun is pressed hard against the skin, and a pattern may be imprinted from a fore-sight or self-loading mechanism. There may be slight escape of smoke, with some local burning of skin and hair, if the gun is not pressed tightly. Bruising around the entry wound is not uncommon.
  - At close range, up to about 20 cm, there will be some smoke soiling and powder burns, and skin and hair may be burnt. The shape of the entry wound gives a guide to the angle that the gun made with that area of skin: a circular hole indicates that the discharge was at right angles to the skin, whereas an oval hole, perhaps with visible undercutting, indicates a more acute angle. Examination of the entry wound will show that the skin is inverted; the defect is commonly slightly smaller than the diameter of the missile because of the elasticity of the skin. Very commonly, there is an 'abrasion collar' or 'abrasion rim' around the hole.
  - Over 1 m or so, there can be no smoke soiling, burning or powder tattooing. At longer ranges (which may be up to several kilometres with a high powered rifle), the entrance hole will have the same features of a round or oval defect with an abrasion collar.
- Exit wounds

  The exit wound of a bullet is usually everted with split flaps, often resulting in a stellate appearance. No burning, smoke or powder soiling will be evident.The internal effects of bullets depend upon their kinetic energy. Other projectiles include air guns and rifles, plastic rounds (rubber bullets) and stud guns.

♦ Injuries due to explosives

- Primary blast effects can cause either physical fragmentation or disruption of the victim or bomber solely from the effects of the wave of high pressure and hot gases striking the body.

- Secondary effects include:
  - burns – directly from the near effects of the explosion and secondarily from fires started by the bomb;
  - missile injuries from parts of the bomb casing, contents or shrapnel or from adjacent objects; peppering by small fragments of debris and dust propelled by the explosion;
  - various types of injury owing to collapse of structures as a result of the explosion;
  - injuries and death from vehicular damage or destruction, such as decompression, intrusion of occupant space, fire and ground impact of bombed aircraft and crash damage to vehicles.

# 69.6 Transportation injuries

## 69.6.1 ROAD TRAFFIC INJURIES

**Motor vehicle injuries**

- Pedestrians
  - Pedestrians suffer primary (from contact with motor vehicle) and/or secondary (from contact with other objects such as the road) injuries.
  - Primary injuries typically form recognizable patterns such as contact with the bumper or other parts of the vehicle.
  - Secondary injuries may be complex and include run-over injuries, contact with other vehicles, contact with road surface, contact with buildings among others.
- Vehicle occupants
  - The position of the vehicle occupant, deployment of airbags and the use of seatbelts are important determines of the type and extent of injuries sustained. These factors should be examined for and documented for all vehicle occupant injuries.
- Drivers
  - Besides the examination as a vehicle occupant, other specific areas to assess and document include steering wheel injuries, accelerator/brake pedal marks, possibility of alcohol intoxication or influence of medicinal or recreational drugs.
- Motor cycles and pedal cycles
  - Most injuries to motorcyclists are caused by falling from the machine onto the roadway. 'Tail-gating' injuries occur when the motorcyclist drives under the rear of a truck, causing severe head injuries or even decapitation.

## 69.6.2 RAILWAY INJURIES

Railway injuries may cause severe mutilation of the body besides other injuries seen in other modes of transport.

### 69.6.3 AIRCRAFT FATALITIES

Aviation incidents can be divided into two main groups: those that involve the crew and the large numbers of passengers of a modern, commercial aircraft, and those that involve the occupants of small, relatively slow, light aircraft.
There should always be a full autopsy on the pilot or suspected pilot, with full microscopic and toxicological examination to exclude natural disease, drugs and alcohol.

- Large aircrafts
  - Large aircrafts are pressurized and loss of cabin integrity may cause injuries due to barotrauma.
  - Large defects may cause the passengers to fall through and fall to their death.
  - Loss of integrity of the fuselage may cause multiple injuries and deceleration injuries.
  - Lesser impacts may cause injuries similar to those of motor vehicle accidents.
  - Fire injuries may result in large number of fatalities.
  - Lighter aircrafts
  - The injuries will be similar to those of the larger aircrafts but to a lesser degree

### 69.6.4 MARINE FATALITIES

Fatalities in the marine setting embrace a range of marine-specific and general injury types. The range of activities include commercial diving, recreational diving, use of powered water sport bikes, sailing, motor cruising and commercial marine transport.
Fatalities result from falling from vessels, hypothermia, drowning, rotating propellers, explosion and fire.

## 69.7 Asphyxia

The literal translation of the word 'asphyxia' from Greek means 'absence or lack of pulsation'. However, asphyxia is commonly used to describe a range of conditions for which the lack of oxygen, whether it is partial (hypoxia) or complete (anoxia), is considered the cause:

- Suffocation is a general term used to indicate death from deprivation of oxygen, either from lack of the gas in the breathable environment or from obstruction of the external air passages.
- Smothering indicates blockage of the external air passages, usually by a hand or soft fabric. A variety of suffocation may be called gagging where fabric or adhesive tape occludes the mouth to prevent speaking or shouting. While the nasal passages remain patent, air can enter, but later blockage by mucus or oedema may lead to death.
- Choking refers to blockage of the upper airways by some foreign body.

- Throttling refers to strangulation, usually by hand.

- Strangulation is the most specific term, indicating the use either of an object, hands or a ligature as a means of applying external pressure to the neck.
- Traumatic asphyxia refers to restriction of chest movement as a result of injury
- Chemical asphyxia refers failure of oxygen transportation (for example carbon monoxide poisoning), failure of oxygen utilization (for example cyanide poisoning)
- Miscellaneous for example drowning where there is physical interference with effective respiration

### 69.7.1 AUTOPSY FINDINGS IN ASPHYXIA DEATHS

These are generally unspecific but include:

- Cyanosis
- Petechial haemorrhages in the skin, the sclera or conjunctivae and under thoracic serous membranes such as the pleura or pericardium.
- Congestion and oedema

Findings at scene of death are an important pointer towards the diagnosis. For example, in an asphyxia death due to use of a plastic bag, the diagnosis will be difficult to arrive at if the bag is removed.

Diagnosis in some of the asphyxia deaths is quite evident for example in the case of 'café coronary' where the food material causing airway occlusion is identified or in a case of hanging where there is a ligature around the neck with features of suspension.

### 69.7.2 IMPORTANT CONSIDERATIONS IN ASPHYXIAL DEATHS

- Demonstration of ante-mortem bruising for example in hanging
- Features suggestive of struggle
- Assessment for alcohol (or other forms of) intoxication as may be seen in postural asphyxia.

## 69.8 Deaths in custody and fatal physical abuse

Fatal physical abuse may occur in vulnerable persons in locations of reduced freedom. These locations could be children's homes, homes for the aged, institutions for the mentally ill, rehabilitation facilities, detention camps, police cells and prisons.

Autopsy appearances of fatal abuse are no different from those by any other homicide, and the confirmation of lethal torture must depend upon circumstantial and other corroborative evidence.

The injuries may be inflicted through beating, burns (of various kinds), cutting or stabbing (by a variety of weapons), clubbing and blunt injury, suffocation, drowning, electrical exposure (from mains electricity or magneto delivering high voltage), 'telefono' (repeated slapping on the sides of the head leading to inner ear injury and tympanic membrane rupture),suspension, shooting, sexual assault, etc

The occurrence of death while a person is either in police custody, or a prison inmate raises public interest and emotions that require careful handling and investigation. A thorough autopsy is required and should be carried out by a forensic pathologist.

Situations that may result to deaths in custody include:

- Traumatic asphyxia may occur where several policemen fall upon a resisting subject to overpower him/her.
- Arm-locks or neck-holds applied by police officers to resisting persons are other causes of deaths during arrest.
- Postural (positional) asphyxia has been reported to have caused sudden deaths in persons after the use of prone maximal restraint or in situations where a person has been placed in a prone position in the rear compartment of a police car.
- Blunt injury may occur from the use of fist, arm or leg or the use of a weapon such as a riot stick or gun butt.
- Alcohol and drugs may cause death in custody.
- Suicide in custody is not uncommon.
- Death in custody may also result from purely natural causes such as cardiovascular diseases.

## 69.9 Unexpected and sudden deaths from natural causes

**General considerations in unexpected and sudden deaths**

- Forensic pathologists deal not only with criminal, suspicious, accidental and suicidal deaths, but also with a wide range of deaths from natural causes. Many of these are sudden, unexpected, clinically unexplained or otherwise obscure, even though there may be no unnatural element in their causation.
- According to the WHO, a sudden death may be defined as one that occurs within 24 hours of onset of symptoms.
- Deaths that occur within 24hours of attending a medical facility and whose pathophysiological process cannot be explained would too require autopsy.

**Causes of unexpected and sudden deaths**

- Cardiovascular disease:
  - coronary atherosclerosis
  - myocardial infarction
  - hypertensive heart disease
  - aortic valve disease
  - aortic aneurysm
  - anomalies of the coronary circulation
  - other coronary artery diseases, such as polyarteritis
  - cardiomyopathies
  - congenital heart disease.

- Respiratory disease
  - Pulmonary embolism including thombo-embolism, amniotic fluid embolism, air embolism, etc
  - Bronchial asthma
  - Respiratory obstruction
  - Hemoptysis

- Gastro-intestinal disease
  - Gastro-intestinal bleeding
  - Mesenteric thrombosis
  - Intestinal strangulation
  - Perforated peptic ulcer with chemical peritonitis

- Genito-urinary disease
  - Complication of pregnancy such as ruptured ectopic pregnancy, incomplete abortion

- Central nervous system disease
  - Cerebral vascular accident
  - Haemorrhage

## 69.10 Emersion and drowning

Many corpses are recovered from water, but not all are due to drowning. Of those that have drowned, pathological proof is often difficult or even impossible to obtain. The autopsy diagnosis of drowning presents one of the major problems in forensic medicine, especially when there is delay in recovering the victim.

Bodies retrieved from water may have:
- died from natural disease before falling into the water
- died from natural disease while already in the water
- died from injury before being thrown into the water

- ♦ died from injury while in the water
- ♦ died from effects of immersion other than drowning
- ♦ died from drowning

Deaths from immersion and drowning may not show any specific features at autopsy and exclusion of natural death and traumatic conditions is important.

## 69.11 Deaths associated with pregnancy

**General considerations**
Maternal deaths are an important indicator of the quality of healthcare in any jurisdiction. Evaluation of maternal deaths requires a good autopsy with full histological examination and other ancillary investigations where necessary.

**Causes**
- ♦ Deaths associated with abortion
  - ▪ Legal abortion may result to death by:
    - ○pulmonary embolism from leg vein thrombosis
    - ○mishaps associated with anaesthesia
    - ○disseminated intravascular coagulation
    - ○cerebral damage
    - ○air embolism following vacuum aspiration
    - ○Haemorrhage
    - ○Sepsis
    - ○Illegal abortion
    - ○Injury from instruments
    - ○Insufflation of air
    - ○Cervical dilatation
    - ○Extreme physical activity
    - ○Intra uterine infection and sepsis
    - ○Toxicity from drugs and chemicals

- ♦ Amniotic fluid embolism
- ♦ Thrombosis and thromboembolism
- ♦ Hypertensive disease of pregnancy
- ♦ Amniotic fluid embolism
- ♦ Early pregnancy deaths
- ♦ Sepsis
- ♦ Haemorrhage
- ♦ Genital tract trauma
- ♦ Anaesthesia

## 69.12 Procedure related deaths

**General considerations**

- Deaths occurring in patients while undergoing medical or surgical procedures are a major concern in healthcare and require adequate investigation as a quality assurance procedure.
- These deaths, which can occur during or within a short time after surgical operation, invasive diagnostic procedure or an anaesthetic, become the subject of a medicolegal investigation.
- Similarly, any death thought to be caused or contributed to, by any of these procedures – irrespective of the interval – may be enquired into if the medical attendants or the relatives consider that a causal relationship exists.
- To be effective such investigations must include an autopsy.
- Ideally the forensic pathologist undertaking the autopsy should be independent of the institution in which either the death occurred or which carried out the procedure.
- Particular challenges the pathologist needs to pay attention to when undertaking these autopsies include the morphological findings, especially in 'anaesthetic deaths', may be minimal or even absent so, expert advice and full clinical information are essential.
- Technically the autopsy on a post-operative death may be difficult as a result of the surgical intervention and its sequelae.
- Numerous surgical and anaesthetic devices may have been introduced into the patient during the procedure, such as airways, endotracheal tubes, indwelling needles, intravascular cannulae, self-retaining catheters, wound drains, chest tubes, monitoring electrodes, and metal or plastic prostheses. It is essential that none of these be removed before autopsy, as their proper placement and patency may need to be checked.
- The hospital laboratory should be requested to retain any ante-mortem blood or body-fluid samples sent to them so that they remain available for analytical checks, such as blood grouping in transfusion mishaps, or creatine phosphokinase activity in malignant hyperthermia.
- Patient's notes including nursing notes, treatment notes, imaging reports and laboratory results should be made available for review.
- Presence of the attending clinicians at the autopsy is recommended as it may provide virtually all the data upon which to base a cause of death, especially in anaesthetic deaths where autopsy dissection may show no or minimal pathologic features.
- Where infusion or transfusion mishaps may be a possibility, especially where gas pressure has been used to hasten the infusion rate, the possibility of air embolism must always be borne in mind.
- Fatalities associated with surgical intervention or invasive diagnostic procedures can be separated into several categories:
- those directly caused by the disease or injury for which the operation or anaesthetic was being carried out. Prior assessment of the patient may prevent these deaths.

- those caused by a disease or abnormality other than that for which the procedure was being carried out. A distinction has to be drawn between those conditions that were known before the operation and those which were unsuspected.
- those resulting from a mishap during, or a complication of, the surgical or diagnostic procedure. This may be inadvertent, from a true 'accident', sometimes caused by unusually difficult operative circumstances, to anatomical abnormalities or even failure of equipment.
- those resulting from a mishap during, or a complication of, the anaesthetic being administered.
    - Hypoxia is also a potent precipitating factor in cardiac arrest.
    - Deaths from respiratory failure also occur, and hypoxia
    - is again an example – either from faults in the apparatus
    - or more commonly to inexperience of the anaesthetist, especially
    - in handling equipment with which he is not familiar.
    - An overdose of the anaesthetic agent may depress the respiratory
    - centres and begin a descending spiral of hypoxia.
    - Excessive premedication and the use of muscle paralyzing agents may also predispose towards respiratory failure unless appropriate measures are taken, such as assisted respiration.
    - Airway obstruction is a recognized danger – from blood, teeth, dentures, faults in the connecting tubing, laryngeal spasm, swabs and an abnormal posture of the neck.
    - Fires are not uncommon, such as ignition of spirit-based skin antiseptics ignited by cautery.
    - Any electrical apparatus is potentially dangerous, and defective cauteries, defibrillators and diathermy equipment may all cause death.
    - Explosions from inflammable gases and vapours, such as cyclopropane and ether, can be catastrophic.
    - Hypersensitivity to anaesthetic agents may too cause death.
    - Malignant hyperthermia is a familial condition in which certain agents, including some anaesthetics and muscle relaxants, precipitate a metabolic change in skeletal muscle with the production of energy, and hence a rapid and sometimes fatal rise in temperature.

