



THE UNITED REPUBLIC OF TANZANIA

**STANDARD TREATMENT GUIDELINES
AND ESSENTIAL MEDICINES LIST**

***MINISTRY OF HEALTH AND SOCIAL WELFARE
FOURTH EDITION
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FOREWORD

The Standard Treatment Guidelines (STG) and the National Essential Medicine List for Tanzania (NEMLIT) was first published in 1991. The fourth edition includes new sections on symptoms and syndrome. The STGs have been updated and are consistent with current national guidelines for diagnosis and management of common diseases. The guidelines also reflect changes in the management of various diseases including asthma and hypertension following recommendations from WHO and experts from international medical associations and agencies. There have been improvements in the format of treatment regimens, showing more clearly the classification of medicines by level of health care within the treatment guidelines, and not just in the NEMLIT.

The STG and NEMLIT aims at providing health practitioners with standardized guidance in making decisions about appropriate health care for specific conditions found in Tanzania. By using STGs, prescribing practices can be rationalized and patient outcomes can be improved while making optimum use of the limited resources for medicines. The NEMLIT attached to the STG retains its purpose of identifying medicines that are considered essential for the treatment of common disease conditions in Tanzania. The medicine list is in line with the World Health Organization (WHO) recommendations under Tanzania conditions. It follows the principles and concepts of essential medicines so as to simplify the management of medicines supply and support a streamlined logistics system.

This set of tools is meant to be a guide for quick reference and its recommendations are valid for most presentations of the conditions covered. Nevertheless, clinical judgment and experience will always prevail for adjustment of treatment in individual cases when necessary.

This new edition of STGs provides Medicines and Therapeutics Committees (MTCs) at our health institutions an opportunity to strengthen their role in improving therapeutics and management of medicines in practice. MTCs are requested to promote the concepts of evidence based selection of medicines and cost-effective treatment protocols and facilitate STGs to be applied in their specific practice settings, translating and incorporating into local guidelines, formularies and in-service training programmes.

The Ministry's policy is that all public and private health workers in Tanzania will promote and adhere to these Standard Treatment Guidelines, and that prescribing, purchasing, labeling and dispensing of medicines should be by generic names as much as possible, and consistent with the level classification in the STGs and NEMLIT.

It is my hope that all health workers in Tanzania will find this document a useful tool in management of patients' illnesses.

Hon. Dr. Hussein Mwinyi (MP)
MINISTER FOR HEALTH AND SOCIAL WELFARE

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HOW TO USE THE DOCUMENT

The guideline covers chapters of common diseases in Tanzania. Most chapters start with a title, a brief description of the topic, common clinical signs and symptoms of each disease, the diagnosis and differentials, investigations, treatments and supportive care. The document comprises the national Essential Medicines List (NEMLIT) which will be used in the public health facilities. The medicines will be used to treat the majority of public health problems and they should be available to health facilities at all time. The guideline also makes provision for referral of patients to higher health facilities.

The indices for all medicines used are found at the back of the guide book, together with the information on how to report the adverse drug reactions. All health care workers are encouraged to report suspected adverse drugs reactions (ADR) when the reaction is potentially serious or clinically significant. The guideline also, makes provision for referral of patients to higher health facilities see the referral form. The last pages of the guideline contain annexes, references as well as the Essential Medicines List.

It is important to remember that the recommended treatments provided in this document are evidence, clinically approved and are in consistent with the already existing WHO guidelines.

Comments that aim to improve these treatment guidelines will be appreciated all the time and the form for that purpose is appended.

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THE NATIONAL ESSENTIAL MEDICINES LIST (NEMLIT)Error! Bookmark not defined.	

ABBREVIATIONS AND SYMBOLS

AA	Alcoholic Anonymous
ACEI	Angiotensin Converting Enzyme Inhibitor
ACR	Albumin Creatinine Ratio
ACS	Acute Coronary Syndrome
AE	Acute Epiglottitis
AF	Atrial Fibrillation
AHF	Acute Heart Failure
AIDS	Acquired Immunodeficient Syndrome
ALU	Artemether Lumefantrine
ANUG	Acute Necrotizing Ulcerative Gingivitis
APH	Ante partum Haemorrhage
APTT	Activated Partial Thromboplastin
ARB	Angiotensin Receptor Blocker
ARDS	Acute Respiratory Distress Syndrome
ARF	Acute Renal Failure
ARI	Acute Respiratory Infections
ATS	Anti Tetanus Serum
BCC	Basal Cell
BCG	Bacillus Calmette – Guerin Vaccines
BG	Blood Glucose
BID	Two Times a Day
BL	Burkitt's Lymphoma
BP	Blood Pressure

CaCx	Carcinoma of the Cervix
CBT	Cognitive Behavioral Therapy
CCB	Calcium Channel Blocker
CCF	Congestive Cardiac Failure
CKD	Chronic Kidney Diseases
CNS	Central Nervous System
CO	Corneal Opacity
COCs	Combined Oral Contraceptives
CPT	Cotrimoxazole Preventive Therapy
CrCl	Creatinine Clearance
CS	Caesarian Section
CT	Computer Tomography
CVD	Cardiovascular Disease
CVP	Central Venous Pressure
CSF	Cerebral-Spinal Fluid
DBP	Diastolic Blood Pressure
DAHf	Decompensate Acute Heart Failure
DC	Direct Current
D&C	Dilation and Curettage
DIC	Disseminated Intravascular Coagulation
DDVAP	Desmopresin
DKA	Diabetes Ketoacidosis
DPM	Drops Per Minute
DRE	Digital Rectal Examination
DSM	Direct Smear Microscope

DT	Delirium Tremens
DTLC	District TB and Leprosy Coordinator
DVT	Deep Vein Thrombosis
EAU	Examination Under Anaesthesia
EBTR	External Beam Therapy
EBV	Epstein Barr Virus
ECG	Electro Cardiogram
ENL	Erythema Nodosum Leprosy
ENT	Ear Nose and Throat
ESR	Erythrocyte Sedimentation Rate
FBP	Full Blood Picture
FFP	Fresh Frozen Plasma
FBC	Full Blood Count
GAS	Group A beta haemolytic Streptococci
GFR	Glomerular Filtration Rate
GIT	Gastro Intestinal Tract
GDM	Gestational Diabetes Mellitus
GI	Gastro Intestinal
GS	Glomerular Diseases
HB	Haemoglobin
HD	Hodgkin Disease
HDCV	Human Diploid Cell Vaccines
HF	Heart Failure
HIV	Human Immunodeficiency Virus
HSV	Herpes Simplex Virus

ICD	International Classification of Disease
ICT	Intracavity
ICU	Intensive Care Unit
IDDM	Insulin Dependent Diabetes Mellitus
IE	Infective Endocarditis
IHD	Ischemic Heart Diseases
I.M/i.m	Intramuscular
INR	International Normalized Ratio
ITP	Idiopathic Thrombocytopenic Purpura
I.V/i.v	Intravenous
IVU	Intravenous urography
IUFD	Intrauterine fetal death
IUGR	Intra Uterine Growth Restriction
JNC	Joint National Committee
KS	Kaposi's Sarcoma
L/l	Litre
LBBS	Left Bundle Branch Block
LDL	Low Density Lipoprotein
MCV	Mean Corpuscular Volume
MDIY	Multiple Daily Insulin Therapy
MDR	Multiple Drug Resistance
MI	Myocardial Infarction
mmHg	Millimeters of Mercury

MRI	Magnetic Resonance Imaging
MU	Mega Unit
MTX	Methotrexate
NGT	Nasal Gastric Tube
NHL	Non Hodgkin’s Lymphoma
NIDDM	Non Insulin Dependent Diabetes Mellitus
NKHS	Non Ketotic Hyperosmolar State
NSCLC	Non Small Cell Lung Cancer
NSAID	Non Steroidal Anti- Inflammatory Drugs
NSTEMI	Non ST Elevation Myocardial Infraction
O/PO	per Oral
OGD	Oesophagoduodenoscopy
ORCI	Ocean Road Cancer Institute
ORS	Oral Rehydration Salts
PB	Paucibacillary
PCR	Protein Creatinine Ratio
PCWP	Pulmonary Capillary Wedge Pressure
PE	Pulmonary Embolism
PEEP	Positive End –Expiratory Pressure
PEM	Protein Energy Malnutrition
PHC	Primary Health Care
PID	Pelvic Inflammatory Disease
PIH	Pregnancy Induced Hypertension
PITC	Provider Initiated Testing and Counseling
POPs	Progesterone Only Pills

PPE	Papular Pruritic Eruption
PPH	Post Partum Hemorrhage
PPROM	Preterm Premature of Rapture of Membrane
PROM	Prolonged Premature Rapture of Membrane
PSA	Prostate Specific Antigen
PT	Prothrombin
PTT	Partial Thrombin Time
RV	Right Ventricle
RT	Radiotherapy
SBO	Systolic Blood Pressure
SC	Subcutaneous
SCC	Squamous Cell Carcinoma
SCLC	Small Cell Lung Cancer
SJS	Steven Johnson Syndrome
SLE	Systemic Lupus Erythematosus
SP	Sulfadoxine Pyrimethamine
SSS	Salt Sugar Solution
STD	Sexually Transmitted Diseases
STEMI	ST Elevation Myocardial Infraction
SVTs	Supraventricular Tachyarrhythmia's
TCC	Transitional Cell Carcinoma
TEN	Toxic Epidemial Necrolysis
T1DM	Type I Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
TIG	Tetanus Immunoglobulin

TSH	Thyroid Stimulating Hormone
TT	Tetanus Toxoid
UFH	Unfractionated Heparin
UTI	Urinary Track Infection
VF/VT	Ventricular Fibrillation/flutter/ Ventricular tachyarrhythmia's
VWD	Von Willebrand Disease
WFI	Water For Injection
WHO	World Health Organization
WPW	Wolff-Parkinson White
µg	Microgram

CHAPTER ONE

SYNDROMIC APPROACH

1.0 PAIN

Pain is the most common symptom of disease. It is an unpleasant sensation localized to a part of the body. It is often described in terms of a penetrating or tissue-destructive process (e.g., stabbing, burning, twisting, tearing, squeezing) and/or of a bodily or emotional reaction (e.g., terrifying, nauseating, sickening). Any pain of moderate or higher intensity is accompanied by anxiety and the urge to escape or terminate the feeling.

Diagnosis

Self-report is the key to pain assessment. In non- or pre verbal children, facial expression is the most valid indicator of pain; therefore use faces pain scale to assess severity. Pain should be assessed by:

- Duration
- Severity, e.g. does the patient wake up because of the pain
- Site
- Character, e.g. stabbing, throbbing, crushing, cramp like
- Persistent or intermittent
- Relieving or aggravating factors
- Accompanying symptoms
- Distribution of pain
- In children pain can be assessed by child's crying voice, posture, movement and colour.

1.1 Treatment for Acute and Mild pain

Aspirin, Paracetamol, and Non-steroidal Anti-Inflammatory Agents (NSAIDs); these drugs are considered together because they are used for similar problems and may have a similar mechanism of action.

Adult

A: Acetylsalicylic acid 600mg every 4 hours until pain subsides

OR

A: Paracetamol 500- 100mg every 6-8 hours until pain subsides.

Children

A: Paracetamol 15 mg/kg/dose 4–6 hourly when required to a maximum of 4 doses per 24 hours;

1.2 Treatment for Severe Pain

Opioids are the most potent pain-relieving drugs currently available. They have the broadest range of efficacy, providing the most reliable and effective method for rapid pain relief.

Adults : C: Tramadol tablets or injection 50-100mg every 6 hours or until pain is controlled.

OR

C: Morphine 10mg IV every 6 hours on a "when necessary" basis;
Children: 0.2mg/kg body weight IV every 6 hours.

For surgery and obstetric conditions

C: Pethidine 100mg IM/ IV every 6 hours when necessary.

CAUTION!! Opioids may cause respiratory depression; therefore use opioids carefully. In case of toxicity, reverse with the narcotic antagonist **naloxone**.

C: Naloxone 0.1-0.2mg IV intermittently. Max. dose 10mg

Do not administer morphine in:

- advanced liver disease
- severe head injury
- acute asthma
- advanced chronic obstructive bronchitis, emphysema or other
- respiratory disease with imminent respiratory failure
- untreated hypothyroidism

Use morphine **with extreme care** if there is:

- Recent or concurrent alcohol intake or other CNS depressants
- Hypovolaemia or shock
- In the elderly

Referral

Refer to Regional and Tertiary care for:

- All children with moderate and acute severe pain
- No response to oral pain control and unable to initiate opioids therapy
- Uncertain diagnosis
- Management of serious underlying conditions

Pain Associated with Trauma or Inflammation

See under Trauma and Injuries section

1.3 Treatment for Chronic Non Cancer Pain

Chronic pain is a pain that persist for more than 4 weeks chronic pain can arise from:

- Tissue damage (nociceptive pain), e.g. arthritis, fibromyalgia's, lower back pain, pleurisy, cancer pain etc

- Injury to nerves (neuropathic pain) e.g. post herpetic neuralgia (pain following shingles), trigeminal neuralgia, diabetic neuropathy, HIV related peripheral neuropathy, drug induced peripheral neuropathy or phantom limb
- Abnormal nerve activity following disease

Psychological evaluation and behaviorally based treatment paradigms are frequently helpful, particularly in the setting of a multidisciplinary pain-management center.

Drug Treatment

Mild Pain

Adult: A: Paracetamol 1000 mg (O) 6 hourly until pain subsides

Pain Associated with Trauma or Inflammation

See under Trauma and Injuries section

Moderate pain (Including neuropathy)

Adults: If still no relief to simple analgesics as above, **add**

C: Tramadol 50 mg (O) 4–6 hourly as a starting dose

May be increased to a maximum of 400 mg daily

Adjuvant therapy

Adults: In addition to analgesia as above **add** antidepressants;

C: Amitriptyline 25 mg (O) at night; Maximum dose: 75mg.

Anticonvulsants and Antiarrhythmics may also be helpful in neuropathic pain. Give Phenytoin or carbamazepine.

Referral

- Pain requiring strong opioids
- Pain requiring definitive treatment for the underlying disease
- All children

1.4 Chronic Cancer Pain

The long-term use of opioids is accepted for patients with pain due to malignant disease. Some degree of tolerance and physical dependence are likely with long-term use. Therefore, before embarking on opioid therapy, other options should be explored, and the limitations and risks of opioids should be explained to the patient (*For detailed information, refer to Malignant Disease chapter*).

2.0 HEADACHE

A Headache is defined as a pain in the head or upper neck. It is one of the most common locations of pain in the body and has many causes. There are three major categories of headaches:

- Primary headaches,
- Secondary headaches, and
- Cranial neuralgias, facial pain, and other headaches

Assessment of headache should be comprehensive for example to include

- Age at onset
- Presence or absence of aura and prodrome

- Frequency, intensity and duration of attack
- Number of headache days per month
- Quality, site, and radiation of pain
- Associated symptoms and abnormalities

2.1 Primary headache

Primary headaches include migraine, tension, and cluster headaches, as well as a variety of other less common types of headache

Migraine Headache

This is characterized by a trial of paroxysmal headache, vomiting and focal neurological events (usually visual). It is more common in females than in males often there is a family history of migraine.

Associated precipitants include:-

- Dietary (cheese, chocolate or red wine)
- Psychological stress

General Measures

- Avoidance of precipitants
- Relaxation to reduce stress

Medicines

In acute attack give analgesics:

A: Paracetamol 1g immediately then every 4 hours; Max 4g per day

OR

A: Aspirin 600mg, repeat after 4 hours if needed.

Plus

C: Metoclopramide oral/IM, 10 mg 3 times daily.

In severe attack give:

C: Ergotamine tartrate 1-2 mg, maximum 4mg in 24hours, not to be repeated at intervals less than 4 days.

For prevention purposes give:

C: Propranolol 80-160mg daily

OR

C: Amitryptiline 10-50mg at night.

Referral

- Patient with additional neurological signs or additional risk factors for an alternate diagnosis, such as immune deficiency. These patients require brain imaging
- Sudden onset of a first severe headache may indicate serious organic pathology, such as subarachnoid hemorrhage
- Acute migraine, not responding to treatment
- Recurrent migraine not controlled with prophylactic therapy

Tension headaches

While tension headaches are the most frequently occurring type of headache, the cause is most likely contraction of the muscles that cover the skull. When the muscles covering the skull are stressed, they may spasm and cause pain. Common sites include the base of the skull, the

temple and the forehead. Tension headaches occur because of physical or emotional stress placed on the body.

Diagnosis

- The pain begins in the back of the head and upper neck and is described as a band-like tightness or pressure.
- Often is described as pressure encircling the head with the most intense pressure over the eyebrows.
- The pain usually is mild (not disabling) and bilateral (affecting both sides of the head).
- The pain is not associated with an aura (see below), nausea, vomiting, or sensitivity to light and sound.
- The pain occurs sporadically (infrequently and without a pattern) but can occur frequently and even daily in some people.
- The pain allows most people to function normally, despite the headache.

Note:

- The key to making the diagnosis of any headache is the history given by the patient
- If the health care practitioner finds an abnormality, then the diagnosis of tension headache would not be considered until the potential for other types of headaches have been investigated.

Treatment

Tension headaches are painful, and patients may be upset that the diagnosis is "only" a tension headache. Even though it is not life-threatening, a tension headache can affect the activities of daily life. The following work well for most people:

A: Aspirin (300-900mg (O) every 4-6 hrs max 4g daily)

OR

A: Ibuprofen (1.2-1.8g daily in 3-4 divided doses preferably after food max dose 2.4g daily, maintenance dose of 0.6-1.2g daily may be adequate.)

OR

A: Paracetamol 1g (O) 8hrly

OR

D: Naproxen 0.5-1g in 1-2 divided daily doses

Massage, and stress management can all be used as adjuncts to tension headaches.

- When pain medications are used for a prolonged period of time, headaches can recur as the effects of the medication wear off. Thus, the headache becomes a symptom of the withdrawal of medication (rebound headache).

Cluster headaches

Cluster headaches are headaches that come in groups (clusters) lasting weeks or months, separated by pain-free periods of months or years. The cause of cluster headaches is uncertain. Some evidence shows that brain scans performed on patients who are in the midst of a cluster headache, shows abnormal activity in the hypothalamus. Cluster headaches:

- May tend to run in families and this suggests that there may be a genetic role
- May be triggered by changes in sleep patterns
- May be triggered by medications (for example, nitroglycerin)

If an individual is in a susceptible period for cluster headache, cigarette smoking, alcohol, and some foods (for example, chocolate) also can be potential causes for headache.

Diagnosis

- Pain typically occurs once or twice daily and last for 30 to 90 minutes
- Attacks tend to occur at about the same time every day
- The pain typically is excruciating and located around or behind one eye. The affected eye may become red, inflamed, and watery

Note: Cluster headaches are much more common in men than women.

Treatment

C: Sumatriptan 6mg; Dose may be repeated after 1 hour. Max dose 12mg a day

OR

C: 100% Oxygen at the rate of 10-15L/min for 10-20 minutes

Prevention of the next cluster headache may include the following:

C: Verapamil 240-960mg (O) 8 -12 hourly divided doses

OR

C: Amitryptiline 25-50 mg (O) daily

Prevention cluster headaches

Since cluster headache episodes may be spaced years apart, and since the first headache of a new cluster episode can't be predicted, daily medication may not be warranted.

Lifestyle changes may help minimize the risk of a cluster headache flare. Stopping smoking and minimizing alcohol may prevent future episodes of cluster headache.

2.2 Secondary headache

Secondary headaches are due to an underlying disease or injury that needs to be diagnosed and treated. Early diagnosis and treatment is essential if damage is to be limited

Examples of Secondary headache:

- **Head and neck trauma**
- **Blood vessel problems in the head and neck**
 1. Stroke or transient ischemic attack (TIA)
 2. Arteriovenous malformations (AVM) may cause headache before they leak
 3. Carotid artery inflammation
 4. Temporal arteritis (inflammation of the temporal artery)
- **Non-blood vessel problems of the brain**

1. Brain tumors, either primary, or metastatic
2. Seizures
3. Idiopathic intracranial hypertension, once named pseudo tumor cerebri,

- **Medications and drugs (including withdrawal from those drugs)**

Infection

1. Malaria
2. Meningitis
3. Encephalitis
4. HIV/AIDS
5. Systemic infections

Diagnosis

- If there is time, the diagnosis of secondary headache begins with a complete patient history followed by a physical examination and laboratory and radiology tests as appropriate
- However, some patients present in crisis with a decreased level of consciousness or unstable vital signs. In these situations, the health care practitioner may decide to treat a specific cause without waiting for tests to confirm the diagnosis

3.0 FEVER

Fever known also as **pyrexia** is a common medical sign of many conditions; characterized by an elevation of temperature above the normal range of 36.5-37.5°C

Diagnosis/Symptoms

Fever is usually accompanied by sickness behavior such as:

- Depression
- Lethargy
- Anorexia
- Sleepiness
- Hyperalgesia
- Inability to concentrate
- Other symptoms include: feeling cold, increased muscle tone and shivering

Treatment guidelines

Give antipyretic medicines: Paracetamol, Ibuprofen or Aspirin (*For dosage, look under pain section above*)

Hyperpyrexia

It is a fever with an extreme elevation of body temperature greater than 41.5° c. Infections are the most common cause of fevers, however as the temperature rises other causes become more general.

Note: Hyperpyrexia is considered a medical emergency as it may indicate a serious underlying conditions.

Management

- Keep the patients adequately hydrated as the most significant risk of complications is dehydration
- If the temperature reads extremely high, aggressive cooling is required

Treatment

- The antipyretic ibuprofen is effective in reducing fever in children
- Ibuprofen and paracetamol may be used together in children

CAUTION!! Aspirin is not recommended in children and young adult, under 16 years due to risk of Reye's syndrome.

4.0 COUGH

Clinical features: Cough is a symptom produced by inflammatory viscid secretions or obstruction of the tracheobronchial system. It may be dry or productive sputum. Cough may be paroxysmal, hacking, explosive, and harsh (brassy).

Treatment

Causative/precipitating factors e.g. CCF, asthma; allergies must be established and treated accordingly. Where causative/precipitating factors cannot be detected, the following treatments may be offered:

For Non-productive irritating cough

A: Cough syrup/Linctus (O) 5-10 ml every 6 hours

Expectorants may be used to liquefy viscid secretions.

A: Cough expectorants (O) 5-10 ml every 6 hours

Note: *Antibiotics should never be used routinely in the treatment of cough*

5.0 CONVULSION

A convulsion is an episode of neurologic dysfunction caused by abnormal neuronal activity that results in sudden change in behavior, sensory perception, or motor activity. For a patient with new onset convulsion the list of possible causes is longer and consists of the following:

- CNS pathologies (stroke, neoplasm, trauma, hypoxia, vascular abnormality)
- Metabolic abnormalities (hypoglycemia/hyperglycemia, hyponatremia/hyponatremia, hypercalcemia, hepatic encephalopathy)
- Toxicological etiologies (alcohol withdrawal, cocaine, isoniazid, theophylline)
- Infectious etiologies (meningitis, encephalitis, brain abscess, neurocysticercosis and malaria)

Approach to a patient:

- Ask for history of epilepsy, if yes; compliance to anticonvulsant
- History of CNS pathology (stroke, neoplasm, recent surgery)
- History of systemic neoplasms, infections, metabolic disorders, or toxic ingestions
- Recent trauma or fall

- Alcohol abuse

Special concerns:

- Eclampsia
- Trauma
- Intracranial hemorrhage
- Alcohol or medication withdrawal (barbiturate, diazepam)
- Drug induced seizures(tricyclic antidepressant and isoniazid overdose)

Laboratory studies:

Clinical information should guide the specific workup of a patient. Some investigations must be ordered:

- Serum glucose level
- Serum electrolyte
- Pregnancy test for women of child bearing age.
- CT scan is indicated as outpatient/inpatient depending on progress of patient after episode of seizure.

For a patient who had previously history of seizure do **CT scan** brain if;

- New focal deficits
- Trauma
- Persistent fever
- New character or pattern to the seizure

ECG should be considered in some patients. Seizure event can be precipitated by cerebral hypoperfusion due to arrhythmia, ECG may identify the following

- Prolonged QTc
- Widened QRS
- Prominent R in aVR
- Heart block

Consider Lumbar Puncture in;

- Immunocompromised
- Persistent fever
- Severe headache
- Persistently altered mental status

Treatment and management

Neurological dysfunction is theorized to occur after 20mn of continuous seizure, so aggressive treatment of any seizure should be done in 5 min. always consider the underlying cause until proved otherwise.

- A, B, C (airway, breathing, circulation)
- Benzodiazepines

A: Diazepam 10-20mg IV at a rate of 0.5ml (2.5mg) per 30 sec. Repeat if necessary after 30-60min. May be followed by intravenous infusion to max. 3mg/kg over 24 hours, per rectum 500mcgrms/kg up to max of 30g)

OR

B: Phenobarbitone 20mg/kg 8 hourly. Max. dose 1.5g

OR

D: Phenytoin 18mg/kg IV stat then 100mg 8 hourly O/IV

6.0 SHOCK

Shock is a life threatening condition characterized by hypotension. If not treated immediately it leads to death.

Diagnosis

- Low blood pressure (systolic BP below 80 mmHg) is the key sign of shock
- Weak and rapid pulse
- Rapid and shallow breathe
- Restlessness and altered mental state
- Weakness
- Low urine output

Note

Signs and symptoms of shock in children must be recognized while still in the compensated state to avoid irreversible deterioration. Therefore, the following are primarily assessed in children:

- Prolonged capillary filling (more than 3 seconds)
- Decreased pulse volume (weak thread pulse)
- Increased heart rate (>160/minute in infants, > 120 in children)
- Decreased level of consciousness (poor eye contact)
- Rapid breathing
- Decreased blood pressure and decreased urine output are late signs and while they can be monitored the above signs are more sensitive in detecting shock before irreversible.

Table 2: Types of Shock

Type of Shock	Explanation	Additional symptoms
Hypovolemic	Most common type of shock Primary cause is loss of fluid from circulation due to haemorrhage, burns, diarrhoea etc.	Weak thread pulse, cold and clammy skin.
Cardiogenic shock	Caused by the failure of heart to pump effectively e.g. in myocardial infraction, cardiac failure etc.	Distended neck veins, weak or absent pulses
Septic shock	Caused by an overwhelming infection, leading to vasodilatation.	Elevated body temperature
Neurogenic shock	Caused by trauma to the spinal cord, resulting in sudden decrease in peripheral vascular resistance and hypotension.	Warm and dry skin
Anaphylactic shock	Caused by severe allergic reaction to an allergen, or drug.	Bronchospasm, angioedema and/or urticaria

I. Emergency treatment

Treatment depends on the type of shock. Intravenous fluid therapy is important in the treatment of all types of shock except for cardiogenic shock. Prompt diagnosis of underlying cause is essential to ensure optimal treatment.

- Maintain open airway
- Administer oxygen with face mask and if needed after intubation with assisted ventilation
- Check for and manage hypoglycemia

II. Fluid replacement (Not for Cardiogenic shock)

Adults:

A: 0.9% Sodium chloride given as the 1L bolus infusion. Repeat bolus until blood pressure is improved.

Transfuse blood and plasma expanders (-) in hemorrhagic shock.

Children:

A: 0.9% Sodium chloride 20 ml/kg as a slow infusion.

Note

- Do not administer IV fluids in case of Cardiogenic shock but maintain IV access
- If patient develops respiratory distress, discontinue fluids
- Septicemia in children: All children with shock which is not obviously due to trauma or simple watery diarrhea should receive antibiotic cover for probable septicemia.

B: Ampicillin 20mg/kg/dose 6 hourly for 7-10 days

OR

C: Ceftriaxone, IM, 50–80 mg/kg/dose immediately as a single dose.

Table 3: Instructions on Mixing Injection with Water

Weight kg	Dose mg	Use one of the following injections mixed with water for injection (WFI)			Age/Months/year
		250mg	500mg WFI 2ml	1000mg WFI 3.5ml	
≥2 – 2.5 kg	125 mg	1 ml	0.5 ml	-	
≥2.5 – 3.5 kg	200 mg	1.6 ml	0.8 ml	-	Birth – 1 month
≥3.5 – 5.5 kg	250 mg	2 ml	1 ml	-	≥ 1 - 3 months
≥5-7 kg	375 mg	3 ml	1.5 ml	-	≥3 - 6 months
≥7-9 kg	500 mg	4 ml	2 ml	-	≥6 – 12 months

≥ 9-11 kg	625 mg	5 ml	2.5 ml	-	≥12 – 18 months
≥11-14 kg	750 mg	6 ml	3 ml	-	≥18 months – 3years
≥14-17.5 kg	1000 mg	-	4 ml	3.5 ml	≥3 - 5 years
>17.5 kg and above	1000 mg	-	4 ml	3.5 ml	5 years and above

! CAUTION!

- Do not administer fluids containing calcium , e.g. Ringer-lactate, within 48 hours of administering ceftriaxone
- Contra-indicated in neonatal jaundice
- Annotate dose and route of administration on referral letter.

7.0 DEHYDRATION

It is defined as the excessive loss of body fluid. There are three types of dehydration: hypotonic or hyponatremic (primarily a loss of electrolytes, sodium in particular), hypertonic or hypernatremic (primarily a loss of water), and isotonic or isonatremic (equal loss of water and electrolytes). In humans, the most commonly seen type of dehydration by far is isotonic (isonatraemic) dehydration which effectively equates with Hypovolemic, but the distinction of isotonic from hypotonic or hypertonic dehydration may be important when treating people who become dehydrated. Physiologically, dehydration, despite the name, does not simply mean loss of water, as water and solutes (mainly sodium) are usually lost in roughly equal quantities to how they exist in blood plasma. In hypotonic dehydration, intravascular water shifts to the extra vascular space, exaggerating intravascular volume depletion for a given amount of total body water loss. Neurological complications can occur in hypotonic and hypertonic states. The former can lead to seizures, while the latter can lead to osmotic cerebral edema upon rapid rehydration.

Hypovolemic

Hypovolemic is specifically a decrease in volume of blood plasma. It defines water deficiency only in terms of volume rather than specifically water.

Signs and symptoms

Symptoms may include headaches similar to what is experienced during a hangover, a sudden episode of visual snow, and dizziness or fainting when standing up due to orthostatic hypotension. Untreated dehydration generally results in delirium, unconsciousness, swelling of the tongue and, in extreme cases, death.

Thirst, dryness of mucous membrane, loss of skin turgor, orthostatic hypotension or tachycardia, reduced jugular venous pressure (JVP) or central venous pressure (CVP) and decreased urine output. In the presence of normal renal function dehydration is associated usually with a urine output of less than 0.5ml kg/hr.

Differential diagnosis

In humans, dehydration can be caused by a wide range of diseases and states that impair water homeostasis in the body. These include:

- External or stress-related causes
 - Prolonged physical activity with sweating without consuming adequate water, especially in a hot and/or dry environment
 - Prolonged exposure to dry air, e.g., in high-flying airplanes (5%–12% relative humidity)
 - Blood loss or hypotension due to physical trauma
 - Diarrhea
 - Hyperthermia
 - Shock (hypovolemic)
 - Vomiting
 - Burns
 - Lacrimation
 - Use of methamphetamine, amphetamine, caffeine and other stimulants
 - Excessive consumption of alcoholic beverages
- Infectious diseases (Refer to gastrointestinal chapter for details)
 - Cholera
 - Gastroenteritis
 - Shigellosis
 - Yellow fever
- Malnutrition
 - Electrolyte disturbance
 - Hypernatremia (also caused by dehydration)
 - Hyponatremia, especially from restricted salt diets
 - Fasting
 - Recent rapid weight loss may reflect progressive depletion of fluid volume (the loss of 1 L of fluid results in a weight loss of 1 kg (2.2 lb)).
 - Patient refusal of nutrition and hydration
 - Inability to swallow (obstruction of the esophagus)

Other causes of obligate water loss

- Severe hyperglycemia, especially in diabetes mellitus
 - Glycosuria
 - Uremia
- Diabetes insipidus
- Acute emergency dehydration event
- Food borne illness

Tests include:

- Blood chemistries (to check electrolytes, especially sodium, potassium, and bicarbonate levels)
- Blood urea nitrogen (BUN)

- Complete blood count (CBC)
- Creatinine
- Urine specific gravity

Other tests may be done to determine the cause of the dehydration (for example, blood sugar level to check for diabetes).

Treatment

For some dehydration oral fluid is the most effective to replenish fluid deficit.

In more severe cases, correction of fluid deficit is best by intravenous therapy. Solutions used for intravenous rehydration must be isotonic or hypotonic.

For severe cases of dehydration where fainting, unconsciousness, or other severely inhibiting symptom is present (the patient is incapable of standing or thinking clearly), emergency attention is required. Fluids containing a proper balance of replacement electrolytes are given intravenously with continuing assessment of electrolyte status.

8.0 HYPOGLYCEMIA

Hypoglycemia is a condition of lower than normal level of blood glucose.

Criteria referred to as Whipple's triad are used to determine a diagnosis of hypoglycemia:

1. Symptoms known to be caused by hypoglycemia
2. Low glucose at the time the symptoms occur
3. Reversal or improvement of symptoms or problems when the glucose is restored to normal

Symptoms of hypoglycemia usually do not occur until the blood sugar is in the level of 2.8 to 3.0 mmol/L (50 to 54 mg/dl). The precise level of glucose considered low enough to define hypoglycemia is dependent on (1) the measurement method, (2) the age of the person, (3) presence or absence of effects, and (4) the purpose of the definition.

Signs and symptoms

Hypoglycemic symptoms and manifestations can be divided into those produced by the counter regulatory hormones (epinephrine/adrenaline and glucagon) triggered by the falling glucose, and the neuroglycopenic effects produced by the reduced brain sugar.

Adrenergic manifestations

- Shakiness, anxiety, nervousness
- Palpitations, tachycardia
- Sweating, feeling of warmth (although sweat glands have muscarinic receptors, thus "adrenergic manifestations" is not entirely accurate)
- Pallor, coldness, clamminess
- Dilated pupils (mydriasis)

- Feeling of numbness "pins and needles" (paresthesia)

Glucagon manifestations

- Hunger, borborygmus
- Nausea, vomiting, abdominal discomfort
- Headache

Neuroglycopenic manifestations

- Abnormal mentation, impaired judgment
- Personality change, emotional lability
- Fatigue, weakness, apathy, lethargy, daydreaming, sleep
- Confusion, amnesia, dizziness, delirium
- Stupor, coma, abnormal breathing
- Generalized or focal seizures

Causes

The circumstances of hypoglycemia provide most of the clues to diagnosis. Circumstances include the age of the patient, time of day, time since last meal, previous episodes, nutritional status, physical and mental development, drugs or toxins (especially insulin or other diabetes drugs), diseases of other organ systems, family history, and response to treatment. When hypoglycemia occurs repeatedly, a record or "diary" of the spells over several months, noting the circumstances of each spell (time of day, relation to last meal, nature of last meal, response to carbohydrate, and so forth) may be useful in recognizing the nature and cause of the hypoglycemia.

Glucose requirements above 10 mg/kg/minute in infants, or 6 mg/kg/minute in children and adults are strong evidence for hyperinsulinism. In this context this is referred to as the *glucose infusion rate* (GIR).

Finally, the blood glucose response to glucagon given when the glucose is low can also help distinguish among various types of hypoglycemia. A rise of blood glucose by more than 30 mg/dl (1.70mmol/l) suggests insulin excess as the probable cause of the hypoglycemia.

For patients who have recurrent hypoglycemia's the following tests might be needed depending on the history and physical examination: insulin, cortisol, and electrolytes, with C-peptide and drug screen for adults and growth hormone in children.

Treatment

Management of hypoglycemia involves immediately raising the blood sugar to normal, determining the cause, and taking measures to hopefully prevent future episodes.

The blood glucose can be raised to normal within minutes by taking 10-20 grams of carbohydrate. It can be taken as food or drink if the person is conscious and able to swallow. This amount of carbohydrate is contained in about 100-120 ml of orange juice or non-diet soda. Starch is quickly digested to glucose (unless the person is taking acarbose), but adding fat or protein retards digestion. Symptoms should begin to improve within 5 minutes, though full

recovery may take 10–20 minutes. Overfeeding does not speed recovery and if the person has diabetes will simply produce hyperglycemia afterwards.

If unconscious or for other reasons can not feed orally secure an IV line and give intravenous dextrose, concentrations varying depending on age (infants are given 2 ml/kg dextrose 10%, children are given dextrose 25%, and adults are given dextrose 50%). Care must be taken in giving these solutions because they can be very necrotic if the IV is infiltrated. If an IV cannot be established, the patient can be given 1 to 2 milligrams of glucagon in an intramuscular injection.

One situation where starch may be less effective than glucose or sucrose is when a person is taking acarbose. Since acarbose and other alpha-glucosidase inhibitors prevents starch and other sugars from being broken down into monosaccharide's that can be absorbed by the body, patients taking these medications should consume monosaccharide-containing foods such as glucose powder, honey, or juice to reverse hypoglycemia.

(For other details, refer to Hypoglycemia, under Endocrine and Metabolic Disease conditions' chapter thirteen)

CHAPTER TWO

ORAL AND DENTAL CONDITION

Oral disease conditions are common and range from dental caries, periodontal conditions, dental abscess and other acute bacterial infections, viral infections, fungal infections, traumatic injuries and tumors. The lesions affecting the maxillofacial region (perioral, jaws and face) are also considered here but for a more detail a relevant text book or manual need to be referred. The clinician should be able to identify conditions requiring immediate attention by the dentist, do the preliminary urgent and life saving measures where possible before referring the patient to a centre with a dentist/dental surgeon. There are some cases which will need the attention of a specialist dental surgeon (like oral and maxillofacial surgeon, orthodontist e.t.c) but in most cases these will be identified by a general dentist.

1. 0 PERIODONTAL CONDITIONS

1.1 Gingivitis

Inflammatory changes in the gingival develop within a couple of days of undisturbed bacterial growth on the cervical portion of the tooth surface.

Diagnostic criteria:

- Inflammation of the gingival which is initially seen as discrete colour and texture changes of the marginal tissues.
- After few days of plaque accumulation overt gingivitis is established, characterized by gingival redness and swelling and increased tendency of the gingival to bleed on gentle probing, during tooth brushing or even on touch.

Prevention

Instructions for proper oral hygiene care

Treatment

Removal of accumulated plaque and oral hygiene instructions on tooth brushing and other adjuvant means of oral hygiene (dental flossing, use of mouth washes)

1.2 Periodontitis

This is the progression of the inflammation of gingivitis into the deep tissue affecting the periodontal membrane causing periodontal pockets, introduction of infection and destruction of periodontium. The damage of the periodontal membrane, periodontal ligaments and eventually alveolar bone leads to formation of pockets which eventually favours more bacterial growth. As the destruction continues the teeth become loose and may eventually fall out.

Diagnostic Criteria

- Reddened, swollen gingiva
- Easily bleeding gingival on gently probing
- Loose/mobile teeth
- Bad breath from the mouth

- Gingival recession
- Periodontal pocket

Investigation: Mainly X-ray (orthopantomogram (OPG)) to determine extent of bone loss

Prevention and Non Pharmacological Treatment

- Instruction and guidance to the patients on proper oral hygiene for proper plaque control
- Plaque control by the dentists by scaling and root planning (this may need several visits as may be found necessary)
- Advanced treatment – if refractory/resistant to treatment or patient has systemic diseases conditions.

Note: Patients with systemic diseases conditions like diabetes mellitus, liver and renal diseases, HIV/AIDS and those who are pregnant or heavy smokers of cigarette are generally at increased risk of periodontal diseases and their management may need referral to a periodontal specialist

Pharmacological treatment

- Mouth washes:
 - A:** Hydrogen peroxide 3% 3-4 times daily
 - OR**
 - A:** Chlorhexidine gluconate 0.2% 3-4 times daily
 - OR**
 - A:** Povidone iodine 0.5% used 3-4 times daily will argument the plaque control treatment.

Use antibiotics only for severe cases and those with evidence of periodontal abscess formation:

- A:** Metronidazole (O) 400mg 8 hourly for 5 days
- Plus**
- A:** Amoxicillin 500 mg 8 hourly for 5 days
- OR**
- A:** Tetracycline 500mg 8 hourly for 5 days.

Note: Tetracycline should not be given to pregnant and lactating mothers to avoid tetracycline stains in for their babies.

1.3 Acute Necrotizing Ulcerative Gingivitis (ANUG)

It is a severe form of gingivitis and it characterized by rapid destruction of gingival tissue, particularly in the area of the interdental papilla. Patients usually present with soreness and bleeding of the gums and foul test (fedor-ex ore). Acute Necrotizing Ulcerative Gingivitis (ANUG) is also called Vincent’s gingivitis or Vincent’s gingivostomatitis. It is common in malnourished children and immunocompromized individuals especially patients with diabetes and HIV/AIDS.

Diagnostic criteria

- Painful and easily bleeding gingival swelling and erythema of the gingival margins
- Yellowish-white ulceration of the gingival
- Fever, malaise and regional lymphadenitis
- In some patients (especially malnourished children), ANUG may presents with extensive destruction of the face and jaws in the severe form known as Cancrum Oris or noma

Treatment

- Professional cleaning with Hydrogen Peroxide 3% (under local anesthesia)

A: Metronidazole 400 mg (O) 8 hourly a day for five days

Plus

A: Amoxicillin 500mg (O) 6 hourly for 5 days

1.4 Stomatitis

This is generalized inflammation of the oral mucosal (including the gingiva) due to different aetiologies. Such aetiologies include infections, chemical burn, radiations. Contact stomatitis (a counterpart of contact dermatitis) also can occur due to allergy.

Diagnosis

Oral sores and ulceration

Treatment

Generally supportive

- Mouth rinse

A: Hydrogen peroxide solution 3% 4-6 hourly

OR

A: Povidone iodine 0.5% mouthwash

OR

C: Chlorhexidine 0.2% Topical oral gel: The best gel is one containing combination of analgesics, anaesthetics and antiseptics (e.g. Choline salicylate, Benzalkonium chloride and Lignocaine hydrochloride)

Note: Mouth washes should not be used at the same time with the gel.

Oral analgesics can be added;

A: Paracetamol 1000mg 8 hourly

OR

A: Diclofenac 50 mg 8 hourly

OR

A: Ibuprofen 400 mg 8 hourly

2.0 DENTAL CARIES

It is a condition whereby the tooth is demineralized by acid which is produced by bacteria in the process of metabolizing sugar. Start slowly with white spots later developing to black/brown spot and cavities in enamel, dentine and eventually the pulp. Dental caries is caused by bacteria of the dental plaque which feed on sugary food substrates producing acid as by-products which dissolve the minerals of the tooth surface. The bacteria which cause dental caries are mainly of streptococcus (*S.mutans*, *S. viridians*)

Diagnostic Criteria

- Early stage – asymptomatic
- Intermediate stage:- black/brown spot which may be visible on any surface of tooth
- Cavities developing on tooth surface
- Pain/toothache elicited by hot, cold or sweet foods/drinks
- Late stage: pain may be spontaneous, intermittent, sharp and severe, even interfering with sleep.
- There is tenderness on percussion of the tooth.
- X-Rays: Periapical x-ray of tooth/teeth may need to be done especially to confirm extent of caries for treatment decision e.g. the caries contained in the dentine can be distinguished from pulpal caries.

Note: The Susceptible sites are those areas where plaque accumulation can occur and be hidden to escape active and passive cleansing mechanisms e.g. pits and fissures of the posterior teeth, interproximal surfaces and teeth in malocclusion.

Prevention

- Proper instruction to avoid frequent use of sugary foods and drinks
- Use fluoridated toothpaste to brush teeth at least once a day

Non-pharmacological measures

- Early lesions presenting as a spot on enamel without cavitation and softening, observe and adhering to preventive measures.
- Lesion with cavitation but confined to dentine – filling/restoration of teeth with suitable filling materials (e.g. amalgam, composite, glass ionomer)
- Lesion involving the pulp (with or without periapical abscess), perform advanced tooth restoration by endodontic treatment wherever possible otherwise tooth extraction is done.

Note: For significantly abscessed tooth see dental abscess]

Pharmacological treatment

Analgesics: for toothache

A: Paracetamol 1000mg 8 hourly

OR

A: Diclofenac 50 mg 8 hourly

OR

A: Ibuprofen 400 mg 8 hourly

3.0 ODONTOGENIC AND NON-ODONTOGENIC OROFACIAL INFECTIONS

3.1 Periapical Abscess

The clinical presentation arises as a complication of inflammation of the dental pulp or periodontal pocket. The condition may be acute and diffuse or chronic with fistula or localized and circumscribed. It is located in the apical aspect of the supporting bone.

Diagnosis

- The patient complain tooth ache
- Pain during intake of hot or cold foods/drinks
- Pain on bringing the tooth on occlusion
- Tenderness on percussion (vertical percussion)
- Swelling of gingiva around the affected tooth

Treatment

- For posterior teeth: Extraction of the offending tooth under local anesthesia

Lignocaine 2% with adrenaline 1:80,000 IU (to establish drainage) is the treatment of choice followed by analgesics.

Adult: Paracetamol (O) 500mg – 1g, 4-6 hourly for 3 days, Child: Paracetamol (O) 10-15 mg/kg 4-6 hourly

- For anterior teeth (incisors, canine and premolars: Extraction is carried out only when root canal treatment is not possible. Give antibiotics:

Adult

A: Amoxicillin (O) 500mg, 8 hourly for 5-7 days;

Children, Amoxicillin (O) 25 mg/kg in 3 divided doses for 5 days.

Plus

A: Metronidazole (O); Adult 400mg 8 hourly for 5-7 days

Children 7-10 years, 100mg every 8 hour

Note: *Periodontal abscess is located in the coronal aspect of the supporting bone associated with a periodontal pocket.*

3.2 Infected Socket

A post extraction complication due to infection of the clot due to contamination (infected socket). The condition is painful and if not managed well could lead to osteomyelitis.

Diagnostic criteria

- Severe painful socket 2-4 days after tooth extraction
- Fever
- Necrotic blood clot in the socket
- Swollen gingiva around the socket
- Sometimes there may be lymphadenopathy and trismus (Inability to open the mouth)

Treatment

- Under local anesthesia with Lignocaine 2% socket debridement and irrigation with Hydrogen peroxide 3%. The procedure of irrigation is repeated the 2nd and 3rd day and where necessary can be extended to 4th day if pain persists. On follow-up visits local anesthesia is avoided unless necessary.
- Patient is instructed to rinse with warm saline (5ml spoonful salt in 200mls cup of warm water) or 3% hydrogen peroxide 3-4 times a day
- Antibiotics prescribed to prevent progression to osteomyelitis:
 - A:** Amoxicillin 500mg (O) 6 hourly for 5 days
 - Plus**
 - A:** Metronidazole 400mg 8 hourly for 5 days.
- *X-Ray:* Periapical X-ray of the socket may be necessary when there are poor progression apart from the above treatment, aim is to check where there are no root remnant, foreign body or any local bone pathology

Referral: is center with maxillofacial unit is considered in case of persistent pain and infection apart from treatment for more than two weeks

3.3 Dry Socket

It is a post extraction complication due to failure to form clot (dry socket). The condition is very painful and it differs from infected socket by lack of clot and its severity of pain.

Diagnosis

- Severe pain 2-4 days post-extraction
- Pain exacerbated by entry of air on the site
- Socket devoid of clot
- It is surrounded by inflamed gingiva

Treatment

Treatment is under local anesthesia with Lignocaine 2% socket debridement and irrigation of hydrogen peroxide 3%. The procedure of irrigation is repeated the 2nd and 3rd day and where necessary can be extended to 4th day if pain persists. On follow-up visits local anesthesia is avoided unless necessary.

3.4 Dental Abscess

Dental abscess is an acute lesion characterizes by localization of pus in the structures that surround the teeth. Dental abscess is a polymicrobial infection. Aerobic Gram positive cocci and anaerobic Gram negative rods predominate among others. The predominant species include; Bacteroides, Fusobacterium, Peptococcus, Peptostreptococcus and Streptococcus viridians.

Diagnosis

- Fever and chills
- Throbbing pain of the offending tooth
- Swelling of the gingiva and sounding tissues
- Pus discharge around the gingiva of affected tooth/teeth
- Trismus (Inability to open the mouth)
- Regional lymphnodes enlargement and tender
- Aspiration of pus for frank abscess

Investigations: Pus for Grams stain, culture and sensitivity and where necessary, perform full blood count.

Treatment

Preliminaries

- Determine the severity of the infection
- Evaluate the status of the patient's host defence mechanism
- Determine the need of referral to dentist/oral surgeon early enough

Non-pharmacological

- Incision and drainage and irrigation (irrigation and dressing is repeated daily)
- Irrigation is done with 3% hydrogen peroxide followed by rinse with normal saline.
- Supportive therapy carried out depending on the level of debilitation (most patients need rehydration and detoxification)

Pharmacology

Drug of choice:

A: Amoxicillin 500mg (O) 6 hourly for 5 days

Plus

A: Metronidazole 400 mg (O) 8 hourly for 5 days.

- Second choice/ severe case

C: Amoxicillin with Clavulanic acid 625mg (O) 12 hourly for 5 days

Plus

A: Metronidazole 400 mg (O) 8 hourly for 5 days.

If allergic to penicillin's:

A: Erythromycin 500 mg (O) 8 hourly for 5 days

Where parenteral administration of antibiotics is necessary (especially when the patient can not swallow and has life threatening infection, consider

C: Ampicillin 500mg IM/IV 6 hourly for 5 days

OR

C: Ceftriaxone 1 gm IV once daily for 5 days

Plus

C: Metronidazole 500 mg IV 8 hourly for 5 days

Note: Incision and drainage is mandatory in cases of deeper spaces involvement followed by a course of antibiotics. The practice of prescribing antibiotics to patients with abscess and denying referral for definitive care until pus has established or resolved has found to lead to more problems for orofacial infections **THEREFORE** early referral for definitive care is important.

Criteria for referral

- Rapidly progressive infection
- Difficulty in breathing
- Difficulty swallowing
- Fascia space involvement
- Elevated body temperature [greater than 39 °C]
- Severe jaw trismus/failure to open the mouth (less than 10mm)
- Toxic appearance
- Compromised host defenses

3.5 Ludwig's Angina

It is a serious life threatening generalized septic cellulitis of the fascia spaces found on the floor of the mouth and tongue. It is an extension of infection from mandibular molar teeth into the floor of the mouth covering the submandibular spaces bilaterally sublingual and submental spaces.

Diagnosis

- Brawny induration
- Tissues are swollen, board like and not pit and no fluctuance
- Respiratory distress
- Dysphagia
- Tissues may become gangrenous with a peculiar lifeless appearance on cutting
- Three fascia spaces are involved bilaterally (submandibular, submental and sublingual)

Treatment

Non-Pharmacological

- Quick assessment of airway

- Incision and drainage is done (even in absence of pus) to relieve the pressure and allow irrigation.
- Only when the airway distress is significant and there is evidence that it is not relieved by incision and drainage then tracheostomy is needed
- Supportive care include high protein diet and fluids for rehydration, detoxification and

Pharmacological

C: Ampicillin 500 mg IV 6 hourly for 5 days

Plus

C: Metronidazole 500mg IV 8 hourly for 5 days

If allergic to penicillin use

A: Erythromycin (O) 500 mg 6 hourly for 5 days

OR

C: Ceftriaxone 1 gm IV once a day for 5 days in case of severe infection

Once the patient is able to swallow the oral replace IV drugs.

Note: For this condition and other life threatening oral conditions consultation of available specialists (especially oral and maxillofacial surgeons) should go parallel with life saving measures.

3.6 Pericoronitis

Inflammation of the soft tissues covering the crown of erupting tooth and occurs more commonly in association with the mandibular third molar (wisdom) teeth. Impaction of food and plaque under the gingiva flap provide a medium for bacterial multiplication. Biting on the gum flap by opposing tooth causes laceration of the flap, increasing the infection and swelling. Then more likelihood of traumatic biting, this may lead to a vicious cycle. Involved bacteria are similar to those causing gingivitis and periodontitis.

Diagnosis

- High temperature,
- Severe malaise
- Discomfort in swallowing and chewing
- Well localized dull pain, swollen and tender gum flap
- Signs of partial tooth eruption or uneruption in the region
- Pus discharge beneath the flap may or may not be observed
- Foetor-ox oris bad smell
- Trismus
- Regional lymphnodes enlargement and tender

Treatment

A: Hydrogen peroxide solution 3% irrigation

If does not help, or from initial assessment the situation was found to require more than that then;

- Excision of the operculum/flap (flapectomy) is done under local anesthesia
- Extraction of the third molar associated with the condition
- Other means include: Grinding or extraction of the opposing tooth
- Use analgesics
- Consider use antibiotics especially when there are features infection like painful mouth opening and trismus, swelling, lymphadenopathy and fever.

Drug of choice

A: Amoxicillin 500mg (O) 6 hourly for 5 days

Plus

A: Metronidazole 400 mg (O) 8 hourly for 5 days

If severe (rarely) refer section 3.4 on treatment of dental abscess

3.7 Osteomyelitis of the Jaw

It is an inflammation of the medullary portion of the jaw bone which extends to involve the periosteum of the affected area. The infection becomes established in the bone ending up with pus formation in the medullary cavity or beneath the periosteum obstructs the blood supply. The infected bone becomes necrotic following ischemia.

Diagnosis

- In the initial stage there is no swelling. The patient has malaise and fever
- There is enlargement of regional lymphnodes.
- The teeth in the affected area become painful and loose, thus causing difficulty in chewing.
- Later as the bone undergoes necrosis the area becomes very painful and swollen.
- Pus ruptures through the periosteum into the muscular and subcutaneous fascia.
- Eventually it is discharged on to the skin surface through a sinus.

Investigation: X-ray – OPG (Orthopantomograph) or mandibular lateral oblique, water’s view for maxilla/midface. The x-ray will show sequestra formation in chronic stage. In early stage features seen in x-ray include widening of periodontal spaces, changes in bone trabeculation and areas of radiolucency. Perform culture and sensitivity of the pus to detect the specific bacteria.

Treatment

Non-pharmacological

- Incision and adequate drainage to confirmed pus accumulation which is accessible
- Culture should be taken to determine the sensitivity of the causative organisms

- Removal of the sequestrum is by surgical intervention (sequestrectomy) is done after the formation of sequestrum has been confirmed by X-ray.

Pharmacological

A: Amoxicillin or cloxacillin 500mg 6 hourly

Plus

A: Metronidazole 400mg gram 8 hourly before getting the culture and sensitivity then change according to results.

For details on antibiotics see section 3.4

- Antibiotic therapy may be continued for about 1-3 months.
- **Referral** is recommended to a zonal referral hospital for any case with long standing pus discharge and sinuses from the jaws

4.0 FUNGAL INFECTIONS

Oral Candidiasis (Thrush)

This is a fungal infection of the oral mucosa caused by *Candidal infection mainly Candida albicans*. *Candida albicans* is yeast and is a normal oral commensally. Under certain circumstances candida becomes pathogenic producing both acute and chronic infection. Acute oral candidiasis (Thrush) is seen most commonly in the malnourished, the severely ill, neonates and HIV-AIDS patients or patients on long term oral corticosteroids use. In chronic oral candidiasis dense white plaques of keratin are formed. Other risks for candidiasis is chronic diseases like diabetes mellitus, prolonged use of antibiotics and ill/poorly fitting dentures.

Diagnosis

Feature of candidiasis are divided according to the types

Pseudomembranous

- White creamy patches/plaque
- Cover any portion of mouth but more on tongue, palate and buccal mucosa
- Sometimes may present as erythematous type whereby bright erythematous mucosal lesions with only scattered white patches/plaques

Hyperplastic

White patches leukoplakia-like which is not easily rubbed-off.

Angular cheilitis (angular stomatitis)

- Soreness, erythema and fissuring at the angles of the mouth
- It is commonly associated with denture stomatitis but may represent a nutritional deficiency or it may be related to orofacial granulomatosis or HIV infection
- Investigation where available: For confirmation cytologic smear in solution of 20% potassium hydroxide for microscopy to see typical hyphae

Treatment

B: Nystatin (suspension) 100,000 IU (1 ml) mixture held in the mouth before swallowing, 4 times a day (after each feed).

OR

C: Miconazole (O) gel 25 mg/ml 5-10 mls in mouth –hold it before swallowing.

The treatment is continued for 5 days after cure/clearance

Where topical application has failed or candidal infection has been considered severe add

B: Fluconazole (O) 150mg once daily for 7-14days

OR

C: Ketoconazole (O) 400mg once daily for 7 days is reserved only for severe cases

Note: Candidiasis has several risk factors; it is recommended that for HIV/AIDS patients with candidiasis the ***HIV guidelines should be referred.***

5.0 VIRAL INFECTIONS

Herpes Simplex Virus

It is a viral infection commonly affecting the lips and perioral soft tissues presenting as papulovesicular lesions which ultimately ulcerate. The condition is recurrent following a primary herpes infection which occurs during childhood leaving herpes simplex viruses latent in the trigeminal ganglia. The primary infection affects mainly the gingiva and palate.

Diagnosis

- A prodrome of tingling, warmth or itching at the site usually precedes the recurrence
- About 12 hours later, redness appears followed by papules and then vesicles
- These vesicles then burst, weep, dry, scab and then heal
- The length of the cycle is variable (5-12 days mean time being 7 days)
- There are no investigation required unless patient has systemic diseases

Treatment

Non Pharmacological Treatment

- Adequate hydration
- Avoid salty and acidic drinks
- Cover lesions on the lips with Petroleum jelly and control any underlying cause

Pharmacological treatment

The disease is otherwise self-limiting condition but sometimes may need drug treatment

Herpes labial

B: Acyclovir Cream apply 4 hourly for 5 days

Herpes Stomatitis

B: Acyclovir 200mg 5 times in 24 hours for 5 days

In immunocompromised

B: Acyclovir 400mg 5 times in 24 hours for 5 days

Pain control by analgesics

A: Paracetamol 1000mg 8 hourly for 3 days

OR

A: Diclofenac 50 mg 8 hourly for 3 days

OR

A: Ibuprofen 400 mg 8 hourly for 3 days

For oral facial lesions of herpes zoster treat with

B: Acyclovir 400 - 800mg 5 times a day for 5 days.

Treatment may require analgesics, topical acyclovir (DOSE AS ABOVE)

6.0 APHTHOUS ULCERATION

Alphthous ulcers or recurrent alphthous stomatitis (RAS) are painful recurrent mucous membrane ulcerations. Usually affect the non-keratinized oral mucous membrane.

Diagnosis

There are 3 types of alphthous ulcers

Minor alphthous ulcers

- Small round or ovoid ulcers 2-4 mm in diameter.
- Surrounded by an erythematous halo and some edema
- Occur in groups of only a few ulcers (i.e., 1-6) at a time
- Found mainly on the non keratinized mobile mucosa of the lips, cheeks, floor of the mouth, sulci, or ventrum of the tongue
- Heal spontaneously in 7-10 days.
- Leave little or no evidence of scarring

Major Alphthous ulcers

Painful ulcers on non-keratinized oral mucous membrane, they are large 1-3 cm edged ulcers, and several may be present simultaneously. There is marked tissue destruction which is sometimes constantly present. Healing is prolonged often with scarring

Herpetiform ulcers

These occur in a group of multiple ulcers which are small (1-5 mm) and heal within 7-10 days
Rationale of treatment: To offer symptomatic treatment for pain, and discomfort, especially when ulcers are causing problems with eating

Treatment

A: Prednisolone 20 mg tid for 3 days then dose tapered to 10 mg tid for 2 days then 5 mg tid for other 2 days.

OR

S: Topical triamcinolone in base used twice daily

Plus

A: Paracetamol 1 gm 8 hourly for three days

IDEAL: Oral gel containing ant inflammatory agent preferably combined with analgesic and antiseptic.

Referral criteria: If the ulcers persist for more than 3 weeks apart from treatment, such lesion may need histological diagnosis after specialist opinion.

7.0 POST EXTRACTION BLEEDING

Commonly due to disturbing the blood clot by the patient through rinsing or inadequate compression on the gauze, though at times may be due to bony/tooth remnants.

Diagnosis

Bleeding socket can be primary (occurring within first 24 hours post extraction) or secondary occurring beyond 24 hours post extraction.

Primary bleeding socket

- Active bleeding from the socket
- The socket may or may not have blood clot
- Patient may be dehydrated and pale if has lost significant amount of blood
- Features of decreased pulse rate and volume, hypotension also if has lost significant amount of blood
- Examine well the socket may be having traumatic area of surrounding bone of the socket

Secondary bleeding socket may show features of infection or trauma

Treatment Guidelines

- After quick survey make sure the patient airway, breathing and circulation are restored if there were derangements
- Check Blood pressure and pulse rate and take quick history
- Give Local anesthesia (lignocaine 2% with adrenaline 1 in 80,000 IU)
- Clear any clot available and examine the socket to identify source of bleeding
- If the bleeding was from soft tissue (which is common) remove any foreign body like bone spicule if found, smoothen any sharp edges
- Suturing of the wound only when necessary (like significantly traumatize gingiva)
- Check and repack the socket with gauze.
- Give proper instructions to follow (bite on gauze pack for 30 minutes, not to rinse or eat hot foods on that day at least of 12 hours and avoid disturbance to the wound)

Packing can be done by material which stimulate blood clotting like oxidized cellulose (e.g. surgicel/gauze) or Thrombin containing gel foam sponges

Medication may be needed especially analgesics example [Paracetamol/diclofenac/ibuprofen] and

D: Tranexamic acid 500 mg (O/IV) 8 hourly for first 24 hours.

Intravenous fluid especially Normal saline 0.9% or Ringer's lactate in case of dehydration then followed by blood transfusion in case of hemoglobin below 7 g/dl in a patient who was otherwise healthy before tooth extract

Rule out bleeding disorders: if bleeding continued after 24 hours despite steps above, consult a hematologist or available physician for further management

8.0 TOOTH SENSITIVITIES

Usually is due to attrition of teeth, abrasion or gingival recession

Treatment

Self care: Tooth brushing with toothpaste for sensitive teeth.

Professional care:

C: Fluoride Gel application

9.0 TOOTH ERUPTION, SHEDDING AND EDENTULOUSNESS

9.1 Eruption of Teeth

Eruption of deciduous /primary teeth usually starts at five months of age. Symptoms associated with it like fever and diarrhea are normal and self limiting unless any other causes can be established. The following conditions usually are associated with tooth eruption and should be referred to dental personnel: eruption cysts, gingival cysts of the newborn and pre/natal teeth.

NOTE: There is nothing like "nylon teeth" what is a myth/believe existing in some traditions instead there are various above mentioned conditions associated with eruption of deciduous/primary teeth

9.2 Shedding of Deciduous/Primary (Milk) Teeth

Phenomenon of loosing of deciduous/primary teeth occurring between aged of 5-12 years is normal physiological changes. Deciduous/primary teeth should be left to fall out on themselves unless the teeth are carious or there is any other indication. Parents should be counseled accordingly and be instructed to assist their children to loosen the teeth the already mobile teeth and when there is no success or the permanent teeth are erupting in wrong direction should consult a dentist. Most of carious teeth will need management by a dentist. Early loss of primary teeth may lead to crowding of permanent teeth.

9.3 Edentulousness

It is the partial or full loss of natural teeth and subsequent resorption of the alveolar bone.

Treatment: It is by designing and constructing dental prosthesis according to aesthetic and functional needs. Materials to be used are many and include: alginate impression materials, calcium chloride powered, acrylic and porcelain, (refer NEMLIT for dental supplies)

10.0 MALOCCLUSIONS

Malocclusion is any variation in the arrangement of teeth leading to abnormal occlusion to the extent that may be functionally harmful or aesthetically objectionable.

Diagnosis

There are several forms of malocclusion

Class 1

The sagittal arch relationship is normal. The anterior buccal groove of the lower permanent molar should occlude with the anterior buccal cusp of the upper first permanent molar.

Class II

The lower arch is at least one half a cusp widths too far distal to the upper.

Class III

The lower arch is at least one half a cusp widths too far mesial to the upper.

Treatment

Rationale for treatment:

- Reduce possibility of temporomandibular joint pain dysfunction syndrome especially in case of cross bites
- Reduce risks of traumatic dental injuries especially in overjet
- Traumatic occlusion and gum diseases and caries especially in crowing
- Avoid psychosocial effects resulting from to lack of self esteem, self confidence personal outlook and sociocultural acceptability

Removable orthodontic appliances are those designed to be removed by the patient then replaced back. They are very useful in our local settings especially for mild to moderate malocclusion in teenagers.

Appliances for active tooth movement fall into two groups

- Simple removable appliances which have mechanical a component to move the teeth
- Myofunctional appliances, which harness the forces generated by the orofacial muscles.

Passive removable appliances may also save two functions:

- Retainers used to hold the teeth following active tooth movement
- Space maintainers, used to prevent space loss following the extraction of teeth.

Fixed orthodontic appliances (braces) are useful in malocclusion which have resulted in relapses of failure after use of removable appliances and moderate to severe malocclusion which can not be managed by removable appliances especially adult patients. Adolescents and adult patients requiring fixed appliances should be referred to an orthodontist.

Preventive orthodontic treatment by serial preventive extraction to create a space for anterior permanent teeth can be done by qualified dental personnel, if in he/she is in doubt it is recommended to consult dental specialist available.

11.0 TRAUMATIC DENTAL INJURIES

It may result to loosening, displacement and or loss of teeth, fracture of teeth and or bone, lacerations and bleeding. The commonest causes are falls (in sports and play) at home or school and motor accidents. Most affected are teeth upper incisors.

Table 1: Diagnosis

Type	Presentation
Tooth Concussion	Injury to supporting tissues of tooth, without displacement.
Subluxation	partial displacement, but is commonly used to describe loosening of a tooth without displacement
Luxation	Displacement of tooth (laterally, labially, or palatally).
Intrusion	Displacement of tooth into its socket. Often accompanied by fracture of alveolar bone
Avulsion	Complete loss of the tooth from the socket

Soft tissue injuries

Abrasion: does a friction between an object and the surface of the soft tissue cause a wound. This wound is usually superficial, denudes the epithelium, and occasionally involves deeper layer.

Contusion: is more commonly called a bruised and indicates that some amount of tissue disruption has occurred within the tissues, which resulted in subcutaneous or sub mucosal hemorrhage without a break in the soft tissue surface.

Laceration: is a tear in the epithelial and sub epithelial tissues. It is perhaps the most frequent type of soft tissue injury, is caused most commonly by a sharp object

Treatment

- Give tetanus toxoid (0.5% IU)
- Check for facial fractures and trauma to other sites, rule out evidence of head Injury (amnesia, loss of consciousness, neurological signs)
- Intra-oral examination: Look for soft-tissue lacerations, dentoalveolar fractures and damage to teeth.
- Check for tooth fragments which may be displaced in soft tissues
- Examine traumatized teeth for mobility and check mobility
- X-rays: (periapical x-ray) especially for suspected root fracture, and OPG x-ray for suspected alveolar bone fracture and jaw fracture
- Suture for any soft tissue wounds
- Wash mouth with warm saline solution of 3% hydrogen peroxide solution. Repeat mouth wash 3 times daily.
- Medication prescribed for elimination of pain; give analgesic (paracetamol or diclofenac or ibuprofen).
- Give prophylactic antibiotics if indicated. Antibiotic cover in cases of suspected contamination or extensive damage (Amoxicillin (oral) 500 mg 8hrly for 5 days).

- Efforts should be made to save the permanent tooth unless there is root fracture. Restoration of aesthetics (composite filling, prosthesis).
- Extraction is treatment of choice for significantly traumatized primary/deciduous teeth with mobility and or displacement. Judge the time which the tooth had to remain before expected exfoliation.

Refer to a dentist, where available orthodontics or endodontic specialist depending on the need of advanced treatment

Note: Referral to oral and maxillofacial surgeon is done to patients with complicated maxillofacial injuries.

Prevention

Proper design of playing grounds, observe road traffic rules, early orthodontic treatment

12.0 TUMOURS AND TUMOUR-LIKE CONDITIONS OF ORAL CAVITY AND FACIAL REGION

Benign Odontogenic Tumors

Ameloblastoma, Calcifying Odontogenic Tumors, Ameloblastic fibroma, Adenomatoid Tumors (Adeno Ameloblastoma), Calcifying Odontogenic Tumors, Ameloblastic Fibro-Odontoma, Odonto Ameloblastoma, Complex Odontoma, Compound Odontoma, Odontogenic Fibroma, Odontogenic myxoma, Cementoma and Cementifying Fibroma.

Non Odontogenic Benign tumors

Benign osteogenic tumors (arise from bone): Osteomas, Myxomas, Chondromas, Ewing's tumor, Central giant cell and Fibro-osteoma. Benign soft tissues non-Odontogenic tumors Papilloma, Fibroma, Fibrous Epulis, Peripheral Giant Cells, Pregnancy Tumors, Hemangioma, Lymphangioma, Lipoma and Pigmented nerves

Treatment: Tumors enucleation or excision in the treatment of choice depending on the type. Can be hemimandibulectomy, total mandibulectomy, hemimaxillectomy or total maxillectomy

Note: The tumors or oral and maxillofacial regions are of wide range and variable presentation, a dental surgeon is trained in identification and diagnosis. Treatment of most of these condition need expertise of oral and maxillofacial surgeon and patients should be referred early enough

Malignant soft and bone tumors

Squamous cell carcinoma, Sarcoma, Lymphosarcoma, Myosarcoma, Chondrosarcoma, Fibrosarcoma, Adenosarcoma, Adenocystic carcinoma and Epidermoid carcinoma.

Treatment

Palliative – but this depends on stage of the tumor: stage I and II surgical excision (squamous Cell carcinoma) with wide margin then curative radiotherapy. Others, surgical excision, radiotherapy followed by chemotherapy, if lesion is not advanced or in stage I and II.

Lymphomas

Burkitt's tumor is an undifferentiated lymphoblastic lymphoma. It shows close association and infection with the Epstein Barr virus. (For management refer to the CANCER/ONCLOGY SECTION

NOTE: Of emphasize is early detection and referral since Burkitt's lymphoma respond very quickly on chemotherapy

CHAPTER THREE

GASTRO INTESTINAL DISEASE CONDITION

1.0 INFECTIONS OF GASTROINTESTINAL TRACT

1.1 Amebiasis

Amebiasis is an infection caused by the protozoa organism *E histolytica*, which can cause colitis and other extra intestinal manifestations, including liver abscess (most common) and pleuropulmonary, cardiac, and cerebral dissemination. This can be through hematogenous spread as septic emboli from the gut wall or sub diaphragmatic abscess rupture into the pleural space or pericardium.

E histolytica is transmitted primarily through the fecal-oral route. Infective cysts can be found in fecally contaminated food and water supplies and contaminated hands of food handlers. Sexual transmission is possible, especially in the setting of oral-anal practices.

Diagnosis of Amebic colitis

- Gradual onset of bloody diarrhea
- Abdominal pain
- Fever
- Spanning several weeks' duration
- Rectal bleeding without diarrhea can occur, especially in children
- Fulminant or necrotizing colitis usually manifests as severe bloody diarrhea and diffuse abdominal pain with evidence of peritonitis and fever.

Treatment

Drug of choice

A: Metronidazole 400 – 800mg (O) 8hourly for 5- 10 days.

Children (below 10 years) 35 – 50mg/kg/d in 3 divided doses, indicatively: 1-3 years 100-200mg 8 hourly for 5 - 10 days ; 3-7 years 100-200mg 6 hourly for 5 -10 days; 7-10 years 200-400mg 8 hourly for 5 -10 days

Second choice

C: Tinidazole (O): Adult 2g daily as a single dose for 3 consecutive days. Children 60 mg/kg as a single dose for 3 consecutive days

OR

D: Secnidazole (O) Adult 2g single dose.

Children (below 12 years to 1 year) 30mg/kg as a single dose

Diagnosis of Amoebic liver abscess

- Fever, right upper quadrant pain, and tenderness of less than 10 days' duration.
- Sub acute presentation can be seen, with concomitant weight loss and anorexia.

- 60% to 70% of patients with amebic liver abscess do not have concomitant colitis, although a history of dysentery within the previous year may be obtained.

Treatment

Drug of choice:

A: Metronidazole ; Adult 400-800mg (O) 8 hourly for 10 days. Repeat course after 2 weeks if necessary.

Children: 1-3 years 100-200mg 8 hourly for 10 days; Children 3-7 years 100-200mg 6 hourly, for 10 days; Children, 7-10 years 200-400mg 8 hourly, for 10 days

Second Choice

C: Tinidazole (PO): Adult 2g daily single dose for 5 consecutive days. Children 50-75mg/kg single dose for 5 Consecutive days

NOTE:

- Metronidazole should be taken with food.
- Aspiration of the abscess may be necessary if there is evidence of impending rupture or a possibility of pyogenic abscess.

1.2 Giardiasis

It is the infection of the upper small intestine caused by the flagellate protozoan *Giardia Lamblia* (or *G. intestinalis*)

Diagnosis

- Infection is mainly asymptomatic
- However when symptoms occur, they include acute and/or chronic diarrhea, without blood or pus. In few cases malabsorption syndrome may occur
- Extra intestinal manifestations are rare and include allergic manifestations such as urticaria, erythema multiform, bronchospasm, reactive arthritis, and biliary tract disease

Investigation: Microscopic stool examination of *Giardia intestinalis* trophozoites or cysts of infected patient, sensitivity increases on serial 3 samples examination.

More specific tests include Stool antigen ELISA or Duodenal biopsy.

Treatment:

Drug of choice

A: Metronidazole (O): Adult and children over 10 years; 2g orally once daily for 3 days OR 400mg 8 hourly for 5 days.

Children below 10 years: 15mg/kg/day in 3 divided dosing for 5- 7days.

Indicatively: 1-3 years 500mg/day; 3-7 years 600-800 mg/day; 7-10 years 1g/day for 3 days.