## 69.13 Verbal autopsies

Verbal autopsies are performed for all deaths occurring in the community and level 1 health facilities. Death certification by a medical practitioner providing direct care. Where external causes are suspected, or where MITS or CA are indicated.

**MITS Procedures**

**Table 66.1:** Materials: **MITS Sample collection Kit Components**

| | |
|---|---|
| Blood spot card | 20G 1.5" and 18G 3.5" spinal puncture needle for CSF collection |
| Photo card | 20G 1.5" and 16G 1" intramuscular needle for blood collection |
| Labels for samples | 20 ml syringes for CSF and blood collection |
| Bard Monopty needle 16G,100 mm | EDTA vacutainer |
| Bard Monopty needle 16G, 160mm | 10 ml sterile tube for CSF and rectal swab |
| Trephine biopsy needle | 2 ml cryogenic vials for tissue microbiology |
| Nasopharyngeal swab with viral transport media | Large 120 ml screw cap jar for storage and transport of tissue cassettes |
| Rectal brush | Tissue cassettes |
| Personal Protective Equipment | 20 ml 10% NBF (neutral buffered formalin) pre-filled jars |

**Methods**

- Appropriate personal protective equipment will be worn by the MITS personnel before commencement of the sample collection.
- The body will be cleaned at the sites of sample collection and left to dry for five minutes between the cleaning steps using clean water, 70% alcohol and finally iodine solution respectively. The sample collection sites include the nares, posterior cervical/occipital region, supra-clavicular region, axillae, right upper quadrant region and the anus.
- Cerebrospinal fluid is collected from the cistern magnum through the occipital region using the 20G spinal needle and delivered to the sterile tube using the 20 ml syringe.
- Nasopharyngeal swab sample is obtained trans-nasally and placed into the nasopharyngeal tube with viral transport media.
- Brain tissue is collected from the occipital region (3 specimens) and transnasally (3 specimens) using the 16G 160 mm Bard Monopty needle. NB: The fontanelles will be used to obtain brain samples in infants. Trans-nasal

brain access is achieved after penetration of the cribriform plate using a trephine needle.

- The first 3 specimens (3 occipital and 3 trans-nasal) are delivered to the cryogenic vial for microbiology tests. A further 6 brain tissue specimens are obtained and delivered to the 20 ml formalin jar for histological analyses.
- Blood sample collection is carried out using 16G and 18G intramuscular needles for infants and adults respectively. The specimen is obtained from supraclavicular or heart puncture. The blood is then delivered to an EDTA vacutainer and blood spot card.
- Lung tissue is collected from the axillary regions using a Bard Monopty 16G 100 mm needle. The first three of the specimens bilaterally are delivered to the cryogenic vials for microbiological tests while the next 6 bilaterally are delivered to 20 ml jars for histological analyses. NB: The specimens are obtained from the different lung lobes through adjusting the angulation of the direction of the needle.
- Liver tissue is obtained from the mid-axillary line at any of the last 3 intercostal spaces using a 16G 100mm Bard Monopty™ needle. The first three specimens obtained are delivered to the cryogenic vials for microbiological tests while the next three are put into a 20 ml formalin jar for histological analyses.
- Rectal stool sample is obtained using a rectal brush and put in a 10ml sterile tube for microbiological analyses.
- After the MITS specimen collection is completed, the sample containers are properly labelled and placed in a MITS cool box for the microbiology samples and MITS kit box for the pathology samples for onward transmission to the laboratory.
- The body is cleaned with 0.5% hypochloride solution and dried. This is then placed into body bags and taken into storage.
- Diagnostic workup depends upon the laboratory evaluation of the specimens submitted. This includes the placenta (in still birth and newborns).

## 69.14 Complete diagnostic autopsy

The procedure for complete diagnostic autopsy is performed, by a qualified pathologist. This applies in all cases of external trauma and selected non forensic deaths. It involves external examination, and internal examination with submission of specimens to clinical/pathology laboratories for evaluation. This is described in detail elsewhere in these guidelines.

**Placental Pathology**

Placental pathology is an essential component of perinatal and neonantal death investigation. Placentas should be submitted in all stillbirths, for evaluation by a qualified pathologist. Where available, placentas should be submitted for evaluation, in all high risk newborns admitted for care. These may be submitted fresh (refrigerated - 2-8 degrees) or fixed (in 10% neutral buffered formal saline). Histopathological evaluation and diagnosis of placental diseases, as defined by

the Amsterdam Classification of Placental Disorders consensus meeting of 2015, is the minimum basic requirement.

**Child abuse, including child sexual assault mortality**
Child abuse, including sexual assault, are largely unrecognized because of a low index of suspicion. All health care providers, in all levels of care, should be competent in identifying cases of abuse, and providing linkage to care, including medico-legal and forensic services. Recognition of injuries among the living and the dead rely upon proper examination of all deceased children. At various levels of care, the following guidelines are necessary.

All child deaths occurring in the community require examination by a medical practitioner or an experienced, trained clinical officer. Prior to certification of death, a verbal autopsy is administered, and a detailed external examination is performed. The autopsy is documented in standard forms. Where injuries are identified, the child is referred for forensic autopsy by a qualified pathologist, as defined by the National Coroner's Service Act no 17 of 2018. Complete Diagnostic Autopsy are indicated in these cases, with collection of appropriate evidence to facilitate medicolegal processes.

**Sexual and gender based violence, of adults, geriatric abuse, mortality**
Sexual and gender-based violence of adults resulting in death are an absolute indication for forensic autopsy. Geriatric abuse is an emerging social issue in Kenya. All health care providers, in all levels of care, should be competent in identifying cases of abuse, and providing linkage to care, including medicolegal and forensic services. Recognition of injuries among the living and the dead rely upon proper examination of all deceased children. At various levels of care, the following guidelines are necessary.

## 69.15 Forensic Odontology

Forensic Odontology deals with the professional handling, examination of dental evidence, expert interpretation and documentation of findings made in the interests of justice. This is particularly done in cases of medico legal proceedings where the dentist is an expert witness.

The aim is to examine and evaluate injuries to the dentition, assess bite marks, maintain and interpret dental records in dental jurisprudence cases such as malpractice or dental fraud. Further, it to identify unknown persons, dead or alive.

**Identification**
The primary means of identification, using the comparative dental identification method (only possible with excellent dental record systems in place).

**Management**

The expert should compare the antemortem and postmortem dental characteristics using written notes, study casts and radiographs, photographs, that would reveal traditions,fillings,etc.

### 69.15.1 DETERMINATION OF AGE

These are based on tooth development and eruption, with better accuracy if root length and degree of mineralization are used.

In patients under 20 years of age:

- Radiological methods :Radiographs are interpreted using standard charts relating age to degree of development e.g. Schour & Massler and Gustafson & Koch.H to give estimates
- Histological methods to assess formation and mineralization of enamel and dentine,neonatal lines using the dentition are useful.Conclusions of dental age analyses are usually accurate to approximately ±1.5 years (with ±4 year's variation for 3rd molars).
- in middle-aged and older adults assess age using periodontal disease progression, attrition and tooth loss. Note the accuracy using these highly-variable markers is in the range of ±10-12 years.

### 69.15.2 DETERMINATION OF GENDER

Anatomical techniques for patients that are past puberty since a lot of similarities occur pre-puberty.

Laboratory based techniques: include DNA testing, identification of the Barr body, and the F body.

### 69.15.3 BITE MARK ANALYSIS

This is in regard to trauma caused by dentition whether on inanimate objects as evidence or bite marks on humans. Often they point to close combat situations, child abuse and sexual assault in relation to interpersonal violence.

**Management**

- Rule out self-inflicted bites, they should be consistent with a contributory history such as falls on the face.
- Determine the size and shape and nature of the dentition to determine whether it is of a child or adult.
- Use the Pretty's Index for Bitemark Severity & Significance.

## 69.16 Exhumation

The term 'exhumation' is usually applied to define the removal of a body buried in a legitimate fashion in a cemetery or graveyard ('inhumation'), rather than the recovery of an uncoffined, clandestinely buried victim of a suspicious death. The

latter is really a ‘scene of crime’ and the pathologist should treat it as such. Exhumations are required for one of the following reasons:

- Where all or part of a graveyard has to be moved for some development of the ground. Often no special examination of each body is made unless there is some historical or anthropological interest.
- Where some civil legal matter needs to be investigated, such as personal injuries for insurance or civil litigation for negligence – usually after a road, industrial or other accident.
- Where new information or substantiated allegations arise to suggest that a death was due to criminal action, either from injury or poison.
- In ancient or historical circumstances to investigate either the individual or a series of individuals for academic interest. A number of such investigations have been carried out on medieval and later inhumations in England, to study disease patterns and nutritional states in old populations – though many of these have been from dry vaults, rather than earth burials.

## 69.17 Mass disasters

The objects of pathological investigation in mass disasters are:

- to retrieve and reconstruct bodies and fragmented bodies decently
- to establish personal identity to conduct autopsies on some or all of those bodies
- to establish the cause of death in some or all, especially air crew and drivers, and to assist in reconstructing the cause of the disaster
- to obtain material for toxicological analysis (especially alcohol and carbon monoxide) where appropriate
- to seek evidence of the cause of the disaster from autopsy examination, such as bomb or detonator fragments that may be embedded in the bodies.

The nursing officer refers the body to the nearest level 3 facility for confirmation and documentation of death.

## 69.18 Death in a level 3 facility

This section applies to persons who die at the facility, persons brought in dead, persons who die on arrival to the facility or persons referred from level 2 facilities for confirmation of death.

The clinical/medical officer takes a clinical history and examines the body to confirm the person has died then prepares clinical notes to that effect and fills out the verbal autopsy form. The clinical/medical officer must determine if the death is of forensic importance from history and examination.

Confirmed deaths of no forensic interest are referred to the chief for issuance of a burial permit, D2, and transfer to the mortuary if required. The chief prepares and submits the register for death.

Confirmed deaths of forensic importance are referred to the police for forensic death investigation.



## 69.19 Death confirmation

Procedure in death confirmation:

- Wash your hands and don personal protective equipment if appropriate.
- Confirm the identity of the patient by checking their wrist band.
- Inspect for obvious signs of life such as movement and respiratory effort.
- Assess the patient's response to verbal stimuli (e.g. "Hello, Mr Smith, can you hear me?")
- Assess the patient's response to pain using one of the following methods:
  - Apply pressure to the patient's fingernail.
  - Perform a trapezius squeeze.
  - Apply supraorbital pressure.
- Assess the patient's pupillary reflexes using a pen torch: after death, the pupils become fixed and dilated.
- Palpate the carotid artery for a pulse: after death, this will be absent.
- Perform auscultation in an attempt to identify any heart or respiratory sounds:
  - Listen for heart sounds for at least 2 minutes.
  - Listen for respiratory sounds for at least 3 minutes.
  - The recommended amount of time to listen for heart and respiratory sounds can vary, but it is generally accepted that a minimum of five minutes of auscultation is required to establish that irreversible cardiorespiratory arrest has occurred.
- Wash your hands, dispose of personal protective equipment appropriately and exit the room, making sure the relevant doors and/or curtains are closed/drawn behind you.

## 69.20 Pediatric pathology

This is concerned with intrauterine fetal deaths of the viable fetus (stillbirths), neonatal deaths and Cchild death. These deaths occur within health facilities or the community. World Health Organizations' International Classification of Disease, version 11 (WHO ICD 11) – Perinatal Mortality, is applied to assign cause and manner of death. All stillbirths must have an independent death investigation and a verbal autopsy form completed. The birth notification alone is insufficient because it does not assign a cause of death and therefore does not input into prevention of such deaths occurring in the future.

Cause of death is assigned following a verbal autopsy, where the best diagnostic category is documented as immediate cause. As a general rule, for perinatal deaths (still births and neonates) the underlying cause of death is always the maternal diagnosis. Among older children (post-neonatal age

group, under 5's), the conventional approach to death notification, using WHO ICD 11, is sufficient.

The WHO ICD 11 standards require that all deaths are certified by a registered medical practitioner. Cause of death documented in the prescribed form, must

include proper diagnostic coding and documentation of the duration of onset of disease, prior to death. The universal cause of death certificate also provides for opinion on manner of death.

Verbal autopsies should be performed for investigation of all pediatric deaths, and the cause of death as determined by assigned verbal autopsy diagnosis. Pathology informed cause of death should be performed in at least 30% of all deaths, using minimally invasive tissue sampling (MITS), or where available, complete diagnostic autopsies (CDA). Where external causes of death are suspected or encountered during the verbal autopsy process, then complete diagnostic autopsies are an absolute indication. The placenta should be available for evaluation, where MITS or CDA are performed, for all stillbirths, and newborns.

## 69.21 Human remains management and mortuary practice

### 69.21.1 HUMAN REMAINS MANAGEMENT

**General Considerations**

- Wash your hands and don personal protective equipment if appropriate.
- Bodies and other human remains should be handled with resepct and dignity, taking into account socio-cultural and religious considerations of the deceased person as well as their wishes and those of their family, subject to existing laws and regulation. Infection prebvention and control practices must be adhered to at all times while handling human remains.
- Occupational health and safety, and personnel training and capacity development must be in tandem with the general expectations in care and service delivery.
- Physical infrastructure and waste management are areas of great importance in human remains management.
- All efforts must be made to identify the deceased persons before disposal of the body.
- Where efforts to identify a body before disposal have been unsuccessful and the decision to dispose of the body is made, all available information that may be useful in later identification of the body should be recorded and preserved by the mortuary and/or authorized persons. This information should be passed on to the relevant department dealing with inventory of unidentified bodies.
- Cremation and other forms of disposal that cause permanent destruction of the body shall not be done for bodies whose identity has not been established.
- Efforts should be made to establish the cause and manner of death before disposal of the body.

- A burial permit should be issued for all bodies before disposal. This may not apply to stillborn fetuses. Whereas a birth notification may be used in place of a burial permit for neonates, this is undesirable as it denies the health care workers and other agencies an opportunity to put in place measures to prevent such deaths in the future.

## 69.21.2 TRANSPORTATION OF BODIES

- The body should remain enclosed within the transport vehicle/gurney/trolley.
- A body transport vehicle/gurney/trolley must be designated solely for this purpose and be easy to decontaminate.
- Body bags may be used for body transport as required.
- Ambulances are not recommended for this purpose.

## 69.21.3 ADMITTING A BODY TO THE MORTUARY

- A mortuary may only admit a body if it has sufficient storage capacity that ensures proper preservation without interfering with any investigation that may be required on the said body.
- A mortuary shall not admit a body suspected of dying an unnatural death unless the death has been reported to the police and written proof availed.
- A mortuary admitting a body must ensure that the body has proper documentation including:
  - A burial permit issued and stamped by an authorized person (a chief -form D2, a registered medical doctor- form D1) or a health facility.
  - A letter signed and stamped by a police officer indicating the OB number and the police station requesting admission of the body.
- Identifying particulars, including name, age and gender of the deceased person should be documented, as well as name and contacts of next of kin, place, date, time and cause of death if known.?
- In the case of unidentified bodies, the mortuary shall only admit the bodies on the written authority of a police officer who shall be present in person.
- Where death has not be confirmed the body shall not be admitted into the mortuary.
- Each body or body part shall be assigned a unique admission number, provisionally identified where possible and correctly labeled with a non-degradable and engraved tag.
- In the case of unidentified bodies, photographs and fingerprints shall be taken at the earliest opportunity and forwarded to the relevant authorities for identification.
- Sampling for DNA and other forms of testing may be considered on a case to case basis as advised by the pathologist-in-charge.
- The mortuary shall be notified in advance of a body that highly infectious (such as ebola) before the said body is transported to the mortuary.
- Whenever possible, a clinical summary should accompany the bodies of all persons dying in health facility.
- All personal effects on the body shall be documented and released to an authorized person (family, guardian or investigating police officer) as appropriate.