Second choice

C: Tinidazole (PO): Adult 2g orally as a single dose during or after meal. Children 50-75mg/kg body weight as a single dose; Repeat once if necessary,

OR

C: Secnidazole (PO) Adult 2g as a single dose.

CAUTION

- Patients on Metronidazole, Secnidazole and Tinidazole should not be taken with alcohol
- Metronidazole, Secnidazole and Tinidazole should be avoided the first trimester of pregnancy.
- Reduce dosing to 50% in significant liver disease.

1.3 Ascariasis

It is an intestinal infection caused by *Ascaris lumbricoides*; *predominates* in areas of poor sanitation and is associated with malnutrition, iron-deficiency anemia, and impairments of growth and cognition.

Diagnosis

- Most patients are asymptomatic
- When symptoms occur, they are divided into 2 categories: early (larval migration) and late (mechanical effects)
- In the early phase (4-16 days after egg ingestion): Fever, Nonproductive cough, Dyspnea, Wheezing.
- In the late phase (6-8 weeks after egg ingestion): Passage of worms (from mouth, nares, anus); diffuse or epigastric abdominal pain, nausea, vomiting; pharyngeal globus, "tingling throat" frequent throat clearing, dry cough; complications - biliary and intestinal obstruction, appendicitis, pancreatitis and malnutrition.

Treatment

Drug of choice

A: Mebendazole (PO): Adult and Children above 2 years 100mg 12 hourly for 3 days **OR** 500mg as a single dose

OR

A: Albendazole 400mg (O) as a single dose

1.4 Ancylostomiasis

It is a hookworm disease caused by infestation of the small intestine with *Ancylostoma duodenale* or *Necator americanus*. It is one of the main causes of anaemia in the tropics which is also the major clinical feature.

Diagnosis

- The majority of patients are asymptomatic

- The major clinical manifestations are iron deficiency anemia and hypoalbuminaemia.

Treatment

Drug of choice

A: Mebendazole: Adult and Children over 2 years 100mg (O) 12 hourly for 3 days
Or 500mg as a single dose

OR

A: Albendazole 400mg (O) as a single dose

Note:

- Both Albendazole and Mebendazole must be chewed. If ova persist, give second course after 3 – 4 weeks.
- Iron replacement and nutritional supplementation (protein and vitamins) should be part of the management strategy.

CAUTION

Albendazole is contraindicated in the first trimester of pregnancy and children below 2 years

1.5 Strongyloidiasis

Intestinal infection caused by two species of the parasitic nematode *Strongyloides*. The most common and clinically important pathogenic species in humans is *S stercoralis*. Distinctive characteristic of this parasite is its ability to persist and replicate within a host for decades while producing minimal or no symptoms in individuals with an intact immune system and its potential to cause life-threatening infection (hyperinfection syndrome, disseminated strongyloidiasis) in an immunocompromised host associated with high mortality rates.

Diagnosis

- The symptoms related to strongyloidiasis may reflect the nematode's systemic passage, its local cutaneous involvement or both.
- During chronic uncomplicated infections, the larvae may migrate to the skin, where they can cause cutaneous strongyloidiasis, known as larva currens because of the quick migratory rate of the larva.
- The intestinal infection is usually asymptomatic but patients may have vague symptoms such as abdominal pain, nausea, flatulence, vomiting, diarrhea and even epigastric pain.
- In malnourished children, strongyloidiasis remains an important cause of chronic diarrhea, cachexia, and failure to thrive.
- Strongyloidiasis can lead to gastrointestinal (GI), pulmonary, dermatologic, neurologic, gram negative bacteremia and other complications especially in patients with hyperinfection.

Treatment

Drug of choice

C: Ivermectin (O): Adults and children over 5 years; 200mcg/kg daily for 2days and Up to 7-10 days for disseminated infection

OR

C: Thiabendazole (O): Adults: 25mg/kg body weight (max.1.5g) 12 hourly for 3 days. Children give the same dose same as for adults

Note: Tablets must be chewed

Alternatively

A: Albendazole: Adults 400mg (O) 12 hourly for 3 days, the medicines may be repeated after 3weeks. For disseminated infection give 7-10 days.

Children over 2 years give 15mg/kg/day in 2 divided doses for 3 days (7-10 days for disseminated infection)

Note:

- Provide antibiotic therapy directed toward enteric pathogens if bacteremia or meningitis is present or suspected
- Provide supportive treatment as indicated (eg, intravenous fluids if volume depletion, blood transfusion if gastrointestinal or alveolar hemorrhage, mechanical ventilation if respiratory failure)
- Symptomatic treatment should be initiated

- Pruritic dermatologic manifestations should be treated with antihistamines
- Inhaled beta-agonists may improve wheezing

1.6 Cestodiasis

Tapeworms disease is acquired from eating raw or undercooked beef infected with *Cysticercus bovis*, the larval stage of *Taenia saginata* (beef tapeworm) or undercooked food containing *Cystercercus cellulosae*, the larval stage of *Taenia solium* (pork tapeworm). Less commonly cestode includes *Diphyllobothrium latum* (poorly cooked fish) and *Hymenolepsis nana* (fecal oral contamination by both human and animals especially dogs).

Diagnosis

- Most tape worm infections are symptomless
- The commonest way of presentation is the appearance of proglottides or segments in the stool
- There may be mild epigastric discomfort, nausea, weight loss and diarrhea
- More specific features depend on the type of the parasite

Laboratory Diagnosis:

Macro and Microscopic stool examination for ova and parasites. It is indicated for some of the cestodes that release eggs or worm segments directly into the stool. Collecting 2-3 stool samples increases the sensitivity. Ultrasonography, CT, MRI, Stool antigen, ELISA tests are valuable in detecting and confirming other forms (i.e. Cystercercosis, Echinococcosis).

Treatment

For *Taenia solium*, *Taenia saginata* and *Diphyllobothrium latum*

Drug of choice

Adults and children over 6 years:

C: Niclosamide 2g (PO) as a single dose after a light breakfast, followed by a purgative (e.g. Magnesium sulphate) after 2 hours.

Children 2-6 years, 1g as a single dose after a light meal, followed by a purgative after 2 hours;

Children under 2 years, 500mg as a single dose after a light meal, followed by a purgative after 2 hours

For Hymenolepsis nana

Adult and children over 6 years

C: Niclosamide 2g as a single dose on the first day, then 1g daily for 6 days.

Children 2-6 years

C: Niclosamide 1g on the first day as a single dose, then 500mg once daily for 6 days.

Children under 2 years, 500mg on the first day as a single dose, then 250mg daily for 6 days

OR

C: Praziquantel 40mg/kg body weight (O) as a single dose

For T.Solium, T.saginata, D. Latum

Adults and children over 2 years

C: Niclosamide 5- 10mg/kg as a single dose.

For H. nana

Adults and children over 2years,

C:Niclosamide 25mg/kg as a single dose.

For Hepatic Echinococcosis

Echinococcosis is treated with Albendazole and surgery or Albendazole and PAIR (puncture aspiration, injection, and re-aspiration).

A: Albendazole 400mg every 12 hours is recommended for 1-3 months before surgical intervention.

Note:

- Administer parenteral vitamin B-12 if evidence of vitamin B-12 deficiency occurs with *Diphyllobothrium* infections
- Tablets should be chewed thoroughly before washing down with water.

CAUTION: Avoid Niclosamide during the first trimester of pregnancy.

1.7 Typhoid and paratyphoid

It is an acute systemic disease resulting from infection by *Salmonella typhi* and *S. paratyphi*, serovar group A and B respectively. Infection is acquired through ingestion of contaminated food and water.

Diagnosis

- The clinical manifestation and duration of illness vary markedly from one patient to another

- The major clinical features are fever, severe headache, drowsiness and muscle pains (myalgia)
- The course of paratyphoid tend be to shorter and less severe compared to typhoid
- Untreated, typhoid fever is a grueling illness that may progress to delirium, obtundation, intestinal hemorrhage, bowel perforation, and death
- Survivors may be left with long-term or permanent neuropsychiatric complications.

Laboratory diagnosis:

The diagnosis of typhoid fever (enteric fever) is primarily clinical. Culture is the criterion standard for diagnosis of typhoid fever with 100% specificity. Culture of bone marrow aspirate; blood and stool cultures should be done within 1 week of onset. Supportive serologic tests: Widal test (rising high titers), indirect fluorescent Vi antibody, ELISA for immunoglobulin M (IgM) and IgG antibodies to *S typhi* polysaccharide.

Treatment

Drug of choice

A: Ciprofloxacin (O): Adult and children over 15 years 500mg 12 hourly for 10 days

Alternatively

A: Chloramphenicol (PO): Adult 500mg 6 hourly for 14 days

Children above 1 years 12.5mg/kg/dose, 6 hourly for 14 days.

CAUTION

Ciprofloxacin is contraindicated in children below 15 years and pregnant women. Chloramphenicol is contraindicated in the third trimester of pregnancy; it may also cause aplastic anaemia which is irreversible.

1.8 Schistosomiasis

Parasitic disease caused by blood flukes (trematodes) of the genus *Schistosoma*. Common species found in Tanzania are *S. haematobium* responsible for urogenital Schistosomiasis and *S. mansoni* responsible for intestinal Schistosomiasis. Infection is through the larval forms of the parasite which is released by freshwater snails. The parasite then, penetrates the skin during contact with infested water. In the body, the larvae develop into adult schistosomes. Adult worms live in the blood vessels, where the females release eggs. Some of the eggs are passed out of the body in the feces or urine to continue the parasite life-cycle. Others become trapped in body tissues, causing an immune reaction and progressive damage to organs.

Diagnosis

Schistosoma mansoni

- There may be abdominal pain and frequent blood stained stool

- In chronic form of *Schistosoma mansoni*; abdominal distention, and vomiting of blood and liver fibrosis (Portal hypertension)
- People co-infected with either hepatitis B or C and *S mansoni* have been shown to have rapid progression of liver disease.

Schistosoma hematobium

- The main clinical feature is painless terminal hematuria
- In chronic and complicated situations can lead to renal failure due to obstructive uropathy, pyelonephritis, or bladder carcinoma (10-20 years after the initial infection)
- In addition, immune complexes that contain worm antigens may deposit in the glomeruli, leading to glomerulonephritis and amyloidosis.

Laboratory diagnosis

Perform stool or urine analysis to identify and specify the eggs in the stool or urine. Kato Katz thick fecal smear technique is needed for chronic disease stage of the intestine and liver. Diagnostic yields are improved by repeated stool samples and from biopsies at sigmoidoscopy. Schistosomal ELISA confirms exposure and if negative reliably excludes infection.

Treatment

Drug of choice

C: Praziquantel: 40mg/kg (O) as a single dose or in 2 divided doses.

NOTE:

- High doses (20mg/kg) as single dose for 2 days for heavy *S. Mansoni* infections
- Medicines will usually arrest progression of clinical features, but will not reverse them
- Surgical interventions may be necessary.

1.9 Shigellosis

Shigella organisms are a group of gram-negative, facultative intracellular bacteria pathogens. They are grouped into 4 species: *Shigella dysenteriae*, *Shigella flexneri*, *Shigella boydii*, and *Shigella sonnei*, also known as groups A, B, C, and D, respectively. Shigellosis is spread by means of fecal-oral, ingestion of contaminated food or water.

Diagnosis

- Sudden onset of severe abdominal cramping, high-grade fever, emesis, anorexia, and large-volume watery diarrhea; seizures may be an early manifestation.
- Abdominal pain, tenesmus, urgency, fecal incontinence, and small-volume mucoid diarrhea with frank blood (fractional stools) may subsequently occur.
- Extra intestinal manifestations associated with *S dysenteriae* may include the following: Severe headache, lethargy, meningismus, delirium, and convulsions involving the CNS; hemolytic uremic syndrome (HUS), microangiopathic hemolytic anemia, thrombocytopenia, and renal failure, profound dehydration and hypoglycemia.

Laboratory diagnosis

Perform microscopic stool examination isolation of *Shigella* from feces or rectal swab specimen.

Stool culture for suspected cases; the yield is greatest early in the course of disease. An enzyme immunoassay (ELISA) for shiga toxin is used to detect *S dysenteriae* type 1 in the stool.

Treatment

Drug of choice

A: Ciprofloxacin (O): Adult, 500mg 12 hourly for 5 days

Children (where the benefit outweighs the risk); 5-10mg/kg/dose. Mmaximum dose 500mg, 12 hourly for 5 days

OR

C:Nalidixic acid (O): Adult, 1g 6 hourly for 7 days

Children over 3months old; 12.5mg/dose 6 hourly for 7 days

OR

A:Erythromycin (O): Adult, 250mg 6 hourly for 5 days

Children, 10mg/kg/dose 6 hourly for 5 days.

Note

- Nalidixic acid is neurotoxic so should be used with caution in older patients; it is contraindicated in epilepsy and renal failure.

1.10 Cholera

Cholera is an acute gastrointestinal infection caused by *Vibrio cholera* organisms (*El Tor* and *Classical biotypes*). In Tanzania only the *El Tor biotype* occurs. Infection occurs through ingestion of contaminated water or food by human feces.

Diagnosis

- After a 24 to 48 hours incubation period, cholera begins with the sudden onset of painless watery diarrhea that may quickly become severe with profuse watery stools (rice water), vomiting, severe dehydration and muscular cramps leading to hypovolemic shock and death
- The stool has a characteristic "rice water" appearance (non bilious, gray, slightly cloudy fluid with flecks of mucus, no blood and inoffensive odor)

Laboratory Diagnosis

Dark field microscopy on a wet mount of fresh stool for identification of motile curved bacillus. Isolation through stool culture is best done through TCBS agar. *Vibrio* serotype can be discerned by immobilization with specific antiserum.

Treatment

- Rehydration, electrolytes and base correction is the most important step
- Management of severely dehydrated patient, IV fluid replacement is preferable
- Oral rehydration is indicated in moderate forms of dehydration but is ineffective in the presence of significant vomiting

For Severe dehydration

- Administer intravenous (I.V) fluid immediately to replace fluid deficit; Use lactated Ringer solution or, if that is not available, isotonic sodium chloride solution.
- For patients older than 1 year, give 100 mls/kg I.V in 3 hours—30 mls/kg as rapidly as possible (within 30 min) then 70 mls/kg in the next 2 hours. For patients younger than 1 year, administer 100 mls/kg IV in 6 hours—30 mls/kg in the first hour then 70 mls/kg in the next 5 hours. Monitor the patient frequently.
- After the initial 30 mls/kg has been administered, the radial pulse should be strong and blood pressure should be normal. If the pulse is not yet strong, continue to give I.V fluid rapidly. Administer ORS solution (about 5 mls/kg/h) as soon as the patient can drink, in addition to I.V fluid.
- If the patient can drink, begin giving oral rehydration salt solution (ORS) by mouth while the drip is being set up; ORS can provide the potassium, bicarbonate, and glucose that saline solution lacks.
- Reassess the hydration status after 3 hours (infants after 6 hrs), In the rare case that the patient still exhibits signs of severe dehydration, repeat the I.V therapy protocol. If signs of some dehydration are present, continue as indicated below for some dehydration. If no signs of dehydration exist, maintain hydration by replacing ongoing fluid losses.
- Start antibiotics (see regimen below) after the patient is rehydrated and vomiting has stopped usually after 4-6 hours. Although the disease is self limiting, an effective antibiotic will reduce the volume of diarrhea and shorten the period during which *Vibrio cholera* is excreted. Antibiotic prophylaxis may be given to all close contacts in the same dosage as for treatment.
- Start feeding 3-4 hours after oral rehydration begins. Preferably, give antibiotics with food to minimize vomiting.

In moderate Dehydration

- Give oral rehydration, approximately 75-100ml/kg in the first four hours
- Reassess after four hours; if improved, continue giving WHO based ORS, in quantity corresponding to losses (eg after each stool) or 10 to 20ml/kg. If not improved, treat as severe

If no signs of dehydration

- For patients who have no signs of dehydration when first observed can be treated at home
- Give these patients ORS packets to take home, enough for 2 days
- Demonstrate how to prepare and give the solution
- Instruct the patient or the caretaker to return if any of the following signs develop; increased number of watery stools repeated vomiting or any signs indicating other problems (eg, fever, blood in stool).

Drugs of choice

A: Doxycycline (O): Adult and child above 12 years; 300 mg as a single dose or 5mg/kg single dose

OR

A:Erythromycin (O): Adult 500mg 8 hourly for 5 days

Children: 40mg/kg/day given in 3 divided doses for 5 days

OR

A: Ciprofloxacin (O): Adult: 30mg/kg single dose (not to exceed 1g) or 15mg/kg 12 hourly for 3 days

NOTE

- A home made ORS equivalent is 6 teaspoons of sugar and one half teaspoon of salt in a liter of water; a half cup of orange juice or some mashed banana can provide potassium.
- Urine output decreases as dehydration develops and may cease. It usually resumes within 6-8 hours after starting rehydration. Regular urinary output (i.e., every 3-4 h) is a good sign that enough fluid is being given.
- In all suspected case notify Ministry of Health and Social Welfare (MoHSW) immediately. For confirmation at the beginning of an outbreak, take rectal swab or stool specimen, handle properly and transport carefully to laboratory.
- Treat on site without referral wherever possible.

CAUTION: Doxycycline should not be used in pregnancy and children below 12 years

1.11 General management of diarrheal diseases

Diarrhea is defined as the abrupt onset of abnormally high fluid content in the stool: more than the normal value of approximately 10 mls/kg/d in the infant and young child, and more than 200 g/d in the teenager and adult. This situation typically implies an increased frequency of bowel movements, which can range from 4-5 to more than 20 times per day. The augmented water content in the stools is due to an imbalance in the physiology of the small and large intestinal processes involved in the absorption of ions, organic substrates, and thus water.

Childhood acute diarrhea is usually caused by infection; however, numerous disorders may cause this condition, including a malabsorption syndrome and various enteropathies. Acute-onset diarrhea is usually self-limited; however, an acute infection can have a protracted course. By far, the most common complication of acute diarrhea is dehydration.

Diarrheal episodes are classically distinguished into acute and chronic (or persistent) based on their duration. Acute diarrhea is thus defined as an episode that has an acute onset and lasts no longer than 14 days; chronic or persistent diarrhea is defined as an episode that lasts longer than 14 days. This distinction is supported by the World Health Organization (WHO).

It is most practical to base treatment of diarrhea on the clinical types of the illness, which can easily be determined when a patient is first examined. Laboratory studies are very useful. Four

clinical types of diarrhea can be recognized, each reflecting the basic underlying pathology and altered pathology:

- Acute Watery Diarrhoea (including Cholera): which lasts several hours or days. The main danger is dehydration and malnutrition if feeding is not continued
- Bloody Diarrhoea (Dysentery): the main dangers are damage of intestinal mucosa, sepsis, and malnutrition. Other complications including dehydration may also occur
- Persistent (Chronic) Diarrhoea: Last for 14 days or longer, the main danger is malnutrition and serious non-intestinal infections, dehydration may also occur
- Diarrhoea with Severe Malnutrition (Marasmus or Kwashiorkor): the main dangers are severe systemic infection, dehydration, heart failure, vitamin and mineral deficiency.

Note: The basis for the management of each type of diarrhoea is to prevent or treat dangers that present.

I. Management of diarrhea in children

- Over 90% of deaths from diarrhea in under-fives would be prevented by:
- Continuing breast feeding and other feeding throughout the attack of diarrhea (prevent malnutrition)
- Making sure mothers know when to take the child to a health facility
- Correct assessment, treatment and continued feeding at the health facility level (See IMCI from the MoHSW manual)
- Treatment of invasive diarrhea (bloody stool) with antibiotics
- Treating or preventing dehydration and electrolyte imbalance with ORS (New osmolarity ORS)
- Reduce the duration and severity of diarrhea and occurrence of future episodes by giving supplemental Zinc
- Referring to hospital for investigation and treatment for severe malnutrition and persistent diarrhea (lasting >14 days)

Other signs which may be useful in assessing severe dehydration and influence management include:

- Weight loss over a short period
- Signs of hypovolemic shock: fast weak pulses, cold extremities, oliguria or anuria
- Hyperventilation, deep and fast breathing indicating acidosis
- Signs of severe malnutrition

II. Management of diarrhea in adults

The principles of management of diarrhea in adult are the same as in children in correction of fluid deficit. As much as possible the cause for diarrhea in adult should be established. Special care should be taken for patients who are immunodeficient e.g. in cases of HIV/AIDS; and/or those with associated chronic disease condition including malignancy. However, the most common cause for diarrhea in adult is food poisoning which is normally self-limiting.

Management of Chronic Diarrhea in Adults.

This may account to 5% but may be under estimation as many patients don't seek medical attention. Causes may include: Medications, mal absorption syndrome, colitides, GI and neuro endocrine tumours, endocroniopathies, chronic infections, and dysmotility bowel syndrome.

Clinical evaluation therefore needs detailed history and examination to determine the pathophysiologic mechanism of diarrhea which will help to stratify the modality of diagnostic evaluation.

Diagnostic evaluation involves;

- Detailed stool analysis: PH, osmolarity, electrolytes, infectious etiologies including clostridium deficile toxin assay, fecal occult blood, fecal fat assay, fecal leucocytes or lactoferin, fecal alpha antitrypsin or elastase, fecal chymotrypsin and laxative screen.
- Full hemogram and ESR.
- Comprehensive metabolic panel: liver functions, renal functions, thyroid functions;
- Urine studies for protenuria and laxative screen, urinary metanephrines and histamines.
- Hormonal assays: serum levels of VIP, gastrin, calcitonin, pancreatic polypeptide, somatostatin, tryptase.
- Serum protein immunoglobulins electrophoresis.

Treatment Guide:

- Correct volume status, electrolyte disturbances and vitamin deficiencies.
- Treat specific underlying cause(s)
- For mild to moderate diarrhea use Loperamide 2- 4mg 6hrly (O) or Diphenoxylate with Atropine 4mg 6hrly.
- In established secretory diarrhea, Octreotide 50 – 250 mcg 12 hrly (SC) is indicated to decrease the volume of stool.
- Empiric treatment with Metronidazole 400mg 8hrly(O) or Ciprofloxacin 500 12 hrly(O) for 5 - 7 days can be considered if patient is at high risk of dehydration, development of systemic complication, or high prevalence of infectious diarrhea in the community.
- Surgical treatment is indicated in established conditions such as, neuro endocrine tumor (NET), severe colitis, or malignancy.

2.0 DISORDERS OF GASTROINTESTINAL TRACT

2.1 Peptic Ulcer Disease

The term peptic ulceration refers to an ulcer in the lower esophagus; stomach and duodenum. They have in common the involvement of acid-pepsin in their pathogenesis leading to disruption of the mucosal integrity causing local defect or excavation due to active inflammation. The common ulcers are duodenal and/or gastric.

Peptic ulcer may present in many different ways, the commonest is chronic, episodic pain present in many different ways, and may persist for months or years. However, the ulcer may come to attention as an acute episode with bleeding or perforation, with little or no previous history. As with duodenal ulcer, epigastric pain is the commonest symptom of gastric ulcer.

2.1.1 Gastro Esophageal Reflux Disease. (GERD)

It is a disorder resulting from gastric acid and other gastric contents into the esophagus due to incompetent barriers at the gastro esophageal junction.

Diagnosis

- Heartburn and regurgitation of sour material into the mouth are specific symptoms
- Symptoms for persistent disease may include odynophagia, dysphagia, weight loss and bleeding
- Extra esophageal manifestation are due to reflux of gastric contents into the pharynx, larynx, tracheobronchial tree, nose and mouth causing chronic cough, laryngitis, pharyngitis. It may also cause or aggravate chronic bronchitis, asthma, COPD, pneumonia, chronic sinusitis and dental decay.

Investigation

Diagnosis clinically by history alone and therapeutic trial of H₂ receptor blocker or proton pump inhibitors (PPI) such as cimetidine 400mg 12hourly **or** Omeprazole 40mg 12 hourly respectively for 1 week, provides support for diagnosis of GERD. Esophagoscopy is valuable but not diagnostic for GERD, double contrast Barium meal is acceptable alternative to patient unwilling to undergo endoscopy. The 24-hours esophageal PH Metry is the specific procedure to confirm presence of GERD.

Treatment

The goals of treatment are to provide symptom relief, heal erosive esophagitis and prevent complication. Life style changes and antisecretory agents may be adequate.

Drug of choice is H₂ Receptor blockers which are effective in symptoms relief and are considered as first line

- C:** Ranitidine 150mg (O) 12 hourly for 14 days;
Children 2 -4mg/kg 12 hourly for 14 days.

Proton Pump inhibitors (PPI) are considered as **second line** and are much more effective in healing ulcers or erosive esophagitis.

Drug of Choice

C: Omeprazole 20mg (O) once daily for 4 -8 weeks

Children 10 -20kg body weight 10mg once daily for 4-8 weeks.

Alternatively

D: Esomeprazole 40mg (O) once daily for 4-8 weeks, then 20mg once daily for maintenance to prevent relapse.

Referral

Refer to specialized centers for all cases with persistent symptoms and/or new complications despite appropriate treatment above.

NOTE

Specific lifestyle changes for patient advice may include

- Reduce spices, and avoid foods and fruits that exacerbate pain in individual patients
- Stop smoking and avoid alcohol
- Low consumption of coffee or tea
- Avoid carbonated drinks
- Avoid medicines such as non-steroidal anti-inflammatory agents (NSAIDS) aspirin, steroids.

2.1.2 Gastro duodenal Ulcers (PUD)

General Management

- Consider peptic ulcer general measures as above
- Referral to a specialist is recommended in presence of persistent symptoms or new onset complications
- Endoscopic biopsy to exclude malignancy in all refractory cases is mandatory
- Evaluation and treatment of *H. Pylori* associated infection is mandatory for effective treatment.

Management of *Helicobacter pylori* infection

Gastric infection with the bacterium *H.Pylori* accounts for majority of PUD. It also plays role in development of gastric mucosal – associated lymphoid tissue (MALT) Lymphoma and Gastric adenocarcinoma.

Laboratory diagnosis

- Perform stool antigen testing; the test should be repeated 3 months after therapy to confirm eradication
- Perform urea breath tests; the test require the patient to be off PPI therapy for 14 days and same days after eradication therapy

- Perform biopsy for urease test; more specific, helpful in cases where antibiotic sensitivity testing is required
- Serology confirms the exposure but not necessarily an active infection

Treatment

Triple therapy is indicated for complete eradication of the organism.

Omeprazole (PO) 20mg twice daily + Amoxicillin (PO) 1g twice daily + Metronidazole (PO) 400mg twice daily for 7 days

OR

Lansoprazole (PO) 30mg twice daily + Clarithromycin (PO) 250mg twice + Tinidazole (PO) 500mg twice daily for 7 days.

2.2 Ulcer Related Conditions

2.2.1 Non-ulcer Dyspepsia (Functional Dyspepsia)

Defined as ≥ 3 months discomforting postprandial fullness, early satiety, and epigastric pain/burning in the absence of organic cause. Most patients follow a benign course, but small number of patients with *H. Pylori* infection or those on NSAIDs progress to ulcer formation. It is the cause of symptoms in more than 60% of patients with dyspepsia.

Diagnosis

Diagnosis clinically as above, plus endoscopic exclusion of esophagitis, peptic ulceration, or malignancy

Treatment

- Eradicate *H. Pylori* if present, if symptoms continue or recurs use H2RB or PPI on per demand basis to control symptoms.
- Use of Prokinetic agents such as Domperidone or Metoclopramide in short course of 2 to 8 weeks, shows beneficial effect at reducing dyspeptic symptoms.

D: Domperidone (PO): Adults: 10 -20 mg 6-8 hourly daily taken 30 minutes before meals; Children: (5- 12 Years) 5 -10mg 6-8 hourly

OR

C: Metoclopramide (PO): Adults: 10mg 8hourly daily

Children: 0.5mg/kg/day in 3 divided doses daily

Counseling and reassurance are important.

2.2.2 Gastritis

Acute gastritis is a term covering a broad spectrum of entities that induce inflammatory changes in the gastric mucosa. The different etiologies share the same general clinical presentation. However, they differ in their unique histological characteristics. The inflammation may involve the entire stomach (e.g., pan gastritis) or a region of the stomach (e.g., antral gastritis). Acute gastritis can be broken down into 2 categories: erosive (e.g., superficial erosions, deep erosions, hemorrhagic erosions) and non erosive (generally caused by *Helicobacter pylori*).

Common etiologies includes certain drugs, alcohol, bacterial, viral, and fungal infections; acute stress, radiation, allergy and food poisoning, bile, ischemia, and direct trauma.

Diagnosis

- Symptoms may include nausea, vomiting, loss of appetite, belching, and bloating
- Occasionally, acute abdominal pain can be a presenting symptom
- Fever, chills, and hiccups also may be present

Note

- The diagnosis of acute gastritis may be suspected from the patient's history and can be confirmed histologically by biopsy specimens taken at endoscopy
- A number of laboratory tests may also be ordered depending on suspected etiology which may include Full hemogram, Liver and Renal functions test

Treatment

- Administer medical therapy as needed, depending on the cause and the pathological findings
- No specific therapy exists for acute gastritis, except for cases caused by *H pylori*
- Administer fluids and electrolytes as required, particularly if the patient is vomiting
- Discontinue the use of drugs known to cause gastritis (e.g., NSAIDs, alcohol)
- Consider short course use of Antacids, H2RB or PPI for relief of symptoms

2.3 Management of GI Bleeding

Acute gastro intestinal (GI) bleeding is common medical emergency resulting in significant morbidity and mortality. It can occur anywhere from mouth to anus; it is therefore subdivided into upper gastrointestinal bleeding(UGIB), anatomically above the ligament of Treitz; and lower gastrointestinal bleeding(LGIB), which is further subdivided to small bowel bleeding(middle GIB) and Colonic bleeding.

Causes for UGIB include, erosive ulcerative disease, esophagitis, portal varices and gastropathy, vascular ectasias, Mallory weiss tear and tumours. Causes for LGIB include, Diverticular disease, hemorrhoids, anal fissures, infectious and radiation colitis, inflammatory bowel disease, polyps, tumours, vascular ectasias and intussusceptions especially in children.

Diagnostic guide

Begin with an assessment of patient's hemodynamic status (normal, orthostatic hypotension, or shock), while trying to localize the acute GIB through focused history and examination. Include the following in history, description of bleeding, duration and frequency, prior bleeding, comorbidities, medications, previous surgery, recent polypectomy or prior radiation.

Assess for the vital signs, stigmata of liver disease, abdominal tenderness, stool colour by rectal examination, nasogastric aspiration may show a positive gastric aspirate.

Diagnostic procedures:

Do baseline investigation, Full hemogram, Coagulopathy profile, liver and renal functions. Specifically, upper and lower endoscopy is appropriately indicated. While Tagged red cell scan and Angiography would be indicated for rapidly or obscure bleeding patients.

Treatment guide

1. Pharmacological

- Intravascular volume replacement should be restored with either ringers lactate or isotonic saline through large bore IV lines.
- Blood transfusion with packed red blood cells should immediately follow. Correct severe thrombocytopenia with packed platelet concentrates, while overt coagulopathy should be corrected with fresh frozen plasma, and Vitamin K S.C injection 5 -10 mg stat given to stable patients.
- Institute (IV) proton pump inhibitors e.g. Esomeprazole 40mg 12hrly. For 3-5 days, then oral therapy up to 6 weeks.
- Add Octreotide 50mcg (IV) stat then 50mcg 8hrly (IV) for 3-5 days specifically for variceal bleeding
- Add ciprofloxacin 400mg 12hrly (IV), or Metronidazole 500 8hrly (IV), or Ceftriaxone 1gm 12hrly (IV) for 3-7 days especially in variceal bleeding.

2. Non Pharmacological

- Endoscopy done within 24 hours could confirm diagnosis and provide sustained hemostasis control. Therapeutic modalities include variceal band ligation, Hemoclipping, sclerotherapy, injectional tamponade therapy, thermocoagulation and angiographic embolization.

3. Surgical Management

- TIPS or shunt therapy is indicated in patients with esophageal varices who have failed pharmacologic and endoscopy therapy or those with bleeding gastric fundic varices.
- Surgical laparotomy for small bowel resection or colectomy is indicated as salvage therapy for small group of patients whom pharmacological, endoscopic, and angiotherapy have failed.

Note: Refer stabilized patients with GIB to specialized centres for expertise management.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is an idiopathic disease, probably involving an immune reaction of the body to its own intestinal tract. The 2 major types of IBD are ulcerative colitis (UC) and Crohn disease (CD). As the name suggests, ulcerative colitis is limited to the colon. Crohn disease can involve any segment of the gastrointestinal tract from the mouth to the anus

2.4.1 Ulcerative colitis

Inflammatory condition that affects the rectum extends proximally to affect a variable amount of the colon. Smoking appears to worsen the disease condition.

Diagnosis

- Active disease is associated with diarrhea, rectal bleeding, tenesmus, passage of mucus, and crampy abdominal pain
- Severity of symptoms correlates with the extent of disease
- Occasionally, diarrhea and bleeding is intermittent and mild that the patient may not seek medical attention, thus though UC can present acutely, symptoms usually have been present for weeks to month
- Complication may present as, Massive hemorrhage (<1%); Toxic megacolon, Perforation with features of peritonitis, stricture.

Note

Diagnosis relies upon the patient's history; clinical symptoms; negative stool examination for bacteria, C.difficile toxin, ova and parasites; sigmoidoscopic appearance; and histology of rectal or colonic biopsy specimen. Single contrast barium enema alternative to sigmoidoscopy but is limited by biopsy access. Supportive laboratory test: CBC for anemia; Thrombocytosis, leucocytosis may reflect active disease.

Treatment and Referral

- Refer patients to specialized centers once disease is suspected for expertise management
- Cure is not available, goals of therapy are to induce and maintain remission

Drug of Choice

C: Sulphasalazine (PO): Adults, 1 gram four times a day for acute disease, reducing to 500mg four times a day for maintenance; Children over 2 years for acute attack use 40-60mg/kg body weight daily.

Maintenance dose 20-30mg/kg body weight daily.

Plus

B: Prednisolone (PO) 30-60mg once daily for severe, acute and extensive disease; reduces gradually according to disease severity.

Note

- Correction of fluid deficit and/or blood is important in acute severe forms which may necessitates hospitalization
- Nutritional therapy should target to replenish specific nutrient deficits
- Life long surveillance is required due to risk of bowel cancer
- Use steroids only when the disease is confirmed, to avoid exacerbation of existing illness.

2.4.2 Crohn's Disease

Crohn's disease is an idiopathic, chronic, transmural inflammatory process of the bowel that often leads to fibrosis and obstructive symptoms and can affect any part of the gastrointestinal tract from the mouth to the anus.

Diagnosis

- Mainly abdominal pain and diarrhea; weight loss, anorexia, and fever may be seen
- Growth retardation in children
- Gross rectal bleeding or acute hemorrhage is uncommon
- Anemia is a common complication due to ileal disease involvement
- Small bowel obstruction, due to stricturing
- Perianal disease associated with fistulization
- Gastroduodenal involvement may be mistaken for *H.Pylori* negative PUD

Diagnostic consideration

- Endoscopy gold standard for diagnosing colonic and terminal ileal disease and readily permits mucosal biopsy and balloon dilatation of any stricture
- Barium follow through is still standard method for evaluating the small bowel, though capsule enteroscopy is superior
- Discriminating features that favours Crohn's from Ulcerative colitis include small bowel disease, mainly right sided colonic disease, rectal sparing, fistulization, and granulomas. Immunological makers: pANCA is predictive in 70% of UC but only 15% in CD; Antibodies to *Saccharomyces cerevisiae* are found in up to 50% CD and less often in UC. When done together specificity is further improved
- Supportive laboratory tests: CBC for anemia; thrombocytosis, leucocytosis, as surrogate sign of inflammation, iron and folate studies, liver functions test, electrolytes/micronutrient deficiency assessment (calcium, magnesium, zinc).

Treatment

- Refer suspected cases to specialized centers for expertise management
- Baseline management as for Ulcerative Colitis above

2.4.3 Pseudomembranous colitis

Clostridium difficile is organism responsible for an infectious colitis that affects 1 of every 200 patients who are admitted to the hospital. Increasingly implicated as a significant cause of morbidity and mortality among hospitalized patients, *C difficile* colitis should also be recognized

among outpatient populations. Prior antibiotic exposure remains the most significant risk factor for development of disease. Antibiotics first seen with clindamycin, but amoxylin and the cephalosporin's are now most frequently implicated. Extreme age, recent GI surgery, malignancy, prolonged hospital stay are other risk factors.

Diagnosis

- Diarrhea and abdominal cramps occurs during first week, but can be delayed up to six weeks
- Nausea, fever, dehydration can accompany severe colitis
- Abdominal examination may reveal distension and tenderness.

Note

- Stool examination is sensitive on anaerobic culture facilities which reveals toxigenic and non toxigenic strains
- Enzyme immunoassays are available for toxins A and B in stool
- Sigmoidoscopy is highly specific if lesion is seen but insensitive compared to the above.

Treatment

Drug of choice

A: Metronidazole (PO): Adults, 400mg 8hourly for 5-days

Children 1 month-12 years: 7.5 mg/kg (max. 400mg) every 8 hours

Second line

D:Vancomycin (PO/IV): Adults, 125mg – 500mg 6hourly for 5- 10days

Children > 1month : 40mg/kg/day in divided doses.