## 69.21.4 BODY STORAGE AND PRESERVATION

- Bodies shall be stored in refrigerated compartments at 2-6$^0$C.
- Chemical preservation (embalming or formalin fixation) should only be performed after postmortem examination/autopsy.

- Temporary body storage may be used at the scene of death especially in mass disasters as arrangements are made to transport the bodies to a mortuary. This temporary storage may take the form of a field mortuary or temporary burial (see body disposal and interement).

## 69.21.5 POST MORTEM EXAMINATION AND AUTOPSY

- External examination (view and grant) may be carried out (at the time and location) of body retrieval depending on the circumstances of death, by a pathologist and/or medical officer. Bodies of forensic interest or where the cause of death cannot be determined by external examination only should be subjected to dissection and internal organ examination, if considered safe. All important external findings shall be document and filed for future reference.
- Verbal autopsies may be carried out to determine cause of death where the services of post-mortem examination or autopsy are not available. Bodies of forensic interest or where the cause of death cannot be determined by verbal autopsy should be subjected to post-mortem and/or dissection and internal organ examination. All important findings shall be document in the verbal autopsy form and filed for future reference.
- When indicated, autopsies should be performed within 24 hours of body reception. Any delays that affect the quality of results should be documented.

## 69.21.6 BODY DISPOSAL

- Common methods for body disposal include burial (interment) and cremation. Other less common methods include aquamation (dissolution), immurement (entombment) and composting.
- Body disposal can only be done after a burial or disposal permit has been issued.
- Burials are done at least 6 feet deep in rural areas and at least four feet deep in public cemeteries.
- Disposal of unclaimed and unidentified bodies:
    - An order for disposal of unclaimed bodies should be sought from a court of law.
    - After an order for disposal has been given, the hospital or mortuary administration shall issue a 30 days' notice to the police for the bodies to be claimed failure to which the mortuary shall be free to dispose them in accordance with cap 241
    - Before disposal autopsy must be performed and documented on all bodies.
    - Unidentified bodies should be buried in marked coded graves for future reference**.** The codes used to mark the grave shall correspond to the records and codes of the deceased held at the mortuary

# 69.22 Mortuary practice

## 69.22.1 GENERAL CONSIDERATIONS IN MORTUARY PRACTICE

- A mortuary is a room or building in which bodies are kept, for safe and dignified storage or for examination awaiting disposal
- Mortuaries, just like other health facilities, are classified in levels based on services offered.
- The mortuary physical infrastructure should be commensurate with services offered.
- The human resource should be well trained and experienced in offering services at the various levels.
- Safety measures including infection prevention and control, and occupational health and safety should be taken into account as well as mental well-being of staff and grieving families.

## 69.22.2 LEVELS OF MORTUARY SERVICES

### LEVEL I

**Description**
This is a temporary body holding facility that holds bodies for a maximum of 24hrs awaiting transfer to a higher level facility.

**Floor Plan**
This is a single room whose size is determined by the anticipated workload

**Services**
To store bodies for a maximum of 24hrs waiting transfer to higher level mortuary. This mortuary should not release bodies for disposa

**Staffing**
The staff in this facility will include (but not be limited to)
- Nurses
- Police officers
- Security guards
- Disaster response team

**Safety**

The safety procedures to be included in this facility shall include
- Personal protective equipment use
- Decontamination
- Clinical waste management including disposal of used gloves, dressings among others (refer to clinical waste management guidelines)

- Other safety procedures will be determined by facility in which this mortuary is housed.
- Biosafety level

**Equipment**

- Gurneys
- Easy to clean tables, trays and trolleys
- Waste disposal bins

## LEVEL II

**Description**

This is a mortuary found in a sub-county (level 4) hospital or a stand-alone mortuary. This also includes most privately run funeral homes. These mortuaries must be supervised by a forensic pathologist.

**Floor Plan**

The mortuary will have the following areas

- Security room at the gate
- Car park
- Clean area for the reception and offices
- Body receiving area
- Cold storage
- Embalming area
- Autopsy suite
- Body dispatch area
- Chapel
- Viewing bay
- Public toilets
- Staff changing rooms
- Staff washrooms
- Staff tea rooms
- Eye wash station and shower
- Skin decontamination areas
- Decomposed bodies handling area

**Services**

The following services shall be offered in level II mortuaries

- Body refrigeration
- Body embalming
- Body viewing
- Basic autopsies limited to road traffic accidents. And any other as may be authorized by the pathologist-in-charge
- Supervision of level in mortuaries
- Data analysis and reporting to level III mortuaries
- Referral services to level III mortuaries

**Staffing**

- Security guards
- Mortuary attendant (minimum certificate holder in mortuary sciences)
- Driver
- Data clerk (minimum certificate in health records)
- Photographer
- Medical officer with forensic training
- Safety officer, may be a mortuary attendant with bio-safety and general safety
- Biomedical engineer (diploma)

**Safety Procedures**

The safety procedures to be included in this facility shall include

- Personal protective equipment
- Decontamination
- Clinical waste management including disposal of used gloves, dressings among others (refer to clinical waste management guidelines)
- Eye wash stations
- Shower cubicles
- Skin decontamination
- Body washing services
- Embalming
- Air filtration system
- Assembly point (s)
- Fire fighting
- Biosafety level II/III

**Equipment**

- Gurneys
- Easy to clean tables, trays and trolleys
- Waste disposal bins
- Autopsy kit
- Oscillator saw
- Embalming machine
- Coolers/refrigerators
- Air conditioning system, in the clean area as required
- Cameras

## LEVEL III

**Description**
This is a mortuary found in a county referral (level V) hospital or an equivalent facility

**Floor plan**
The mortuary will have the following areas
- Security room at the gate
- Clean area for the reception and offices
- Body receiving area
- Cold storage
- Embalming area
- Autopsy suite
- Body dispatch area
- Chapel
- Viewing bay
- Public toilets
- Staff changing rooms
- Staff washrooms
- Staff tea rooms
- Eye wash station
- Skin decontamination
- Decomposed bodies handling area
- Lecture /classrooms
- Cadaveric organ harvesting room

**Services**
The following services shall be offered in level III mortuaries
- Body refrigeration
- Body embalming
- Basic autopsies limited to road traffic accidents. And any other as may be authorized by the chief government pathologist
- Supervision of level II mortuaries
- Data analysis and reporting to level IV mortuaries
- Referral services to level IV mortuaries
- Other forensic autopsies except in death due to gunshots and explosives
- Cadaveric cornea harvesting
- Teaching at certificate level
- Clinical autopsies
- Biosafety level II/III

**Staffing**
- Security guards and drivers
- Mortuary attendant (minimum certificate holder in mortuary sciences)
- Data clerk (minimum certificate in health records)
- Photographer
- Medical officer with forensic training
- Safety officer, may be a mortuary attendant with bio-safety and general safety
- Mortuary technologists with minimum diploma training

**Safety**

The safety procedures to be included in this facility shall include

- Personal protective equipment use
- Decontamination
- Clinical waste management including disposal of used gloves, dressings among others (refer to clinical waste management guidelines)
- Eye wash stations
- Shower cubicles
- Skin decontamination
- Body washing services
- Embalming
- Air filtration system
- Assembly point (s)
- Fire fighting

**Equipment**

- Gurneys
- Easy to clean tables, trays and trolleys
- Waste disposal bins
- Autopsy kit
- Oscillator saw
- Embalming machine
- Coolers/refrigerators
- Air conditioning system, in the clean area as required
- Cameras

## LEVEL IV

**Description**

- Staff tea rooms
- Eye wash station
- Skin decontamination
- Decomposed bodies handling area
- Lecture /classrooms
- Cadaveric organ harvesting room
- Radiology room and radiology reporting area
- Entomology laboratory
- Histology lab
- Isolation room for bodies with confirmed or suspected highly infectious diseases

**Services**

The following services shall be offered in level III mortuaries

- Body refrigeration
- Body embalming
- Basic autopsies limited to road traffic accidents. And any other as may be authorized by the chief government pathologist
- Supervision of level I mortuaries
- Data analysis and reporting to level IV mortuaries

- Referral services to level IV mortuaries
- Other forensic autopsies except in death due to gunshots and explosives
- Cadaveric cornea harvesting
- Teaching at certificate level
- Clinical autopsies
- Forensic radiology
- Forensic entomology
- Teaching for certificate and diploma level
- Referral services to level V

**Staffing**

- Security guards
- Mortuary attendant (minimum certificate holder in mortuary sciences)
- Driver

Data clerk (minimum certificate in health records This is a regional mortuary that provides referral services to several mortuaries in a specified region or an equivalent. This serves as a regional forensic center.

**Floor Plan**

The mortuary will have the following areas

- Security room at the gate
- Clean area for the reception and offices
- Body receiving area
- Cold rooms
- Embalming area
- Autopsy suite
- Body dispatch area
- Chapel
- Viewing bay
- Public toilets
- Staff changing rooms
- Staff washrooms
- Photographer
- Medical officer with forensic training
- Safety officer, may be a mortuary attendant with bio-safety and general safety
- Mortuary technologists with minimum diploma training
- Mortuary scientist, graduate
- Forensic pathologist
- Clinical pathologist
- Police officer(s)
- Quality manager, graduate
- Safety manager, graduate
- Forensic photographer, graduate

**Safety**

The safety procedures to be included in this facility shall include
- Personal protective equipment use
- Decontamination
- Clinical waste management including disposal of used gloves, dressings among others (refer to clinical waste management guidelines)
- Eye wash stations
- Shower cubicles
- Skin decontamination
- Body washing services
- Embalming
- Air filtration system
- Assembly point (s)
- Fire fighting
- Isolation for highly infectious diseases
- Bio-safety cabinets
- Biosafety level III

**Equipment**

- Gurneys
- Easy to clean tables, trays and trolleys
- Waste disposal bins
- Autopsy kit
- Oscillator saw
- Embalming machine
- Coolers/refrigerators
- Air conditioning system, in the clean area as required
- Radiology
- Cameras
- Bio safety cabinets

## LEVEL V

**Description**
This is the National Forensic Referral, Teaching and Research Centre.

**Floor Plan**
The mortuary will have the following areas
- Security room at the gate
- Clean area for the reception and offices
- Body receiving area
- Cold rooms
- Embalming area
- Autopsy suite
- Body dispatch area
- Chapel
- Viewing bay
- Public toilets
- Staff changing rooms

- Staff washrooms
- Staff tea rooms
- Eye wash station
- Skin decontamination
- Decomposed bodies handling area
- Lecture /classrooms
- Cadaveric organ harvesting room
- Radiology room and radiology reporting area
- Entomology laboratory
- Isolation room for bodies with confirmed or suspected highly infectious diseases
- Cadaveric organ harvesting theatre
- Research laboratories
- Lecture rooms
- Teleconference centre
- Teaching laboratories
- Library
- Board room

**Services**
- Body refrigeration
- Body embalming
- Supervision of level IV mortuaries
- Data analysis and reporting to level IV mortuaries
- Referral services to level IV mortuaries
- Forensic autopsies
- Cadaveric cornea harvesting
- Teaching at certificate level
- Clinical autopsies
- Forensic radiology
- Forensic entomology
- Referral services from level IV
- Research in forensic sciences
- Cadaveric organ harvesting
- Library services
- Training
- Telemedicine and teleconferencing

**Staffing**
- Security guards
- Mortuary attendant (minimum certificate holder in mortuary sciences)
- Driver
- Data clerk (minimum certificate in health records)
- Photographer
- Medical officer with forensic training
- Safety officer, may be a mortuary attendant with bio-safety and general safety
- Mortuary technologists with minimum diploma training
- Mortuary scientist, graduate
- Forensic pathologist
- Forensic scientist

- Clinical pathologists
- Police officer(s)
- Quality manager, graduate
- Safety manager, graduate
- Forensic photographer, graduate
- Researchers
- It experts
- Statisticians
- Librarians
- Surgeons and other specialists in cadaveric organ harvesting



**Safety**

The safety procedures to be included in this facility shall include

- Personal protective equipment use
- Decontamination
- Clinical waste management including disposal of used gloves, dressings among others (refer to clinical waste management guidelines)
- Eye wash stations
- Shower cubicles
- Skin decontamination
- Body washing services
- Embalming
- Air filtration system
- Assembly point (s)
- Fire fighting
- Isolation for highly infectious diseases
- Bio-safety cabinets
- Biosafety level IV

**Equipment**

- Gurneys
- Easy to clean tables, trays and trolleys
- Waste disposal bins
- Autopsy kit
- Oscillator saw
- Embalming machine
- Coolers/refrigerators
- Air conditioning system, in the clean area as required
- Cameras
- Bio safety cabinets
- Specialized surgical equipment
- Radiology

# *69.23* Mortuary layout

## 69.23.1 LOCATION

- Adequate vehicular access from the service road
- Away from clinical, kitchen and dining areas if hospital based
- Located at ground level
- Near histopathology laboratory Services
- Separate access for staff, undertakers and visitors

### LAY-OUT

For the purpose of infection control, facility should have clean area, transitional area and dirty area. Workflow should be planned to minimize movement from dirty to clean areas

**Dirty area**

- Post mortem room
- Utility room
- Instrument room
- Body store

**Clean area**

- Reception
- Waiting rooms
- Interview/counseling room
- Bier rooms
- Offices
- Observation area
- Staff changing area
- Specimen store

**Transit area**

- The body handling area
- The disposal room
- The PM transit area

Post mortem room should be connected to the body storage area, the dirty utility room/ instrument store and PM transit area, through which access is gained to the staff changing area and from the circulation routes. It may also be connected to the disposal room.

The disposal area should be organized in such a way that clinical wastes, linen and domestic waste are not mixed together prior to collection.

**Staff changing rooms**

Staff and non-mortuary personnel must remove outer clothes before entering the PM room. This will take place in the staff changing area.

Boots and stocks of protective garments (as prescribed by local policy) should be stored in the PM transit area, leading off the staff changing area and from where access is gained to the PM room. Staff should change into boots and protective garments in the PM transit area before entering the PM room.

Staff and others should discard used protective clothing and boots within the PM suite, and change into slip-on footwear before moving into the connecting staff changing area. Reusable protective clothing should be bagged up as appropriate before transferring to the disposal room.

## 69.23.2 MORTUARY DESIGN REQUIREMENTS



### ENTRANCES AND SIGNPOSTS

- The mortuary entrance should be easy to access and bear relevant signage.
- For hospital based mortuaries, the number of entrances depend on whether staff, relatives and the arrival of bodies from the hospital share a common approach and then follow separate traffic routes to the individual entrances to the relevant parts of the mortuary, or whether there is direct access from a hospital street to the different parts of the mortuary. In either case, an entrance will be needed for collection of bodies by undertakers and, if appropriate, bodies arriving from outside the hospital.