2.5 Irritable Bowel syndrome

Irritable bowel syndrome (IBS) is a functional GI disorder characterized by abdominal pain and altered bowel habits in the absence of specific and unique organic pathology.

Diagnosis

- Abdominal discomfort of at least 3 months duration
- Bloating or feeling of distension
- Altered bowel habits (constipation and/or diarrhea)
- Exacerbations triggered by life events.
- Coexistence of anxiety and depression.

Diagnostic Considerations

- Hematology and biochemistry studies
- Stool microscopy
- Colonoscopy with biopsy

Treatment

- Refer patients to specialized centers for proper evaluation and management.
- Supportive therapies;
- Reassurance and explanation are essential.
- High fibre diet and eating a healthy diet.
- Relief of pain due to abdominal cramps

A: Hyoscine butyl bromide 20mg (O) four times a day

- Relief of anxiety that may be making symptoms worse

C: Diazepam 5-10 mg (O) 8 hourly

Give short and infrequent courses only, in order to avoid dependence.

- If constipation is predominant in IBS encourage high fiber diet.
- If diarrhea predominant in IBS

C: Loperamide 4mg (O) stat, followed by 2mg after each unformed stool until diarrhoea is controlled.

- Explore psycho-social factors in resistant cases and counseling.

2.6 Malabsorption syndrome

Malabsorption is a clinical term that encompasses defects occurring during the digestion and absorption of food nutrients by and infections of the gastrointestinal tract. Although presenting symptoms, such as diarrhea and weight loss may be common, the specific causes of malabsorption are usually established based on physiologic evaluations. The treatment often depends on the establishment of a definitive etiology for malabsorption. Etiologic examples include pancreatic insufficiency, bacterial overgrowth, celiac disease, tropical sprue, lactase deficiency, diabetic enteropathy, thyroid disease, radiation enteritis, gastrectomy and extensive small bowel resection.

Diagnosis

Depending on etiology, presentation may collectively include:

- Diarrhoea a commonest symptom which is frequently watery
- Steatorrhea due to fat malabsorption; characterized, by the passage of pale, bulky, and malodorous stools. Stools often float on top of the toilet water and are difficult to flush
- Weight loss and fatigue
- Flatulence and abdominal distention
- Edema due to hypoalbuminemia, and with severe protein depletion ascites may develop
- Anemias which can either be microcytic iron deficiency (celiac disease) or macrocytic vitamin B-12 deficiency (chrohn's disease or ileal resection).
- Bleeding disorders (Ecchymosis, melena, and hematuria) due to vitamin K malabsorption and subsequent hypoprothrombinemia.
- Metabolic defects of bones (osteopenia or osteomalacia) due to vitamin D deficiency. Bone pain and pathologic fractures may be observed. Malabsorption of calcium can lead to secondary hyperparathyroidism.

- Neurologic manifestations: Electrolyte disturbances, such as hypocalcaemia and hypomagnesaemia, can lead to tetany. Vitamin malabsorption can cause generalized motor weakness (pantothenic acid, vitamin D) or peripheral neuropathy (thiamine), a sense of loss for vibration and position (cobalamin), night blindness (vitamin A), and seizures (biotin).

Treatment

- Patients should be referred to specialized centers for proper evaluation and definitive management
- Two basic principles underlie the management of patients with malabsorption, as follows:
 - The correction of nutritional deficiencies
 - When possible, the treatment of causative diseases
- Nutritional support
 - Supplementing various minerals, such as calcium, magnesium, iron, and vitamins, which may be deficient in malabsorption, is important
 - Caloric and protein replacement also is essential
 - Medium-chain triglycerides can be used as fat substitutes because they do not require micelle formation for absorption and their route of transport is portal rather than lymphatic
 - In severe intestinal disease, such as massive resection and extensive regional enteritis, parenteral nutrition may become necessary.
- Treatment of causative diseases
 - A gluten-free diet helps treat celiac disease
 - A lactose-free diet helps correct lactose intolerance; supplementing the first bite of milk-containing food products with Lactaid also helps
 - Protease and lipase supplements are the therapy for pancreatic insufficiency
 - Antibiotics are the therapy for bacterial overgrowth
 - Corticosteroids, anti-inflammatory agents, such as mesalamine, and other therapies are used to treat regional enteritis.

2.7 Pancreatitis

Pancreatitis is an inflammatory process in which pancreatic enzymes auto digest the gland. It may present as acute pancreatitis, in which the pancreas can sometimes heal without any impairment of function or any morphologic changes, or as chronic pancreatitis, in which individuals suffer recurrent, intermittent attacks that contribute to the functional and morphologic loss of the gland.

2.7.1 Acute Pancreatitis

It is due to sudden inflammation of the pancreas due to pancreatic enzymes auto digestion. Common risk factors which trigger the acute episode are presence of gallstones and alcohol intake.

Diagnosis

- Severe, unremitting epigastric pain, radiating to the back
- Nausea and vomiting

- Signs of shock may be present
- Ileus is also common
- Local complications: inflammatory mass, obstructive jaundice, gastric outlet obstruction
- Systemic complication: sepsis, acute respiratory distress syndrome, acute renal failure

Diagnostic considerations

- Serum amylase, in counts over 1000U/L, but poor correlates with disease severity. Serum Lipase twice the normal limit has superior sensitivity and specificity.
- Complete blood counts, Urea and electrolytes, bicarbonate levels, liver transaminases and albumin, LDH, glucose, calcium, CRP, and lipid profile for modified Glasgow criteria to disease severity and outcomes.
- Abdominal ultrasound, Plain abdominal X-ray, Chest X-ray, CT Abdomen.

Treatment

- Prompt referral to specialized centers with intensive care facilities is recommended
- Principles of management include expertise supportive therapy:
 - Nil per oral regimen for few days up to weeks is indicated depending on severity.
 - Intravascular volume expansion (colloids/crystalloid)
 - Opiates analgesia and antiemetics usually required.
 - Prophylactic antibiotics in severe state, useful when there is evidence of sepsis(IV) ceftriaxone 1g 12hrly + Metronidazole 500mg 8hrly or Meronem 1g 8hrly
 - ERCP + Sphincterotomy when gallstones are present in the CBD.

2.7.2 Chronic Pancreatitis

Chronic pancreatitis is long-term (chronic) inflammation of the pancreas that leads to permanent damage. The most common cause for such a condition is long-term excessive alcohol consumption.

Diagnosis

- The most common symptom is upper abdominal pain that may be accompanied by nausea, vomiting and loss of appetite
- As the disease gets worse and more of the pancreas is destroyed, pain may actually become less severe
- During an attack, the pain often is made worse by drinking alcohol or eating a large meal high in fats.
- Because a damaged pancreas can't produce important digestive enzymes, people with chronic pancreatitis may develop problems with digesting and absorbing food and nutrients. This can lead to weight loss, vitamin deficiencies, diarrhea and greasy, foul-smelling stools.
- Over time, a damaged pancreas also can fail to produce enough insulin, which results in Diabetes.

Diagnostic Consideration:

- Abdominal X-ray, for evidence of pancreatic calcifications
- CT, MRCP, ERCP, and Endoscopic ultrasound are complementary
- Biochemical; Glucose tolerance test, serum vitamins (A, D, E, K), hemoglobin and calcium levels,
- Pancreatic function tests: Secretin /CCK – secretory test, fecal elastase1 concentrations

Treatment

Referral is recommended for expertise evaluation and management in specialized centers. Because chronic pancreatitis cannot be cured, direct the treatment towards:

- **Relieving pain with pain-killers-** In rare cases, surgery/ ERCP to open blocked ducts or remove part of the pancreas may be done to relieve pain.
- **Improving food absorption** - The patient should be recommended to follow a low-carbohydrate, high-protein diet that also restricts some types of fats. Once digestive problems are treated, patient will usually gain back weight and diarrhea improves. Another way is by giving the patient pancreatic supplements containing digestive enzymes.
- **Treating diabetes** - Treat diabetes with careful attention to diet to help keep blood sugar levels stable. In some people, insulin injections and other diabetic medications are needed.

2.8 Peritonitis

Refers to inflammation of the peritoneum; it may be localized or diffuse in location, acute or chronic in natural history, infectious or aseptic in pathogenesis. Acute peritonitis is most often infectious usually related to a perforated viscus (secondary peritonitis); primary or spontaneous peritonitis refers to when no intraabdominal source is identified. Acute peritonitis is associated with decreased intestinal motility, resulting in distention of the intestinal lumen with gas and fluid. The accumulation of fluid in the bowel together with the lack of oral intake leads to rapid intravascular depletion with effects on cardiac, renal, and other systems.

Chronic peritonitis refers to longstanding inflammation of the peritoneum. Causes include repeated attacks of infection such as from pelvic inflammatory disease (PID), Metastatic lesions or foreign substances that induce inflammation, and chronic infections within the abdomen such as Tuberculosis.

Diagnosis

- Acute peritonitis is usually characterized by acute abdominal pain and tenderness, dehydration, fever, hypotension, nausea and vomiting and tachycardia.
- Complications include abscess formation, oliguria and shock.
- Similar features may be seen in spontaneous bacterial peritonitis (SBP), which occurs in cirrhotic patients with ascites. Bacterial translocation, bacteraemia and impaired antimicrobial activity contribute to its development. Gram negative bacilli (E. coli) commonly are a causative microbe.

Diagnostic considerations: (specific)

- Peritoneal fluid analysis for microscopy, microbiology, culture and sensitivity
- Macroscopic evaluation of the peritoneal fluid will exclude hemoperitoneum in trauma cases
- Blood cultures due to bacterimia
- Scanning procedures (ultrasound and/or CT scan) facilitates the diagnosis, Abdominal having the highest diagnostic yield.

Treatment considerations

Surgery remains a cornerstone of peritonitis treatment.

Antimicrobial therapy is adjunctive to surgical correction of underlying lesion or process and treatment will depend on causative agent.

Where cause is not known antibiotics of choice are:

C: Ampicillin (I.V) 1g every 6hours for 5-10 days

Plus

A: Gentamicin (I.V) 4 mg/kg/24 hours in 3 divided doses for 5-10 days

Plus

C: Metronidazole (I.V)/ (O) 400-600mg every 8 hours for 5-10 days.

Referral

- Patient needs referral to centers where surgical intervention is adequate (i.e. expertise and medical facility)
- Refer to TB section for TB peritonitis management.

2.9 Constipation

According to the Rome III criteria for constipation, a patient must have experienced at least 2 of the following symptoms over the preceding 3 months: Fewer than 3 bowel movements per week; straining; lumpy or hard stools; sensation of anorectal obstruction; sensation of incomplete defecation, manual maneuvering required defecating.

Constipation is a symptom, not a disease. Contributory factors may include inactivity, low fiber diet and inadequate water intake. Specific causes may include, conditions associated with neurologic dysfunction, scleroderma, drugs, hypothyroidism, hypokalemia, hypercalcemia, Cushing's syndrome, colonic tumours, anorectal pain, and psychological factors.

Diagnosis

- Fewer than three bowel movements per week, small, hard, dry stools that is difficult or painful to pass, need to strain excessively to have a bowel movement, frequent use of enemas, laxatives or suppositories are characteristic.
- Other features may include; abdominal bloating, rectal bleeding, spurious diarrhea, low back pain, feeling of incomplete evacuation, and tenesmus.

Referral

The following signs and symptoms, if present, are grounds for urgent evaluation or referral:

- Rectal bleeding
- Abdominal pain
- Inability to pass flatus
- Vomiting
- Unexplained weight loss.

Diagnostic guides:

An extensive work up of the constipated patient is performed on an outpatient basis and usually occurs after approximately 3-6 months of failed medical management. It is advised to refer the patient at this juncture to specialized centers.

Laboratory evaluation may include a complete blood count (CBC), fecal occult blood especially in middle-aged or elderly adults; Thyroid function tests, serum chemistry to exclude metabolic causes of constipation.

Imaging studies are used to rule out acute processes that may be causing colonic ileus or to evaluate causes of chronic constipation. Lower gastrointestinal (GI) endoscopy, colonic transit study, defecography, anorectal manometry, surface anal electromyography (EMG), and balloon expulsion may be used in the evaluation of constipation.

In the acute situation with a patient at low risk who usually is not constipated, no further evaluation is necessary. Consider sigmoidoscopy, colonoscopy, or barium enema for colorectal cancer screening in patients older than 50 years. Colonoscopy represents the current criterion standard.

Treatment guide:

- Find out the type of food taken by patient.
- Exclude other organic causes of partial bowel obstruction.
- Encourage high fibre diet, adequate fluid intake.
- Give laxatives as required but avoid chronic use.

Stimulant laxative

A: Bisacodyl (PO) 5-10mg

OR

A: Bisacodyl suppository (PR) 10mg at bed-time

Osmotic laxative

C: Lactulose solution (3.1 – 3.7g/ml) (O); Adults 15ml, 12 hourly; Children under one year 2.5ml, 12 hourly; Children 1 – 5 years 5ml, 12 hourly and Children 5 – 10 years 10ml, 12 hourly.

2.10 Hemorrhoids

Hemorrhoid disease is due to enlargement or thrombosis of the veins in the external or internal hemorrhoidal plexus. The internal hemorrhoids are graded into four groups:

- Bleeding with defecation
- Prolapses with defecation but return naturally to their normal position
- Prolapses any time especially with defecation and can be replaced manually
- Permanently prolapsed.

Diagnosis

The most common presentation of hemorrhoids is rectal bleeding, pain, pruritus, or prolapse. However, these symptoms are nonspecific and may be seen in a number of anorectal diseases. A thorough history is needed to help narrow the differential diagnosis and adequate physical examination to confirm the diagnosis.

Diagnostic considerations

- Anoscopy is mandatory for viewing internal hemorrhoids
- Flexible sigmoidoscopy is performed to exclude proximal disease

Treatment

The following is a quick summary of treatment for internal hemorrhoids by grade:

- Grade I hemorrhoids are treated with conservative medical therapy and avoidance of non steroidal anti-inflammatory drugs (NSAIDs) and spicy or fatty foods
- Grade II or III hemorrhoids are initially treated with nonsurgical procedures (sclerotherapy, band ligation)
- Very symptomatic grade III and grade IV hemorrhoids are best treated with surgical hemorrhoidectomy
- Treatment of grade I.V internal hemorrhoids or any incarcerated or gangrenous tissue requires prompt surgical consultation

External hemorrhoid symptoms are generally divided into problems with acute thrombosis and hygiene/skin tag complaints. The former respond well to office excision (not enucleation), while operative resection is reserved for the latter. Therapy is directed solely at the symptoms, not at aesthetics.

Supportive management

- Treat any identified causative condition
- Encourage high fibre diet
- Careful anal hygiene
- Saline baths
- Avoid constipation by using stool softener.

Drugs of choice

Steroids and local anesthetics aims to reduce inflammation and provide relief during painful defecation. Refer preparations and dosing in cap. 2.10 below.

2.11 Anal fissures

These are painful linear ulcers in the anal canal. Young and middle aged adults most commonly affected. Primary fissure occur in the posterior midline. It can also be secondary to Crohn's disease, anal cancer, or infection such as syphilis, TB in which case they occur more lateral. Passage of hard stools is a common predisposition to primary fissures.

Diagnosis

The hall mark is severe sharp pain during and after defecation with/out bright red bleeding.

Diagnostic consideration

Perform digital rectal examination or proctoscopy, which must be done with topical anesthesia.

Treatment Guide

- Stools must be made soft and easy to pass; ensure high fluid intake, use osmotic laxatives such as Lactulose 20 mls 12 hrly (O)
- Topical anesthetics (Lidocaine jelly 2% - applied 12 to 8 hrly anal area with frequent seat baths reduces sphincter spasm.
- Vasodilator treatment with topical isosorbide mononitrate 1% or diltiazem 2% - applied 12 hrly anal area, is effective at increasing fissure healing rate and it is the first line of management.

- If the fissure in few weeks surgical sphincterotomy is indicated to lower the sphincter tone.

2.12 Pruritus Ani

Also known as anusitis is the irritation of the skin within perianal region, the intensity of anal itching increases from moisture, pressure, and rubbing caused by clothing and sitting. At worst, anal itching causes intolerable discomfort that often is accompanied by burning and soreness. Causes include:

- Benign anorectal condition such as hemorrhoids or anal fissure
- Neoplasia such as anal cancer, Paget's disease
- Dermatological disease e.g. dermatitis, lichen sclerosus
- Infection: Candida, thread worm
- Some dietary components e.g. coffee

Treatment guides

- Treat underlying condition
- Short term use of steroid - Prednisone Caproate Ointment applied 12 hrly or suppository applied once daily is recommended.
- Proper hygiene and to wear cotton underwear
- Avoid hot and spicy foods.

3.0 DISORDERS OF THE LIVER AND BILIARY TRACT

3.1 Hepatitis

This is a medical condition defined by the inflammation of the liver and characterized by the presence of inflammatory cells in the tissue of the organ. The condition can be self-limiting or can progress to fibrosis and cirrhosis. Hepatitis may occur with limited or no symptoms, but often leads to jaundice, anorexia and malaise. Hepatitis is **acute** when it lasts less than six months and **chronic** when it persists longer. A group of hepatotropic viruses cause most cases of hepatitis worldwide, but it can also be due to other viral infections (e.g. Cytomegalovirus, Epstein-Barr, Coxsackie viruses), toxins notably alcohol, certain medications, some industrial organic solvents and plants, autoimmune diseases and metabolic disease.

3.1.1 Acute Viral Hepatitis

It is a systemic infection affecting the liver predominantly. Almost all cases of acute viral hepatitis are caused by one of five hepatotropic viral agents: Hepatitis A virus (HAV), Hepatitis B virus (HBV), HBV – associated delta agent or Hepatitis D virus (HDV), and Hepatitis E virus (HEV).

Diagnosis

Acute infection with a hepatitis virus may result in conditions ranging from subclinical disease to self-limited symptomatic disease to fulminant hepatic failure.

Collectively patients may develop fever, anorexia, malaise, jaundice, abdominal pain after specific incubation periods; and in severe forms signs of acute liver failure including altered consciousness may be present.

Diagnostic guides

Quantitative analysis for presence of specific antibodies and/or antigenemia is mandatory for establishing a specific causative viral agent. The severity of liver injury is determined by transaminases levels (ALT) in particular, and more precisely by liver biopsy. The viremia is determined through PCR method and for some viral subtypes through genotyping.

Treatment guides

Acute infection is usually self limiting, especially for HAV, HEV, and only 80% of HBV, and 20% of HCV cases. Supportive management is all that is required during acute illness, except in fulminant cases where specific antiviral medication may be required.

Note: Refer all cases of suspected Hepatitis to referral centers for expertise management.

3.1.2 Chronic viral Hepatitis

There is an on going inflammatory reaction in the liver for at least 6 months. The most common causative hepatotropic viral agents are HBV, HCV, and HDV. Non viral cause may include, drugs (methyldopa, Isoniazid), autoimmune hepatitis, Wilson's disease, hemochromatosis, α -antitrypsin deficiency. Notably disease chronicity can progress into liver cirrhosis and hepatocellular cancer in span of years if no early treatment is initiated.

Diagnosis

- There is a wide clinical spectrum ranging from asymptomatic serum amino-transaminases elevations to apparently acute and even fulminant hepatitis.
- Common symptoms include, fatigue, malaise, anorexia, low grade fever; jaundice is frequent in severe disease.
- Some patients may present with complications of cirrhosis: ascites, variceal bleeding, encephalopathy, coagulopathy, and hypersplenism. Some extra hepatic features may also predominate.(urticaria, arthritis, vasculitis, polyneuropathy, glomerulonephritis, thyroditis)

Diagnostic guides:

In addition to the above guides, surveillance studies for development of cirrhosis and its complications or HCC include ultrasonography, CT scan, serum α - feto protein.

Treatment

B: Lamuvidine 150mg (O) once daily.

OR

B: Tenofovir 300mg (O) once daily

Treatment is long term (48- 96 weeks)

Combination therapy is indicated in HIV co infected patients.

- HCV is treated by Inj. Pegylated interferon (180 μ g S.C) in combination with Tabs Rebaivirin 800mg/day (O) in devided dose for genotype 2&3 or 1000mg/day(O) in devided dose for genotype 1,4,5 up to 48 weeks.
- **Note:** Referral of these patients to specialized centers for expertise management is highly recommended.

3.2 Liver Cirrhosis

This is a common end point of many causes of liver diseases; commonly caused by chronic hepatitis B & C and alcoholic liver disease. Other causes include autoimmune hepatitis and metabolic liver disease. It is a histological diagnosis characterized by hepatic fibrosis and nodule formation. Depending on etiologic process the progression of liver injury to cirrhosis may occur over weeks to years. Clinical classification of the disease using Child- Tourcotte- Pugh score is used to determine a 1-year mortality and need for liver transplantation.

Diagnostic features

- Include jaundice, hepatomegaly, ascites, features of increased estrogen levels in men, while in women there are features of increased androgen levels. Loss of libido, testicular atrophy and impotence are common among male cirrhotic.
- In women predominant features are breast atrophy, menstrual disturbances including amenorrhea. Features of portal hypertension like splenomegaly, ascites, distended abdominal wall vessels and variceal bleeding are common.
- Hepatic encephalopathy and renal dysfunction is a sequel of associated complications.

Diagnostic guides

To include, complete blood count, liver functions, serum electrolytes, viral hepatitis panel (B, C, and D), autoimmune markers (AMA,ANA), and makers for associated metabolic disease (ceruloplasmin, ferritin), alpha fetoprotein, Imaging (ultrasonography with Doppler studies, CT, MRI) and Liver biopsy.

Treatment Guide

In compensated cirrhosis:

- Treat the cause and associated complications.
- Encourage high calorie diet and protein intake.

In decompensate cirrhosis:

- Treat specifically the manifestation of hepatic decompensation. e.g. ascites, hepatic encephalopathy, hepatorenal syndrome, GI bleeding, spontaneous bacterial peritonitis.
- Liver transplantation is definitive treatment once an episode of decompensation has occurred.

Note

It is advisable to refer patients with this condition to specialized centers for proper evaluation and treatment. A planned supportive management can then be continued at the referring centers.

3.3 Ascites of chronic liver disease

There is accumulation of fluid into peritoneal cavity; contributing factors includes portal hypertension, hypoalbuminemia, hepatic lymph, hepatorenal syndrome.

Diagnosis

- May be asymptomatic if small amounts
- Abdominal distension and discomfort in increasing amounts, anorexia, nausea, early satiety, heartburn, flank pain, and respiratory distress.

Treatment guide:

Salt restriction < 2gm per day

C: Spironolactone 100- 200mg/ day(O); increase dose up to 400mg if fluid not mobilized despite low sodium diet – This is the first line therapy.

A: Furosemide 40mg/day (O) is added to spironolactone at ratio 2.5:1 up to maximum dose 160mg/day.

Note: Dose of each medication can be increased every 1- 2 weeks to the maximum doses indicated.

Monitor weight reduction (targeted at 0.5 and 1kg/day if peripheral edema is present), urinary Na and K, serum electrolytes and creatinine.

- If ascites still present despite the above measures, manage the condition as refractory ascites where large volume paracentesis is indicated with concurrent infusions of albumin (10g/L of ascites removed)
- Liver transplantation is the definitive management.

3.4 Cholestatic Jaundice

Cholestasis is a symptom of many diseases. It is defined as a pathologic state of reduced bile formation or flow. The mechanisms of cholestasis can be broadly classified into hepatocellular (Intrahepatic), where an impairment of bile formation occurs, and obstructive (extra hepatic), where impedance to bile flow occurs after it is formed.

Intrahepatic causes of cholestasis include viral hepatitis, alcohol, primary biliary cirrhosis, drug toxicity, Hodgkin's lymphoma and pregnancy. Extra hepatic causes which may be amenable to surgical correction include choledocholithiasis and carcinoma of the biliary tree. Parasitic infections such as Ascariasis may also cause cholestatic jaundice

Diagnosis

- The prominent features include jaundice, dark urine, pale stools, and itching/pruritis.

Diagnostic considerations

- Liver functions; for elevated serum levels of total bilirubin, direct bilirubin, alkaline phosphatase, gamma-glutamyl transferase, bile salt concentration
- Elevated serum cholesterol
- Elevated fecal fat levels.
- Imaging and endoscopic studies. (USS, MRI, MRCP, ERCP, PTC)
- Liver biopsy.

Treatment

- Identify and treat specific cause
- Some medical care is directed at cholestasis and its consequences:

D: Cholestyramine (O) 4 -16g daily relieves itching

OR

D: Ursodeoxycholic acid (PO) 8-10 mg/kg/day (in 2 or 3 divided doses)

Surgery is indicated for extrahepatic cholestasis.

Note

Refer patients with cholestatic liver disease to specialized centres, particularly if it is severe or prolonged.

3.5 Hepatic encephalopathy

A clinical state associated with alteration in mental status and cognitive function occurring in the presence of liver failure; it may be acute and reversible or chronic and progressive. Precipitants of the condition include, GI bleeding, azotemia, constipation, high protein meal, hypokalemic alkalosis, CNS depressant drugs (benzodiazepines and barbiturates.), hypoxia, hypercarbia, sepsis.

Diagnosis

Confusion, slurred speech, flapping tremors, change in personality that can include being violent and hard to manage to being sleepy and difficult to arouse (refer to grades of hepatic encephalopathy by West Haven Criteria – grade 1-4).

Diagnostic guides

- Evaluation for extent and cause of liver injury need to be established especially for patients in whom the diagnosis of liver disease has not been previously made
- Investigate to include: liver functions, complete blood count, serum electrolytes, blood sugar
- Seek the precipitants including septic screen (culture of blood, urine, sputum, ascites); exclude GI bleed (↓HB, history of melena), or evidence of renal impairment (↑↑urea, ↑creatinine)
- Abdominal U/S or CT scan may show evidence of portal hypertension.

Treatment

General measures

- Identify and if possible eliminate the cause (e.g drugs, viral hepatitis, septicaemia)
- Toxins, alcohol or upper G.I bleeding)
- Avoid use of all unnecessary drugs including diuretics and sedatives
- Provide non protein containing high calorie food (2000kCal/day)

Drug Treatment

Antibiotic treatment of choice:

D: Neomycin 4-12g/day (PO/NGT);

OR

A: Metronidazole 400-500mg (PO)/ (IV) 8 hourly

Give laxatives to provoke diarrhea:

C: Magnesium sulphate (O) 4g with water twice daily

OR

C: Lactulose solution 60 mls/day in 2-3 divided dose to ensure 2-4 soft stools passed daily and carry out high bowel washout.

- Give dextrose 10% (I.V infusion) 3 litres/day with 2g (26mmol) potassium chloride added to every litre bag (if renal function is satisfactory).
- Check for any infection and treat immediately
- If signs of bleeding are present give
 - D:** Vitamin K (I.V) 10mg
 - Plus**
 - S:** Fresh Frozen Plasma initially
 - Add**
 - Platelets if count <20 x 10g/l and patient is still bleeding
- If ethanol etiology is suspected give:
 - C: Thiamine** (I.V) 10mg before dextrose infusion and continue daily for 3 days.

Note: Hepatic encephalopathy is a medical emergency and requires referral to specialized and equipped centers for proper evaluation and management.

CHAPTER FOUR

RESPIRATORY DISEASE CONDITIONS

1.0 ACUTE RESPIRATORY INFECTIONS (ARI)

1.1 Pneumonia

Pneumonia is the inflammation of the lung tissue. Pneumonia can either be primary (to the causing organism) or secondary to pathological damage in the respiratory system. The common causative organisms for pneumonia are bacterial (for example *Streptococcus pneumoniae*, *Hemophilus influenza*, and *Staphylococcus aureus*, and *Mycoplasma pneumoniae*, viral or parasitic e.g *Pneumocystis jirovecii*. The important clinical features are high fever 39°C, dry or productive cough, central cyanosis, respiratory distress, chest pain and tachypnea.

1.1.1 Pneumonia in Children

For more details, refer also *Integrated Management of Childhood Illness (IMCI) guidelines*

Diagnosis

For children under five years of age the important symptoms are coughing or difficult breathing. Classification of pneumonia in children is based on respiratory rate which is fast breathing and chest in-drawing.

Fast breathing is defined as

- Respiratory rate > 60 age less than 3 months
- Respiratory rate > 50 age between 3 months and 5 years
- Chest indrawing is when the lower part of the chest moves in when the child breathes in.

Table 1: Important clinical features of pneumonia in underfives

Age	Signs	Classification
<i>Infants less than 2 months</i>	<ul style="list-style-type: none">• Severe chest in-drawing Or	Severe pneumonia (all young infants with pneumonia are classified as severe)
	<ul style="list-style-type: none">• 60 breaths per minute or more• No severe chest in-drawing• Less than 60 breaths per-minute	No pneumonia: Cough or cold
<i>Children from 2 months to 1 year</i>	<ul style="list-style-type: none">• Chest in-drawing	Severe pneumonia
	<ul style="list-style-type: none">• No chest in-drawing• 50 breaths per minute or more	Pneumonia
	<ul style="list-style-type: none">• No chest in-drawing• Less than 50 breaths per minute	No pneumonia Cough or cold
<i>Children from 1 year to</i>	<ul style="list-style-type: none">• Chest in-drawing	Severe pneumonia

5 year	<ul style="list-style-type: none"> No chest in-drawing 40 breaths per minute or more 	Pneumonia
	<ul style="list-style-type: none"> No chest in-drawing Less than 40 breaths per minute 	No pneumonia Cough or cold

General management

- Oxygen therapy if available
- Supportive care
 - Lower the temperature if $\geq 38.5^{\circ}\text{C}$, give Paracetamol
 - If wheezing giving rapid-acting bronchodilator: Nebulized Salbutamol
 - Ensure that the child receives daily maintenance fluid appropriate for the child's age but avoid over-hydration refer to IMCI/ STG & Essential medicines List for Children

Treatment of very severe pneumonia:

A: Ampicillin 50 mg/kg I.V/I.M every 6 hours
Plus

A: Gentamicin (7.5 mg/kg I.V/I.M once a day) for 5 days;

If child responds well, complete treatment at home or in hospital with

A: Amoxicillin (15 mg/kg three times a day)
Plus

A: Gentamicin 7.5 mg/kg I.M once daily for a further 5 days.

Alternatively,

B: Chloramphenicol (25 mg/kg I.M or I.V every 6 hours) until the child has improved. Then continue orally 4 times a day for a total course of 10 days.

If the child does not improve within 48 hours, switch to

A: Gentamicin (7.5mg/kg I.V/IM once a day)
Plus

A: Cloxacillin (50 mg/kg IV or IM every 6 hours), then continue Cloxacillin orally 4 times a day for a total course of 3 weeks.

- If the child is not improving use ceftriaxone (80 mg/kg I.V or I.M once daily) for 10 days.
- For children above 5 years, atypical pneumonia should be considered e.g. mycoplasma. A macrolide (Erythromycin OR Azythromycin) should be considered as a drug of choice in addition to the above antibiotics or as a second line treatment.

Severe pneumonia

A: Benzyl Penicillin 50 000 units/kg I.V or I.M every 6 hours for at least 3 days
THEN

A: Amoxicillin 15 mg/kg 8 hourly for 7 days.

- If the child does not improve within 48 hours, or deteriorates, look for complications and treat accordingly. If there are no apparent complications, switch to

B: Chloramphenical (25 mg/kg every 6 hours I.V or I.M) until the child has improved. Then continue orally for a total course for 10 days.

Non-severe pneumonia

A: Amoxicillin 25 mg/kg 12 hourly for 5 days

- Give the first dose at the clinic and teach the mother how to give the other doses at home.
- Encourage breastfeeding and feeding.

1.1.1 Pneumonia in Adults Community Acquired Pneumonia

First Line management

- Chest X-ray not necessary but preferable for in-patient

First Line Treatment

Table 2: Treatment of Typical Community Acquired Pneumonia

Condition	Treatment	Duration
Mild pneumonia (treated on out-patient basis)	Amoxycillin (O) 500 – 1000 mg every 8 hours	5 days
	<i>Or</i> Erythromycin 500 mg every 8 hours	5 days
Severe pneumonia (in-patient)	Ceftriaxone 1g mg every 12 hrs	7-10 days

Second line treatment

If patient is in respiratory distress, or no response after 3 days of first line treatment, or patient's condition deteriorates, then investigate. For interpretation of X-ray and management algorithm, see Section HIV related respiratory conditions (applicable to HIV negative patients with difficult to treat bacterial pneumonias).

Table 3: Treatment of Atypical Community Acquired Pneumonias

Condition	Treatment	Duration
Atypical Pneumonias	Erythromycin (O) 500 mg every 6 hours	7 to 10 days
<i>Pneumocystis jirovecii</i> Pneumonia (PJP)	Co-trimaxazole (O) 3 to 4 tabs of 480mg every 6 hours PLUS Folic acid if cytopenic <i>Alternatively in sulphur allergy:</i> Clindamycin	21 days

	450-600 mg (O) every 6 hours	
<i>Staphylococcus aureus</i> Pneumonia	Cloxacillin (IV) 1 to 2mg every 6 hours Or *Clindamycin (IV/O) 600mg every 6 to 8 hours	14 days 14 days
Klebsiella Pneumonia	Chloramphenicol (IV) 500 mg every 6 hours +/- Gentamicin (IV) 4 to 5 mg/kg/24 hrs in 2 divided doses	10 to 14 days 10 to 14 days

NOTE: In severe *Pneumocystis jirovecii* pneumonia (PCP), add 30 – 40mg prednisolone for 14 days.

Alternative in Staphylococcal and Klebsiella Pneumonia

D: Ceftazidime 1g (IV/IM) every 8 hours. Max. dose 6g daily

Hospital Acquired Pneumonia

This is defined as pneumonia that occurs more than 48 hours after hospital admission but that was not incubating at the time of admission.

Table 4: Treatment of Hospital acquired Pneumonia

Condition	Treatment	Duration
Empirical treatment until bacteriology available	Ampicillin (IV) 1g every 6 hours	7 to 10 days
	PLUS Gentamicin (IV) 4 to 5mg/kg/day in 2 divided doses	7 to 10 days

2.0 Bronchospasm

This is a contraction of smooth muscle in the walls of the bronchi and bronchioles, causing narrowing of the lumen

2.1 Wheezing

Wheezing is a high-pitched whistling sound heard near the end of expiration. It is caused by spasmodic narrowing of the distal airways. Sometimes children with pneumonia present with wheeze.

In a young infant below 3 months, wheezing is a sign of serious illness - REFER IMMEDIATELY to a higher level if the condition cannot be managed at your facility.

Wheezing for infants between 3 and 12 months may be due to bronchiolitis, a viral infection – REFER to a higher level if the condition cannot be managed at your facility .

In Children more than 1 year wheezing may be due to asthma, *refer to section on asthma*. If the child is in distress, give a rapid-acting bronchodilator (*see section on asthma*) and REFER to a higher level if the condition cannot be managed at your facility.

General management

- *If the child has fever (>39° C) give Paracetamol*
- Give Oxygen to all children with wheezing and severe respiratory distress
- Give daily maintenance fluids appropriate for the child's age
- Encourage breast-feeding & oral fluids
- Encourage the child to eat as soon as food can be eaten
-

Treatment

Bronchodilator in Children 1-5 years

If a rapid acting bronchodilator is required drugs of choice: Adrenaline (1:1000) 0.01 ml/kg body weight by subcutaneous (SC) injection up to maximum of 0.25 ml may be repeated after 20 minutes.

Oral bronchodilator (for Children 1-5 years) Salbutamol (O) 0.4 mg/kg/day divided in 3-4 doses for 5 days.

2.2 Asthma

Diagnosis/ Clinical features: Asthma is a reversible obstructive airways disease of varying severity. The symptoms are caused by constriction of bronchial smooth muscle (bronchospasm), oedema of bronchial mucous membrane and blockage of the smaller bronchi with plug of mucus. It can be triggered by factors like allergens, infections, exercise, drugs e.g. Aspirin, tobacco smoke, inhaled chemicals etc. It is characterized by dyspnea, wheezing, tightness of the chest and cough.

Management guidelines

- Maintenance therapy should be adequate
- Treatment of acute attacks
- Avoid heavy exercise

NOTE: The management of asthma in children is similar to that in adults. Infants under 18 months, however, may not respond well to bronchodilator

Asthma attack/ acute asthma

Acute asthma is a substantial worsening of asthma symptoms. The severity and duration of attacks are variable and unpredictable. Assessment of the severity of asthma must be rapidly evaluated using the following clinical criteria (not all signs are necessarily present)

Table 5: Assessment of severity of asthma attack in children ≥2 years & adults

	Clinical Presentation	Treatment (Children & Adults)
MILD-MODERATE ATTACK	<p>Able to talk in sentences</p> <p>Respiratory rate</p> <p>Child 2-5 yrs ≤40/min</p> <p>Child >5 yrs ≤30/min</p> <p><i>And</i></p> <p>No criteria of severity</p>	<p>Salbutamol inhalation¹</p> <p>Give 2-4 puffs every 20-30 min up to 10 puffs if necessary during 1st hour</p> <ul style="list-style-type: none"> - If symptoms completely subside observe for 1-4 hrs, give Salbutamol for 24-48 hrs (2-4 puffs every 4-6 hours) for 3 days - If attack is only partially resolved give 2-4 puffs of Salbutamol every 3-4 hrs if attack is mild; 6 puffs every 1-2 hrs if the attack is moderate, until symptoms subside. When attack completely resolved proceed as above - If symptoms worsen or do not improve, treat as SEVERE ATTACK
SEVERE ATTACK	<p>Cannot complete sentences in 1 breath</p> <p><i>Or</i></p> <p>Too breathless to talk/ feed</p> <p>Respiratory rate</p> <p>Child 2-5 yrs >40/min</p> <p>Child >5 yrs >30/min</p> <p>Adult ≥25/min</p> <p>Pulse</p> <p>Child 2-5 yrs >140/min</p> <p>Child >5yrs >125/min</p> <p>Adult ≥110/min</p> <p>O₂ saturation ≥92%</p>	<p>Admit the patient, place in semi-sitting position</p> <p>Oxygen continuously 5L/min (maintain O₂ saturation between 94-98%)</p> <p>Salbutamol inhalation² 2-4 puffs every 20-30 min up to 10 puffs if necessary in children <5 yrs, up to 20 puffs in children >5 yrs and adults</p> <p>Hydrocortisone injection (IV) 5mg/kg in children, 100mg in adults every 6 hrs until the patient stabilizes, then switch to oral Prednisolone 1-2mg/kg once daily to complete 3 - 5 days of treatment</p> <p>If attack is completely resolved continue with Salbutamol inhalation 2-4 puffs every 4 hrs for 24-48 hours and oral Prednisolone 1-2mg once daily to complete 3-5 days of treatment.</p> <p>If not improving or condition worsens,treat as LIFE-THREATENING ATTACK</p>
LIFE-THREATENING ATTACK	<p>Altered level of consciousness (drowsiness, confusion, coma)</p> <p>Exhaustion</p>	<p>Admit the patient, place in semi-sitting position</p> <p>Oxygen continuously 5L/min (maintain O₂ saturation between 94-98%)</p>

¹ Use a spacer to increase effectiveness. If conventional spacer not available, take a 500ml plastic bottle, insert the mouth piece of the inhaler into a hole on the bottom of the bottle (the seal should be as tight as possible). The child breathes from the mouth of the bottle in the same way as he would with a spacer

Silent chest	Salbutamol nebulizer 2.5 mg for children <5 yrs and in children >5 yrs & adults 2.5-5 mg every 20 -30 min then switch to Salbutamol aerosol when clinical improvement is achieved
Paradoxical thoracoabdominal movement	
Cyanosis	Hydrocortisone injection (IV) 5mg/kg in children, 100mg in adults every 6 hrs
Collapse	
Bradycardia in children or arrhythmia/ hypotension in adults	In adult administer a single dose of Magnesium Sulphate (Infusion of 1 to 2g in 0.9% Sodium Chloride over 20 minutes)
O ₂ saturation <92%	In children use continuous nebulization rather than intermittent nebulisation.

Nocturnal Asthma

Patients who get night attacks should be advised to take their medication on going to bed.

Chronic Asthma in Adults

The assessment of the frequency of daytime and nighttime symptoms and limitation of physical activity determines whether asthma is intermittent or persistent. There are 4 categories (see table).

Therapy is step-wise (Step 1-4) based on the category of asthma and consists of:

- Preventing the inflammation leading to bronchospasm (*controllers*)
- Relieving bronchospasm (*relievers*)

Controller medicines in asthma

- Inhaled corticosteroids e.g. Beclomethasone

Reliever medicines in asthma

- β_2 agonists e.g. Salbutamol (short-acting)

Table 6: Long-term treatment of asthma according to severity

Categories	Treatment
STEP 1 Intermittent asthma - Intermittent symptoms < once/week - Night time symptoms < twice/ month - Normal physical activity	No long-term treatment Inhaled Salbutamol <i>when symptomatic</i>
STEP 2 Mild persistent asthma - Symptoms > once/ week but < once/ day - Night time symptoms > twice/ month - Symptoms may affect activity	Continuous treatment with inhaled Beclomethasone in children <5 yrs 50-200 mcg twice daily; in children >5 yrs and adults 100-250 mcg twice daily Plus

	Inhaled Salbutamol <i>when symptomatic</i>
STEP 3 Moderate persistent asthma <ul style="list-style-type: none"> - Daily symptoms - Symptoms affect activity - Night time symptoms >once/ week - Daily use of Salbutamol 	Continuous treatment with inhaled Beclomethasone in children <5 yrs 200-400 mcg twice daily; in children >5 yrs and adults 250-500 mcg twice daily Plus Inhaled Salbutamol 1-2 puffs four times/day
STEP 4 Severe persistent asthma <ul style="list-style-type: none"> - Daily symptoms - Frequent night time symptoms - Physical activity limited by symptoms 	Continuous treatment with inhaled Beclomethasone in children <5 yrs >400 mcg twice daily; in children >5 yrs and adults >500 mcg twice daily +Inhaled Salbutamol 1-2 puffs four – six times/day

3 0 BRONCHITIS

3.1 Acute bronchitis

It is a self-limited inflammation of the bronchi due to upper airway infection. Acute bronchitis is one of the most common conditions associated with antibiotic misuse. This respiratory condition is generally caused by a virus. Pertussis is the only indication for antibacterial agents in the treatment of acute bronchitis.

Diagnosis

- Patients with acute bronchitis present with a cough lasting more than five days (typically one to three weeks), which may be associated with sputum production.
- Acute bronchitis should be distinguished from chronic bronchitis (see below), it is not a form of COPD.

Symptomatic treatment

- with non-steroidal anti-inflammatory drugs: Paracetamol, Aspirin
- cough suppressant syrups
- There is NO benefit from antibiotic use

3.2 Chronic Bronchitis

It defined by a chronic productive cough for three months in each of two successive years in a patient in whom other causes of chronic cough have been excluded. Patients may get secondary bacterial infection with development of fever and production of thick smelly sputum.

Non Pharmaceutical Treatment

- Stop smoking and/or remove from hazardous environment
- Prompt treatment of infective exacerbations

- Controlled oxygen therapy
- Physiotherapy
- Bronchodilator may give some benefit

Pharmaceutical Treatment

- Give β -agonist e.g. Salbutamol (O) 4-8mg 6 – 8 hourly
OR
D: Ipratropium bromide aerosol 20 – 80mg, 6 – 8 hourly
- Trial of steroids if there is any possibility of reversible airways obstructions
A: Prednisolone (O) 20mg once daily for 5 days

4.0 Chronic obstructive pulmonary disease (COPD)

Definition.

Chronic obstructive pulmonary disease (COPD) is a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible (WHO)

Clinical symptoms and signs.

- Chronic cough and sputum production often precede the development of airflow limitation by many years.
- Abnormal shortness of breath and increased forced expiratory time

A COPD diagnosis

- **Diagnosed** based on factors such as signs/symptoms, patient history, physical examination, chest X-rays.
- Is confirmed by a simple test called spirometry, which measures how deeply a person can breathe and how fast air can move into and out of the lungs.

COPD Exacerbations: *a sustained worsening of the patient's condition, from the stable state and beyond normal day-to-day variations, that is acute in onset and necessitates a change in regular medication in a patient with underlying COPD.*

Additionally, a generalized sub classification of exacerbations based on health-care utilization is proposed.

- Mild: patient has an increased need for medication, which he/she can manage in his/her own normal environment.
- Moderate: patient has an increased need for medication, and he/she feels the need to seek additional medical assistance.
- Severe: patient/caregiver recognizes obvious and/or rapid deterioration in condition, requiring hospitalization.

Clinical signs and symptoms.

- Increased dyspnoea,
- Productive cough with altered sputum
- Fever.
- Alternatively, the symptoms may be more nonspecific, such as malaise, fatigue, insomnia or sleepiness, and depression.

The major diseases included in this category are:

- **Chronic bronchitis** - a chronic, inflammatory condition of the bronchi characterized by coughing and expectoration (spitting-up) of sputum (mucous coughed-up from the lungs) occurring on most days and lasting 3 months or longer for at least two consecutive years.
- **Emphysema** - a respiratory disorder that is characterized by enlargement and eventual destruction of the air sacs (alveoli) in the lungs, through which oxygen passes from the lungs into the bloodstream.
- **Bronchiectasis** is characterized by inflamed and easily collapsible airways, obstruction to airflow, and frequent hospital visits and admissions. (See below).
- Although **asthma** is also a condition that is associated with airway obstruction, and many people with COPD also suffer with asthma, as a general rule, asthma is not included under the category of COPD.

Non pharmacological treatment:

The role of **supplemental treatments** in the management of patients with COPD, including:

- Pulmonary rehabilitation
- Patient education
- Psychosocial support
- Nutrition
- Supplemental oxygen therapy

Pharmacological treatment

- The major types of **medications** that are often prescribed for patients with stable COPD, which include:
- Inhaled bronchodilators
- Inhaled corticosteroids
- Theophylline.

Surgical treatment options for the treatment of patients with **advanced emphysema**, which include:

- Bullectomy
- Lung-volume reduction surgery
- Lung transplantation

5.0 OTHER RESPIRATORY INFECTIONS

5.1 Acute laryngo-tracheobronchitis/Croup

Clinical features: Croup is acute inflammation of the larynx, trachea and bronchi which occurs in young children (usually between 6 months to 3 years of age). It arises as a result of narrowing of the airway in the region of the larynx. The most common cause is viral infection (particularly parainfluenza viruses) but may also be due to bacterial infection. The obstruction is due to inflammation and oedema.

Diagnosis

- The symptoms include paroxysmal “barking” cough, inspiratory stridor, fever, wheezing, hoarseness of voice and tachypnoea
- Such symptoms usually occur at night
- Respiratory failure and pneumonia are potentially fatal complications.

General management

- Prevent asphyxiation
- Treat inflammatory edema
- Humidification of inhaled air
- Hospitalization may be necessary

Note

- No stridor at rest, give no antibiotics
- Stridor at rest or chest in-drawing or fast breathing REFER IMMEDIATELY to hospital

Mild Croup

- Only stridor when upset, no moderate/severe ARI
- Likely of viral origin
- Home care – steam inhalation
- Antibiotics NOT required

Severe Croup

- Likely bacterial origin
- Stridor in a calm child at rest
- Chest in drawing
- Antibiotics are NOT effective and should not be given

Treatment

Admit to hospital, give Oxygen therapy to all patients with chest in-drawing (using nasal prongs only, DO NOT use nasopharyngeal or nasal catheter) until the lower chest wall in-drawing is no longer present

C: Dexamethasone 0.6 mg/kg orally daily in 1-2 divided doses

Plus

C: Nebulized Adrenaline (400 mcg/kg) every 2 hours if effective; repeat after 30 min if necessary.

5.2 Laryngeal Diphtheria

Is an infection caused by *Corynebacterium diphtheria*; it is directly transmitted from person to person by droplets. Children between 1-5 years of age are most susceptible although non-immune adults are also at risk.

Diagnosis

Diphtheria is characterized by grayish-white membrane, composed of dead cells, fibrin, leucocytes and red blood cells as a result of inflammation due to multiplying bacteria.

General management

- Isolate the child
- Gently examine the child's throat – can cause airway obstruction if not carefully done.
- NGT for feeding if unable to swallow
- Avoid oxygen unless there is incipient airway obstruction
- May need tracheostomy if there is incipient airway obstruction

Treatment

Drug of choice

A: Penicillin V (250 mg four times daily) for a total treatment course of 14 days

OR

A: Erythromycin 125-250 mg every 6 hours for 14 days

OR

C: Azithromycin 125-500mg daily for 3 days

OR

A: Penicillin G (Benzyl Penicillin) 25,000 to 50,000 units/kg to a maximum of 1.2 MU IV every 12 hours until the patient can take oral medicine)

Plus

Diphtheria antitoxin (IM or slow IV) dose depends upon the site and severity of infection:

- First give a test dose of 0.1ml of 1 in 10 dilution of antitoxin in 0.9% Sodium Chloride intradermally to detect hypersensitivity
- It should be given immediately because delay can lead to increased mortality
- The dose should be administered intravenously over 60 minutes in order to inactivate toxin rapidly
- 20,000 to 40,000 units for pharyngeal/laryngeal disease of <48 hours duration,
- 40,000 to 60,000 units for nasopharyngeal disease
- 80,000 to 120,000 units for >3 days of illness or diffuse neck swelling ("bull-neck")

NOTE Tracheostomy may be required for airways obstruction

5.3 Whooping Cough

It is a highly infectious childhood disease caused by *Bordetella pertussis*. It is most severe in young infants who have not yet been immunized.

Diagnosis

- After an incubation period of 7 –10 days, the child develops fever, usually with a cough and nasal discharge which are clinically indistinguishable from a common cough and cold

- In the second week, there is paroxysmal coughing which can be recognized as pertussis
- The episodes of coughing can continue for 3 months or longer
- The child is infectious for a period of 2 weeks up to 3 months after the onset of illness
- The main clinical feature is paroxysmal cough associated with a whoop.

General management

- During paroxysms of coughing, place the child head down and prone, or on the side, to prevent any inhaling of vomitus and to aid expectoration of secretions.
- Care for the airway but avoid, as far as possible, any procedure that could trigger coughing, such as application of suction, throat examination
- Do not give cough suppressants, sedatives, mucolytic agents or anti-histamines.
- If the child has fever ($>38.5^{\circ}\text{C}$) give paracetamol.
- Encourage breastfeeding or oral fluids
- Whooping cough is preventable by immunization with pertussis vaccine contained in DPT triple vaccine.
- Admit infants aged less than 6 months to hospital; also admit any child with pneumonia, convulsions, dehydration, severe malnutrition, or prolonged apnoea or cyanosis after coughing.

Treatment

Antibiotics

A: Erythromycin 12.5 mg/kg (PO) every 6 hours for 10 days.

This does not shorten the illness but reduces the period of infectiousness

If there is fever or if erythromycin is not available

A: Chloramphenicol 25 mg/kg (PO) every 8 hours for 5 days

Oxygen

Give oxygen to children who have spells of apnoea or cyanosis, or severe paroxysms of coughing.

- Use nasal prongs, not a nasopharyngeal catheter or nasal catheter which can provoke coughing.

5.4 Bronchiectasis

Bronchiectasis is characterized by inflamed and easily collapsible airways, obstruction to airflow, and frequent hospital visits and admissions.

Diagnosis

The diagnosis is usually established clinically on the basis of chronic daily cough with viscid sputum production, and radiographically by the presence of bronchial wall thickening and luminal dilatation on chest x-rays.

General management

- Antibiotics are used to treat an acute exacerbation and prevent recurrent infection by suppression or eradication of existing flora.
- Physiotherapy and postural drainage
- Avoid smoking

- Respiratory care during childhood measles helps prevent the development of bronchiectasis in children

Treatment

Management of bronchiectasis is aimed at treating the underlying cause (eg, removal of an airway foreign body or treatment of aspiration or humoral immunodeficiency), improving mucocilliary clearance, treating and preventing infection, and controlling inflammation.

Acute exacerbation

Adults

A: Ciprofloxacin 500mg every 12 hours for 7-10 days

Plus

A: Metronidazole 500mg every 8 hours for 7-10 days

Children:

A: Amoxicillin 40mg/kg (O) in 3 divided doses for 5-7 days

Plus

A: Metronidazole 7.5 mg/kg every 8 hours for 5-7 days

Prevention of infection

A: Ciprofloxacin 500mg (PO) once daily for 7 – 14 days/ month

OR

A: Erythromycin (PO) once 250-500mg for 7-14days/month

5.5 Lung abscess

Lung abscess is a cavity within the lung parenchyma filled with necrotic tissues which occurs as a result of tissue-destroying infection.

Diagnosis

It is characterized by high fever, breathlessness, cough productive of large amounts of foul-smelling sputum and haemoptysis.

General management

Postural drainage

Treatment

C: Ampicillin (start with IV then oral) 500-1000mg every 8 hours for 4-6 weeks (children 50mg/kg/dose)

Plus

C: Metronidazole start with IV then oral 500 mg every 8 hours for 4-6 weeks (children 7.5mg/kg)

CHAPTER FIVE

OBSTETRICAL/GYNAECOLOGICAL DISEASE CONDITIONS &CONTRACEPTION

1.0 INFECTION OF THE GENITAL-URINARY TACT

1.1 Urinary Tract Infection during Pregnancy

Diagnosis

Whenever possible urine specimen for microscopy, and/ or culture and sensitivity tests should be carried out before drug are initiated, except on acute conditions.

First Line:

A:Amoxycillin (**O**)500 mg every 8 hours for 5 days

Second Line:

A: Nitrofurantoin (**O**) 100 mg every 6 hours for 5 days with food
Plus

C: Amoxicillin +Clavulanic acid 625mg (O) 8hrly for 5 days

For Positive RPR or Syphilis during pregnancy

B: Benzathine penicillin B (IM) 2.4 MU weekly 3 doses.

For Penicillin **allergic patients**

A: Erythromycin (O) 500 mg every 6 hours a day for 14 days
OR

C: Azithromycin 500mg daily for 3 days

1.2 Vaginal Discharge during Pregnancy

Vaginal discharge during pregnancy can be physiological or due to infection.(Bacterial, fungal or both). The infection is usually polymicrobial and necessitates the use of combined drugs.For bacterial infections treatment options are:

A: Erythromycin (O) 500 mg every 8 hours for 10 days
OR

C: Azithromycin 500mg daily for 3 days
Plus

A: Metronidazole (O)400 – 500 mg every 8 hours for 8 hours for 7 days

For fungal infection (vaginal candidiasis) give:

A: Clotrimazole vaginal pessaries one noct for 6 days
OR

C: Miconazole vaginal pessaries once daily for 3 days

CAUTION!!

- Avoid taking both drugs concomitantly if sides effects are intolerable
- Avoid metronidazole in the first trimester
- Avoid alcohol while taking metronidazole

2.0 ABORTION

It is interruption of pregnancy (expulsion of a fetus) before it is viable, legally at 28th week of gestation. Clinical types are recognized according to findings when the patient is first seen. These include: Threatened abortion, inevitable abortion, incomplete abortion, complete abortion and missed abortion.

Diagnosis

- Clinical features will depend on the types of abortion
- Viginal bleeding which may be very heavy in incomplete abortion, intermittent pain which ceases when abortion is complete and cervical dilation in inevitable abortion
- In missed abortion, dead ovum retained for several weeks while symptoms and signs of pregnancy disappear
- When infected (septic abortion) patient presents with fever tachycardia, offensive vaginal discharge, pelvic and abdominal pain.

Puerperal/Post abortal Sepsis

Pyrexia in women who has delivered or miscarried in the previous 6 weeks may be due to puerperal or abortal sepsis and should be managed actively. Abdominal pain in addition to pyrexia is strongly suggestive. The uterus may need evacuation however parenteral antibiotics must be administered before evacuation.

C: Ampicillin (I.V)1gm start

Plus

A: Metronidazole 500mg

Plus

A: Gentamycin 80mg stat

Patient should continue with the following oral antibiotics after evacuation for 5 to 7days

For Mild/moderate

A: Amoxicillin (O) 500mg every 8 hours for 10 days

Plus

A: Metronidazole (O)400 mg every 8 hours for 10 days

Plus

A: Doxycycline (O)100 mg every12hrs for 10 days

Treatment Guidelines for severe cases

- Body temperature higher than (38⁰C)
- Marked abdominal tenderness are signs of severe post abortal sepsis

Drug of Choice:

A: Benzylpenicillin (I.V)2MU every 6 hours

Plus

B: Chloramphenicol (I.V) 500 mg every 6 hours
Plus
A: Metronidazole (O) 1 g twice daily

Note: If patient cannot swallow continue with parenteral treatment give Metronidazole 1 gm (PR) twice daily or IV/500 mg every 8 hours

Choice for parenteral antibiotics:

C: Ampicillin (IV) 500 mg every 6 hours
Plus
A: Gentamicin (IM) 80 mg every 8 hours
Plus
A: Metronidazole (O) or (PR) 1 g twice daily for the duration of 5 to 7 day

Note: Pelvic abscess may be suspected if after 48 hours no response, in this case laparotomy or referral may be necessary

3.0 PREMATURE RAPTURE OF MEMBRANE

A) Prolonged Premature Rapture of Membrane (PROM): Rupture of membranes before onset of labour.

B) Pre – term premature rupture of membrane (PPROM): Rupture of membranes before term i.e. 37 completed weeks

Diagnosis/ clinical features

It characterized by leakage of watery fluid per vagina confirmed by performing a sterile speculum examination.

General management

Give (IV) fluids Ringer’s Lactate OR Normal saline

Prolonged PROM for more than 12 hrs is a risk of ascending infection which leads to chorioamnionitis (infection of chorion amnion and amniotic fluid)

Treatment

- PROM at term: Delivery with 24hrs
- PPRM: If no sign of infection, wait for foetal maturity and give prophylaxis

A: Amoxyllin 500mg (O) 6 hourly x 10days

OR

A: Erythromycin 500mg (O) 6 hourly 10 days.

If there are signs of infections-pyrexia, foul smelling liquor (chorioamnionitis)

C: Ampicillin 1g (IV) stat then 500mg 6 hourly for 5 to 7 days

OR

D: Ceftriaxone 1g (IV) daily for 5 days

OR

A: BenzylPenicilline (IV) 2MU every 6hrs

OR

C: Chloramphenicol (I.V) 500mg every 6 hours

Plus

A: Metronidazole 500mg 8hrly for 5 days

For urgent Delivery irrespective of gestational age

A: Benzylpenicillin (I.V) 2MU every 6 hours

Plus

C: Chloramphenicol (I.V) 500 mg every 6 hours until the patient is able to take oral medication.

4.0 PROPHYLAXIS FOR CAESARIAN SECTION

Prophylactic use of antibiotics in women undergoing caesarean section reduces the risk of infection-related complications and serious infection post operation.

Thirty minutes before operation

C: Ampicillin 1 g (I.V)

Plus

C: Metronidazole 500mg (I.V) start

OR

D: Ceftriaxone 1g (I.V) start.

Immediately before operation give

A: Benzylpenicillin (I.V) 5MU as a single dose

Plus

C: Chloramphenicol (I.V) 1 g as single dose. Continue with antibiotics after delivery for 3-5 days

Note: Use of antibiotics for prophylaxis during surgery, should be evaluated from situation to situation and not generalized

5.0 NAUSEA AND VOMITING IN PREGNANCY

Nausea and vomiting of pregnancy is the most common medical condition in pregnancy women. It commonly occurs between 5 and 18 weeks of pregnancy.

Management

- If vomiting is not excessive, advise to take small but frequent meals and drinks
- If persistent, vomiting cases, search for other reasons e.g. malaria, UTI, Multiple pregnancy or molar pregnancy and gastritis
- Otherwise give:-

Drug of Choice:

A: Promethazine (O) 25 mg at night

OR

C: Metochlopramide (O)10mg 8hrly

OR

A: Chlorpheniramine (**O**) 4mg at night

In Severe cases

General management

Give Ringers Lactate depending on severity of dehydration; If possible check for electrolyte imbalance.

Medicine of choice:

A: Promethazine (I.V) 25 - 50 mg 12 hrly

OR

C: Metochlopramide 10mg (I.V/I.M) 8hrly

PLUS

C: Omeprazole 20mg 12hrly (caution of its use in first trimester)

OR

D: Prochlorperazine (O) 5 mg up to 3 times per day

For Hyperemesis Gravidarum (Vomiting and dehydration): Admit and give

A: Dextrose 5% IV then Ringer lactate + Dextrose normal saline

Plus

A: Promethazine (I.M) 25 mg twice daily

OR

D: Prochlorperazine (I.M) 12.5 mg twice daily.

6.0 ANAEMIA DURING PREGNANCY

Definition: Hemoglobin level less than 11g/dl; Mild anaemia 9 – 11 g/dl; Moderate 7-8.9 g/dl; Severe less than 7g/dl

Investigate for the following in case of anaemia

- Stool for ova and parasites
- Full blood count (FBC)
- Peripheral blood film for malaria parasites
- Urine for microscopy, culture and sensitivity test
- HIV test

Prophylaxis in antenatal Care

A: Ferrous sulphate (**O**) 200 mg 2-3 times per day

Plus

A: Folic acid (**O**) 5mg once daily

CAUTION!! -Ferrous sulphate should be taken with or after food
-Where vomiting is experienced reduce dosage to tolerable level

Treatment for Mild to moderate anaemia

A: Ferrous sulphate (O) 200 mg 2-3 times per day

Plus

A: Folic acid (O) 5mg once daily

General management for Severe Anaemia

- Admit to the hospital
- Give blood transfusion slowly
- Give frusemide 40mg- 80mg before blood transfusion
- Continue with haematinics as above

If patient has severe anemia in pregnancy the following clinical investigation should be done:

- Stool for ova and parasites
- Full blood count (FBC)
- Peripheral blood film for malaria parasites
- Urine for microscopy, culture and sensitivity test
- HIV test

7.0 HYPERTENSION IN PREGNANCY

7.1 Chronic Hypertension

This is also called primary hypertension / chronic hypertension where elevation of blood pressure occurs before pregnancy. systolic pressure raises to 140 – 159 mmHg and/or diastolic pressure of 90 – 99 mmHg. The underlying cause of primary hypertension is not clear.

Drug of Choice:

A: Methyldopa 250 – 500 mg (O) every 6-8 hours daily

7.2 Pregnancy Induced hypertension (PIH)

- Rise in blood pressure during pregnancy of $\geq 140/90$
- **Pre eclampsia:** Rise in blood pressure during pregnancy **PLUS** proteinuria
- **Eclampsia** Occurrence of convulsion (fits) in patient with pre eclampsia where other causes of convulsion have been excluded

Treatment of Mild to moderate pre eclampsia

General measures

- Regular check of BP
- Monitoring of foetal wellbeing
- Monitoring of proteinuria
- Advice on adequate rest
- Advise on regular use of cocoa containing food
- Exclude UTI
- Check urine for protein
- Count this as a high risk antenatal patient

Medicine

A: Methyldopa 250-500mg 8 hrly

OR

C: Nifedipine 10 mg 12 hourly

Severe pre eclampsia

Criteria for diagnosis: Blood pressure \geq 160/110; Severe headache, Epigastric/ retrosternal pain, Blurring of vision, Hyperreflexia, Oliguria, Proteinuria \geq 5g/ 24hrs collection (\geq +3 in dip stick) and Intra uterine growth restriction (IUGR).

General measures

- Admit in the hospital
Give

B: Normal saline

Plus

C: Nifedipine 10-20 mg 12 hrly;

Plus

C: Hydralazine 10 mg (I.V) slowly

Plus

B: Magnesium sulphate 4gm (IV) in 20 mls of normal saline for 10-15 min followed by 5gm of 50% MgSO₄ in each buttock; Followed by 4gm of MgSO₄ in 250 mls of normal Saline to run over 4hrs. Maintenance dose: 4gm of MgSO₄ (IM alternative buttock) 4hourly for 24hrs.

Deliver as soon as the BP is controlled.

Note: MgSO₄ regimen should continue until 24 hrs after the last fit.

Eclampsia

General principle

- Control fits
- Control Blood pressure
- Deliver

General measures

- Keep the airway clear
- Fluid and electrolyte balance

Treatment

- Give magnesium sulphate as above
- Give antihypertensive as above
- Fluid management as above
- Deliver vaginally unless there is another obstetric indication for caesarean delivery

Mild PIH

Diastolic: 90 – 100 mm and no proteinuria

Advice bed rest

- Weekly antenatal clinic visits
A: Acetylsalicylic acid (O) 75 mg once daily

Moderate PIH

Diastolic: 100-110 mm, no proteinuria

Treatment

A: Acetylsalicylic acid (O) 75 mg once daily. Plan immediate delivery at gestation > 37 weeks

Admit and monitor BP up to 6 times per day, and give

A: Methyldopa (O) 250 – 500 mg every 6-8 hours daily

Severe PIH

Diastolic>110

Treatment

C: Nifedipine (Sublingually) 10 mg

The need for more doses indicates the urgency for delivery.

Pre-Eclámptica Toxemia (Proteinuria PIH)

Management

- Exclude UTI
- Check urine for protein daily
- Plan delivery at 37 weeks or before

Treatment

A: Acetylsalicylic acid 75 mg once daily

Plus

C: Hydralazine (IM) 12.5 mg

OR

C: Nifedipine (sublingual) 10 mg.

Imminent Eclampsia

This is proteinuria PIH characterized by visual disturbance or epigastria pain and or signs of brisk reflexes.

Management

- Plan urgent delivery
- Prevent convulsions by
 - A:** Diazepam (I.V – infusion) 40 mg diluted in 1 litre of Sodium chloride 0.9% over 6 hours

Treatment

If diastolic pressure still >110 mm give antihypertensive:

C: Hydralazine 12.5 (I.M) intermittently

OR

C: Nifedipine (sublingually) 10 mg.

Eclampsia (Proteinuria PIH with Fits)

Treatment

A: Diazepam (IV infusion) 40 mg diluted in 1000 ml of normal saline infused over 6 hours

- If diastolic pressure > 110 mm give antihypertensive as above
- Plan urgent delivery

8.0 DIABETES IN PREGNANCY

Gestational diabetes develops in women during pregnancy because of insulin resistance or insensitivity due to steroid hormones produced from the placenta. High blood sugar levels in the mother's body are passed through the placenta to the developing baby. This can cause health problems. Gestational diabetes usually begins in the second half of pregnancy and goes away after the baby is born.

Management

- Diabetic pregnant women require management before and throughout pregnancy
- Diabetes should be controlled by diet, oral hypoglycaemics and or Insulin
- Throughout pregnancy blood sugar should strictly be within the range of 4-6 mmol/L
- Insulin requirement will increase as pregnancy progresses
- During labour check blood sugar 4hourly in order to detect hypoglycaemia and manage accordingly
- When labour induced give half the usual insulin dose first and start on IV infusion of dextrose 5% at 125 ml per hour
- Manage the patient on a sliding scale of insulin after labour
- Continue to monitor blood sugar after delivery in order to adjust insulin requirement

9.0 HEART BURN IN PREGNANCY

Heartburn (also called acid indigestion or acid reflux) is a burning sensation that often extends from the bottom of the breastbone to the lower throat. It's caused by some of the hormonal and physical changes in pregnant women.

Management

Pregnant women should avoid:

- Food and beverages that cause gastrointestinal distress
- Tobacco and alcohol
- Eating big meals; should eat several small meals throughout the day
- Drinking large quantities of fluids during meals
- Eat close to bedtime; they should give themselves two to three hours to digest food before they lie down
- Sleep propped up with several pillows or a wedge. Elevating upper body will help keep the stomach acids where they belong and will aid food digestion.

Treatment

A: Magnesium trisilicate (O) as needed

OR

C: Omeprazole 20 -40 once a day

10.0 RESPIRATORY DISTRESS SYNDROME

Respiratory Distress Syndrome is likely to occur in newborn and in premature labour before 36 weeks gestation.

Drug of choice

B: Hydrocortisone (IV) 250 mg repeats after 24 hours

OR

D: Dexamethasone (IV) 12 mg, two doses at an interval of 12 hours.

Note: If no delivery the course can be repeated after one week

CATION!!: Anemic patients under Beta stimulants and steroids are inclined to congestive cardiac failure

11.0 STIMULATION OF LABOUR AND MYOMETRIAL RELAXATION

- Myometrial stimulants should be used with great care before delivery especially in porous women
- Use in obstructed labour should be avoided
- Oxytocics are indicated for:-
 - augmentation of labour
 - Induction of labour
 - Active management of third stage of labour.
 - Uterine stimulation after delivery

11.1 Labour Induction

For induction of labour use: Oxytocin IV the dose will depend on parity.

- Primigravida:
 - A:** Oxytocin IV 5 IU in 500mls of fluid titrate at 15, 30, 60 drops per minute until desired uterine contractions are attained
- Multiparous:
 - A:** Oxytocin IV Starts with low dose eg 1.25 IU in 500mls of fluid titrate as above. Regulate the dose according to response.

If no progress of labour is achieved give;

A: Oxytocin (IV) Initially 1 unit then 4 units in 1 litre Normal Saline at 15, 30, 60 drops per minute until regular contractions lasting for more than 40 seconds are maintained

When 4 units are not enough to cause maintained contractions, and it is first pregnancy, the dose can be increased to 16, 32 then 64 units in litre of Normal Saline each time increasing the delivery rate through 15, 30 and 60 dpm.

11.2 Augmentation of Labour

If labour progress is not optimum labour augmentation is necessary. Can be achieved by:

A: Oxytocin as above

OR

Artificial rupture of membranes and Oxytocin

- If the membranes already ruptured and no labour progressing, the steps above should be followed
- Obstructed labour could be the cause of labour failure.

Note: Rule out obstruction before augmenting labour with oxytocin

11.3 Myometrial Stimulation after Delivery

Post partum hemorrhage (PPH)

It is an excessive bleeding of more than 500ml after the third stage of labour and a major cause of maternal morbidity and mortality.

Major causes are;

- Uterine atony
- Tears of the vagina/vulva
- Retained products of conception
- Rarely rupture of the uterus
- Bleeding disorder (e.g coagulopathies, DIC)

Management

In order to prevent the occurrence of this condition, active management of the third stage of labour (ATMSL) is mandatory. This involves the injection of an oxytocic after the delivery of the foetus followed by controlled cord traction and uterine massage.

Treatment

Drugs of Choice:

A: Oxytocin (I.M) 10 I.U.

OR

A: Ergometrine (I.M) 0.25 – 0.5 mg

OR

A: Misoprostol 800 -1000 microgram (mcg) orally/rectally

Give **Oxytocin (I.M)** 5 units after delivery of the infant; when no response gives Oxytocin (I.V infusion) 10-20 units in 1 litre of NS running at 10-20 drops per minute (dpm)

Second Choice: Ergometrine (IM) 0.5 mg after delivery of the infant, in the absence of myometrial contraction and to prevent postpartum hemorrhage

Note: Use Ergometrine cautiously in hypertensive heart disease patients.

11.4 Myometrial Relaxation

This is done to relax the uterus in order to:

- Relieve fetal distress immediately prior to cesarian section
- Stop contraction of uterine in premature labour
- Prevent uterine rupture
- Perform external cephalic version

Drug of Choice **A:** Salbutamol 4 mg (O) every 8 hours

Note

β -stimulants should **NEVER** be used if the patient had an antepartum hemorrhage
 β -stimulants are **CONTRA-INDICATED** for the following

- With cardiac disease
- Severe anemia in pregnancy

12.0 TERMINATION OF PREGNANCY

Abortion is illegal in Tanzania except under the following legal conditions:

- Where there is a substantial threat to the woman's health or life in continuing the pregnancy
- Where there is a significant risk or it is known that the foetus has a serious medical conditions or malformation
- Where the pregnancy results from rape and there is no intention to keep the pregnancy.

Recommended methods

- Routine Dilation and curettage - up to 7 weeks since last menstrual period
- Suction termination – Between 7-12 weeks since the last menstrual period
- Prostaglandin termination – after 12 weeks since the last menstrual period.

13.0 PREGNANCY AND LACTATION

General Guidelines

- All drugs, if possible, should be avoided during the first trimester
- Well known medicine and their use in pregnancy and lactation, which have been documented as safe, should be preferred – AVOID new drugs
- Absence from a list of medicine not to be used in pregnancy or lactation does not guarantee safety
- During pregnancy and lactation, medicines should be prescribed only if benefit overweighs risk to the foetus or neonate.

14.0 PELVIC INFLAMMATORY DISEASES

Pelvic inflammatory disease (PID) occurs when there is infection in the female reproductive organs. The infection can happen as an ascending infection from the vagina, after delivery (puerperal sepsis), after an abortion (septic abortion), postmenstrual or after Dilation and Curettage (D&C) operation. The common causative organisms are *Neisseria gonorrhoea*, *Chlamydia trachomatis* and *Mycoplasma hominis*. Endogenous bacteria e.g. gram-negative aerobes and anaerobes like bacteroides, streptococcus, anaerobic streptococcus and E. coli may also cause PID. The condition can either be acute, sub-acute or chronic.

Diagnosis

The main clinical features are lower abdominal pain, backache, vomiting, vaginal discharge, menstrual disturbance, dyspareunia, fever, infertility and tender pelvic masses. PID predisposes to ectopic pregnancy.

Treatment

In acute PID gives Intravenous fluid (Ringers Lactate or Normal saline)

A: Ciprofloxacin (O) 500mg single dose,

Plus

A: Doxycycline (O) 100 mg every 12 hourly for 10 days

Plus

A: Metronidazole (O) 400 – 500 mg every 8 hours for 10 days.

Give an appropriate analgesic depending on the severity of the disease:

A: Diclofenac 50-100 mg every 8 hours preferably after food

OR

A: Acetylsalicylic acid (O) 600 mg every 8 hours preferably after food

OR

A: Paracetamol (O) 500mg, 8 hourly.