### BODY STORAGE AND PROCEDURE ROOM

- Should be adjacent to the PM room and adjoin the bier room. Space is required in the body handling area for parking and maneuvering trolleys. Body weighing facilities are required. Space is also required for the reception of bodies on trolleys from the hospital, the labeling or identification of bodies and entering details in a record book or computer, , the transfer of bodies to the refrigerated body store, the removal and transfer of bodies from the body store to the PM room or to the bier room, the removal of bodies from the store, and confirmation of identity before handing over to undertakers or for police identifications. Consideration should be given to the use of mobile and fixed hoists, which will have implications on space requirements.
- The body store consists of a number of labeled compartment bays, (refrigerated at approximately 4°C), each containing between three and five racks for holding the body trays upon which bodies are stored. Individual compartment bays may either be physically separated from one another or may be open between one another in a continuous run. Compartment bays may either have a door at one end or may be double-ended in the case of pass-through fridges.
- All doors to the refrigeration compartment bay must open to give access to the body trays. All doors should be fitted with locks. High quality hinges and

locks are an important consideration. All compartment bays should be capable of being drained. Internal rollers and racking holding body trays should be removable to permit clear entry to the compartment bay for cleaning purposes. The refrigeration plant must be fully accessible for maintenance.

- Hand hygiene facilities and wash down points must be provided in the body handling area.
- Lockers for the storage of personal effects removed from bodies should be provided in a secure area.

### Finishes

The floor of the body handling area must be hardwearing, non-slip and impervious to water and disinfectant. The walls should be capable of withstanding regular washing or hosing down and should meet the raised junction with the floor at a waterproof joint. Ceilings, and where relevant ceiling suspension grids, should be capable of withstanding frequent washing down.

### Ventilation

Mechanical ventilation should be provided to the body handling area so that air flows from this area into the PM room. Where there is direct access from outside to the body handling area, it will be necessary to provide some form of lobby, with two sets of doors.

## AUTOPSY ROOM

1. The autopsy room should place be direct from body storage area in order to allow transfer bodies to the autopsy table and have ample space for movements.
2. Autopsy table should be of adjustable height and have drainage and water supply. Consideration should be given for rotating tables. The table should have a hot and cold water supply and a waste outlet of about 75mm diameter fitted with a suitable, readily accessible tap and drain pipe.
3. There should be an organ dissection bench. The dissecting bench should have raised edges and slope to a sink(s), which should be deep enough for the washing of organs. There should be provision for running water over the bench itself. The drainage flow of water should be checked and confirmed. The positioning of sinks along the dissecting bench should suit the pattern of working agreed upon by the staff. A sluice is required for the opening of intestines and disposal of contents. A low pressure water pipe should be provided, preferably in the wall of the sink(s). A standing waste is required. A filter trap is necessary. The bench should be easily cleanable and have no traps for infected material and should preferably be wall mounted.
4. Most PM rooms will require a minimum of two PM tables to permit the pathologist to carry out several examinations at one attendance.
5. Post-mortem tables should be easily cleanable and free from traps for potentially infected material.

6. Walls and floors must be finished with hard and durable surfaces, easy to clean, impervious to liquids and resistant to disinfectants. Floors should be very hard wearing an non slip.

## Lighting

Postmortem room should have ample daylight and distributing of and location of windows should take into account privacy and prevent glare and excess sunlight. Artificial lighting should provide good general illumination with higher levels for task lighting over the post mortem tables and dissecting benches.

## Ventilation

Negative control ventilation system



## DIRTY/ UTILITY/ INSTRUMENT ROOM

- Should open directly off the PM room and it serves as a dirty utility room and for storage of instruments.
- An automated washer- disinfector should be provided for the cleansing and disinfection of instruments after use.
- Where sterilization is required the equipment should be transported to the sterilization department.
- Sinks for washing and disinfecting bowls and instruments.
- Flash sluice.

## POST MORTEM TRANSIT AREA

- Entry to the PM room will be via the PM transit area, which leads off the staff changing area and separates clean and dirty activity areas.
- Staff entering the PM room will need to change into protective clothing. Suitable shelving, racks and hooks should be provided within the PM transit area for the storage of protective clothing and boots.
- Staff should discard used protective clothing within the PM transit area or PM room. Separate bins for the disposal of single-use items and collection of re-usable items pending cleaning should be provided.
- Hand hygiene facilities with hands-free tap control should be provided for the washing of hands following the removal of protective clothing.
- Staff must pass through a boot wash before entering and upon leaving the PM room. Boots should be stored in the PM transit area.

## STAFF CHANGING AREAS

Two identical sets of WCs/showers and lockable storage spaces should be provided in the staff changing area to allow for flexible use by either sexes or different staff groups (according to local policy). Hand washing facilities should be provided.

## OBSERVATION AREA

Should be physically separate from the post mortem room

## SPECIMEN STORE

- Shelves made from impervious material will be required for holding jars or containers of various sizes.
- Floor space, or space below high benching, may be required for formalin containers. The room must be continuously ventilated because of the hazard arising from formalin used in the specimen containers.

## PATHOLOGIST'S OFFICE

The function of the pathologists' office is to provide space for consultations and writing reports. It should have a window for natural ventilation and light, and should be entered from the circulation route leading to the staff changing area and the body handling area.

## TECHNICIANS' OFFICE/ REST ROOM

- Should have access to the body viewing facilities .It should be situated near the body handling area and the undertakers' entrance so that bodies may be registered and labeled before being deposited in the body store. It should be entered from the circulation route leading to other parts of the mortuary.
- The staff call bells for undertakers and visitors will need to be located here
- Apart from clerical functions, the office will be used for relaxation between work periods.
- It should be furnished with a desk(s), chairs, shelves and filing cabinet.
- Lockers should be provided to enable technicians to store clothing and personal effects in this room.

## DISPOSAL ROOM

A disposal room is required with adequate space for the temporary storage of securely packed refuse and dirty linen bags (appropriately color coded) with easy access for their collection.

## CLEANER'S ROOM

- A cleaners' room should be provided to service the whole accommodation.
- There should be lockable cupboard space for secure storage of stock and shelves for holding in-use materials.
- There should be adequate space for maneuvering machines, for emptying and filling buckets and bowls, and the routine servicing and cleaning of equipment.
- There should be unrestricted access to the sink, and to a wash-hand basin.

### GENERAL PURPOSE LINEN STORE

- A general purpose store will be needed for a wide variety of stock items and linen that do not require specialized environmental conditions. As stock dimensions vary considerably, adjustable shelving would be an advantage. Adequate floor space should be allowed for the storage of bulky goods.
- The store must be accessible to staff servicing both the body handling and viewing areas, and the PM room activity requirements.

## 69.24 Crime scene support



### 69.24.1 GENERAL CONSIDERATIONS

Forensic pathologist may be called upon by investigating agents to support in crime scene analysis and interpretation as well as evidence collection. In this regard, the pathologist may require a basic understanding of processes and protocols in crime scene management. These include:

- Note taking
- Securing a crime scene
- Evidence management
- Scaling the investigation to the even

### 69.24.2 THE ROLE OF THE FORENSIC PATHOLOGIST IN CRIME SCENE MANAGEMENT

- When a forensic pathologist is requested by police authorities to attend the scene of a suspicious death, she/he is 'briefed' as to the circumstances of the case by the Senior Investigating Officer (SIO), or his/her representative.
- A strategy for approaching the body, the collection of trace evidence from, and around, the body and ultimately the recovery of the body from the scene, is agreed with crime scene investigators, forensic scientists and the SIO.
- The forensic pathologist examines the body, noting its disposition, the surroundings in which the body lies and the presence of injuries that can be seen without disturbing the body or the scene.
- The pathologist supervises recovery of the body by crime scene investigators and funeral directors.

## 69.25 Forensic anthropology

- Forensic anthropology is a special sub-field of physical anthropology (the study of human remains) that involves applying skeletal analysis and techniques in archaeology to solving criminal cases.

- A forensic anthropologist can assist in the identification of deceased individuals whose remains are decomposed, burned, mutilated or otherwise unrecognizable, as might happen in a plane crash.
- Forensic anthropologists are also instrumental in the investigation and documentation of genocide and mass graves.
- Along with forensic pathologists, forensic dentists, and homicide investigators, forensic anthropologists commonly testify in court as expert witnesses.
- Using physical markers present on a skeleton, a forensic anthropologist can potentially determine a person's age, sex, stature, and race.
- In addition to identifying physical characteristics of the individual, forensic anthropologists can use skeletal abnormalities to potentially determine cause of death, past trauma such as broken bones or medical procedures, as well as diseases such as bone cancer

## 69.26 Forensic entomology

- Forensic entomology is the scientific study of the colonization of a dead body by arthropods.
- It involves the identification of insects and other arthropods associated with human remains as an aid to determining the time and place of death.
- This includes the study of insect types commonly associated with cadavers, their respective life cycles, their ecological presences in a given environment, as well as the changes in insect assemblage with the progression of decomposition.
- Insect succession patterns are identified based on the time a given species of insect spends in a given developmental stage, and how many generations have been produced since the insects introduction to a given food source.
- Insect development alongside environmental data such as temperature and vapor density, can be used to estimate the time since death, due to the fact that flying insects are attracted to a body immediately after death.
- The identification of postmortem interval to aid in death investigations is the primary scope of this scientific field. However, forensic entomology is not limited to homicides, it has also been used in cases of neglect and abuse, in toxicology contexts to detect the presence of drugs, and in dry shelf food contamination incidents.
- Equally, insect assemblages present on a body, can be used to approximate a given location, as certain insects may be unique to certain areas.

# Part IX: COVID-19

The coronavirus disease 2019 (COVID-19) is an acute respiratory infection caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). Coronaviruses are a large family of enveloped RNA viruses, some of which cause illness in people (e.g., common cold, SARS, MERS) and others that circulate among mammals (e.g., bats, camels) and birds. Rarely can animal coronaviruses spread to humans and subsequently spread between humans. Similar to SARS and MERS, it is thought that human transmission occurs via respiratory droplets produced when a person sneezes or coughs and aerosol in certain circumstances, including airway manipulation. Aerosol generation occurs in coughing, nebulization, tracheal intubation and airway suctioning.

WHO first declared COVID-19 a public health emergency of international concern on 30 January 2020 and subsequently declared it a pandemic on 11 March 2020. The pace at which COVID-19 spread worldwide and in Kenya was unprecedented. Kenya discovered the first documented case of COVID-19 within its borders on 13 March 2021. COVID-19 is highly transmissible and infectious and runs the risk of overwhelming the health system's capacity, with the need to support not just those with COVID-19 but also those with other illnesses. A lot of efforts have gone into reducing transmission of the virus, including restrictions on gatherings, contact tracing, quarantine and isolation. With the spread of COVID-19 in the communities, preventive public health measures such as hand washing and proper use of face masks cannot be overemphasized. The most common symptoms of COVID-19 include cough, loss of smell and/or taste, fever, difficulty breathing, headache, sore throat, running nose, chest pain, myalgia, fatigue, general weakness and diarrhoea. The most common clinical presentation is a respiratory infection with a symptom severity ranging from a mild common cold-like illness (estimated to be 80% of cases) to severe viral pneumonia in approximately 14%, leading to acute respiratory distress syndrome potentially fatal in about 5%. Current estimates of the incubation period range from 1 to 14 days, with a median incubation period of five to six days. Transmission can occur during the incubation period, even without symptoms.

Certain groups of people are at higher risk for transmission and severe disease, including healthcare workers who work with COVID-19 patients. In addition, vulnerable and marginalized groups such as people with disabilities may face challenges in accessing healthcare and have worse outcomes from COVID-19.
People of any age can catch COVID-19, but it most commonly affects middle-aged and older adults. The risk of developing severe COVID-19 disease increases with age from age  Some conditions can result in higher severity of illness in adults of any age;

- Diabetes Mellitus (Type 1 or 2)
- Heart Conditions ( such as heart failure, coronary artery disease, cardiomyopathies or hypertension)
- Overweight and obesity

- Smoking
- Chronic kidney disease
- Chronic lung diseases, including COPD (chronic obstructive pulmonary disease), asthma (moderate-to-severe), interstitial lung disease, cystic fibrosis, and pulmonary hypertension
- HIV/AIDS
- Immune Suppression
- Liver disease
- Pregnancy
- Sickle cell
- Solid organ or blood stem cell transplant
- Cerebrovascular disease

Clinical manifestations of COVID-19 are generally milder in children compared with adults. Symptomatic children may present with non-respiratory symptoms such as gastroenteritis more frequently than adults. An acute hyperinflammatory syndrome leading to shock or multi-organ failure has been described, known as the Multisystem Inflammatory Syndrome (MIS-C), which is temporally associated with COVID-19 in children and adolescents.

A significant challenge in the war against the pandemic appears to be the rate at which mutations occur, resulting in several variants resulting in more infections and increased disease severity. This highlights the importance of strengthening public health measures and vaccination strategies early in the response.

# 70. CASE DEFINITION

Suspected case of SARS-CoV-2 infection:

**1. A person who meets the clinical AND epidemiological criteria:**

**Clinical criteria:**

- Acute onset cough AND fever;
- Acute onset of ANY TWO OR MORE of the following signs or symptoms:

Cough, fever, loss of taste or smell, difficulty breathing, sore throat, running nose, chest pain, fatigue/general weakness, headache, diarrhoea, altered mental status (Children may present with atypical symptoms)

**AND**

**Epidemiologic criteria:**

- Where there is widespread community transmission in several regions of the country, then all patients will be considered to have met epidemiologic criteria

**2. A patient with severe acute respiratory illness (SARI)**

(SARI: Acute respiratory infection with or without fever; and cough; with onset within the last 10 days; and requires hospitalization)

**Probable case of SARS-CoV-2 infection**

- A patient who meets the clinical criteria above AND is a contact of a probable or confirmed case or linked to a COVID-19 cluster

- A suspected case with chest imaging showing findings suggestive of COVID-19 disease
- Recent onset loss of taste or loss of smell with no other identified cause (Common imaging findings include bilateral peripheral opacities with lower lung distribution. Opacities usually ground glass opacities that may progress to consolidations)
- Unexplained death in an adult with SARI prior to death AND had contact with a probable or confirmed case or linked to a COVID-19 cluster

**Confirmed case of SARS-CoV-2 infection**

- A person with a positive SARS-CoV-2 PCR test
- A person with a positive SARS-CoV-2 Antigen RDT AND meeting criteria for either suspected or probable case; OR has contact with a probable or confirmed case.

# 71. Infection Prevention and Control (IPC) plan in response to COVID-19

**Introduction**

The main aim of IPC is to prevent or limit the spread of SARS-COV2 at all levels of healthcare

**Facility preparedness**

All facilities should have the following:

- An IPC program or a dedicated IPC focal person
- A functional screening and triage area for early case identification
- A holding area for cases awaiting results or transfer
- A mechanism to ensure standard and transmission-based precautions.
- Adequate healthcare workers to provide 24-hour patient care without exhaustion.
- A plan to conduct health worker exposure risk assessment
- Continuous training and refresher courses for the existing staff and any new staff
- Adequate IPC supplies and equipment

**Quarantine and Isolation**

- Limit the number of visitors
- Continue to observe respiratory hygiene and cough etiquette
- Observe hand hygiene by either use of soap and water or an alcohol-based hand rub
- Ensure proper ventilation of the facility or home
- Observe for fever or other symptoms daily
- Watch for danger signs or signs or signs of deterioration like dyspnea and report to a health facility
- Use of either separate utensils or disposable utensils

**Quarantine**

Quarantine is the separation and restricted movement of healthy persons who have been exposed to persons with COVID-19. It can be applied at the individual, family or community level. All persons who have had contact with a confirmed case of COVID-

19 should quarantine for 14 days and get a COVID-19 test if they develop any symptoms. The quarantine can either be self-quarantine or carried out at a designated facility.

**Isolation**

Isolation is separating sick people with a contagious disease from those who are not ill. All confirmed COVID-19 cases identified should be isolated. The isolation location can be in a health facility for those with severe illness, at home for those who meet the self-isolation criteria or at a community isolation facility. Isolation precautions may be dropped 10 days after the onset of symptoms, provided that one has had no fever without antipyretics for at least 24 hours.

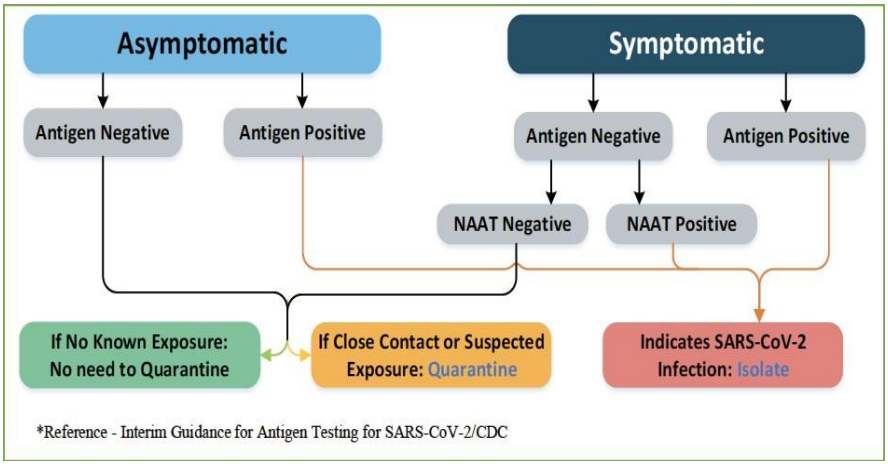
**Diagnosis of COVID-19**

This section aims to provide guidance on who to test for COVID-19 and the preferred tests to use in the clinical setting. Testing is only recommended for diagnosis and not as an indicator of recovery from COVID-19. Testing should be offered to all persons meeting the case definition

**Preferred Initial Tests**

Nucleic Acid Amplification Tests (NAATs) such as the SARS-CoV-2 Polymerous Chain Reaction (PCR) are the preferred initial tests. Where access to a PCR test is limited or too costly then SARS-CoV-2 antigen testing can be utilized. Turn-around times for antigen tests are generally shorter than for PCR testing and thus an antigen test can help with quick identification of COVID-19 cases. Sensitivity of antigen tests is lower than that of NAATs. Therefore, a negative test may warrant confirmation by a PCR test in symptomatic patients. A positive antigen test does not warrant confirmation unless the patient is asymptomatic and the diagnosis is in doubt.

Serological tests i.e., SARS-COV-2 antibody detection tests should not be used for diagnosis of COVID-19. They can only be used to check for previous infection for example in the setting of serological surveys. Indeterminate PCR test results usually indicate that only one of the 2 or more target genes being tested for was identified. These tests should be considered presumptively positive.




**Colletion of Specimens**

- Collect specimens from the upper respiratory tract (URT; nasopharyngeal and oropharyngeal) AND, where clinical suspicion remains and URT specimens are negative, collect specimens from the lower respiratory tract when readily available (LRT; expectorated sputum, endotracheal aspirate, or broncho-alveolar lavage in ventilated patient) for SARS-CoV-2 testing by RT-PCR and bacterial stains/cultures.

- Use appropriate PPE for specimen collection (droplet and contact precautions for URT specimens; airborne precautions for LRT specimens). When collecting URT samples, use viral swabs (sterile Dacron or rayon, not cotton) and viral transport media. Do not sample the nostrils or tonsils. In a patient with suspected SARS-CoV-2, especially with pneumonia or severe illness, a single negative URT sample does not exclude the diagnosis, and additional URT and LRT samples are recommended. LRT samples are more likely to be positive and for a longer period. Clinicians may elect to collect only LRT samples when these are readily available (for example, in mechanically ventilated patients). Sputum induction should be avoided due to increased risk of increasing aerosol transmission.

- Samples should be collected in a timely manner for clinical management and outbreak control. Ensure that staff responsible for collection of samples are well trained and available. Samples should be transported to the laboratory using Viral Transport Media and should be triple packaged. For further details on sample collection please refer to the Ministry of Health Targeted Testing Strategy for Coronavirus disease 2019 (COVID 19) in Kenya.

**Specimens for testing**

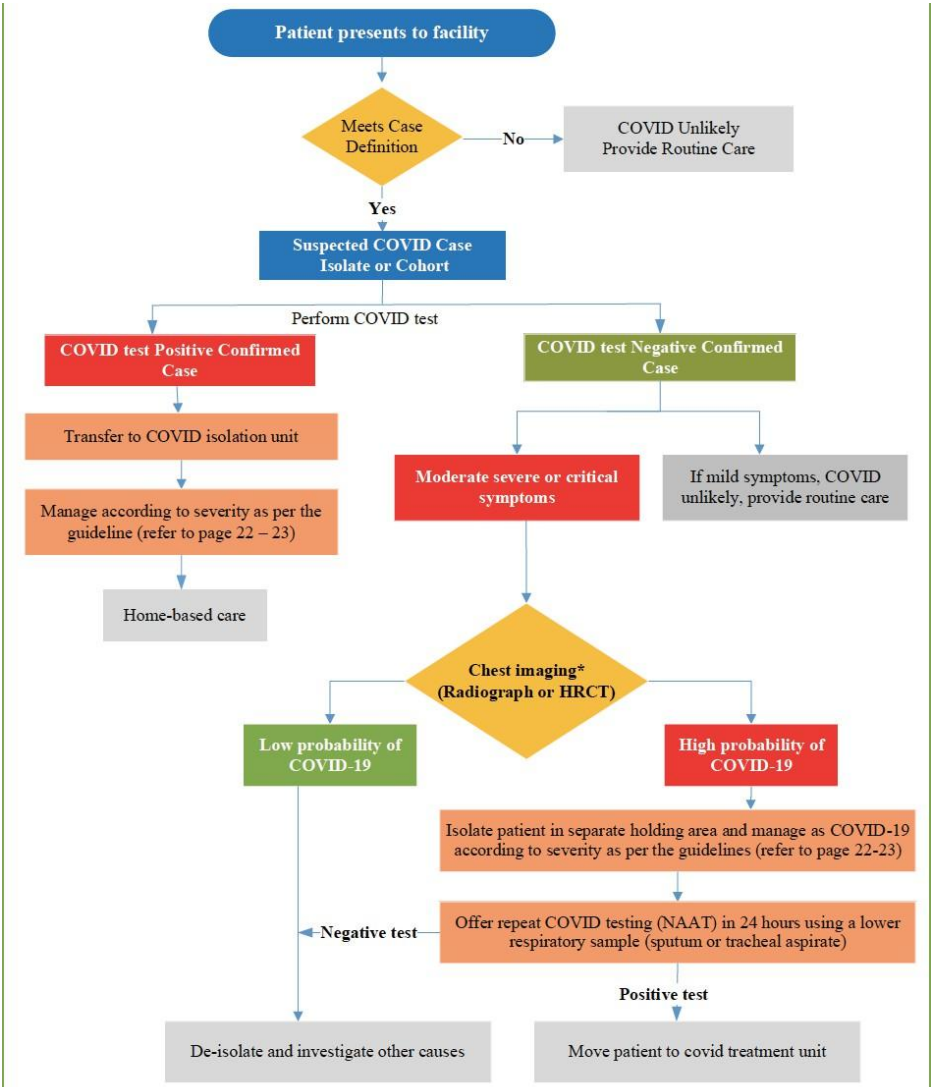
Specimens can be taken from the upper respiratory tract or the lower respiratory tract. Upper respiratory tract samples include nasopharyngeal swabs, oropharyngeal swabs and nasopharyngeal aspirates. Lower respiratory tract specimens include bronchoalveolar lavage specimens and expectorated sputum.

**Collection of specimens for laboratory diagnosis**

**NB:**

**The role of radiological tests for diagnosis of COVID-19**

Imaging including chest radiographs and high-resolution CT scans are useful in monitoring of the clinical course and evaluating disease severity. Chest CT scan images from patients with COVID 19 typically demonstrate bilateral peripheral ground glass opacities which are non-specific. These can be found other kinds of pneumonia. This makes the diagnostic value of chest CT scan in COVID 19 low and dependent on radiographic interpretation. Given the variability in chest imaging findings, chest radiograph or CT scan alone is not recommended for the diagnosis of COVID 19.


Patient presents to facility
Meets Case Definition
No
COVID Unlikely Provide Routine Care
Yes
Suspected COVID Case Isolate or Cohort
Perform COVID test
COVID test Positive Confirmed Case
COVID test Negative Confirmed Case
Transfer to COVID isolation unit
Manage according to severity as per the guideline (refer to page 22 – 23)
Home-based care
Moderate severe or critical symptoms
If mild symptoms, COVID unlikely, provide routine care
Chest imaging* (Radiograph or HRCT)
Low probability of COVID-19
High probability of COVID-19
Isolate patient in separate holding area and manage as COVID-19 according to severity as per the guidelines (refer to page 22-23)
Negative test
Offer repeat COVID testing (NAAT) in 24 hours using a lower respiratory sample (sputum or tracheal aspirate)
Positive test
De-isolate and investigate other causes
Move patient to covid treatment unit

**Management of COVID-19**

The management of patients with COVID-19 depends on severity of disease at presentation.

Once patient is CONFIRMED positive by a PCR or rapid antigen test categorize them into the following groups based on presentation

**Table 71:1 COVID-19 severity categorization in adults and adolescents**

| CATEGORY | FEATURES |
|---|---|
| 1. Mild illness | Fever, cough, sore throat, malaise, headache, muscle pain<br>BUT<br>**No dyspnoea (shortness of breath)** and **No abnormalities on chest imaging** |
| 2. Moderate illness | Clinical features of pneumonia (fever, cough, **dyspnoea)**<br>AND/OR<br>**radiological features of pneumonia**<br>BUT<br>**Oxygen saturations (SPO2) greater than or equal to 94% on room air** |
| 3. Severe illness | Clinical and radiological features of pneumonia, tachypnea with RR>30<br>AND<br>**oxygen saturation (SPO2) less than 94% on room air** |
| 4. Critical illness | Features of severe illness<br>AND<br>Any of the following:<br>• **respiratory failure**<br>• **sepsis/septic shock**<br>• **multiorgan dysfunction**<br>• **acute thrombosis** |

**Table 71:2 severity categorization in children**

| CATEGORY | FEATURES |
|---|---|
| 1. Mild illness | Fever, cough, sore throat, malaise, headache, muscle pain<br>**BUT**<br>No dyspnoea (shortness of breath and No abnormalities on chest imaging |
| 2. Moderate illness | Clinical signs of non-severe pneumonia (cough or difficulty breathing)<br>**AND**<br>Fast breathing*<br>**AND/OR**<br>[redacted] 350; 1-5years: 340 |

| | |
|---|---|
| 3. **Severe illness** | Child with clinical signs of pneumonia (cough or difficulty in breathing) + at least one of the following:<br>• Central cyanosis or SPO2 <90%;<br>• Severe respiratory distress (e.g., fast breathing*, grunting, very severe chest indrawing);<br>• General danger sign: inability to breastfeed or drink, lethargy or unconsciousness, or convulsions<br><br>*Fast breathing (breaths/min): <2months: ³60; 2-11months: ³50; 1-5years: ³40 |
| 4. **Critical illness** | Features of severe illness<br>AND<br>Any of the following:<br>• Acute respiratory distress syndrome<br>• Respiratory failure requiring mechanical ventilation<br>• Sepsis/Septic shock<br>• Other organ failure requiring ICU care |
| 5. **MIS-C** | Preliminary case definition: Children and adolescents 0–19 years of age with fever > 3 days<br>**AND**<br>Two/more of the following:<br>• Rash or bilateral non-purulent conjunctivitis or muco-cutaneous inflammation signs (oral, hands or feet);<br>• Hypotension or shock;<br>• Features of myocardial dysfunction, pericarditis, valvulitis, or coronary abnormalities;<br>• Evidence of coagulopathy,<br>• Acute gastrointestinal problems;<br><br>**AND**<br>No other obvious microbial cause of inflammation<br>**AND**<br>Evidence of COVID-19 (RT-PCR, antigen test or serology positive), or likely contact with patients with COVID-19. |

**Supportive care**

Supportive care should be offered to all patients diagnosed with COVID-19. This includes the following:

1. Counselling and psychosocial support
2. Symptomatic treatment
3. Adequate nutrition and hydration

**Table 71:3 Management of asymptomatic, mild and moderate COVID-19**

| | |
|---|---|
| Asymptomatic or mild illness | **Assess for eligibility for home-based care**<br>Patient qualifies if they have no risk factors for disease progression or poor outcomes (see below) and a suitable space is available at home (separate room with separate bathroom), has resources to access basic PPE for family members e.g., face masks and gloves, no house members who are increased risk of severe illness if exposed e.g., see below<br>**Risk factors for poor outcome:**<br>Age >60, coronary artery disease, stroke, diabetes, hypertension, cancer, chronic lung disease, frailty, pregnancy, immunosuppression, chronic kidney disease<br>**Management**<br>Symptomatic treatment for mild disease (paracetamol, antihistamines). **Steroids should NOT be used for patients with asymptomatic, mild or moderate disease.**<br>(Isolation precautions as outlined in the IPC section) |
| Moderate Illness | • Baseline tests - blood count, renal and liver function, HIV test, random blood sugar.<br>• symptomatic treatment:<br>  • Fever - Paracetamol<br>  • Sore throat - gargles<br>  • cough, nasal congestion - antihistamine<br>• VTE prophylaxis with Enoxaparin 40mg once a day if admitted to a health facility<br>  • Where enoxaparin is not available, use low dose unfractionated heparin at 5000units subcutaneous BD)<br>  • Where patient unable to use standard anticoagulation therapy, consider use of direct-acting anticoagulants<br>  • Consider prophylaxis for children older than 5 years with comorbidities and not ambulant. Refer to BNF for dosage guidelines for pediatrics<br>Where there is pressure for space for isolation of patients, the following patients with moderate illness can be managed at home:<br>• Young <60 years<br>• Oxygen saturations >94% on room air<br>• No comorbidities<br>• Have easy access to a health facility in case of worsening of symptoms<br>• Physically active |

**Table 71:4 Management of severe and critical COVID-19**

| | |
|---|---|
| **Severe illness** | • Baseline Tests (Total blood count, renal and liver function, HIV test, random blood sugar)<br>• Symptomatic treatment<br>• Oxygen supplementation to maintain SPO2s above 90% and above 92% in pregnant women (oxygen supplementation can be via nasal prongs, masks, non-rebreather masks or high flow nasal cannula - see below)<br>• Dexamethasone 6mg per day for up to 10 days (where dexamethasone is not available, consider using prednisone 40 mg OD or methyl prednisone 32mg OD. This short duration of dosing does not require tapering)<br>  • For children - Dexamethasone 0.15mg/kg iv/PO OD to a maximum of 6mg or prednisolone 1mg/kg OD maximum 40mg OD, methylprednisolone 0.8 mg/kg IV OD maximum 32mg OD<br>• VTE prophylaxis Enoxaparin 40mg OD once a day for the duration of hospitalization (Where enoxaparin is not available, use low dose unfractionated heparin at 5000units subcutaneous BD)<br>• Self proning for 12 to 16 hours a day (see self-proning guide below) as tolerated |
| **Critical Illness** | • Baseline tests- total blood count, renal and liver function tests, HIV test, random bold sugar<br>• Symptomatic treatment<br>• Admit to a **Critical Care Unit.**<br>• Mechanical Ventilation if no improvement in oxygenation with maximal oxygen flows with other modalities - see guide to noninvasive ventilation, tracheal intubation and ventilation below<br>• Prone for 12 to 16 hours per day<br>• Conservative fluid management i.e., give IV fluid only if hypovolemic<br>• Closed suctioning of secretions where available<br>• Give Dexamethasone 6 mg per day for up to 10 days (where dexamethasone is not available, consider using prednisone 40 mg OD or methylprednisolone 32mg OD. This short duration of dosing does not require tapering)<br>  • For children - Dexamethasone 0.15mg/kg iv/ PO OD to a maximum of 6mg or prednisolone 1mg/kg OD maximum 40mg OD, methylprednisolone 0.8 mg/kg IV OD maximum 32mg OD<br>• VTE prophylaxis 40mg Enoxaparin OD SC (Where enoxaparin is not available, use low dose unfractionated heparin at 5000units subcutaneous BD)<br>**Where possible, document advance directives for all patients e.g., do not resuscitate for patients who are unlikely to do well or have another terminal condition** |

**Baseline Tests**

- Should be done for all patients who are admitted and all patients with risk factors for poor outcomes: total blood count, random blood sugar, Urea Electrolytes Creatinine, Liver function tests. HIV testing should be offered to all patients.
- Chest imaging is recommended in patients with severe illness who fail to improve on standard therapy and in all patients with critical illness. Include an ECG if indicated.
- Where available a C-Reactive Protein (CRP) may be useful in managing patients who acutely deteriorate.