In chronic PID

Give an appropriate analgesic diclofenac, ibuprofen aspirin or paracetamol depending on the severity of the pain. Do not give antibiotics.

15.0 HORMONAL CONTRACEPTION

Oral contraceptives (oestrogen – progestogen combinations) are used primarily for prevention of conception. It may also be used in treatment of dysfunctional uterine bleeding, dysmenorrhea or endometriosis.

The goal of therapy in the use of these products for contraception is to provide optional prevention of pregnancy while minimizing the symptoms and long term risks associated with excess or deficiency of the oestrogen and progestogen components. The eligibility for hormonal contraception can be obtained from nearest family planning clinic or unit.

15.1 Oral Contraceptives

They fall into two major categories:

Combined oral contraceptives) COCs)

A: Oestrogen 30 – 35 micrograms (as ethinyloestradiol)

“Low Dose”

A: Oestrogen 50 micrograms + progestogen

“ High Dose”

“Triphasic pills” – contain phased levels which closely mimic normal cyclical hormonal activity

- Lower oestrogen dose pills cause fewer side effects than higher dose pills
- Mid-cycle spotting in patients on 30 microgram COCs can be managed by changing to 50 microgram COCs
- Menstruation on COCs will be regular, light and short

Progestogen Only Pills (POPs)

These contain norethisterone, or norethindrone or levonorgestrel. This type is suitable for lactating mothers or women with mild or moderate hypertension. Menstrual irregularity is a more common side effect.

Management

Follow up:

- Instruct women always to inform the doctor or nurse that they are on contraceptives while attending clinic or hospital.
- Women on Oral Contraceptives need regular physical check-ups including blood pressure measurement every six months e.g. if women develop depression after starting OC.

Need to Withdraw COCs or POPs

- Pregnancy
- Severe headaches especially associated with visual disturbances
- Numbness or paresis of extremities
- Unexplained chest pain or shortness of breath
- Severe leg pains
- Development of any of the absolute contra-indication conditions

Medicines Reducing the Effect of Oral Contraceptives

The following drugs are likely to reduce the effectiveness of OCs and woman may become pregnant so the woman should be advised to use additional prevention method such as condom.

- **Hypnotic/sedatives** anti-migraine medication, barbiturates, chloral hydrate, diazepam
- **Antacid:** Aluminium hydroxide, magnesium hydroxide, magnesium trisilicate
- **Anti TB as rifampicin**
- **Antiretroviral** as Nevirapine and Ritonavir
- **Certain antibiotics** as Ampicillin and other Penicillins and Tetracyclines.

Note

- For long term use of these drug "High Dose" COCs – 50 micrograms should be used or other method of contraception

Drug made less effective by Oral Contraceptives

Prescribers might consider increasing the doses of the following drugs, known with careful monitoring

- Anticonvulsant
- Ant diabetic agents
- Anticoagulants
- Antihypertensive agents (methyldopa)
- Corticosteroid
- Hypnotics, sedatives or other CNS depressants

Post Coital Contraception ("morning-after pill")

The method is applicable mostly after rape and unprotected sexual intercourse where pregnancy is not desired. Within 3 days (72 hours) of unprotected sexual intercourse, give

A: Combined oral Contraceptive ethinyloestradiol 100 mcg and levonorgestrel 500 mcg (2 high dose COC tablets)

OR

A: Ethinyloestradiol 30-35 mcg and levonorgestrel 150-250 mcg -3 tablets (3 low dose COC tablets).

- Repeat this dose after twelve hours
- Advice to return to physician if menstruation does not occur within 3 weeks
- Give advice on contraceptive use
- Rape victims should also be given Erythromycin (O) 250 mg every 6 hours for 5 days
- Offer counseling

Long Term Hormonal Contraceptives

These contraceptives should be prescribed by Medical Doctors only or trained family planning staff.

Injectable Contraceptive:

A: Medroxyprogesterone acetate IM 150 mg every three months

CAUTION!! Avoid use in for severe hypertension and in women without proven fertility

Implant Contraceptive (see FP manual for current implants in use)

“Norplant” Containing levonorgestrel in six silastic capsules is implanted in the left upper arm made local anesthesia.

“Norplant” Is effective for five years and is recommended for women who have completed their family or nor ready for sterilization or those not able to take oestrogen containing contraceptives.

Contraindications for Norplant

- Severe hypertension
- Thromboembolism
- Active liver disease
- Sickle cell anaemia
- Undiagnosed genital bleeding
- Severe headaches

16.0 ANTEPARTUM HAEMORRHAGE (APH)

CAUTION!! all patients with APH must be managed in the hospital setting

Diagnosis

- Bleeding from the birth canal after the 28th week of gestation
- Main forms are placenta praevia and abruptio placenta
- Bleeding is painless in placenta praevia
- Bleeding may be visible or concealed in abruptio placenta
- Pain and shock in abruptio placenta correspond with degree of separation
- **Placenta praevia**

Placenta attached at the lower segment characterized by painless vaginal bleeding

- **Abruption placenta**

Premature separation of the placenta characterized by severe abdominal pain, shock, foetal distress or foetal death.

General management

If patient is bleeding heavily or in shock:

- Vital signs (BP, pulse, temperature)
- IV line (two are better than one)
- Take blood (Grouping and cross matching, FBC, platelet count)
- Give (I.V) fluids quickly Ringer 's Lactate
- Give oxygen
- Send somebody for two or more units of blood
- Indwelling catheter
- Do ultrasound; if no placenta praevia, speculum and vaginal examination
- If rapid vaginal delivery is considered, prepare vacuum
- Add Oxytocin and amniotomy
- In most cases of placenta praevia CS is indicated. Give antibiotic prophylaxis before CS: Ampicillin 1g I.V (single dose)PLUS Metronidazole 500mg I.V (single dose)

If patient in good condition:

- Observe closely for signs of worsening
- Do ultrasound; if no placenta praevia, speculum and vaginal examination
- Consider prolongation of pregnancy to term

Follow Up after delivery

- Close monitoring (vital signs, shock symptoms, uterus size and consistency)
- Check Hb 48 hours after delivery
- Inform patient about history (risks for further pregnancies, mode of delivery)
- Discuss possible modes of contraception before discharge

Management of Abruptio Placentae

- Open 2 IV lines
- Give Ringer Lactate or Normal Saline quickly
- Catheterization
- Blood grouping and crossmatch order enough blood
- Bed side clotting time
- In most cases there is already IUFD Induce with amniotomy and oxytocin infusion
- Give adequate analgesia

NB if the baby is alive at term or near term consider CS

- Expectant therapy
- Allow bed rest
- Blood grouping and cross-matching
- Active therapy delivery if foetus viable. If a major placental separation has occurred, emergency delivery to minimize the possibility of disseminated
- Intravascular coagulation

- Give blood when indicated.

17.0 DYSMENORRHOEA

Dysmenorrhoea is painful menstruation preventing normal activities and require medication.

There are 2 types of dysmenorrhoea:

- Primary (no organic cause). Typically, in primary dysmenorrhoea pain occurs on the first day of menses, usually about the time the flow begins, but it may not be present until the second day. Nausea and vomiting, diarrhoea and headache may occur.
- Secondary (pathological cause) e.g. PID and uterine polyposis and membranous (cast of endometrial cavity shed as a single entity (rare).

Treatment

- Allow bed rest
- Give Analgesics such as
 - A:** Ibuprofen 200-600 mg every 8 hours (maximum 2.4 g/day)
 - OR**
 - A:** Acetylsalicylic acid 300-600 mg every 4 hours
 - OR**
 - A:** Diclofenac 50-100mg 8-12 hourly
 - OR**
 - C:** Mefenamic acid 500mg 8 hourly
 - Plus**
 - A:** Hyoscine butylbromide 20mg 8hourly

Women with regular complaints can easily detect length of use during their periods (2-3 days usually sufficient)

- Treat the underlying condition if known

Note: For primary dysmenorrhoea patients may be advised to start taking Ibuprofen one or two days before menses and continue for three to four days during menses to minimize painful menstruation

18.0 INFERTILITY

This is failure to conceive after one year of regular coitus without contraception. It is classified as primary when there has never been a history of pregnancy or it is secondary when there is previous history of at least one conception.

Treatment

Treatment in all cases depends upon correction of the underlying disorder(s) suspected of causing infertility whether primary or secondary.

Note: Refer infertile couple to Obstetrics/Gynecologist/ infertility specialists

CHAPTER SIX

CARDIOVASCULAR DISEASE CONDITIONS

1.0 INFECTIVE ENDOCARDITIS (IE)

The infective process of endocardial layer of the heart can involve native or prosthetic valve and congenital defects/shunts.

Alpha-haemolytic streptococci are the most common causes of native valve endocarditis but *Staphylococcus aureus* is more likely if the disease is rapidly progressive with high fever, or is related to a prosthetic valve (*Staphylococcus epidermidis*)

Diagnosis: Use Modified Dukes Criteria below and consult microbiologist where possible. Three sets of blood cultures should be taken before starting treatment.

Modified Dukes Criteria

Major Criteria

- Positive blood cultures of typical organism for IE from at least two separate blood cultures
- Evidence of endocardial involvement by Echocardiogram (Trans-thoracic Echo/Trans-oesophageal Echo)

Minor Criteria

- Fever > 38°C
- Presence of Rheumatic Heart Disease, Congenital heart diseases
- Vascular phenomena; Major arterial emboli, Septic pulmonary infarcts, mycotic aneurysm, Intracranial haemorrhage, Conjunctival hemorrhage, Janeway lesions
- Immunological phenomena; Glomerulonephritis, Osler's nodes, Roth's spots, Rheumatoid factor.
- Serologic evidence of active infective endocarditis or blood culture not meeting major criterion.

Definitive IE

- Two Major Criteria **or**
- One Major and three minor criteria **or**
- Five Minor Criteria

Possible IE

One major and one minor or three minor criteria

Empirical Treatment

Table 1: Treatment for Native valves

Antibiotics	Dosage & Route*	Duration
Benzyl Penicillin G	18 -24million Units/24 hours IVI, 4hourly in equally divided dose	4 – 6 weeks
<i>or</i> Ceftriaxone	2mg once daily IVI	4 – 6 weeks
<i>plus</i> Cloxacillin	2g IVI 6hourly	4 -6 weeks
<i>plus</i> Gentamicin **	1-1.5mg/kg IVI every 8hours	2-4 weeks
Methicillin-Resistant Staphylococci (MRSA) add Anaerobes		
Vancomycin	30mg/kg/24hours IVI in two equally divided dose, not to exceed 2gm/24 hours unless serum levels are monitored	4 -6 weeks

**Dosage patient with normal renal function **It is important to assay serum gentamicin levels every 3-4 days. One-hour peak concentration should not exceed 10mg/l and trough concentration (2 hour pre-dose) should be less than 2mg/l.*

Table 2: Prosthetic Valve Empirical treatment

Antibiotics	Dosage & Route*	Duration
Benzyl Penicillin G (X-Pen)	18 -24million Units/24 hours IVI, 4hourly in equally divided dose	>6 weeks
<i>or</i> Ceftriaxone	2mg once daily IVI	>6 weeks
<i>plus</i> Cloxacillin	2g IVI 6hourly	>6 weeks
<i>plus</i> Rifampicin	300 -600mg every 8hourly	>6 weeks
and Gentamicin**	1mg/kg IVI every 8hours	2 weeks

**Dosage patient with normal renal function **It is important to assay serum gentamicin levels every 3-4 days. One-hour peak concentration should not exceed 10mg/l and trough concentration (2 hour pre-dose) should be less than 2mg/l.*

At any stage, treatment may have to be modified according to:

- *detailed antibiotic sensitivity tests*
- *adverse reactions*
- *allergy*
- *failure of response*

Endocarditis leading to significant cardiac failure or failure to respond to antibiotics may well require cardiac surgery.

Referral

All patients with IE should be referred at specialized care center for treatment

Prophylaxis of Endocarditis Infective

To reduce the risk of bacterial endocarditis, antibiotic prophylaxis should be given to patients with congenital heart disease; acquired Valvular Heart disease (notably rheumatic heart disease), prosthetic heart valves that undergo any of the following:

- Dental procedures
- Upper respiratory tract surgery, e.g. tonsillectomy
- Urinary tract instrumentation and surgery
- Dilatation and Curettage (D & C) in presence of infection
- Surgery through infected tissues eg skin

Prophylaxis against endocarditis – Low risk group: Dental procedures, upper respiratory tract, obstetrics and gynaecological procedures under local or general anaesthesia

Table 3: Prophylaxis against Endocarditis

Dose	Frequency
Adult Amoxicillin 3g po Paediatric Amoxicillin 50mg/kg	One hour before procedure
Or Penicillin allergy or recent penicillin administration <one month Erythromycin Adult 1.5 g and then 500mg Paed 20 mg/kg body weight then 10mg/kg body Or Clindamycin 600mg >5 years – 150mg 5 -10 years 300mg	One hour before operative procedure then Six hourly after operation, as long as necessary One hour before procedure

Dental procedures, upper respiratory tract, obstetrics and gynaecological procedures under general anaesthesia

Table 4:

Dose	Frequency
Adult Ampicillin IV 1g then 500mg OR Amoxicillin po 3g, then 1g	Half an hour before operation or During induction, then after 6hrs
Paediatric Cloxacillin (IV) 50 mg/kg body weight Plus Ampicillin (IV) 50 mg/kg body weight Plus	4hrs before anaesthesia then 6 hours post-op. Half an hour before operation or During induction

Gentamicin (IV) 1.5-2 mg/kg body weight Penicillin allergy or recent penicillin administration <one month <i>see under special risk groups below.</i>	
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Prophylaxis against endocarditis -†**Special high risk group;** Dental procedures, upper respiratory tract, obstetrics and gynaecological procedures **or** genitourinary

Table 5:

Dose	Frequency
Adult Ampicillin (IV) 1g Cloxacillin (IV) 2g And Gentamicin) 5mg/kg body weight or 120mg.	Half an hour before operation or During induction Single dose,
Paediatric Cloxacillin (IV) 50 mg/kg body weight <i>Plus</i> Ampicillin (IV) 50 mg/kg body weight <i>Plus</i> Gentamicin (IV) 1.5-2 mg/kg body weight	Half an hour before operation or At induction single dose
If penicillin allergy or administration of penicillin in the past month Clindamycin IV* 300mg and Gentamicin IV 120mg	Half an hour before operation or At induction single dose

Do not use clindamycin for urological/gynaecological procedures because it will not prevent enterococcal infection. In these cases replace clindamycin with Vancomycin iv [Specialist-only drug] 1g over at least 100 minutes 1-2 hours before procedure. † Prosthetic cardiac valve or prosthetic material used for cardiac valve repair, Previous IE, Congenital heart disease (CHD) **and Cardiac transplantation recipients who develop cardiac valvulopathy*

2.0 RHEUMATIC FEVER

It is a non-suppurative sequela of a group A β haemolytic streptococcal (GABHS) pharyngeal infection.

Diagnosis Use the Jones Criteria updated 1992 see table below

- Two major criteria or
- One major criterion with two minor criteria, with evidence of antecedent streptococcal infection

Table 6: Criteria for Rheumatic Fever Diagnosis

Major Criteria	Minor Criteria
Carditis Migratory polyarthrititis Sydenham's chorea Erythema Marginatum	Clinical <ul style="list-style-type: none"> • Fever • Arthralgia Laboratory <ul style="list-style-type: none"> • Elevated Acute phase Reactants eg CRP • Prolonged PR interval
Plus Supporting evidence of recent group A streptococcal infection e.g. positive throat culture or antigen detection and/or elevated streptococcal antibody tests*	

**Anti -Streptolysin O, Anti -Deoxyribonuclease B*

Treatment

Non pharmacological

Acute stage:

- Bed rest and supportive care until all evidence of active carditis has resolved
- Patient education.
- Intensive health education for prevention of sore throats.

Pharmacological treatment

Treatment of acute attack for eradication of streptococci in throat: Regardless of the presence or absence of pharyngitis at the time of diagnosis.

B: Benzathine Penicillin 1.2MU single dose im

Paediatric > 5 years 0.3MU, 5-10 years 0.6 MU > 10 years 1.2.mu single dose IM.

OR

A: Penicillin V 500mg two to three times daily for 10 days orally.

Children > 10years 500mg, 5-10 years 250mg, < 5years 125mg two to three times daily for 10 days orally

If allergic to Penicillin

A: Erythromycin 500mg or 40mg/kg 4 times per day for 10 days orally

Treatment of acute Arthritis and Carditis:

A: Aspirin orally 25mg/kg* 4 times a day as required.

Aspirin should be continued until fever, all signs of joint inflammation and the ESR have returned to normal and then tapered gradually over 2 weeks. If symptoms recur, full doses should be restarted.

**dose should be reduced if tinnitus or other toxic symptoms develop*

In severe carditis with development of increasing heart failure or failure of response to aspirin,
Plus

A: Prednisolone 1-2mg/kg once a day for 3-4 weeks.

Then reviews *Gradual reduction and discontinuation of prednisolone may be started after 3-4 weeks when there has been a substantial reduction in clinical disease.*

Heart failure should be managed in the usual way see Heart Failure Section.

Treatment of Sydenham's chorea:

Adult

B: Haloperidol 1.5-3mg (O) 8hourly a day as required.
Paediatrics 50mcg/kg in 2 divided doses.

Referral: Ideally all patients should be referred to specialized care

- where surgery is contemplated
- management of intractable heart failure or other non-responding complications
- pregnancy

Antibiotic prophylaxis after rheumatic fever:

Prophylaxis should be given to all patients with a history of acute rheumatic fever and to those with rheumatic heart valve lesions. The optimum duration of prophylaxis is controversial, but should be continued up to at least 21 years of age.

Note: Specific situations requiring prophylaxis for longer periods (up to 30 years as a guide):

- definite carditis in previous attacks
- high risk of exposure to streptococcal infection at home or work (crowded conditions, high exposure to children)

Medicines

B: Benzathine Penicillin IM Adult 2.4MU monthly or every three weeks*
Paediatrics <12yrs 1.2MU every 4 weeks or 3 weeks* up to 21-30yrs

OR

A: Penicillin V (PO) 250mg 12 hourly
Paediatric<12yr 125-250mg 12 hourly a day up to 21-30yrs

OR

A: Erythromycin 250mg 12hourly a day, Paediatric <12yr 125-250mg 2 times a day up to 21-30yrs

**Every 3 week regimen is more effective*

3.0 VALVULAR HEART DISEASE AND CONGENITAL STRUCTURAL HEART DISEASE

Valvular Heart Disease

These are chronic sequelae of acute Rheumatic fever or acute sequelae of infective endocarditis or ischaemic heart disease, consisting of valvular damage, usually left heart valves, with varied progression of severity and complications.

Congenital Heart Disease

It is a congenital chamber defects or vessel wall anomalies

Valvular Heart Disease and Congenital structural Heart Disease may be complicated by:

- Heart failure
- Infective endocarditis

- Atrial fibrillation
- Systemic embolism eg Stroke

General measures

- Advise **all** patients with a heart murmur with regard to the need for prophylaxis treatment prior to undergoing certain medical and dental procedures
- Advise patients to inform health care providers of the presence of the heart murmur when reporting for medical or dental treatment

Referral

- All patients with heart murmurs for assessment
- All patients with heart murmurs not on a chronic management plan
- Development of cardiac signs and symptoms
- Worsening of clinical signs and symptoms of heart disease
- Any newly developing medical condition, e.g. fever
- All patients with valvular heart disease for advice on prophylactic antibiotic treatment prior to any invasive diagnostic or therapeutic process

4.0 HYPERTENSION

Hypertension is elevation of blood pressure (B.P) noted on at least three separate occasions.

Diagnosis

If blood pressure measurements performed on three separate occasions when either

- The initial SBP is ≥ 140 mmHg or
 - The DBP is ≥ 90 mmHg **measured** on three separate occasions, a minimum of 2 days apart and/or taken over period of two months
- a minimum of 3 blood pressure readings must be taken at the first visit to confirm hypertension
 - If SBP is ≥ 160 mmHg or DBP ≥ 100 mmHg Stage II of JNC –VII – especially when SBP > 180 mmHg and/or DBP >110 mmHg Immediate drug treatment is needed - See Hypertensive crisis - Urgency/Emergencies section
 - Consider Secondary hypertension with identifiable cause in young patients < 30 years or elderly patient > 60 years presenting for first time with hypertension.

KEY POINTS

- Hypertension control has shown to have significant benefit for patients.
- Co-existent risk factors should be detected and treated.
- Assess cardiovascular risk.
- Lifestyle modification and patient education are essential in all patients.
- Drug treatment for SBP >140 mmHg; DBP > 90 mmHg.
- Antihypertensive treatment is required for life in truly hypertensive patients
- Hypertension often has no symptoms: the aim of treatment is to lower the risk of End-organ damage, especially stroke
- Compliance is the most important determinant of blood pressure control.
- Explanation, education and minimizing side-effects of drugs are important
- Extra care should be taken with antihypertensive drugs administered to those over 60 years of age, because of increased side-effects. Lower doses are needed
- Recommended an alternative contraceptive method for women using oestrogen

Containing oral contraceptive

- Evidence of end organ damage, i.e. cardiomegaly, proteinuria or uraemia, Retinopathy or evidence of stroke, dictates immediate treatment
- Patients should be reviewed every 1-3 months, till blood pressure controlled the every 6months and more often if necessary
- Urgent blood pressure reduction may precipitate stroke or blindness. It is only Indicated in those patients with hypertensive emergencies (see below)
- The aim of treatment is to bring the diastolic BP below 90 mm Hg, without unacceptable side effects

Note: Patients should be evaluated for Risk Stratification - Major Risk Factors, Target Organ Damage and Associated Clinical Cardiovascular Condition and Co-morbidity

Figure 1: Hypertension Management flow diagram

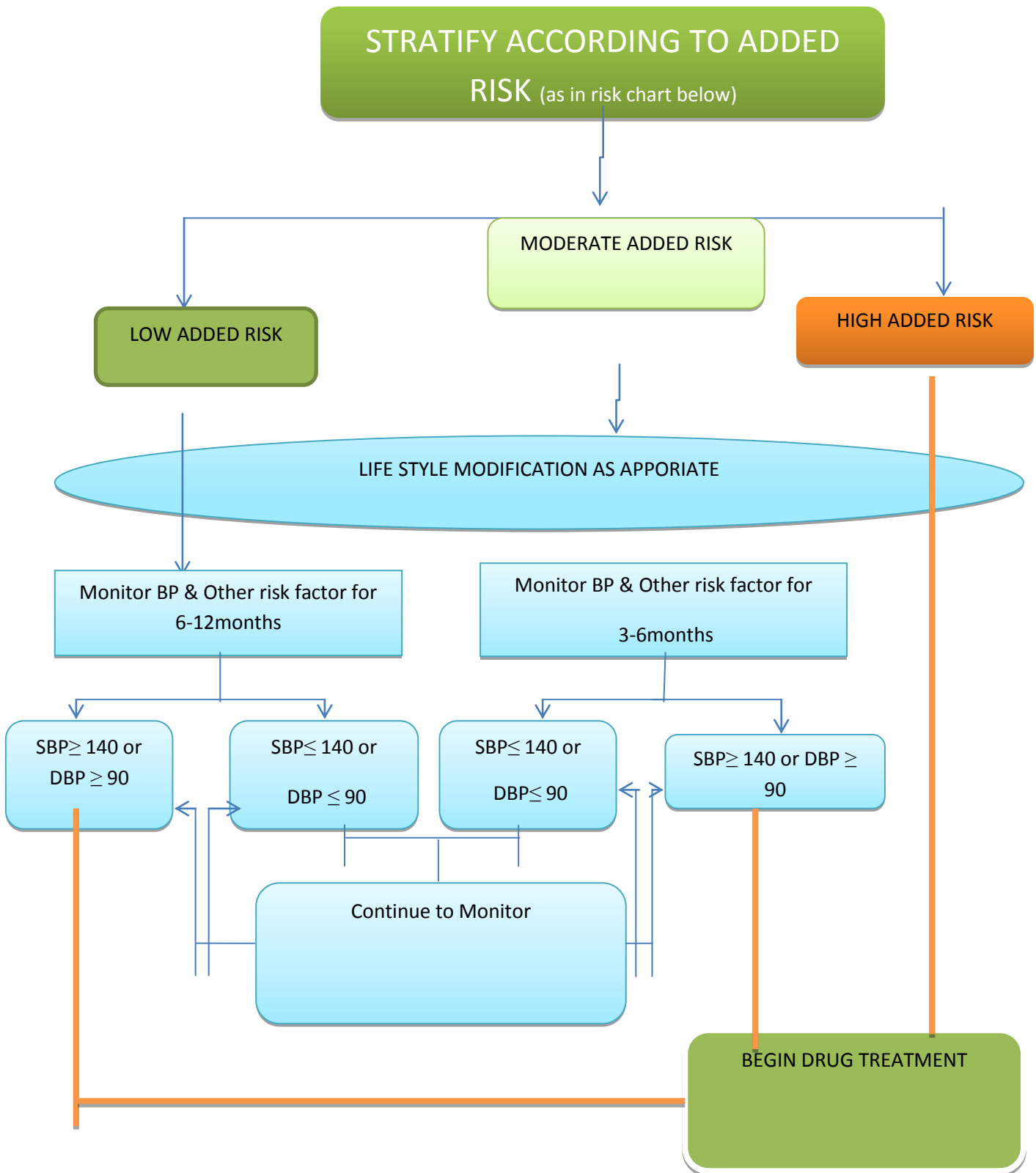


Table 7: Major Risk factors, Target Organ Damage and Associated Clinical Condition

Major Risk factors	Target organ damage	Associated Clinical condition
Level of SBP & DBP	Left Ventricular Hypertrophy based on the ECG	Coronary Artery Disease
Smoking		Heart Failure
Dyslipidemia <ul style="list-style-type: none"> Total Cholesterol < 5mmol/l or LDL >3.0mmol/l or HDL < 1mmol/l men, <1.2mmol/l women 	Micro-Albuminuria: Albumin/Creatinine ratio 3 - 30mg/mmol	Chronic Kidney Disease Albumin Creatinine ratio >30mg/mmol
Diabetes Mellitus	Slightly elevated Creatinine Men 115 - 133µmol/l Women 107 – 124µmol/l	Stroke or Transient Ischaemic Attack
Family history of premature Ischaemic Heart Disease/Coronary Artery Disease Men <55 years, Women <60years		Peripheral Vascular Disease
Waist Circumference – Abdominal Obesity Men ≥ 102cm Women ≥ 88cm		Advanced retinopathy Haemorrhage, or Exudates Papilloedema

Treatment

Objective:

Achieve and maintain the target BP: In most cases the target BP should be: systolic below 140 mmHg and diastolic below 90 mmHg. **Achieve target BP** in special cases as: In diabetic patients and patients with cardiac or renal impairment, target BP should be below 130/80 mmHg; Prevent and treat associated cardiovascular risks such as dyslipidemia and lifestyle modification

Non – pharmacological therapy

Lifestyle modification:

- Weight Reduction; Maintain ideal body weight BMI 18.5 – 24.9kg/m²
- Adopt DASH* eating plan; Consume a diet rich in fibre - fruits, vegetable, unrefined carbohydrate and low fat dairy products with reduced content of saturated and total fat
- Dietary Sodium; Reduce dietary sodium intake no more than 1000mmol/l (2.4gm sodium or 6gm sodium chloride)
- Physical Activity; Engage in regular activity such as a brisk walking at least 30min/day most days a week
- Stop using all tobacco products
- Moderation of alcohol consumption; Limit consumption to no more than 2 drinks per day in men and no more than one drink per day in Women and light person

*DASH – Dietary Appropriate to Stop Hypertension

Pharmacological therapy

First line treatment without compelling indications:

Low Dose Thiazide diuretics + Potassium sparing e.g. Bendroflumethiazide 2.5 -5mg/d,
Hydrochlorothiazide 12.5 -25mg/d + Spironolactone 25mg daily.

Second line treatment with compelling indications:

Compelling indications	Drug class
Angina	• β -blocker or Long acting calcium channel blocker
Prior or Post-myocardial infarct	• β -blocker and ACE inhibitor • If β -blocker contraindicated: Long acting calcium channel blocker eg verapamil
Heart failure For volume overload:	• ACE inhibitor and Carvedilol • Diuretics – Spironolactone Furosemide
Left ventricular hypertrophy (confirmed by ECG)	ACE inhibitor or ARB
Stroke: secondary prevention	Hydrochlorothiazide or Indapimide and ACE inhibitor
Diabetes Mellitus	ACE inhibitor or ARB, usually in combination with diuretic
Chronic kidney disease	ACE inhibitor, usually in combination with diuretic
Isolated systolic hypertension	Hydrochlorothiazide or Long acting calcium channel blocker
Pregnancy	Methyldopa or Hydralazine (Avoid ACEI/ARB tetragenic)
Prostatism	alpha-blocker
Elderly	CCB

Recommended Medicines for Treatment of Hypertension

S/N	CLASS	DRUG	DOSAGE
01.	Thiazide Diuretics	Bendroflumethiazide	5mg once daily
		Hydrochlorothiazide	12.5mg daily
02.	Loop Diuretics	Furosemide	40mg- 80mg daily
		Torsemide	2.5mg – 5mg daily
03.	Potassium Sparing Diuretics	Spirinolactone	25mg once daily
		Eplerenone	25mg once daily
04.	Central Adrenergic Inhibitor	Methyldopa	250mg 12hrly

		Clonidine	50µg 8hrly
05.	Beta Blockers <ul style="list-style-type: none"> • Non selective • Selective • Alpha& Beta blockers 	Propranolol Atenolol Metoprolol Carvedilol	80mg 12 hrly 50 – 100mg once daily 100mg 12hrly 12.5 -25mg daily
06.	ACE Inhibitors	Captopril	12.5mg- 25mg 12hrly
		Enalapril	5- 20mg daily
	ARB's	Losartan	50 -100mg daily
07.	Calcium channel blockers –CCB	Nifedipine SR	10- 20mg 12hrly
		Amlodipine	5 – 10mg once daily
08.	Direct Vasodilators	Hydralazine	25mg twice daily

Referral

Referral is dynamic and patients can be referred up to a specialist or down to PHC when controlled. Consultation without referral may be all that is necessary.

Referrals are indicated when:

- Resistant (Refractory) Hypertension
- All cases where secondary hypertension is suspected
- Complicated hypertensive urgency/emergencies
- Hypertension with Heart Failure
- When patients are young (<30 years) or blood pressure is severe or refractory to treatment.

Resistant (Refractory) Hypertension

Hypertension that remain >140/90mmHg despite the use of 3 antihypertensive drugs in a rational combination at full doses and including a diuretic. Consider all correctable causes of refractory hypertension, before you refer.

Hypertensive urgency

Symptomatic severe hypertension BP DBP >110 mmHg and/or 180mmHg with evidence of Target Organ Damage or grade III/IV Retinopathy with no immediate life-threatening neurological or cardiac complication such seen in emergencies

Note; All patient hypertensive urgency should be treated in hospital

Treatment goal to lower DBP to 100mmg slowly over 48 -72 hour this can be achieved with two oral agents preferably

- Long acting Calcium Channel Blocker
- ACE Inhibitor use in low dosage initially
- Beta Blocker
- Diuretic – Thiazide or Loop diuretics Furosemide beneficial in renal insufficiency & pulmonary oedema and potentiate above other classes

Hypertensive Emergency

A marked elevated blood pressure systolic BP ≥ 180mmHg and/or a diastolic BP ≥130mmHg **associated with life threatening situations** one or more of the following:

- Unstable angina/Myocardial Infarction

- Hypertensive Encephalopathy e.g. severe headache, visual disturbances, confusion, coma or seizures which may result in cerebral haemorrhage
- Acute left ventricular failure with severe pulmonary oedema (extreme breathlessness at rest)
- Excessive circulating catecholamines: e.g. pheochromocytoma – rare cause of emergency; food or drug interaction with monoamine oxidase inhibitors
- Rapidly progressive renal failure
- Acute aortic dissection
- Eclampsia and severe pre-eclampsia

Treatment goal require immediate lowering of BP usually with parental therapy preferably Intravenous agents as infusion with strictly monitoring of haemodynamics in high care depended unit or intensive care unit in the hospital

Preferable intravenous drugs are

- Nitroglycerin (glyceryl trinitrate)
- Hydralazine or Dihydralazine

5.0 HEART FAILURE

5.1 Acute Heart Failure (AHF) or Decompensated Acute Heart Failure (DAHf)

AHF defined as rapid or gradual onset of signs & symptoms of heart failure that result on urgent unplanned hospitalization or Emergency Medicine Department visits. The Clinical Signs & symptoms are significantly life threatening.

If the above features occurs in patient diagnosed with structurally heart disease categorize as **Decompensated Acute Heart Failure (DAHf)**.

The cause and immediate precipitating factor(s) of the AHF must be identified and treated to prevent further damage to the heart.

Causes

- Decompensation of pre-existing chronic Heart Failure eg Cardiomyopathy, Peripartum Cardiomyopathy
- Acute Valvular Regurgitation – AR, MR 2^o endocarditis, rupture of chordae tendinae
- Worsening pre-existing Valvular Disease– MS MR AR AS
- Severe Aortic Stenosis
- Hypertensive crisis
- Acute Coronary Syndrome - NSTEMI/STEMI, RV infarction, Mechanical complication of ACS
- Acute arrhythmias – VT /VF AF/flutter or other SVTs
- Acute Severe Myocarditis
- Aortic Dissection - Acute/chronic
- Pericardial Effusion with Cardiac tamponade

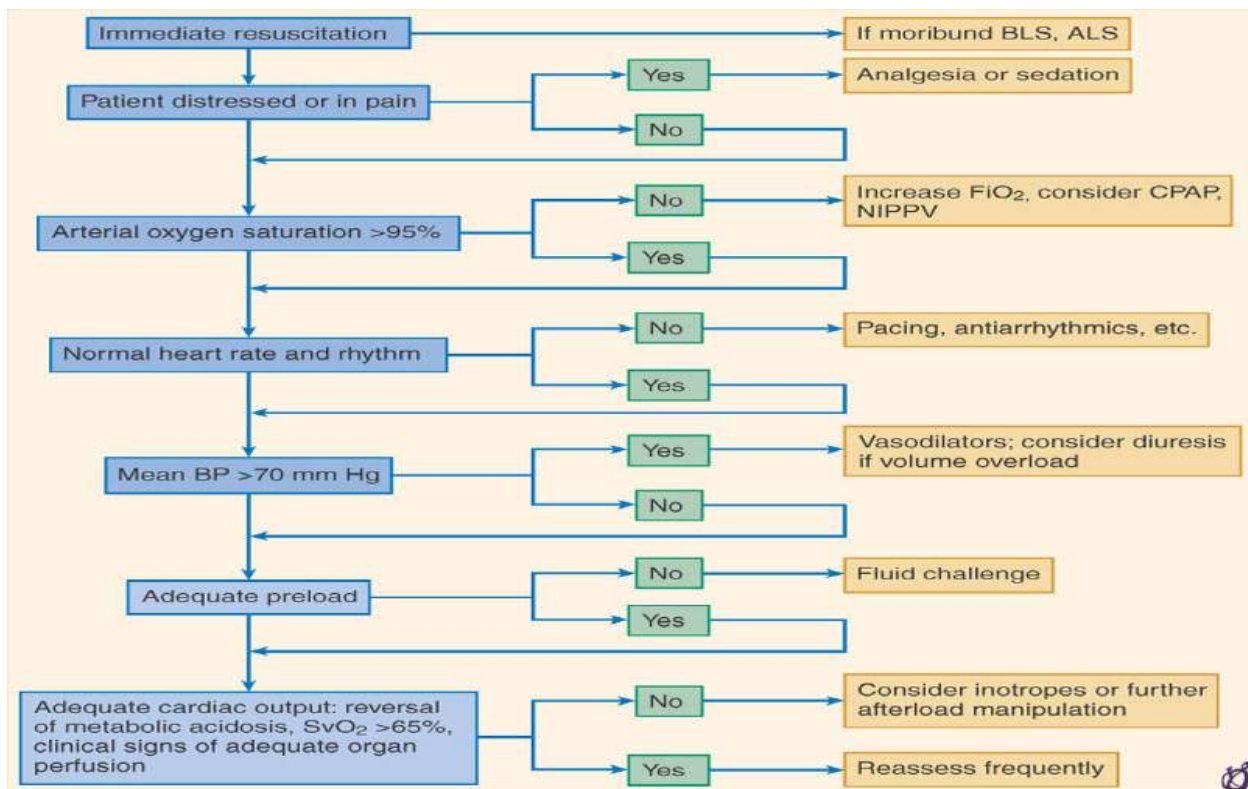
Precipitating factors

- Lack of Compliance with medical therapy
- Infections –Pneumonia, UTI, septicemias
- Anaemia
- Arrhythmias – Rapid AF other SVTs
- Thyroid disease – hypothyroidism
- Pulmonary Embolus
- Volume overload - iatrogenic
- Drug abuse/Alcohol – eg thiamine deficiency

Treatment Goals

To improve clinical symptoms and Outcome, management strategy should be based on Clinical, laboratory and haemodynamic findings. All patient with AHF should be cared and admitted high care dependent unit or Intensive Care Unit

Figure 2: Management of Acute Heart Failure



(Modified from Nieminen MS, Bohm M, Cowie MR, et al: Executive summary of the guidelines on the diagnosis and treatment of acute heart failure: The Task Force on Acute Heart Failure of the European Society of Cardiology. Eur Heart J 26:394-416, 2005.)