**Other Therapeutic Agents**

The following drugs (Table 65.5) may have a role in the management of COVID-19. Specialist input would be required in defining the appropriate patient population, weighing benefit against risk, and cost considerations. These agents are still investigational and under emergency use authorization. This means that a patient must be educated on the evidence around their use and must consent to their use prior to prescription. Their use should be reported to the Pharmacy and Poisons Board

**Table 71:5 Other Therapeutic Agents**

| Drug | Mechanism of Action | Potential Indications |
|---|---|---|
| **Tocilizumab** | monoclonal antibody against IL-6 | Hospitalized patients with severe and critical COVID-19 with disease progression and elevated markers of systemic inflammation (CRP >75) despite steroid use. |
| **Baricitinib (with remdesivir)** | Janus Kinase (JAK) 1 and 2 selective inhibitor | Hospitalized patients with severe COVID-19 with disease progression and elevated markers of systemic inflammation despite steroid use (Baricitinib alone) or in patients with severe COVID-19 in whom steroids are contraindicated (Baricitinib with remdesivir) |
| **Remdesivir** | an antiviral agent that inhibits SARS-Co-V-2 replication | Hospitalized patients with severe but not-critical COVID-19 who are within 10 days from the onset of symptoms.<br><br>There is conflicting data on the use of remdesivir, with most clinical trials showing no mortality benefit. Some studies have shown that remdesivir may reduce duration of illness by few days and only if initiated very early after disease onset rather than at the time a patient is deteriorating. |

**Table 71:6 Adult Covid-19 Patient Care**

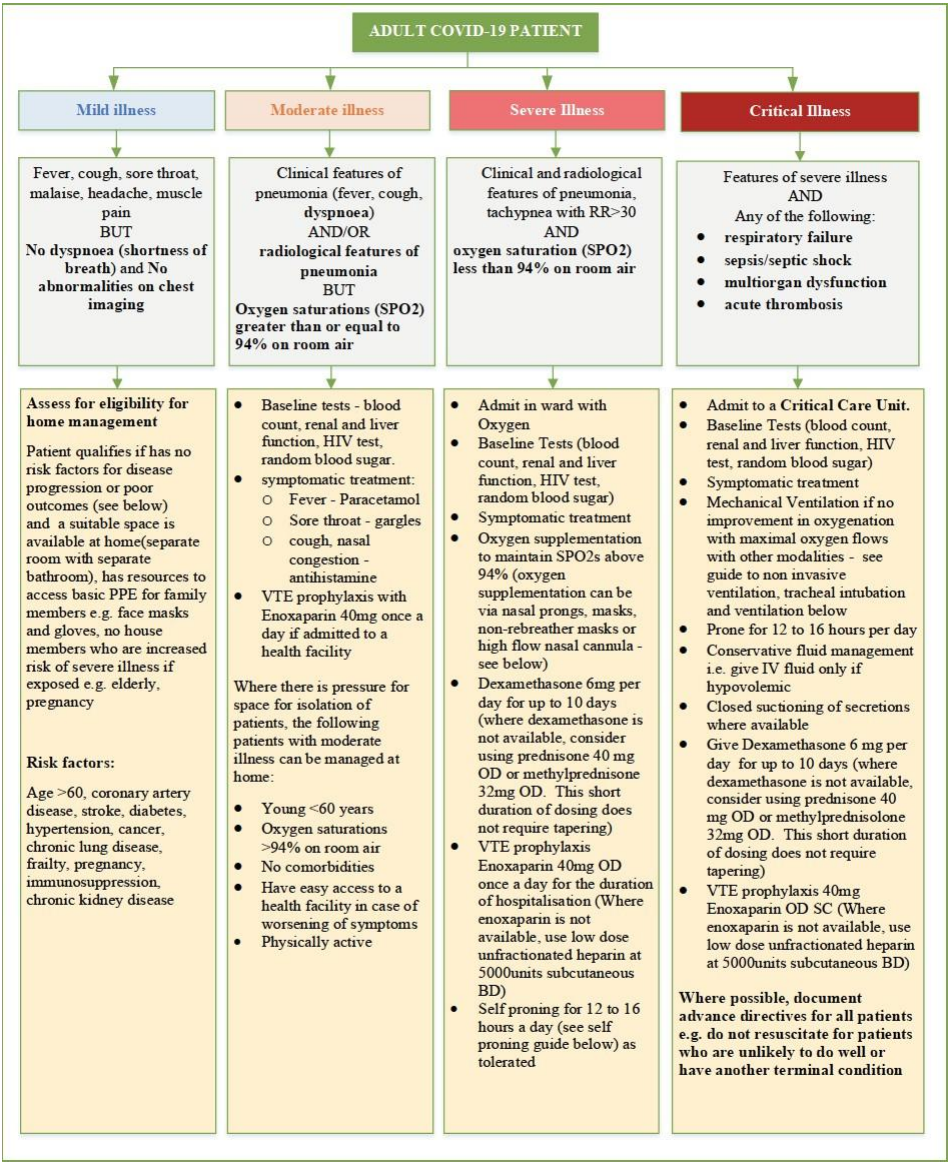
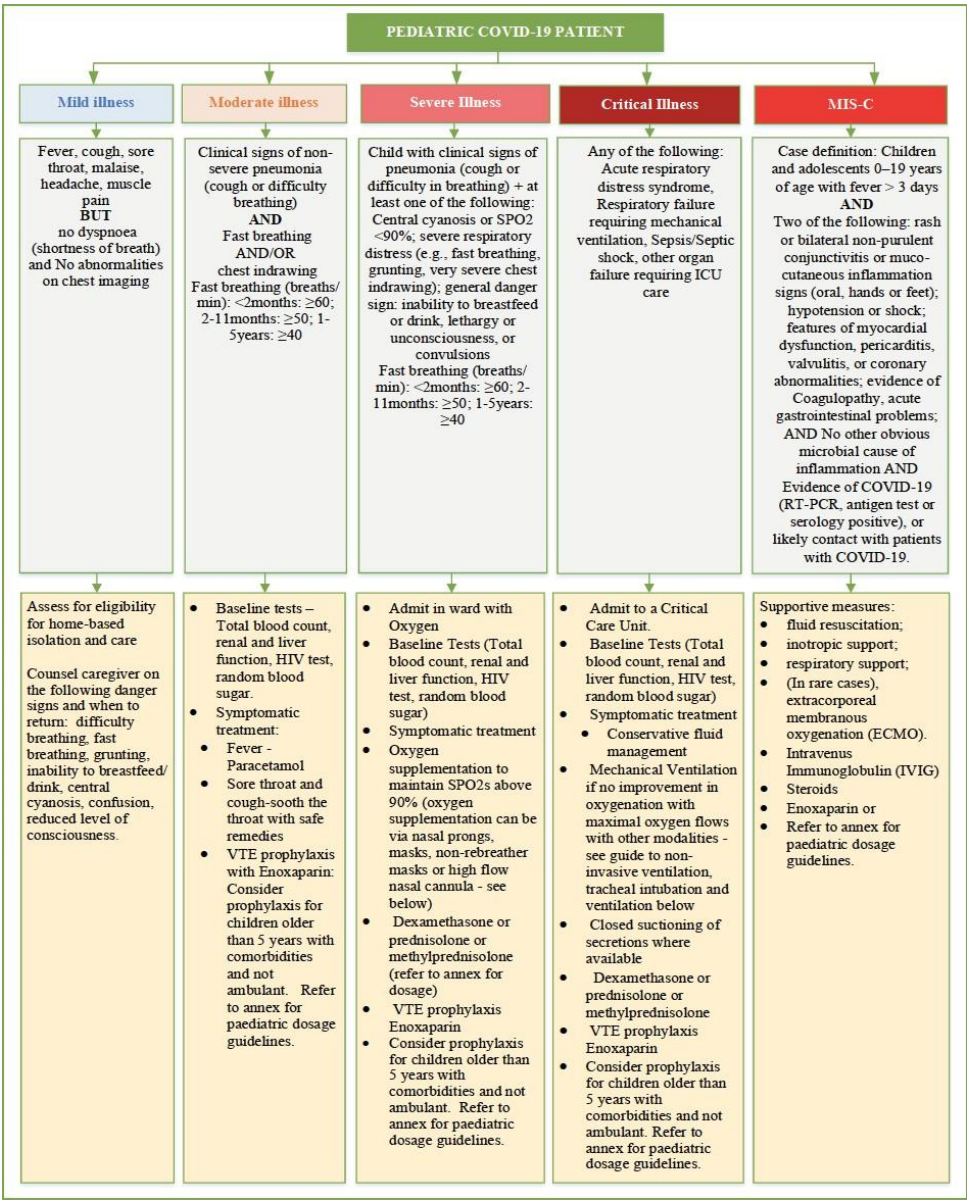


**Table 71:7 Pediatric Covid-19 Patient Care**



**Oxygen therapy:**

Oxygen therapy: Oxygen via nasal cannulae is indicated in those with saturations of 94% or below. Up to 4 litres of oxygen can be administered via this route. Monitoring of response can be done both via pulse oximetry and arterial blood gases.

If the patient continues desaturating despite this, higher flow oxygen will be required. Current delivery systems available include the face mask (5-10L/min) and the non-rebreather mask (up to 15 L/min). High flow nasal cannula can support flows of up to 60L/min. If the patient requires high flow oxygen, please contact critical care/pulmonology team as escalation to the ICU may be necessary.

Remember that the risk of aerosolization increases once oxygen flows of above 4L/min per minute are required and an N95 mask should be used in addition to other precautions.

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# APPENDIX

## COMMON SKIN CONDITIONS

**A. Inflammatory Conditions**

| | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
|---|---|---|---|---|
| | **ECZEMA:** A common inflammatory, genetic skin disease that can be acute & chronic. The common types are:<br><br>**i) Atopic** - is a type of eczema, occurring in atopic individuals and often presents in early childhood. Is associated with other allergic disease like Rhinitis and Asthma<br><br>**ii) Nummular (Discoid)** - a recurrent form of eczema with rounded coin-shaped lesions<br><br>**iii) Dyshidrotic (Asteotic)**<br><br>**iv) Stasis Eczema** – Is due to chronic venous insufficiency and heart disease. | Acute eczema presents with Itching, papules & plaques, erythema, swelling, oozing, to frank blistering or vesicles and bullae with subsequent scaling and/or crusting.<br>Chronic eczema consists of lichenified lesions (skin thickening with increased markings), excoriations and hyper-or hypo-pigmentation. Secondary bacterial infection can occur.<br>The lesions may be single or few on the extensor surfaces of extremities, buttocks and back<br><br>Has circular, tender pruritic vesicular rashes on palms and sides of fingers but may also affect the soles and toes. The lesions usually subside by peeling. It is more common in summer and is commonly associated with hyperhidrosis, hence the name. Stress is an important triggering factor.<br><br>Consists of itchy rashes and pigmentation on the medial side of one or both lower legs above the medial malleolus, and followed by edema, weeping, vesiculation, crusting & ulceration.<br><br>Develops on lower limbs due to poor venous return or heart disease with itchy, scaling rashes and discolouration around | Skin Biopsy for histopathology: acute type shows intercellular edema (spongiosis) and intraepidermal vesicles, while in chronic eczema there is acanthosis and hyperkeratosis (thickened stratum malpighii and stratum corneum). | This involves use of:<br>**Emollients** – White soft paraffin, Glycerine or Organic oils & lotions<br>**Drying/ antiseptic solutions:** soaks or compresses e.g. Aluminum acetate, **Potassium permanganate** and Normal saline Solution.<br>Nutrition: Ensure balanced diet and adequate hydration<br><br>**Topical corticosteroids:** Moderate potency in cream or Ointment form with or without local antibiotic cream eg **Betamethasone & Clobetasole.**<br>Systemic treatment: involves the use of several agents including;<br>**Antihistamines –** Cetrizine, Terfenadine, Promethazine and Chlorpheniramine<br>Topical or systemic immune modulators & Suppressants eg Tacrolimus or Pimecrolimus, Ciclosporin A, Azathioprine & Methotrexate<br>**Broad-spectrum antibiotics** – if there evidence of bacterial infections (Oral or Injectable).<br>**Phototherapy (UVB)**- short band for short periods<br><br>Patients are given health education on the disease and things he/she should use or avoid like wearing of cotton clothes and hand gloves, avoidance of stress and dry skin |

| | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | **v) Seborrhoeic Eczema**- occurs in two distinct forms; infantile and adult.<br>Adult type affects hairy areas (Dandruf) while infantile on the nappy area.<br><br>**vi) Contact dermatitis –** this has 2 forms; Primary irritant and Allergic contact. May be occupational in origin | ankles, on the shin. Varicose veins and ulcers occur.<br><br>Patient develops greasy yellowish scales usually on the scalp, diaper area and intertriginous folds e.g. groins, neck and retro-auricular areas. Facial skin, eyebrows and eyelashes may be involved. A yeast, Pityrosporon ovale plays an important role in the pathogenesis.<br><br>Rashes develop according to the site of exposure. Is caused by various allergens eg for primary – soaps and detergents, **rubber** in shoes and gloves and **nickel** in neck laces, earings and watch straps for 2nd type. | | **Follow up**, and if no improvement refer to dermatologist |
| 2. | **PSORIASIS** | | | |
| | A common, genetic, inflammatory and proliferative skin disease that presents with silvery and loosely adherent scales<br>The common types are:<br>Plaque type, Nummular, Guttate, Pustular and Erythrodermic | Affects both males and females & has injury, hormones, weather, metabolic disease and drugs as the main precipitating factors | **Skin biopsy:** for histopathology shows-Parakeratosis; retention of nuclei in horny layer.<br>- Thinning or absence of granular cell layer.<br>- Epidermal hyperplasia, elongated and clubbed rete ridges.<br>- Epidermal micro abscesses in the stratum corneum. | **I) General measures:**<br>- Rest, mild sedation, anxiety and depression mnx.<br>- Take care of general, physical and psychological health.<br>- Reassure and give emotional support stressing the non-<br>**II) Specific topical therapy:** used in mild and localized cases<br>**Tar preparations: Crude coal tar 2-5%,** avoid on the face, genitalia, and flexures or in pustular and erythrodermic psoriasis.<br>**Gockerman Technique: Crude tar bath + UVL.**<br>**Anthralin (dithranol): 0.1 up to 0.5% Ointment;**<br>**Mild corticosteroids:** use especially for the face, neck, flexures and genitalia.<br>**Salicylic acid: 3-5% ointment** as keratolytic agent.<br>**Vitamin D3: Calcipotriol** and **Dithranol** Ointment or cream.<br>**For severe & generalized forms, use: Phototherapy:** |
| | **i) Plaque type -**<br>Is commonest type and forms plaques that are raised from thickened skin<br><br>**ii) Nummular psoriasis -** | **Erythematous papules** covered with silvery, dry, loosely attached scales.<br>The **papules** may coalesce forming **well-defined plaques**<br>Lesions are usually bilateral, symmetrical on extensor surfaces of upper and lower limbs, elbows, knees, lumbosacral, scalp, flexures, nails, glans and tongue<br><br>Is characterized by round lesions, spots or coins, hence the term "nummular". Where these plaques appear on the body but, some areas are more common than others, like the limbs. | | |

| | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | **iii) Guttate** – Affects children and young adults<br><br>**iv) Pustular psoriasis**<br>Is one of the severe types and patients require admission<br><br>**v) Erythrodermic psoriasis -** Involves more than 90% of body surface area.<br><br>**vi) Other forms** – those affecting joints, scalp and nails | A type of psoriasis that appears as red, scaly, small, teardrop-shaped spots. It doesn't normally leave a scar.<br>Causes the formation of sterile pustules (no organisms, only PMNs). It may be generalized or localized to hands and feet.<br><br>It starts on top of plaque psoriasis after the withdrawal of systemic or very potent topical corticosteroid therapy<br><br>**Psoriasis involving joints;** either the distal interphalangeal joints or less commonly large joints.<br>**Scalp psoriasis:** forms thick scaling lesions on the scalp, with coronal pattern (corona psoriatica).<br>**Nail psoriasis:** has changes eg colour change, lifting of nail plate (sub-ungual keratosis), ridging & pitting | | UVB alone may be given thrice weekly<br>UVB narrow band (311 nm).-<br>**Topical retinoids** – Isotretinoin Ointment/Gel<br>**Laser therapy:** eg Excimer 308 nm or dye laser.<br>III) **Systemic therapy** – is for severe and extensive psoriasis vulgaris, generalized pustular, erythrodermic or **Methotrexate:** a Folic acid antagonist- Dose: 0.2-0.4 mg / kg / week.<br>Also give **Health education and Counseling** for Psycho-social support.<br>**Regular Follow up** & **Refer** severe, unresponsive or worsening cases to dermatologist |
| 3. | **PITYRIASIS ROSEA** | | | |
| | Is a self-limiting skin condition that presents as discrete scaly papules and plaques along the Langer lines (cleavage lines) over the trunk and limbs | The classic feature is a Herald (mother) patch on the trunk in up to 90% of cases. The patch is erythematous with slightly elevated scaling borders and a lighter depressed center, measuring 3 cm or more in diameter and associated with generalized smaller lesions<br>(secondary eruption), presents on the trunk along the Langer lines of the skin usually on the torso, upper arms, thighs or neck. | Skin biopsy for histology usually shows focal parakeratosis, spongiosis, and acanthosis in the epidermis, and extravasated red blood cells with perivascular infiltrates of lymphocytes, monocytes, and eosinophils in the dermis.<br><br>Differential diagnosis includes secondary syphilis, seborrheic dermatitis, nummular eczema, pityriasis lichenoides chronica, tinea corporis, viral exanthems, lichen planus | Topical medications - **Calamine lotion or zinc oxide gel** are used.<br>**Mild topical corticosteroids** eg Hydrocortisone and Betamethasone are applied twice daily for several weeks<br>**Antihistamines – Cetrizine or Terfenadine** for allergy and reducing itching.<br>**Taking baths** using lukewarm water or soaking in bathing oils.<br>**Drugs, such as Azithromycin and Acyclovir (Valtrex, Zovirax)** are used for severe cases |
| 4. | **LICHEN PLANUS** | | | |
| | LP is a chronic, inflammatory, autoimmune disease that affects the skin, oral mucosa, genital mucosa, scalp, and nails.<br><br>The different forms – | Lichen planus lesions are described using the six P's (planar [flat-topped], purple, polygonal, pruritic, papules, plaques).<br>Onset is acute, affecting the flexor surfaces of the wrists, forearms, and legs. | | **High-potency topical corticosteroids** are first-line therapy for all forms of lichen planus, cutaneous, genital, and mucosal erosive lesions.<br>In addition to **clobetasol, topical tacrolimus** appears to be an effective treatment |

| | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | i) **Linear lichen planus** manifests as aggregated linear lesions on the limbs that may develop the Koebner phenomenon on areas of injury.<br><br>**ii) Annular lichen planus**<br><br>**iii) Bullous lichen planus** on the buttocks | The lesions are often covered by lacy, reticular, white lines known as **Wickham striae.**<br>Lesions occur on the skin, mucous membranes (oral) and on other extra-oral sites eg the vulva and vagina, involvement of the nails, scalp, esophagus, or eyes though less common.<br>There are 4 forms of oral lichen planus: reticular, atrophic, bullous, and erosive. | | **Systemic corticosteroids** given for severe, widespread lichen planus involving oral, cutaneous, or genital sites.<br>**Referral to a dermatologist** for systemic therapy with acitrecin or an oral immunosuppressant should be considered. |
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**B. Bacterial Skin Infections**

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| **5.** | **BACTERIAL INFECTIONS (SUPERFICIAL)** | | | |
| | Bacterial skin infections often begin as small, red bumps that increase in size. Some bacterial infections are mild and easily treated with topical antibiotics, but other infections require an oral antibiotic. Different types of bacterial skin infections include:<br>**Superficial** –<br>**Boils, carbuncles, Ecthyma**, and<br><br>**Folliculitis**<br>**Causes**<br>**1. Staph. aureus**<br>**2. Pseudomonas spp.** – responsible for outbreaks<br>**3. Other organisms** - include; Klebsiella spp., Enterobacter spp. and Proteus ssp.<br>4. **Malassezia (folliculitis)** -is caused by a dermatophyte | A boil, also called a furuncle, begins as a painful infection of a single hair follicle. Boils can grow to become larger (3-5 cm) and they commonly occur on the buttocks, face, neck, armpits and groin while, a carbuncle is a deeper skin infection that involves a group of infected hair follicles. (Staph. aureus & Strept. Group A and Epidermidis)<br>Carbuncles are found on the back of the neck, shoulders, hips and thighs, and they are especially common in middle-aged or elderly men or people with diabetes or HIV infection.<br><br>Is the inflammation of hair follicles due to an infection, injury or irritation.<br>It is more common in children, whilst folliculitis of the beard area is more common in adult males. Histologically, there is presence of inflammatory cells within the wall and ostia of the hair follicle. | The common tests done include; Blood for CBC, Acute phase proteins and Pus swab/aspirate for Gram stain and culture.<br><br>**The tests are similar;**<br><br>CBC, Acute phase proteins and<br>Pus swab/aspirate for Gram stain and culture | **Incision and drainage:** is done for large boils or carbuncles and the wound is cleaned and dressed<br>**Analgesics:** depending on severity. Paracetamol, Ibuprofen, Meloxicam or Tramadol for short periods<br>**Antibiotics: for** severe or recurrent infections.<br>The commonly used are Cloxacillin, Ampiclox, Cephalexin and Erythromycin or Azithromycin<br>NB: If there is any underlying condition then treat it as well.<br><br>For folliculitis, simple cases will improve with good hygiene (Chlorhexidine) **but for severe ones** use systemic antibiotics as above or Topical or antifungal drugs – Clindamycin, Mupirocin, Fusidic acid plus hair removal creams |
| **6.** | **ACNE** | | | |
| | It is an inflammatory condition involving pilo-sebaceous structures and presents with multiple papules, cysts or nodules. It is caused by infection by a bacterium, Propioni bacterium acnes. Occurs worldwide in all races, sexes and is estimated to affect 5-15% of general population | The common types include:<br>• Acne vulgaris – non inflammed<br>• Inflammed<br>• Excoriated<br>• Cystic<br>• Nodulo-cystic<br>• Acne conglobata<br>• Infantile type<br>**Predisposing factors:** Genetics, diet – rich in low density fats, | Differential diagnoses include; Folliculitis,<br>Rosacea ie due to over steroid use, Drug rash & Pityriasis rubra pilaris<br><br>Investigations done are: CBC plus ESR<br>Pus swab for microscopy<br>Serum antibody levels<br>Skin biopsy for histology | Topical agents are:<br>**Keratolytic agents** eg Salicylic acid, Benzoyl peroxide, Adapaline creams or gel<br>**Vitamin A analogues** eg. Acitracin, Tretinoin<br>**Antibiotics** – Doxycyline, Minocycline, Clindamycin, Erythromycin, etc for 1-3 months<br>Sulphur drugs are used for those not fit for sotretinoin<br>Intralesional orticosteroids eg Triamcinolne , Dexamethasone, etc |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | | hormonal activity – adolescence & menses or pregnancy, Type of skin, stress and hot weather or sunshine<br>**Grade 1**- is the mildest with little inflammation, papules (comedos) on the face, mid upper chest, upper back & on whole trunk. Blackheads and milia occur. **Grade 2** – has papules, pustules and some nodules. **Grades 3 & 4** – are severe forms with nodules, cysts, scars and even keloids | | **Phototherapy** – use of short-wave UV light<br>**Minor surgery** – physical extraction and laser therapy |
| | | | | |
| **7.** | **BACTERIAL (DEEP)** | | | |
| | The deep infections include:<br>**i) Cellulitis -** is a common bacterial infection of the dermal and subcutaneous tissue. | Due to Group A Streptococcus (pyogenes and Staphylococcus aureus. Others are from Haemophilus influenza & pneumococcus.<br>The risk factors include: - gravitational eczema, leg ulcers, lymphoedema, tinea, trauma and<br>‣ Previous episodes of cellulitis<br>‣ Obesity<br>‣ Immuno-compromise<br>The usual signs and symptoms are:<br>‣ Redness of the skin<br>‣ Swelling of the skin<br>‣ Tenderness<br>‣ Warm skin<br>‣ Pain<br>‣ Bruising<br>‣Blisters | • Useful tests include blood cultures, complete blood cell (CBC) with differential, and levels of creatinine, bicarbonate, creatine phosphokinase, and C-reactive protein (CRP) and Antinuclear antibodies. | General supportive:<br>• Rest the area.<br>• Elevate the area to ease swelling and discomfort.<br>• **Use analgesics** like acetaminophen or ibuprofen<br>• Specific:<br>• **Antibiotics** eg Ceftriaxone, Cephalexin, Cloxacillin, etc<br>• **Antibiotic therapy** will also depend on culture results<br>• **Surgery** – that involves draining of an abscess or pus |
| | | | | |
| | **ii) Erysipelas** | | | |
| | Is a skin infection which affects only affects the upper layers of the skin. However, it can overlap with cellulitis that involves deeper layers of the skin. | The condition is usually acute in onset, following cuts to the skin, ulcers, or bed sores, insect or animal bites and wounds from surgery<br>The skin is usually affected in a particular area and can occur with the following features:<br>• Swollen and shiny<br>• Redness<br>• Warm and tender to the touch<br>• Blisters in severe cases | The investigations are similar to those for Cellulitis:<br>**Blood** -CBC, C-reactive Proteins and **Radiological (X-rays, CT Scans & MRI)** in case of trauma or osteomyelitis | **Analgesics** like acetaminophen or ibuprofen<br>**Specific:**<br>**Antibiotics** eg Ceftriaxone, Cephalexin, Cloxacillin, etc<br>• Antibiotic therapy will also depend on culture results<br>• **Surgery** – may be needed and involves draining of an abscess |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | | • Sharp edges between the affected area and unaffected skin<br>• Red streaks above the affected area<br>• It can turn purple or black in severe cases | | |
| | **iii) Necrotizing Fasciitis** | | | |
| | Is a rare but potentially fatal infection involving the subcutaneous tissue and fascia and rapidly progressive.<br>It is also known **as flesh-eating disease**. Deaths from NF can be sudden and sensational. | The causative organisms include;<br>• Aeromonas hydrophila<br>• Clostridium ssp<br>• E. coli<br>• Klebsiella ssp<br>• Staphylococcus aureus<br><br>A **painful, red swelling** which, becomes larger over hours. Pain becomes worse unless treatment is started in time.<br><br>There may **be discharge** from the infected area, or it may become discolored as it decays. Blisters, bumps, black dots, or other skin lesions might appear.<br>**Other symptoms** of necrotizing fasciitis include: fatigue, weakness, fever with chills and sweating. Nausea, vomiting,<br>Dizziness and infrequent urination | **Blood tests** can show evidence of damaged muscles and subcutaneous tissue.<br>**A skin biopsy** – may be useful.<br>**Radiological exams** eg CT Scans and MRI scans | **Use strong IV antibiotics**: Ceftriaxone, Metronidazole, etc<br>**Potent analgesics** – Tramadol and NSAIDs are given to control pain<br>**Intravenous immunoglobulin** (IVIG) is currently recommended<br><br>**Prompt Surgical Treatment:**<br>Involves performing debridement of necrotic tissue. |