Specific Pharmacological treatments

Diuretics: Relief of Congestive symptoms

- **Loop diuretic :**

C: Furosemide 40-120mg I.V

OR

S: Torsemide 5 -20mg orally

- Potassium Sparing Agents:

C: Sprinolactone 25 -50mg

OR

S: Eplerenone 25 – 50mg orally

Vasodilators – Mainstay of treatment of AHF/DAHF preferable therapy intravenous vasodilators

Vasodilator	Indication	Dosing	Side effect
Nitroglyceride Glycerly trinitrate, 5- mononitrate	AHF with SBP>90- 100mmHg	Initial dose 20µg/min Effective dose range 40 - 400 µg/min	Hypotension, headache tolerance with continuous use after 24 hours

Monitor blood pressure keep SBP >90-100mmHg (Mean BP 60 - 65mmHg)

Consider oral vasodilators in case intravenous Vasodilator not available or unavailability of intensive care or high dependent unit care

C: Isosorbide mononitrate 10 - 20mg (O) 12 hourly

OR

C: Hydralazine 25 mg 6-8 hourly.

Maximum dose: 200 mg/day

Inotropic Agents indicated in AHF/DAHF with hypotension or cardiogenic shock ie SBP <90mmHg

S: Dobutamine infusion 2 -20 µg/kg/min

OR

D: Dopamine infusion <3 µg/kg/min (renal effect), 3-5 µg/kg/min (inotropic effect), >5 µg/kg/min (vasodilator effect)

Special consideration:

1. Add ACEI – Captopril 6.25 - 25mg three times a day
Enalapril 5 -20mg three times a day

When patient is out of congestion state and renal function (Urea & Creatinine, K+) is normal

2. Add Beta blocker – Carvedilol 6.25 -25mg twice a day

When patient is out of congestion state and SBP above 90mmHg

3. All admitted patients with Acute heart failure should be given anticoagulation

Unfractionated Heparin 5,000u subcutaneous twice a day or

Low molecular weight Heparin - Enoxaparine 40mg – 80mg subcutaneous twice a day

In case patient admitted with beta blocker continue with Carvedilol unless is contraindicated

Note that

- Patients admitted with beta blocker have lower rate of ventricular arrhythmias, a shorter length of stay in hospital, reduced 6-month mortality compared those not receiving beta blocker
- Those who were maintained on them has significant lower rate of rehospitalization and death within 6 month after discharge
- Patient should continue their beta blocker during admission of AHF unless significant hypotension or cardiogenic shock present

Referral

- All patients with AHF should be treated at centre where at least can perform Echocardiographic assessment
- Conditions requiring Cardiac surgery refer to Muhimbili Cardiovascular Institute/Centre

5.2 Chronic Heart Failure

CHF is a clinical syndrome and has several causes.

Diagnosis

The diagnosis of Chronic Heart failure requires the following features:

- Symptoms of heart failure, typically breathlessness or fatigue, at rest or during exertion
- Objective evidence of cardiac dysfunction preferably by Echocardiography (Systolic and/or Diastolic)
- A clinical response to treatment is supportive but not sufficient for diagnosis

Hence diagnosis and management of CHF should be sought at referral centres where at least echocardiography assessment can be performed.

Asymptomatic left ventricular dysfunction is considered as precursor of symptomatic HF and is associated with high mortality.

Treatment**Treatment of Systolic Heart Failure (LVEF < 45 - 50%)**Aims of Treatment

- Prevention of
 - A) Disease leading to cardiac dysfunction and heart failure eg hypertension, coronary artery disease, valve disease etc
 - B) Progression to HF once cardiac dysfunction is established
- Maintenance or Improvement in quality of life
- Improve survival

Non pharmacological management

- **Patient and family education**
 - Explain what HF is and why symptoms occurs, cause of HF, how to recognize symptoms and what to do when they occur, daily self-weighing and what to in case of weight gain.
 - Rationale of treatment, importance of adhering to drug & non drug prescription
 - Refrain from smoking

- Prognosis
- Drug counseling – Effects, doses and times of administration, side effects and adverse effects
- Dietary and social habit – control sodium intake when necessary, avoid excessive fluid intake in severe HF Limit fluid intake to 1–1.5 L/day if fluid overloaded despite diuretic therapy. Avoid excessive alcohol intake
- Regular exercise within limits of symptoms.
- Sexuality counsel regarding the risk of pregnancy and the use of oral contraceptives & Phosphodiesterase-5 inhibitor are not recommended in advanced HF, if used nitrate should be avoided < 24 -48hours of nitrate intakes
- Drug to avoid or used with caution
 - NSAIDs & Coxibs
 - Class I anti-arrhythmics
 - Calcium antagonists
 - Lithium
 - Tricyclic anti-depressants
 - Corticosteroid

Pharmacological treatment

Combination of

- Diuretics – loop diuretics & Aldosterone antagonist (potassium sparing agents)
- ACE-inhibitors or ARB
- Beta blocker especially Carvedilol- improve Morbidity & Mortality in CHF.

Add if patient in NYHA class III/IV

- Vasodilator agents: The combination of hydralazine/Nitrate
- Cardiac Glycosides – Digoxin, give with caution has narrow therapeutic index see below under section of Cardiac Glycosides

Consider Anti-thrombotic agents – Heparin &/or warfarin under special indications see below

Diuretics: Essential for symptomatic treatment when fluid overload is present as manifest as pulmonary congestion and/or jugular vein congestion and/or peripheral oedema

Loop Diuretics –

C: Furosemide 40 – 80mg 2-3times/day

OR

S: Torasemide 5 – 40mg 2times/day

Thiazide

A: Hydrochlorothiazide 12.5 - 25mg (O) once a day

OR

S: Metolazone 0.1 – 10mg day

Aldosterone antagonist (potassium sparing agents)- Recommended in addition to ACEIs,β – Blocker and loop diuretics in advanced heart failure (NYHA-III/IV) and in patient with a recent myocardial infarction to improve survival and morbidity.

D: Spironolactone 25 -50mg once a day

OR

S: Eplerenone 25 -50mg once a day

Note:

Diuretic should be administered in combination with ACEIs and beta blocker

Replace potassium loss in case of hypokalaemia if Furosemide given alone without aldosterone antagonist.

Major side effects are hypokalemia, hypomagnesaemia, hyponatraemia, acid-base disturbance, and hyperuricaemia and glucose intolerance

ACE-inhibitors improve survival, symptoms, functional capacity and left ventricular remodeling and reduce hospitalization in patients with chronic heart failure

Recommended as first line therapy in patients with reduced LV systolic function with or without symptoms.

Important adverse effects are dry cough, hypotension, renal insufficiency, hyperkalemia, and angioedema.

- Contraindicated in the presence of ACEI induced cough, bilateral renal artery stenosis and angioedema

C: Captopril 6.25mg 25mg 3 times a day

OR

D: Enalapril 2.5 10mg 2 times a day

Note:

- Recommended as first line therapy in all patients with stable, mild, moderate and severe CHF from ischaemic or non ischaemic cardiomyopathies and reduced LVEF (with or without symptoms) on standard treatment in combination with diuretics & ACEIs unless contraindicated.

S: Carvedilol first dose 3.125mg 12 hourly, then increments (mg/day) 6.25 mg 12 hourly, 12.5 mg 12 hourly, 25mg mg 12 hourly up to maximum dose of 50mg 12 hourly as tolerated

Note: Beta Blockers is contra- indication to patients with

- Bronchial Asthma or Severe Pulmonary disease
- Symptomatic bradycardia or hypotension

Angiotensin II Receptor Blockers (ARBs)

Note:

- ARBs can used as an alternative to ACEI to improve morbidity and mortality
- Avoid combination therapy of ARBs & ACEIs have been associated with increased morbidity & mortality in stable CHF patients, however cautiously can considered in CHF patients who remain symptomatic to reduce mortality and hospital admissions in combination with ACEIs and β -Blockers

S: Losartan 50 – 100mg/day

OR

S: Candesartan 4- 32mg/day

Cardiac Glycosides - Digoxin has only been shown to reduce morbidity- re-hospitalization, but has narrow therapeutic index/range then toxicity, give with caution. Monitor digoxin level - trough blood levels (before the morning dose) should be maintained between 0.65 and 1.5 nmol/L.

C: Digoxin 0.125mg -0.25mg/day

Patients at high risk of digoxin toxicity are:

- the elderly
- patients with poor renal function
- hypokalaemia
- low body weight

Vasodilator agents: The combination of hydralazine/Nitrate has been shown to improve morbidity – quality of life and mortality can be added on above standard combination CHF or can be used on patient intolerant to ACEI and/or ARBs

C: Hydralazine 25 mg 3 times a day.

OR

C: Isosorbide Dinitrate/Mononitrate 10- 20 mg 2 times a day.

Anti-thrombotic agents – Heparin &/or warfarin – firmly indicated on CHF with atrial fibrillation, previous thromboembolic events or a mobile LV thrombus
Heparin for DVT prophylaxis for patients admitted to hospital, unless contraindicated:

D: Heparin 5 000 units (SC) 8 hourly

OR

D: Warfarin (O) 5 mg daily.

Control with INR to therapeutic range, i.e. between 2.0 and 2.5

Thiamine Supplement Consider in all unexplained heart failure

Referral

Ideally all patients with CHF should be managed on dedicated HF clinics/units with devoted HF expert staffs (nurses & doctors). The following patients should be referred for specialized care

- Severe HF class III/IV
- HF of unknown origin
- Relative contraindication: asymptomatic bradycardia and/or low blood pressure
- Intolerance to low doses
- Previous use of β –blockers and discontinuation because of symptoms
- Bronchial asthma or severe pulmonary disease

6.0 PULMONARY OEDEMA

Common cause of pulmonary oedema

Cardiac/Fluid overload

- Cardiac Failure

- Fluid overload (eg renal failure, iatrogenic)

Non Cardiac Pulmonary Oedema

Increased capillary permeability (ARDS); many causes including

- Systemic Sepsis – particular gram negative infection
- Pancreatitis
- Head injury
- Aspiration of gastric contents
- Amniotic embolus

Conditions predisposing to Acute Respiratory Distress Syndrome (ARDS) includes; infections, shock, trauma (eg fat embolism, lung contusion) liquid aspiration (eg acid, drowning) drug overdose (eg heroin, barbiturates), inhaled toxins (eg Chloride gas) haematological disorders (eg DIC, massive blood transfusions, post cardiopulmonary bypass) metabolic disorders eg uraemia, hepatic failure) miscellaneous (eg increased intracranial pressure, eclampsia, pancreatitis, paraquat poisoning)

Referral

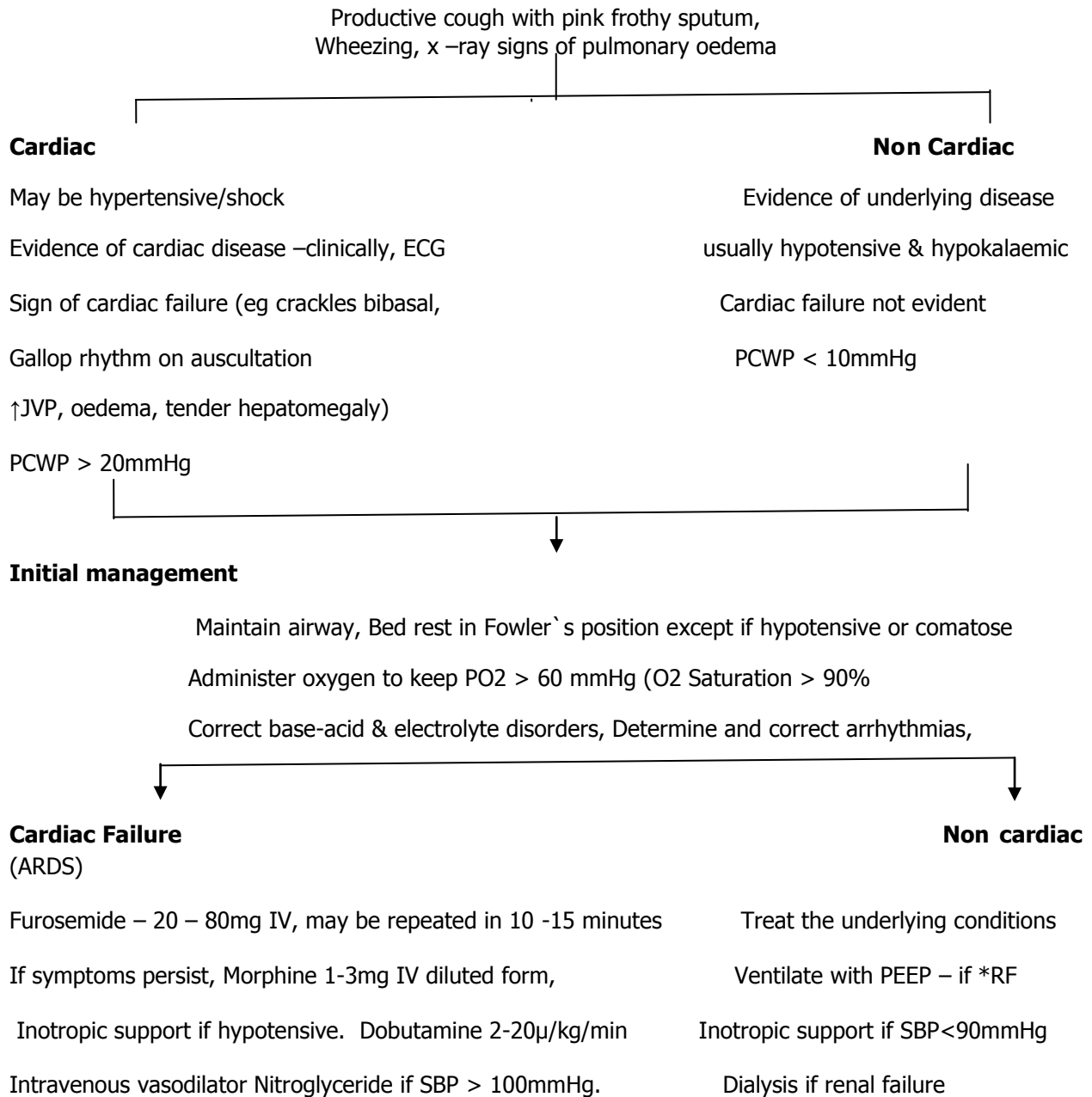
All cases of suspected pulmonary oedema should be referred to a specialized care with High care unit or ICU hospital. Patient should be stabilize first before referral see approach below

Clinical approach of pulmonary oedema

Common presentation

Dyspnoea/tachypnoeic/orthopnea, Respiratory failure

Figure 4: Clinical Approach of Pulmonary Oedema



Note: Echo mandatory if available to determine Aetiology & guide treatment

***RF – Respiratory Failure**

7.0 ACUTE CORONARY SYNDROMES (ACS)

ACS is divided into

- ST Elevation Myocardial Infarction (STEMI)
- Non ST Elevation Myocardial Infarction (NSTEMI)
- Unstable Angina (UA)

7.1 ST Elevation Myocardial Infarction (STEMI)

Classical MI present with triad of typical chest pain*, typical ST elevation on the ECG or new LBBB and elevated cardiac biomarkers

**exclude or consider other cause pericarditis, pulmonary embolus, fractured ribs, and Aortic dissection, oesophageal spasm*

Treatment

Non Pharmacological

- Admit ICU or CCU for monitoring
- Bed rest in Fowler's position and reassurance.
- Oxygen via canular or mask
- Establish IV line
- ECG monitor & rhythm strip

Drug Management

Adjunctive therapy

Control cardiac pain

C: Glyceryl trinitrate sub-lingual/ spray 0.5mg (make sure patient hasn't taken phosphodiesterase-5 inhibitor).

For persistent pain and if oral therapy is insufficient

S: Glyceryl Trinitrate IV, 1–2 mcg/kg/min titrated with chest pain over 8 – 24 hours.

OR

C: Morphine, IV, 1–2 mg/minute dilute 10 mg up to 10 mL with sodium chloride solution 0.9%. Total maximum dose 10 mg, repeat after 4 hours if necessary.

But Pain not responsive to this dose may suggest ongoing unresolved ischaemia; appropriate measure should be taken to reverse the ischaemia.

Anti-platelets therapy

A: Aspirin 300mg stat (O) then followed by 150 mg daily

OR

D: Clopidogrel 300 -600mg stat then followed by 75mg daily next day

Statin high dose

C: Simvastatin 40mg daily

Heparin UFH 5,000 -12,500U sc/iv a day

OR

S: Enoxaparin 1mg/kg sc bid

β –Blockers –Early use within 6 hours results in reduction of infarct size, decrease mortality, incidence of re-infarction and sudden death.

In case of LV dysfunction

S: Carvedilol initial dose 6.25mg twice daily preferred, titrate dose upward. Max. dose 25mg twice daily

Others in the settings of Normal systolic function

A: Atenolol 12.5 – 50mg once a day,

OR

S: Metoprolol 12.5 -50mg once a day

ACEIs early use within 24 hours of index event is beneficial in decreasing mortality especially in large infarct and if there is cardiac failure or LV dysfunction present eg

S: Perindopril 4 -8mg once a day,

OR

D: Enalapril 10mg bid

OR

C: Captopril 6.25 -12.5mg tid

Reperfusion therapy – Definitive management of STEMI

I. Thrombolytic Therapy:

Thrombolytic agents have shown significant reduction in mortality and should be used in all eligible patients, most beneficial if given first 6 hours but can be given up to 12 hours after onset of chest pain. Check for contraindications before you administer thrombolytics

S: Streptokinase, I.V, 1.5 million units diluted in 200 mL sodium chloride 0.9%, infused over 30– 60 min

OR

S: Alteplase TPA 15mg as bolus, 0.75mg/kg over 30min, then 0.5mg/kg over 60min

OR

S: Tenecteplase 40mg IV bolus (70 -79kg body weight) 30 -35mg < 70kg body weight

Contraindications:

Absolute

- Previous allergic to streptokinase or used within the last year for streptokinase only
- Stroke CVA within the last 3 months
- History of recent major trauma
- Bleeding within the last month
- Aneurysms
- Surgery or head injury within the preceding month
- Active bleeding or known bleeding disorder

Relative

- Refractory hypertension
- Warfarin therapy
- Pregnancy
- Traumatic resuscitation
- Recent retinal laser treatment

- Subclavian central venous catheter
- TIA in the preceding 6 months

II. Primary Percutaneous Intervention (1^o PCI) – Only in centre where the 1^o PCI, Coronary angioplasty/stenting can be performed and has been shown to have superior outcomes compared to thrombolytic therapy

Note: STEMI patients in Cardiogenic Shock start immediately inotropic support with Dobutamine and urgently transfer to cardiac Catheterization laboratory for Intra-Aortic balloon counterpulsation and urgent 1^o PCI

Referral

All patients with STEMI should be referred Specialized Cardiology Centre – Muhimbili Cardiovascular Institute/centre for further management

7.2 NON-ST Elevation Myocardial Infarction (NSTEMI) and Unstable Angina (NSTEMI/UA)

Non-ST Elevation MI: Chest pain that is increasing in frequency and/or severity or occurring at rest. The chest pain is associated with elevated cardiac enzymes and ST segment depression or T wave inversion or normal ECG on ECG.

Unstable Angina: Angina that is increasing in frequency and or severity, or occurring at rest. It also encompasses post-infarct angina. The chest pain may be associated with ST segment depression or T wave inversion or normal ECG. There is no rise in cardiac enzymes.

Treatment - General Measures See STEMI Section above

Drug Management - Adjunctive therapy See STEMI Section above

Referral

All NSTEMI/UA patients are required to have Coronary angioplasty and/or stenting has been shown to have superior outcomes compared to medical therapy should be referred to Specialized Cardiology Centre/Cardiovascular Institute for further management

7.3 Post myocardial infarction

Non-medical therapy

- Risk stratification and modification, including attention to smoking and lipid lowering strategies
- Appropriate risk reduction diet.
- Rehabilitation programme.

Medical therapy

- Continue medical management.

Anti-platelets therapy

A: Aspirin 300mg (O) stat then followed by 150 mg daily

OR

D: Clopidogrol 300 -600mg stat then followed by 75mg daily next day

Statin HMGCoA reductase inhibitors

C: Simvastatin 20 - 40mg daily with a goal to achieve LDL level \leq 1.8mmol/l

β –Blockers

S: Carvedilol 6.25 - 25mg 12 hourly in Heart Failure and/or asymptomatic LV dysfunction in combination with diuretics – loop and/or aldosterone antagonists

A: Atenolol 12.5 – 50mg once a day

OR

S: Metoprolol 12.5 -50mg once a day

OR

ACEIs

S: Perindopril 2 -8mg once a day,

OR

D: Enalapril 10mg bid

OR

C: Captopril 6.25 12.5mg tid

Referral

- Myocardial infarction related mitral regurgitation or VSD
- Ongoing chest pain or post-infarct angina
- Refractory ventricular tachyarrhythmias

8.0 CHRONIC STABLE ANGINA PECTORIS

Characteristic chest pain due to myocardial ischaemia usually occurring on exercise and relieved by rest but stable in nature

Treatment

Non pharmacological therapy

- Lifestyle modification.
- Intensive health education.
- Modify reversible risk factors – optimal control of glucose in Diabetic patient, optimal control of blood pressure, stop smoking.

Pharmacological therapy

C: Aspirin oral, 75 -150 mg (O) daily

Plus

A: Atenolol 12.5 – 100mg once a day,

OR

S: Metoprolol 12.5 -50mg once a day,

OR

If β -blocker cannot be tolerated or is contraindicated, consider long acting calcium channel blocker

Long acting calcium channel blocker

e.g.

S: Amlodipine 5 -10mg (O)

OR

C: Nifedipine SR 20 -40mg (O) daily,

OR

D: Verapamil CR 120- 240mg once a day
OR
D: Diltiazem 60mg once a day

Nitrates:

C: Isosorbide mononitrate, 10 -20mg twice daily
OR

C: Isosorbide dinitrate, oral, 20–40 mg, twice daily

At 8:00 and 14:00 for both drugs in order to provide nitrates free period to prevent tolerance.

Statin - HMGCoA reductase inhibitors

C: Simvastatin 20 - 40mg daily with a goal to achieve LDL level $\leq 1.8 - 2.7$ mmol/l

Note:

- This therapy requires good initial evaluation, ongoing support for patients and continuous evaluation to ensure compliance.
- Therapy should be initiated together with appropriate lifestyle modification and adherence monitoring.

REFERRAL

- When diagnosis is in doubt
- High risk patients poorly controlled hypertension, diabetic patients to evaluate severity of inducible ischaemia
- Failed medical therapy

9.0 ATHEROSCLEROTIC PERIPHERAL DISEASE

Diagnosis

History and palpation of pulses confirms diagnosis

Non pharmacological therapy

- Smoking cessation is essential and is the single most important intervention to prevent Progression
- Exercise within exercise tolerance and other lifestyle modifications.

Pharmacological therapy

C: Aspirin 150 mg (O) daily
Plus

C: Simvastatin 10 mg (O) day.

REFERRAL

- Ongoing vascular insufficiency, which may be surgically reversible

10.0 ACUTE PULMONARY EMBOLISM

Clinical Spectrum less than two weeks

- Sudden onset of dyspnoea often with unexplained anxiety (most common)
- Pleuritic chest pain and haemoptysis

- Massive embolism: pleuritic chest pain, cyanosis, right heart failure and shock. Minor emboli or pulmonary infarction may herald massive embolism and must be treated vigorously
- Source of embolus may be found – deep vein thrombus

Investigations

- **ECG** – Not reliable test for diagnosis may be normal. Sinus tachycardia most common, acute right ventricular strain – ie right axis shift, **S1Q3T3** occurs in small percentage of cases, may develop acute bundle branch block – right or left, may simulate right ventricular infarction, may develop arrhythmias – eg atrial fibrillation
- **Arterial blood gases;** not diagnostic, the pO₂ decreased <60mmHg due ventilation/perfusion mismatch. pCO₂ decreased due to hyperventilation, pH increased but may decrease in shocked patient
- **D-dimer test** – Very Sensitive blood test, but not specific. A negative test d-dimer test excludes an embolus in majority of cases
- **Chest X-ray** – Not very reliable usually normal, diaphragm may be raised on affected area, atelectasis may occur, peripheral wedge shaped shadow & plural effusion
- **Cardiac Echocardiography;** Useful in diagnosis, features suggestive or support evidence of massive embolus acute right ventricular strain
- **Computed Tomography Pulmonary Angiogram Scan (CTPA);** Useful can demonstrate the presence and extent of proximal pulmonary emboli
- **Ventilation/Perfusion Scan;** Useful in stable patient to confirm the diagnosis. The presence of a perfusion defect with normal ventilation not corresponding to an x-ray abnormality is characteristic
- **Pulmonary Angiography;** Still gold standard investigation may necessary establish diagnosis and catheter based embolectomy in the catheterization lab.

Treatment

I. General

- Administer O₂ – maintain pO₂ > 60mmHg,
- Treat shock
- Correct electrolyte & acid base abnormalities and arrhythmias
- Ventilate if patient in respiratory failure

I. Anticoagulation

S: Heparin (UFH) 10,000units IV bolus, then maintenance infusion starts with 6,000U over 6hours to keep PTT or clotting time 2-3 times above baseline. PTT should be performed 12hourly according to lab instruction.

OR

S: Enoxaparin 1mg/kg twice daily

Start warfarin after 24hours of heparin

II. Thrombolytic (Fibrinolysis)

Indicated in proximal massive pulmonary emboli & haemodynamically unstable if no contra-indication exists

S: Streptokinase 250,000IU infusion over 30minutes, then 100,000IU per hour for 24hours

OR

S: Alteplase (rtPA) 100mg IV infusion over 2hours

Referral

All cases suspected of pulmonary embolus should be referred to a specialized hospital care

11.0 CARDIAC ARRHYTHMIAS/ DYSRHYTHMIAS

Exclude underlying structural cardiac disease in all patients with cardiac dysrhythmias

Tachyarrythmias:

A) Narrow QRS Complex Tachyarrythmias (SVTs)

Definition

Sustained (> 30 seconds) or non-sustained narrow QRS (\leq 0.1 seconds) tachycardias.

Atrial Fibrillation

Acute onset (< 48 hours)

- Assess clinically, e.g. heart failure, mitral stenosis, thyrotoxicosis, hypertension, age and other medical conditions.
- Consider anticoagulation with heparin or warfarin
- Synchronized DC cardioversion is occasionally necessary in emergency especially haemodynamics instability or consider if is the first episode.

Non-acute/chronic (> 48 hours)

As above, but not immediate DC cardioversion is indicated, unless in hypotensive emergency case. Anticoagulation with oral warfarin 2mg – 5mg orally once a day for at least a month, then perform elective cardioversion at specialized hospital.

Atrial Flutter

- P waves visible before QRS, commonly occurs, usually 2:1. (150 per minute). P waves, usually negative in Lead II precede QRS, blocked P in ST segment or hidden by QRS.
- Vagal stimulation with ECG may reveal blocked P waves.

AV Junctional Re-Entry Tachycardias

- Usually paroxysmal, Often young with normal heart.
- AV nodal re-entry or WPW syndrome. P waves usually not visible (hidden by QRS).

Atrial Tachycardias

- Rare, Often incessant P before QRS (often long PR) or hidden in T
- May cause heart failure (tachycardia cardiomyopathy).

Atrial Fibrillation

Pharmacological Treatment

Initial

- Anticoagulate with warfarin.
- Control the ventricular rate with **one** of the following:

C: Digoxin oral, 0.25mg daily; use only in heart failure.

A: Atenolol, oral, 50–100 mg daily (contraindicated in asthmatics; caution in Heart failure).

- DC cardioversion in selected cases, after 4 weeks Warfarin anticoagulation.

Long – term

- Continue Warfarin anticoagulation long-term, unless contra-indicated: Warfarin, oral, 5 mg daily.
Control with INR to therapeutic range: INR between 2–3: patient is stable do 3 monthly monitoring
If INR < 1.5 or > 3.5: do monthly monitoring

Use:

- Prophylaxis in chronic atrial fibrillation
- Prior to cardioversion to sinus rhythm
- In lone atrial fibrillation of persons 65 or older.

If the patient has a prosthetic valve, **ADD**

C: Aspirin, soluble, oral, 150 mg daily

CAUTION

Use Warfarin only if INR can be monitored regularly. If not, consider use of aspirin.

Rate control

Continue as above.

Digoxin only controls rate at rest and is insufficient on its own.

If used long-term, combine with s-blocker.

In the elderly and patients with renal impairment:

C: Digoxin (O) 0.125 mg initial dose

Adjust dosages according to trough levels within the therapeutic range.

Do levels only if the patient has been on the drug for at least 10 days.

A: Atenolol (O) 50–100 mg daily

Prevention of recurrent paroxysmal atrial fibrillation

Only in patients with severe symptoms despite the above measures:

D: Amiodarone 200 mg (O) 8 hourly for 1 week, followed 200 mg twice daily for one week and thereafter 200 mg daily. Specialist initiated.

Precautions:

- halve dosage of warfarin and monitor INR closely, until stable
- avoid concomitant digoxin
- monitor thyroid function every 6–12 months as thyroid abnormalities may develop

Atrial Flutter

Non pharmacological Treatment – Electrical Cardioversion

Synchronised DC cardioversion, 200 J, after sedation with:

A: Diazepam 10–20 mg IV

If flutter has been present longer than 48 hours, defer cardioversion for 4 weeks after anticoagulation with warfarin, unless severe symptoms or heart failure require urgent conversion.

Pharmacological Treatment

None is nearly as effective as DC cardioversion.

Most drugs have serious side effects. Do not use verapamil as it will **not** convert flutter to sinus rhythm and may cause serious hypotension.

Anticoagulants if sustained.

Long term treatment: Recurrent atrial flutter is an indication for referral. Many can be cured by radiofrequency catheter ablation.

AV Junctional Re-Entry Tachycardias

Non Pharmacological Treatment

Vagal manoeuvres: Valsalva or carotid sinus massage. The patient should be supine and as relaxed as possible, to avoid competing sympathetic reflexes.

Pharmacological Treatment

If vagal manoeuvres fail:

D: Adenosine, rapid IV bolus, 6 mg through a good IV line, followed by a bolus of 10 mL Sodium chloride 0.9% to ensure that it reaches the heart before it is broken down.

Run the ECG for 1 minute after the injection.

If 6 mg fails, repeat with 12 mg.

If the drug reaches the central circulation before it is broken down the patient will experience flushing, sometimes chest pain and anxiety. If the tachycardia fails to terminate without these symptoms, the drug did not reach the heart.

If none of the above is effective, or if the patient is hypotensive, consider DC shock.

CAUTION!! Verapamil and digoxin are contraindicated in WPW syndrome.

Long – term Treatment

Teach the patient to perform vagal manoeuvres, Valsalva is the most effective.

For infrequent, non-incapacitating symptoms:

β –Blockers e.g.:

A: Atenolol 50–100 mg (O) daily (If asthmatic)

OR

D: Verapamil (O) 80–120 mg three times daily (Normal heart)

Referral

NARROW QRS COMPLEX (SUPRAVENTRICULAR) TACHYARRHYTHMIAS (SVTs)

- Poor rate control
- Severe persistent symptoms
- Patients with severe symptoms

REGULAR NARROW QRS (SUPRAVENTRICULAR) TACHYCARDIAS

- Frequent or severe symptoms for curative radiofrequency catheter ablation
- **all** WPW syndrome (sinus rhythm ECG shows delta waves) for radiofrequency catheter ablation

B) WIDE QRS (VENTRICULAR) TACHYARRHYTHMIAS (VTs)

Definition

Sustained (> 30 seconds) or non-sustained wide QRS (> 0.12 seconds) tachycardias

A REGULAR WIDE QRS TACHYCARDIAS

Are **ventricular** until proved otherwise.

Regular wide QRS supraventricular tachycardias are uncommon.

B SUSTAINED (> 30 SEC) IRREGULAR WIDE QRS TACHYCARDIAS

Are usually due to atrial fibrillation with bundle branch block, or pre-excitation (WPW syndrome).

C NON-SUSTAINED (< 30 SEC) IRREGULAR WIDE QRS TACHYCARDIAS

Are usually ventricular.

They are common in acute myocardial infarction.

D TORSADES DE POINTES VENTRICULAR TACHYCARDIA (VT)

Has a twisting pattern to the QRS complexes and a prolonged QT interval in sinus rhythm.

It is usually due to a QT-prolonging drug, ± hypokalaemia.

(A) REGULAR WIDE QRS TACHYCARDIAS

- Refer all cases after resuscitation and stabilisation.
- Emergency DC cardioversion is mandatory with a full protocol of CPR.

Non Pharmacological Treatment

- Cardio-pulmonary resuscitation (CPR).

If no cardiac arrest:

- DC cardioversion, 200 J, after sedation with:
Diazepam, I.V, 10–20 mg
If 200 J fails, use 360 J.

If cardiac arrest:

- Defibrillate (not synchronised).

Pharmacological Treatment

DC cardioversion is first line therapy for regular wide QRS tachycardias. Drugs are needed if VT recurs after cardioversion or if spontaneous termination/recurrence.

D: Amiodarone, IV, 5 mg/kg infused over 30 minutes

Plus

D: Amiodarone 800 mg (O) once daily for 7 days then 600 mg/day for 3 days followed by a maintenance dose of 200–400 mg/day

CAUTION!! Amiodarone may cause a serious long-term side effects and long half-life. Therefore, patients require regular monitoring by specialist.

D: Lidocaine 50–100 mg (1–2 mg/kg) IV initially and at 5 minute intervals if required to a total of 200–300 mg,

Thereafter, for recurrent ventricular tachycardia only

D: Lidocaine, IV infusion, 1–3 mg/minute for 24–30 hours. Lidocaine will only terminate \pm 30% of sustained ventricular tachycardias, and may cause hypotension, heart block or convulsions.

CAUTION!! Never give verapamil IV to patients with a wide QRS tachycardia.

Note: For emergency treatment of ventricular tachycardia, DC cardioversion is first-line therapy, even if stable

(B) SUSTAINED (> 30 SECONDS) IRREGULAR WIDE QRS TACHYCARDIAS

If the QRS complexes have a pattern of typical right or left bundle branch block, with a rate < less than 170/minute, treat as for atrial fibrillation. See Section for Atrial fibrillation.

If the rate is > 170 per minute, and/or the complexes are atypical or variable, the likely diagnosis is WPW syndrome with atrial fibrillation, conducting via the bypass tract, DC conversion.

Do not treat with drugs

Verapamil and digoxin may precipitate ventricular fibrillation by increasing the ventricular rate.

(C) NON-SUSTAINED (< 30 SECONDS) IRREGULAR WIDE QRS TACHYCARDIAS

Most are ventricular.

In acute myocardial infarction, only treat non-sustained ventricular tachycardia if it causes significant haemodynamic compromise. Ensure the serum potassium level is above 4 mmol/L

Pharmacological Treatment

D: Amiodarone, IV, 5 mg/kg infused over 30 minutes. Specialist initiated.

Plus

D: Amiodarone 800 mg (O) once daily for 7 days then 600 mg/day for 3 days followed by a maintenance dose of 200–400 mg/day

OR

Only in a haemodynamically stable patient:

D: Lidocaine, IV, 50–100 mg (1–2 mg/kg) initially and at 5 minute intervals if required to a total of 200–300 mg.

Thereafter, for recurrent ventricular tachycardia only:

D: Lidocaine, IV infusion, 1–3 mg/minute for 24–30 hours

In the absence of acute ischaemia or infarction, consider torsades de pointes, due to QT prolonging drugs.

(D) TORSADES DE POINTES VENTRICULAR TACHYCARDIA (VT)

Non Pharmacological Treatment

- Cardioversion/defibrillation, as necessary.
Torsades complicating bradycardia: temporary pacing.

Pharmacological Treatment

Stop all QT-prolonging drugs.

Correct serum potassium.

B: Magnesium sulphate 2 g I.V over 5–10 minutes

If recurrent episodes after initial dose of magnesium sulphate:

B: Magnesium sulphate 2 g I.V over 24 hours

Torsades complicating bradycardia:

A: Adrenaline infusion to raise heart rate to > 100 per minute (if temporary pacing unavailable).

Referral

All cases of wide QRS tachycardia, after resuscitation and stabilisation

12.0 HEART BLOCK (SECOND OR THIRD DEGREE)

The majority of cases occurs in patients over 60 years and is idiopathic, with an excellent long-term prognosis, provided a permanent pacemaker is implanted. Acute, reversible AV block commonly complicates inferior myocardial infarction. The condition may also be induced by metabolic and electrolyte disturbances, as well as by certain medicines.

Non Pharmacological Treatment

- Emergency cardio-pulmonary resuscitation.
- External pacemaker should be available in all secondary hospitals and must be preceded by appropriate analgesia.