**C. Fungal and Parastic Skin Infections**

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| **8.** | **FUNGAL INFECTIONS (SUPERFICIAL)** | | | |
| | Fungal skin infections are grouped as either superficial or deep (including the systemic infections) The superficial infections are called Dermatophytosis (Trichophyton, Microsporum, and Epidermophyton). | The infections are benign and involve the skin, scalp and nails. Candida albicans usually cause mucosal infections plus the skin<br>Commonest types are: Tinea capitis, T. facie, T. corporis, T. cruris, T, manuum, T. unguium (also called onychomycosis) & T. pedis (Athletes foot).<br><br>Clinical presentation is similar except on nails and scalp. | The usual investigations done include;<br>Woods light examination for P. versicolor. KOH Test, Skin scrapings or nail clippings for culture | Treatment modalities include oral and topical agents. Good personal hygiene is an important adjunct to antifungal therapy. Decision regarding the appropriate therapy in a given patient takes into account the extent and location of the infection, the benefits and risks.<br>A **large variety of topical treatments** are used including: Clortrimazole, Miconazole, Selenium sulfide, Tolnaftate, haloprogin, and sodium thiosulfate.<br>**For extensive infections, oral or systemic drugs** are used - Griseofulvin, Ketoconazole, Itraconazole and Terbinafine. |
| **9.** | **FUNGAL INFECTIONS (DEEP)** | | | |
| | **i) Candidiasis**<br><br>**ii) Aspergillosis**<br><br>**iii) Cryptococcosis** | Presents with:<br>• Single or widespread lesions<br>• Small red papules or larger nodules<br>• Purpuric lesions can resemble ecthyma gangrenosum or purpura fulminans.<br><br>• Few or many lesions<br>• May result in rapidly spreading red patch with a necrotic centre (blackened dead tissue)<br>• May resemble pyoderma gangrenosum.<br><br>• Most often a skin rash is the first sign of infection | Diagnosis is made by history, careful physical examination and investigations including Skin biopsy and skin scrapings for fungal cultures. | For treatment, a number of antifungal agents are now available for treating deep fungal infections, including:<br>Amphotericin B in conventional and liposomal formulations, and<br>the Triazoles eg Itraconazole (Sporanox) and Fluconazole (Diflucan) given for 4-6 weeks.<br><br>**Surgical procedures** – may be done for extensive lesions or damage to skin |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | | • Extremely varied appearance<br>• Papules, abscesses, plaques, blisters, sinuses, ulcers, cellulitis or purpura (bleeding into the skin). | | |
| | **iv) Blastomycosis**<br>**v) Histoplasmosis** | • Papules, nodules, warty lesions<br>• Pustules, abscesses, ulcers and scars<br>• May cause oral lesions. | | |
| | **vi) Mycetoma (Eumycetoma)** | • Scaly (psoriasis-like) or soft papules, nodules, abscesses, sinuses or ulcers<br>• May cause mouth ulcers<br>• May cause erythema multiforme, erythema nodosum or toxic erythema.<br>• Causes masses that are painless but may extend to the bone and cause destruction to tissues. | | |
| **10.** | **PARASITIC SKIN INFESTATIONS** | | | |
| | Epidermal parasitic skin diseases (EPSD) are a heterogeneous category of infectious diseases confined to the upper layer of the skin.<br>The six major EPSD are scabies, tungiasis and pediculosis or liceThe typical primary lesions are multiple papules called burrows accompanied with itching, worse at night (capitis, corporis and pubis),<br><br>i) Scabies<br>a) Usual scabies | Scabies is a cutaneous parasitosis due to the presence of a mite, Sarcoptes scabiei hominis within the epidermis. It exists in two forms: ordinary scabies, relatively benign and moderately contagious; and crusted scabies, favoured by immune deficiency.<br><br>The primary lesions are multiple papules called **burrows** accompanied with itching, worse at night.<br><br>Lesions are often seen in the interdigital spaces of the hand and flexor aspect of the wrist, areolae, buttocks, elbows, axillae. The back and the face are spared.<br><br>Secondary skin lesions: resulting from scratching (excoriations, crusts) or super-infection<br><br>Has thick, scaly, erythematous plaques, generalized or localized, with or without itching | The only useful test is to squeeze material from a burrow and take for direct microscopy or staining.<br><br>Skin biopsy may be taken to help rule out other conditions | All clothing and beddings (including that of contacts) are changed after each treatment. They are washed with hot water at ≥ 60 °C then dried in the sun, or exposed to sunlight for 72 hours, or sealed in a plastic bag for 72 hours.<br><br>Topical scabicides namely, **Benzyl benzoate emulsion** is applied OD for 3 days, and patient is advised not to wash on 2nd day but continues to apply, or 1% **Gammabenzene hexachloride (Lindane)** lotion is applied over the entire body only once.<br><br>Treatment of secondary bacterial infection, if present, should be done. |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | b) Crusted (Norwegian) scabies | (50% of cases). Delay in diagnosis may lead to a scabies epidemic. | | |
| | ii) Jiggers (Tungiasis) | | | |
| | Tungiasis is an infestation by the sandfly flea Tunga penetrans or related species. The flea has many other names like jigger, sand flea or chigoe. | It inflicts misery upon tens of millions of people, mostly children and the elderly.<br>The female **sand flea burrows** into the skin and sucks the host's blood before producing eggs and causes intense itching and pain.<br>The feet are the most involved but other parts can be affected.<br>**Bullous-type lesions** leads to Fissures, ulcers, lymphangitis, lymphoedema, ascending neuritis, deformation and loss of nails, and tissue necrosis. People get difficulties in walking. | | The standard treatment is surgical extraction of burrowed sand fleas, which is usually done by the patients themselves or a caregiver.<br>**Surgical extraction** - is only be performed in an appropriately equipped health facility or by an experienced health worker using sterile instruments.<br>**Topical drugs –** Permethrin, Metrifonate, Thiabendazole and ivermectin have been with varying success.<br>Prevention includes – improving hygiene and always wearing of shoes. |
| | **iii) Lice (Pediculosis)** | | | |
| | Is a benign contagious parasitic infection due to 3 species of lice specific to humans: head lice, body lice and pubic (Crab) lice. | Transmission from person to person occurs through direct or indirect contact.<br>Body lice are potential vectors of relapsing fever, typhus (Eruptive rickettsioses), and trench fever.<br>– Head lice mainly affect children: itching and scratch marks (nape of neck and around the ears), which may become secondarily infected (impetigo) in prolonged infestation; presence of live lice and/or live (shiny, grey) nits attached to the hair shaft within 5 mm of the scalp.<br>– **Body lice** mainly affect mostly refugees, prisoners & the homeless: itching and scratch marks (back, belt line and armpits), often inflammed and infected; presence of lice and nits in the clothing.<br>– **Pubic lice (also called, Crab lice)** are considered to be sexually transmitted: itching and scratch marks and lice and nits are at the base of the hair shaft.<br>**-Head lice:** affect the scalp but nits are on hairs causing itching and scaling. | Investigations for lice **are not necessary** but one can use a special light, **called a Wood's light examination**, to check for nits. This special light makes the nits be seen as **pale blue**. | **Apply 4% Dimeticone to scalp and dry hair**, paying particular attention to the areas behind the ears and around the nape of the neck **for children 6 months and over and adults:** leave on hair for 8 hours, then rinse thoroughly. Repeat after 7 days.<br><br>**Oral Ivermectin PO 200 micrograms/kg single dose** is an alternative.<br>Eradication of lice requires that **hairy areas are shaved clean** to remove nits. Decontaminate combs, headwear and bedding (wash using hot water ≥ 60 °C for 30 minutes, iron or dry in the sun or seal in a plastic bag for 2 weeks.<br>– Treat as all contacts with live lice and/or live nits. |
| **11.** | **VITAMIN DEFICIENCIES** | | | |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | Deficiencies of Vitamins A and B deficiency can wreak havoc on the skin, causing acne, rashes, dry and flaky skin, cracked lips, and wrinkles.<br>The common conditions are Pellagra and Flacky paint dermatitis. | **i) Pellagra** is caused by **deficiency of niacin, also known as vitamin B-3.** It is marked by dementia, diarrhea, and dermatitis, also known as **"the three Ds".** If left untreated, pellagra can be fatal.<br><br>The main symptoms of pellagra are **3Ds (dermatitis, dementia, and diarrhoea).** This is because niacin deficiency is most noticeable in body parts with high rates of cell turnover, such as your skin or gastrointestinal tract.<br><br>**ii) Flacky Paint Dermatitis:** is common in Kwashiorkor resulting from inadequate protein or fat intake. It is characterized by **generalized edema with a "flaky paint" dermatosis.** Rice milk intake during infancy has been proved to cause kwashiorkor in the developing and under developed countries.<br>**Lesions are darkly pigmented patches form**, and these may peel or desquamate, rather like old, sun-baked blistered paint. T | Useful tests for Vitamin deficiencies include **Complete blood count & A vitamin panel blood test** checks the levels of these thirteen essential vitamins to identify any deficiencies and determine if supplements are needed. | Treatment consist of provision of **Vitamin supplements for 2-3 months, and a balanced diet rich in proteins,** minerals and vitamins<br><br>**Specific vitamins like B3, Vitamin B-complex or Multivitamins** may be used, |
| **12.** | **BENIGN PIGMENTARY DISORDERS** | | | |
| | Pigmentation disorders affect many people. Some of the most **benign** common are pigmented birthmarks, macular stains, hemangiomas, port wine stains, while disorders include albinism, melasma, vitiligo and pigmentation loss due to skin damage (Post-inflammatory). | **Birthmarks**<br>These appear at birth or just a few weeks after birth. While most birthmarks are non-cancerous, certain birthmarks can pose health risks.<br><br>**Pigmented birthmarks**<br>**Nevus of Ota -** is marked by bluish or grayish discoloration of the face and sometimes the white part of the eye (sclera). Patients with this type of birthmark are at a higher risk of developing a melanoma and glaucoma and needs examinations by a neurologist as well as an ophthalmologist.<br>**Mongolian spots**. These birthmarks appear bruised or bluish in color and usually develop on the back or buttocks of babies. They disappear by age 4 and does not need to be treated. | Not many investigations are available but genetic studies may be done, hormonal assays and skin biopsy for histopathology. | The following agents can be used to treat epidermal melanosis or hypomelanosis singly or, more effectively, in combination:<br>• **Hydroquinone cream 1-3%**<br>• **Topical retinoids eg Isotretinoin Cream 0.1 %**<br>• **Topical corticosteroids**<br>• **Glycolic acid and other fruit acids**<br>• **Azelaic acid**<br><br>Patients are given health education on the diseases and reassurance. |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | | **Café-au-lait spots (French for "coffee with milk").** These are light brown-to-dark brown flat spots with smooth or irregular borders.<br>About 10% can be associated with the genetic disorder **neurofibromatosis.** These birthmarks may be treated with a laser for cosmetic purposes<br><br>**Nevi (moles):** are flesh-colored to light-to-dark brown. They may be flat or raised. Some may change and become a skin cancer called a melanoma. For this reason, moles should be watched for bleeding, pain, itch, color, shape, symmetry, even borders, and size changes. | | |
| **13.** | **SERIOUS PIGMENTATION DISORDERS** | | | |
| | Pigmentary disorders typically indicate an increased amount of melanin, leading to darker color of the skin, called hypermelanosis or hyperpigmentation.<br>Notable examples are: Vitiligo, albinism, piebaldism and Melasma and Post-infmmatory. | **i) Vitiligo:**<br>In this condition, the body's immune system attacks melanocytes, causing pigment loss. **Other immune system diseases** associated with vitiligo include diabetes, pernicious anemia, thyroid disease, and Addison's disease.<br>The **white skin patches**, occur around the mouth and eyes, or on the back of the hands. In some people, these patches can appear **all over the body**.<br><br>**ii) Piebaldism**<br>This results from congenital absence of melanocytes in affected areas of the skin and hair (depigmentation) that may have spontaneous expansion and contraction<br>It is autosomal dominant mutation of the c-kit or SNA12 gene, rare<br>Pathophysiological changes that occur include;<br>1. Hypomelanosis or loss of colour on affected parts<br>2. Histologically, absence or markedly decreased melanocytes in affected areas<br><br>**iii)Post-inflammatory:** | Some of the investigations that are available include; genetic, hormonal assays and skin biopsy for histopathology. | For this category of disorders, in addition to the topical preparations, a wide variety of systemic drugs can be used including:<br>ablative procedures (e.g., **chemical peels, cryotherapy, intense pulsed light, lasers**)<br><br>**Skin grafting** using skin obtained from large and flat parts of the body with normal skin.<br>• **Minigrafts or pinch grafts:** most successful, and<br>• **Culture grafts:** not always cosmetically acceptable<br>Photo-chemical method using - PUVA: not always easily available. |

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| | | In this type, there is a loss of skin color after the skin heals from an injury like burns that damaged the melanocytes). | | |

**D. Dermatological Emergencies**

| S. NO | DISEASES | CLINICAL FEATURES | INVESTIGATIONS | TREATMENT |
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| 1. | **DERMATOLOGICAL EMERGENCIES** | | | |
| | Dermatological emergencies are caused by allergic reactions or infections (bacterial or viral). Rapid management may be required in the case of severe drug reactions, infections, allergic reactions and flares of inflammatory dermatoses.<br><br>The common emergency conditions include; Eczema herpeticum, Erythroderma, Stevens-Johnson syndrome, toxic epidermal necrolysis, Acuite drug reactions and Acute Skin Failure Syndrome. | Typical symptoms of SJS and TEN include:<br>• Prodromal flu-like/non-specific upper respiratory tract illness.<br>• A painful rash starting on the trunk which then spreads over several hours to days onto the face and limbs.<br>• Mouth ulcers or soreness.<br>• Painful or irritated eyes.<br>Other important things to enquire about in the history include:<br>• Drug history (e.g. newly commenced medication – symptoms can occur 5-28 days after starting the causative medication).<br>Other signs: macules which then progresses to blisters and eventually sheets of desquamation.<br>• **Positive Nikolsky's sign:** gentle rubbing of the skin causing desquamation or scaling.<br>• Ulceration, erythema and blistering in the oral cavity<br>• Conjunctivitis or corneal ulceration<br>**Classification of SJS SJS-TEN and TEN is that;**<br>SJS has <10% of body surface, SJS/TEN is between 10-30% and TEN is >30%. | **Viral and bacterial swabs** can be taken from the base of a new blister.<br>**Swabs for viral studies and Tzank smears:** show the presence of HSV type 1 or 2 would confirm the iagnosis.<br><br>**Other tests include;** daily arterial blood gas analysis, complete blood count, serum urea, electrolytes & creatinine, glucose, albumin, LFT, complete urine examination and chest x-rays are essential. Culture from skin lesions.<br><br>For SJS, TEN and EM skin biopsy is a must. | The mainstay of treatment is **oral antivirals (e.g. Aciclovir or Vancyclovir) and may be combined with topical Acyclovir cream.**<br>In serious cases, **intravenous Acyclovir** can be considered.<br>**Dose of Acyclovir** – 800mg x 5 per day for 5-7 days.<br>Treat secondary bacterial **Potent analgesics** like Tramadol and others are used.<br>**Specific therapy** depends on the underlying cause.<br>**Prompt initiation of appropriate treatment** on the lines of a 100% burns patient and excellent double barrier nursing care are the twin principles of management that can salvage many lives. Emollients are used in abundance and cool, wet dressings are the mainstays of treatment.<br>**Treat any underlying causes (**e.g. discontinuing suspected trigger medication).<br>**Patients should be managed in a warm room (~30°C)** and have their fluid balance, electrolytes and body temperature monitored closely. |

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| | | **Observe for complications** of SJS and TEN include:<br>1. Dehydration/hypovolaemic shock<br>2. Secondary infection of the skin or mucous membranes<br>3. Other infections<br>4. Disseminated intravascular coagulation<br>5. Thromboembolism, and<br>6. Death (mortality rates are approximately 10%). | | **Careful and close Observation**s of vital signs.<br>**Daily arterial blood gas analysis, complete blood count, blood urea, creatinine, glucose, electrolytes, albumin, LFTs, complete urine** analysis.<br>**Take chest x-rays, culture from skin lesions and venous line alternate day**s is desirable for antibiotic therapy. |

REPUBLIC OF KENYA

MINISTRY OF HEALTH