Pharmacological Treatment

Analgesia if external pacemaker:

C: Morphine 10–15 mg IM 3–6 hourly

AV nodal block with narrow QRS complex escape rhythm only:

B: Atropine, I.V bolus, 0.6–1.2 mg, May be repeated until a pacemaker is inserted.

Use in a patient with inferior myocardial infarct and hypotension and second degree AV block. It is temporary treatment of complete AV block before referral (urgently) for pacemaker.

OR

For resuscitation of asystole:

A: Adrenaline 1:10 000, slow IV, 5 mL (0.5 mg)

Used as temporary treatment of complete heart block when other drugs are not effective

Referral

**CAUTION!! HEART BLOCK IS A MEDICAL EMERGENCY.
REFER URGENTLY!**

- All cases with a heart rate below 40 beats/minute after resuscitation and stabilisation
- All cases of second or third degree AV block, whether or not myocardial infarct or other reversible cause is suspected, and whether or not the patient is thought to be symptomatic
- A permanent pacemaker is the definitive form of treatment. This service is only available in Muhimbili Cardiovascular Institute (tertiary institutions) for now.

13.0 SINUS BRADYCARDIA & SINUS ARREST

This rhythm does not require treatment, unless they are causing symptoms, i.e. syncope, dizziness, tiredness and poor effort tolerance.

Sinus bradycardia < 50/minute or sinus arrest with slow escape rhythm, accompanied by hypotension, strongly suggests a treatable underlying cause:

- Acute inferior myocardial infarct
- Hyperkalaemia, especially if wide QRS and/or peaked T waves
- Drugs, especially combination of verapamil and β -blocker or digoxin
- Hypothermia
- Hypoxia

Treat the cause. Consider atropine if inferior infarct.

SINUS ARREST

- Refer all to a cardiologist specialists.

CHAPTER SEVEN

SKIN DISEASES AND ALLERGIC REACTIONS

1.0 BACTERIAL SKIN INFECTION

Bacterial skin infections can range from impetigo, folliculitis, furunculosis, erysipelas, cellulitis to recurrent boils. All these are caused by either staphylococcus alone or together with streptococcus but rarely streptococcus alone. There are other non-bacterial skin infections i.e.viral (warts, herpes simplex, herpes zoster and varicella, kaposi varicelliform eruption), fungal (candidiasis, tinea corporis, pityriasis versicolor), skin infestations (scabies and pediculosis)

1.1 Impetigo

It is bacterial infection of subcorneal layer of epidermis with characteristic honey-colored serous crusts. It is usually caused by a staphylococcus aureus. It occurs commonly in school children, usually starting on the face, especially around the mouth or nose. May form bullous lesions (bullous impetigo) characteristically flaccid.

Treatment Options

- Remove crusts
- Wet dressings: weak PP soaks, 1:40000 (0.025%) solution 0.5% GV paint
- Apply Topical mupirocin 2% b.d for 5 to 7days OR Topical fusidine b.d for 5 to 7days
- Simply wash with soap and water
- Keep infected areas clean and prevent spread to others [care with towels, clothes, beddings; change frequently)
- If severe, or systematic symptoms are present (e.g. Pyrexia) add an oral antibiotic.

Drug of Choice is

A: Phenoxymethylpenicillin (O)for 7-10 days

Adults	250 – 500mg every six hours
Children	25mg/kg/24 hrs every six hours

Second Choice

A: Erythromycin (O) for 7-10 days

Adult	250 – 500mg every 6 hours
Children	25-50mg/kg/24 hrs in 4 divided doses

OR

A: Cloxacillin (O) for 7 – 10 days

Adults	250 – 500mg four times daily (every 6 hours)
Children	50 – 100 mg/kg/24hrs every 6hours in equal doses

1.2 Folliculitis

It is the inflammation of the hair follicle. The most common forms are caused by invasive staphylococcus but other bacteria, viruses, and fungi may also be responsible. Other forms (eosinophilic folliculitis in HIV/AIDS) are non infectious. Mechanical irritation is also a factor, such as prolonged sitting. Deep follicular inflammation often occurs in the bearded areas of the face (Sycosis barbae).

Treatment

- Suspected irritants should be avoided
- Use of suitable disinfecting and cleansing agents should be encouraged
- Appropriate anti-infective skin preparations (Neomycin sulphate, gentamycin oxytetracycline cream/ointment or mupirocin ointment 2% can be used
- If severe, or systematic symptoms are present (e.g. Pyrexia) add an oral antibiotic or systemic antibiotics (penicillinase-resistant penicillins or first-generation cephalosporins for 7–10days).

1.3 Furunculosis

It is deep follicular infection that starts as a firm red nodule which rapidly becomes painful and fluctuant in a few days. Healing with scarring it follows over several weeks. In some individuals it is chronic and recurrent.

Treatment

- Usually resolves spontaneously, but is improved by placing hot compresses over the boil until it breaks
- In a healthy person, review after 2 days, if not improving consider surgical incision and drainage

Note: If the boil causes swollen lymph nodes and fever, consider systemic antibiotics

Drugs of Choice

Adults

C: Flucloxacillin (O) 500mg four times daily for 7 -10 days;

OR

A: Erythromycin (O) 500mg 8hrly for 7-10days;

Children

C: Flucloxacillin (O) 50-100mg/24hrs every 6hours in equal doses

OR

A: Erythromycin (O) 25-50mg/kg/every 8 hours in a day.

For recurrent furuncles (furunculosis): Give systemic antibiotics (often clindamycin 300mg B.D. for 7–10days), search for predisposing factors (diabetes mellitus, immunosuppression, perineal or nasal carriage of *Staphylococcus aureus*)

1.4 Erysipelas

It is bacterial infection of upper half of dermis with lymphatic vessel involvement, due to streptococcal infection Group A B Hemolytic Strep (Group C, G and B). The disease begins as a small break in the skin or umbilical stump (infants). The affected area has growing redness, accompanied by high fever and pains.

Treatment

- Bed rest
- Lifting the affected part

A: Potassium permanganate solution 1:4000

OR

C: Mupirocin ointment 2% application may be beneficial

Plus

A: Phenoxymethylpenicillin or Amoxicillin (O) 500mg 6hrly for 7-10 days

Children

A: Phenoxymethylpenicillin or Amoxicillin (O) 25-50mg/kg 6hrly for 7-10 days

Note: Erysipelas has a tendency to recur in the same area, especially if there are predisposing factors such as chronic lymphatic oedema. In recurrent episodes, increase the **duration of antibiotics to 10 – 14 days**

1.5 Acute Cellulitis

It is a deep inflammation involving lower half of dermis and subcutaneous tissue most commonly caused by streptococci or staphylococci. Acute cellulitis should be differentiated from erysipelas as follows:

- Raised, sharply demarcated margins from uninvolved skin erysipelas;
- Indistinct borders – acute cellulitis and accompanied with systemic symptoms

Treatment

- Immobilise
- Limb elevation
- NSAIDS
- Systemic antibiotics:

C: Flucloxacillin (O) for 10-14 days 500mg four times daily, Children Flucloxacillin 50-100mg/24hrs every 6hours in equal doses

OR

A: Erythromycin (O) 500mg 8hrly for 10-14days, Children (O) 25-50mg/kg/every 8 hours in a day for 10 -14days

Note: Acute cellulitis can be serious if not treated early (spreads through lymphatics and bloodstream). Refer to dermatologist.

1.6 Acne

It is a multifactorial disease primarily of teenagers with follicular plugging and inflammation. Polymorphic lesions include open and closed comedones, papules, pustules nodular and cystic lesions involving the face, chest, shoulders and back.

Treatment

- Seek underlying cause e.g. stress, overuse of ointments on skin, steroids or anticonvulsant drugs etc.
- Encourage a healthy lifestyle – exercise, sunshine, diet, etc
- Use ordinary soap (harsh antibacterial cleansers or iodine-containing preparations may aggravate the acne)

Treatment of choice:

A: Benzoyl peroxide 2.5% gel topically at night for 3-6 months

Plus

A: Doxycycline (O) 100 mg once daily for 2-4 months.

OR

S: Retinoic acid topically 0.025-0.05% at night

If unresponsive refer to specialist for oral retinoids (isotretinoin 0.5 -1mg/kg) and further assessment.

Note: The acne may initially worsen with treatment. If too irritant, use every second or third night. Patients should be encouraged to persist with treatment.

1.7 Paronychia

It is inflammation of the nail fold characterized by painful red swellings of the nail folds which may be due to bacteria or yeast.

Acute Paronychia

Treatment

Tenderness and presence of pus indicates the need for systemic antibiotics

Drug of choice

A: Phenoxymethylpenicillin (O) 500mg 6hrly for 7-10 days

Second choice

Adults **C:** Flucloxacillin (O) 500mg 6hrly for 7-10 days

Children **C:** Flucloxacillin (O) 25-50mg/kg every 6hrs for 7-10days

Chronic Paronychia

Often it is a fungal infection, due to candida. Avoid excessive contact with water, protect from trauma and apply:

A: Miconazole or Clotrimazole cream, apply twice daily for 7-10days

OR

B: Fluconazole (O) 200mg-400mg weekly for 3-6months (pulse therapy)

OR

S: Itraconazole (O) 200mg once daily for 14days

Treat secondary infection with antibiotics as above

Note: For both acute and chronic paronychia, incision and drainage may be needed

2.0 FUNGAL SKIN INFECTIONS

2.1 Dermatophytosis (Ringworm)

It is a chronic fungal infection determined by the nature of the dermatophyte, by the tissue it invades i.e. skin, hair or nails and by the degree of host response. Infections with dermatophytes are usually called *tinea*; for further description, the anatomical site is added. The clinical infection usually starts from an inoculation site and spreads peripherally hence the annular lesions with an active border. In non medical jargon, the diagnosis is often known as "ringworm". It is sometimes accompanied by loss of hair, itching and pustules.

2.1.1 Tinea Corporis (Body Ringworm)

Annular, expanding lesions with central healing and distinct borders on the body or face. A fine scale may be present.

Treatment

Drug of choice

A: Compound benzoic acid (Whitfield's ointment) applied two times a day for up to 4 weeks.

Second choice

A: Clotrimazole cream 1% applied thinly two times a day, continue for 5 to 7 days after clearing of lesions

OR

C: Miconazole cream 2%, and apply thinly two times a day. Continue for 5-7 days after clearing of lesions.

2.1.2 Tinea Capitis (Scalp Ringworm)

In this case, the fungus has affected the hair follicle. Topical treatment is not effective. Treat with:

B: Griseofulvin (O) 500mg daily for 6 week, **together with fatty meals**
Children 15-20mg/kg once daily

Note: Do not crush the tablet (micronised tablet)

2.2 Pityriasis Versicolor

Common fungal infection caused by yeast. Hypopigmented/hyperpigmented confluent patches of varying size with fine scale on the chest, back, arms and occasionally neck and face.

Treatment

- Apply whitfield ointment, miconazole **or** clotrimazole cream into scales twice daily for 2 weeks

OR

- **C:** Sodium thiosulphate solution 20% twice daily for 2 weeks
- Oral ketoconazole may be used in more widespread lesions, 200mg once a day for 2 weeks.

2.3 Tinea Pedis (Athlete's Foot)

This is a very common fungal infection and is often the source of infection at other sites. Treat any bacterial superinfection first:

First choice:

A: Whitefield's lotion twice daily for 2 weeks

Second choice: If fails to respond, try

A: Clotrimazole cream 1% twice daily for 2 weeks.

OR

C: Miconazole cream 2%

OR

C: Tolnaftate solution twice daily

In severe infections use

D: Terbinafin 250mg once or twice for 2 weeks to 1 month.

ADVISE: Frequent change of socks/footwear, use of cotton socks, thorough drying between toes after bathing, separating the opposing skin surfaces (e.g. with a piece of gauze) will help speed up healing

2.4 Candidiasis

It is caused mainly by candida albicans. Clinical features depend on the site of infection. The skin lesions are characterized by an erythematous, moist exudate in the skin folds. Patients may develop subcorneal and satellite pustules.

Involvement of the nails lead to painful swelling of the nail bed and folds which may discharge pus and is made worse by contact with water. There may be destruction of the nail plate.

Oral lesions are characterized by white, adherent mucosal plaques in buccal cavity including tongue which may be forcibly removed. May extend to oesophagus and lower GIT.

Vulval-vaginal candidiasis is characterized by itchy, curd-like whitish vaginal discharge, dysuria and dyspareunia.

Candidiasis is usually precipitated by prolonged use of contraceptive pills, pregnancy, diabetes, prolonged antibiotic and corticosteroid use AND immunosuppressive treatment.

Treatment

I. Gastrointestinal Tract (G.I.T) candidiasis

B: Nystatin oral suspension- gargle and swallow 4 times a day

-Newborns: 200,000-400,000 Units/day

-<2 years old 400,000-1,000,000 Units/day

->2 years old 1,000,000-2,000,000 Units/day

OR

C: Miconazole oral gel apply every 8 hours for 7 days

OR

B: Fluconazole 200mg once daily for 14 days in adults

For angular cheilitis-

A: Nystatin cream or ointment 12 hrly for 2-4 weeks

II. Vaginal infections

B: Nystatin vaginal pessaries; insert 1 at night for 14 days

OR

A: Clotrimazole vaginal pessaries; insert 1 at night for 6 days

OR

C: Miconazole vaginal pessaries insert/apply once at night for 3 days

OR

B: Fluconazole 150mg stat

Referral

If recurrent or unresponsive to treatment, refer to specialist

2.5 Deep fungal infections

The common clinical entities of deep fungal infections are Nocardiosis and Madura foot which may be a Mycetoma or an actinomycetoma. Mycetoma is caused by *Mycetozoa* and Actinomycosis by actinomyces. The clinical features depend on the infected site and can last months to years.

- **First lesion:** nodule
- **Localisation:** feet, legs, arms, buttocks, scalp, trunk
- **Discharging sinuses:** Grains may be visible usually black for Eumycetomas and white yellow for Actinomycetomas. Patients usually experience pain before rupture of discharging sinus.

Treatment

For Actinomycetomas

A: Co-trimoxazole 960mg every 12 hours

Plus

S: Rifampicin 300mg every 12 hours for 2-4 months

Alternative drugs

for Adults:

A: Phenoxymethylpenicillin(O) 500 mg every 6 hours 2-4 months;

for Children: Phenoxymethylpenicillin (O) 25 mg/kg body weight 6 hourly for 2-4 months.

For Eumycetomas

Trial of antifungals e.g. itraconazole, voriconazole, ketoconazole is recommended. Usually necessitates long term treatment, at least one year.

CAUTION: Doxycycline should not be given to pregnant women and children under 12 years of age

NOTE: Regular blood examination must be done when Co-trimoxazole is used for more than 14 days

Referral

- For **Radical surgery**, refer to the specialist for the initial management
- In complicated cases of eumycetoma refer to specialist for further management. Surgery is often necessary and includes wide margins, sometimes amputation.

Alternative drug for Nocardiosis

Adult:

S: Dapsone 100 mg every 24 hours for 2-4 months

Children: **Dapsone** 25 – 50 mg every 24 hours for 2-4 months

3.0 PARASITE INFECTION

Scabies

Scabies is an intensely pruritic and highly contagious infestation of the skin caused by a mite *Sarcoptes scabie* burrowing into the skin; affecting humans and other animals. The main clinical features are, a short elevated serpiginous (S-shaped) track in the superficial epidermis, known as a burrow, this is pathognomonic of a scabies infestation. A small vesicle or papule may appear at the end of the burrow or occur independently. Norwegian scabies presents with extensive crusting (psoriasiformlike lesions) of the skin with thick, hyperkeratotic scales overlying the elbows, knees, palms, and soles.

Treatment

A: Benzoyl Benzoate Emulsion 25% (12.5% for children) apply every 12 hours for 3 days. Repeat treatment after 1 week.

Note

- Treat **all** close contacts, especially children in the same household with
- Wash clothes and beddings, leave in the sun to dry followed by ironing.
- Secondary bacterial infection, (septic scores'') treat with antibiotics as in impetigo for 5 days.
- Explain that the itch may continue for several weeks after treatment. In case of itching apply steroid

4.0 VIRAL INFECTION

4.1 Herpes Simplex

It is an acute viral infection characterized by superficial vesicles containing clear fluid in the skin and mucous membranes, particularly of the buccal area, on the conjunctiva, corneas or genitalia. It is caused by the herpes virus homines. The main clinical features are: prodromal symptoms of tingling discomfort or itching, followed by vesicular formation.

Treatment

B: Acyclovir (O) 400mg 8 hourly for 7 – 10 days

Note: Use of systemic Acyclovir is optimum when given within the first 48

4.2 Herpes Zoster (Shingles)

It is due to the resurgence of the varicella-zoster virus which also causes chickenpox. Severe burning pain precedes the appearance of grouped vesicles overlying erythematous skin and following a dermatome; does not cross the midline. The disease may heal with scarring.

Treatment

A: Acyclovir cream 5% applied until vesicles disappear.

Plus

B: Acyclovir (O) 800 mg 5 times a day until no new lesions appear

- **Wound care:**
 - A:** Potassium Permanganate soaks (1:4000)

- For Secondary infection (bacterial) apply 12 hrly topical
 - B:** Gentamycin 1% ointment
 - Or
 - C:** Mupirocin 2% cream

Post-Herpetic Neuralgia

After the lesions have resolved:

C: Amitriptyline (O) 25 mg at night, may be increased to 150 mg at night

OR

C: Carbamazepine (O) 100 mg at night; may be gradually increased to twice a day according to response.

CAUTION: Refer if there is no improvement of severe neuralgia. Refer immediately in case of herpes zoster ophthalmicus for atropinization

4.3 Chicken Pox

Chicken pox like herpes zoster is caused by the varicella zoster virus. Lesions are preceded by fever and characteristically vesicular in different stages of development. It is self limiting.

Treatment complications

Adult

A: Paracetamol 1 g every 8 hours

Plus

A: Calamine lotion with 1% phenol, apply over the whole body every 24 hours

Children

A: Paracetamol 10 mg/kg body weight every 8 hourly

Plus

A: Calamine lotion with 1% phenol in adults

5.0 ALLERGIC CONDITIONS

5.1 Allergic Contact Dermatitis

It is a delayed hypersensitivity following skin contact with a particular chemical (dye, perfume, rubber, nickel or drugs, skin preparations containing lanolin, iodine, antihistamines, neomycin, vioform etc).

Management

Avoid contact if allergic.

5.2 Eczema

Atopic Dermatitis/Eczema: Often a personal or family history of atopic disease (asthma, hay fever or atopic dermatitis). Exact cause is not known. These persons are also more susceptible to herpes simplex and vaccinia (but not varicella-zoster).

The clinical form may differ according to age

I. Infantile eczema ("milk crust"): usually appears at 3 months of age with oozing and crusting affecting the cheeks, forehead and scalp.

IMPORTANT: If generalized exfoliative dermatitis develops, refer to a specialist

II. Flexural eczema: starts at 3-4 years, affecting the flexure surface of elbows, knees and nape of neck (thickening and lichenification). In adults any part of the body may be affected with intense itching, particularly at night.

Note: Eczema may evolve through acute (weepy), subacute (crusted lesions), and chronic (lichenified, scaly) forms.

Treatment of Eczema

- Remove any obvious cause e.g. skin irritants or allergens (avoid irritants e.g. medicated soap, wool and extremes of temperature).
 - D:** Apply Emulsifying ointment - the equivalent of cream E45, Sofderm cream
- Treat itching with an oral antihistamine:
 - A:** Chlorpheniramine (O) 4-16 mg at night
 - OR**
 - A:** Promethazine (O) 25mg at bedtime increased to 50mg if necessary
 - OR**
 - C:** Cetirizine 10mg
 - OR**
 - C:** Loratadine 10mg once daily

CAUTION: Never use topical antihistamines

- Treat any infection (usually bacterial, but occasionally viral). Choice of skin preparations depends on whether lesions are wet (exudative) or dry/lichenified (thickened skin with increased skin markings).
- If eczema is "weepy", use saline baths or bathe in:
 - A:** Potassium permanganate 1:4000 (0.025%) solution once daily for 2-4 days until dry. Where large areas are involved give a course of antibiotics for 5-10 days (as for impetigo)
- After the lesions have dried, apply an aqueous cream for a soothing effect. A topical corticosteroid cream may be useful in the acute phase. Use the mildest topical corticosteroid which is effective, start with:
 - C:** Hydrocortisone 1% cream for wet, ointment for dry skin. Apply thinly, initially, two times a day.

CAUTION: Only use 1% hydrocortisone on the face unless prescribed by a specialist

Note: Potent topical corticosteroids may cause harmful cutaneous and systemic side effects especially if use is prolonged or involves extensive body surface. Striae, acne, hyperpigmentation and hypopigmentation, hirsutism and atrophy may result. Avoid long term use; don't use on weepy or infected skin. Advise patients NOT to use them as cosmetics

- If the skin starts scaling (condition becomes chronic), add/apply an emollient such as: emulsifying ointment or liquid paraffin.

5.3 Urticaria

It may be allergic, toxic or physical in origin. In many cases the cause is unknown (idiopathic). Allergic urticaria may be caused by: drugs (e.g. penicillin), infection, contact with plants, pollen, insect bites, or foodstuff (e.g. fish, eggs, citrus fruits, nuts, strawberries, tomatoes). Physical urticaria may be caused by mechanical irritation, cold, heat, sweating.

Treatment

- If acute (existing for less than 3 months), exclude drug reactions (e.g. penicillin), or infection
- Give oral antihistamines:
 - A:** Chlorpheniramine (O) 4-16 mg once at night
 - OR**
 - A:** Promethazine (O) if sleeplessness is a feature: **Adults**, 25 -50 mg at night
 - OR**
 - C: Cetrizine (O)** 10mg once daily
 - OR**
 - C: Loratadine (O)** 10mg once daily
 - Children 0.1 – 0.2 mg/kg 2-4 times a day or 0.5 mg/kg at bedtime
- Deworm patients with Albendazole (O) 400mg stat in adults.

Note: Warn about drowsiness. If no improvement after 1 month or chronic problem, refer to specialist for combination therapy (H1, H2 inhibitors).

5.4 Psoriasis

An inherited inflammatory condition of the skin characterized by thick, silvery white scaly plaques affecting mainly scalp and extensor body surfaces usually symmetrically distributed with a chronic relapsing course.

Note: Exclude precipitating factors e.g. alcohol, deficiencies of B12 or folate, stress, infections.

Treatment

- Sun exposure to the lesions for half an hour or one hour daily may be of benefit
 - C:** Crude Coal tar 5% in Vaseline in the morning
 - Plus**
 - C:** Salicylic acid 5% in Vaseline to descale
 - Plus**
 - C:** Betamethasone ointment 0.025% in the evening.
- Alternatively:
 - C:** Dithranol 0.1% once a day
 - OR**
 - C:** Calcipotriol 0.05% ointment OD (vitamin D derivative)

Note: Systemic steroids are discouraged in this condition due to their rebound effect. If not responding well, refer to specialist for appropriate systemic treatment with methotrexate, cyclosporine, azathioprine etc.

6.0 OTHER SKIN DISEASES

6.1 Pellagra

Syndrome caused by deficiency of a variety of specific factors, nicotinic acid being the most important. Cardinal signs: diarrhea, dermatitis (sites exposed to sun and pressure) and dementia.

Important skin findings include:

- Casal's necklace; hyperpigmented scaling involving the neck region
- Hyperpigmented scaly lesions on sun exposed areas

Treatment

Treat both adults and children with:

C: Nicotinamide (O) 500mg once daily for four weeks or until healing is complete;

Children give 5mg/kg per day for children.

Advice on Diet: The diet should be rich in protein (meat, groundnuts, and beans)

6.2 Vitiligo

It is a condition that causes patches depigmentation of skin. It occurs when melanocytes, the cells responsible for skin pigmentation die. Clinical features include depigmentation of patches of skin that occurs on the face, neck, trunk and extremities

Treatment

There is no cure for vitiligo, but there are a number of treatments that improve the condition. Treatment options generally fall into four groups:

- Sub block
- Skin camouflage
- Corticosteroids
- Depigmentation

Note: Counsell the patient about the condition

6.3 Brucellosis (Undulant fever)

Brucellosis is an infection caused by Brucella organisms. Man gets infected through exposure to infected tissue and milk or milk products. It is characterized by sweating, weakness, headache, anorexia, fever, malaise, arthralgia, weight loss, and pain in the limbs, back and rigorous. There is splenomegaly, lymphadenoraphy and hepatomegaly.

Treatment

Adults:

A: Doxycycline (O)100mg once daily for 4 weeks
Plus

A: Co-trimoxazole (O) 960 mg every 12 hours for 4 weeks.

Children: 6 weeks – 5 years

A: Co-trimoxazole (O)0.5ml syrup/kg every 12 hours for 4 weeks;

Children 5-12 years

A: Co-trimoxazole 480 mg every 12 hours for 4 weeks
Plus

S: Streptomycin 20- 40 mg/kg body weight for 10 days

CAUTION: Doxycycline should not be used in children under 12 years or during pregnancy

6.4 Lichen Planus

It is a chronic inflammatory skin condition, extremely pruritic. Primary lesions are characterized by violaceous, shiny flat topped papules which may coalesce and evolve into into scaly plaques distributed over inner wrists, arms and thighs as well as sacral area. Post inflammatory hyper pigmentation is common. Scarring alopecia may result from lichen planopilaris (severe)

Treatment

A: Chlorpheniramine (O) 4mg 6 hourly

Plus

A: Betamethasone valerate ointment 0.1% twice daily

Plus

D: Clobetasol propionate ointment 0.05% -0.1% twice daily

In severe case refer to specialist for systemic corticosteroid and topical application under occlusion

6.5 Drug Reactions

Drug reactions can be classified in many ways. One useful approach is to separate predictable reactions occurring in normal patients from unpredictable reactions occurring in susceptible patients.

Predictable adverse reactions

- Overdosage (wrong dosage or defect in drug metabolism)
- Side effects (sleepiness from antihistamines)
- Indirect effects (antibiotics change normal flora)
- Drug interactions (alter metabolism of drugs; most commonly the cytochrome P-450 system)

Unpredictable adverse reactions

- Allergic reaction (drug allergy or hypersensitivity; immunologic reaction to drug; requires previous exposure or cross-reaction).
- Pseudoallergic reaction (nonimmunologic activation of mast cells).
- Idiosyncratic reaction (unexplained reaction, not related to mechanism of action, without known or suspected immunologic mechanism).

Note:

- Although we will concentrate on cutaneous drug reactions, remember that every organ system can be affected.

- Almost every drug can cause almost every type of reaction. Clinically, one must learn which reactions are most likely to produce certain findings.
- 80% of allergic and pseudoallergic drug reactions are caused by β -lactam antibiotics, aspirin, NSAIDs, and sulfonamides

Types of Drug Reactions

The most common types of drug reactions are macular and maculopapular exanthems along with urticaria and angioedema; Fixed drug eruption and erythema multiforme/toxic epidermal necrolysis

I. Exanthemous Reaction

. Main differential diagnostic consideration is viral exanthem or on occasion acute exanthem such as guttae psoriasis or pityriasis rosea.

Drugs commonly responsible: Ampicillin, amoxicillin, aminoglycosides, allopurinol, barbiturates, benzodiazepines, carbamazepine, co-trimoxazole, gold salts, penicillin, phenytoin, piroxicam

II. Fixed drug Eruption.

It is a cutaneous drug reaction that recurs at exactly the same site with repeated exposure to the agent. Clinical features include typically red-brown patch or plaque; occasionally may be bullous. Most common sites are genitalia, palms, and soles, as well as mucosa. Lesions typically 5–10cm in diameter but can be larger; often multiple. It starts as edematous papule or plaque; later becomes darker. Frequently resolves with postinflammatory hyperpigmentation. It is uncommon in children.

Note: When confronted with hyperpigmented macule on genitalia, always think of fixed drug eruption

Management: Avoidance of triggering agent; topical corticosteroids may speed resolution

III. Severe Skin Reactions

a. Erythema multiforme

Most erythema multiforme is caused by herpes simplex virus, especially if recurrent. The classical clinical findings are iris or target lesions, most often on the distal limbs. Lesions caused by mycoplasma or especially drugs are more often on the trunk and less like to have a target pattern. We prefer the term *erythema multiforme-like* for such lesions, which carry the risk of developing into severe skin reactions.

b. Stevens Johnson Syndrome (SJS)

It is a combination of erythema multiforme with mucosal lesions as well as systemic signs and symptoms whereby more than 90% of the skin area is involved

Clinical features:

- Patients almost invariably have prodrome with fever, malaise, or arthralgias.
- Abrupt development of erythema multiforme
- Mucosal involvement

- Mouth (100%): Erosions, hemorrhage and crusts on lips, and erosions in
- Mouth covered by necrotic white pseudomembrane.
- Eyes (70–90%): Erosive conjunctivitis, can lead to scarring.
- Genitalia (60–70%): Painful erosions.
- When mycoplasma is trigger, pulmonary involvement is possible (20%).

Management

- Short burst of systemic corticosteroids helpful in many cases but two problems:
- Exclude or treat underlying infection, which could be worsened by immunosuppression..
- Routine topical care: disinfectant mouth washes, antibiotic or corticosteroid eye drops (after ophthalmologic consultation).

c. Toxic epidermal necrolysis

It is a severe life-threatening disorder with generalized loss of epidermis and mucosa

Clinical features:

- Prodrome depends on underlying disease and triggering drug
- Sudden onset of either diffuse maculae (erythema multiforme–like drug reaction) or diffuse erythema without maculae
- Then prompt progression towards widespread erythema and peeling of skin; skin lies in sheets and folds on the bedding.
- Extensive mucosal erosions.
- Possible loss of hair and nails, as well as extensive postinflammatory hypopigmentation.
- Multiple systemic problems because of fluid and protein loss, difficulties in temperature regulation, fever, leukocytosis, and risk of secondary infections.

Treatment

Systemic corticosteroids, if employed, should be used early to attempt to abort the immunologic reaction. Later in the course, they probably increase risk of infection and slowhealing.

A: Prednisolone 80–120mg daily.

Note: Ophthalmologic monitoring is essential, as risk of scarring and blindness is significant

d. Pruritic papula eruptions (PPE)

This is a skin condition characterized by itchy papular eruptions on the extensor area of the upper and lower limbs which is associated with HIV infection.

Treatment

C: Betamethasone valerate 0.025% 12 hourly for 3-4 weeks

OR

S: Dapsone 100mg once a day for one month

6.6 Albinism

Definition

Albinism is an inherited condition present at birth, characterized by a lack of pigment that normally gives color to the skin, hair, and eyes. Many types of albinism exist, all of which involve lack of pigment in varying degrees. The condition, which is found in all races, may be accompanied by eye problems and may lead to skin cancer later in life if not well prevented at early childhood.

Diagnosis

It's not always easy to diagnose the exact type of albinism a person has; there are two tests available that can identify only two types of the condition. Recently, a blood test has been developed that can identify carriers of the gene for some types of albinism; a similar test during amniocentesis can diagnose some types of albinism in an unborn child. A chorionic villus sampling test during the fifth week of pregnancy may also reveal some types of albinism.

The specific type of albinism a person has can be determined by taking a good family history and examining the patient and several close relatives.

The "hairbulb pigmentation test" is used to identify carriers by incubating a piece of the person's hair in a solution of tyrosine, a substance in food which the body uses to make melanin. If the hair turns dark, it means the hair is making melanin (a "positive" test); light hair means there is no melanin. This test is the source of the names of two types of albinism: "ty-pos" and "ty-neg."

The tyrosinase test is more precise than the hairbulb pigmentation test. It measures the rate at which hair converts tyrosine into another chemical (DOPA), which is then made into pigment. The hair converts tyrosine with the help of a substance called "tyrosinase." In some types of albinism, tyrosinase is not active and hence melanin production breaks down.

Prevention

- Genetic counseling is very important to prevent further occurrences of the condition.
- Mechanical preventions such as long sleeve shirt, bouze , skirt and trousers and wide brim hat to prevent skin cancers

Adults and Children

C: SPF 30+(Contains-Titanium Dioxide 9% , Zinc Oxide 8%)

Apply twice a day at 8am and 2pm Daily.

Treatment

There is no treatment that can replace the lack of melanin that causes the symptoms of albinism. For the eye problems that often accompany the lack of skin color, glasses which are tinted should be worn to ease pain from too much sunlight.

There is no cure for involuntary eye movements (nystagmus), and treatments for focusing problems (surgery or contact lenses) are not effective in all cases.

Senile Pruritus

Itching associated with degenerative changes that occur in aging skin.

Treatment

Skin lubrication twice daily with Glycerin

CHAPTER EIGHT

MUSCULO SKELETAL AND JOINT DISEASE CONDITIONS

1.0 INFECTIONS

1.1 Osteomyelitis

Osteomyelitis denotes infection of the bone and is most common in children under 12 years. Staphylococci are the most frequent responsible organisms. Salmonella osteomyelitis infection is a common complication of sickle cell anaemia. Tuberculous osteomyelitis occurs in association with having tuberculosis

Diagnosis

- Common symptoms are fever, malaise and severe pain at the site of bone infection
- If the infection is close to a joint there may be a 'sympathetic' effusion

Table 1:Types of Bone Infection and Treatment

Condition	Treatment	Duration
Acute Osteomyelitis	Surgical drainage (recommended in all cases presenting with history > 24 hours) Cloxacillin (I.V) 1 to 2 g 4 times a day Or Clindamycin (IV) 600 mg three times a day. See Notes on Acute Osteomyelitis in text.	6 weeks or stop at 3 weeks if X-ray normal
Chronic Osteomyelitis	Surgery. Antibiotics not generally recommended	
Osteomyelitis in patient with sickle cell anemia	Ampicillin (I.V) 2 g four times a day plus Cloxacillin (I.V) 1 to 2g four times a day Plus Chloramphenicol (I.V) 500 mg four times a day (if salmonella is suspected)	5 to 12 weeks 6 to 12 weeks 2 to 3 weeks
Septic Arthritis	Surgical drainage Cloxacillin or Clindamycin as for acute osteomyelitis	
Gonococcal Arthritis	Benzympenicillin (I.V) 2.5 to 5 MU four times a day or (if penicillin resistant) Kanamycin (I.M) 2 g once daily	6 days 7 days
Compound Fracture (no infection established)	Cloxacillin (I.V) 1 g four times a day Or Clindamycin (I.V) 600 mg 3 times a day Ceftriaxone 1 gram 3 times a day	3 days

Note: Acute Osteomyelitis

- Culture and sensitivity tests are essential to determine further treatment
- For Osteomyelitis, treatment may be completed orally after 4 weeks, if fever and toxicity have resolved.
- ESR useful as guide of efficacy of treatment
- **Fusidic acid** may be a better alternative in the very sick patients.

Treatment of Acute Osteomyelitis

Adults:

C: Cloxacillin 2-3 g IV every 6 hours for 7 days then orally for 4 weeks

OR

S: Clindamycin 0.3 – 0.6 g I.V every 6 hours for 7 days and treat orally for a total of 4 weeks.

Children:

C: Cloxacillin 25 mg/kg body weight IV initially every 6 hours for 7 days and then orally for 4 weeks.

Treatment for in-patients with sickle cell osteomyelitis

Adults:

C: Ampicillin 2 g IV every 6 hours for 7 days then orally 4 weeks

Plus

C: Cloxacillin 2 g IV every 6 hours for 7 days then orally 4 weeks.

Children:

C: Ampicillin 50mg/kg body weight IV every 6 hours

Plus

C: Cloxacillin 25mg/kg body weight IV every 6 hours for 7 days then orally 4 weeks

Further treatment should be influenced by results of culture and sensitivity.

In case of salmonella being identified then give:

B: Chloramphenicol 500 mg IV every 6 hours for at least 21 days

Plus

A: Benzyl Penicillin 1.2 MU IV or IM every 6 hours for at least 21 days

In chronic osteomyelitis: surgery may be indicated. In all cases of osteomyelitis, pain should be treated with an adequate analgesic

A: Paracetamol 1000 mg every 6 hours

In severe cases

C: Pethidine 1 mg/kg body weight I.M when necessary.

C: Morphine syrup (PO)

Children

A: Paracetamol 10mg/kg body weight every 8 hours.

1.2 Tropical Pyomyositis

The cause of tropical pyomyositis is uncertain since abscesses explored early are sterile but later culture of the pus usually yields *Staphylococcus aureus*.

Diagnosis

The main clinical features are fever and painful induration of one or more of the large muscles, mostly in the lower limbs

Treatment

- Drain the pus from abscess

Adults:

A: Cloxacillin give 500 mg every 6 hours for 14 days

OR

A: Erythromycin give 500 mg every 6 hours for 14 days;

Children

A: Cloxacillin 25 mg/kg body weight every 6 hours for 14 days

OR

A: Erythromycin 10 mg/kg body weight every 6 hours for 14 days

2.0 INFLAMMATORY CONDITIONS

General Guidelines

- The first line treatment for most of these conditions is a non-steroidal anti-inflammatory drug (**NSAID**). This group includes **Aspirin, Indomethacin** and **Ibuprofen**, but does NOT include **Paracetamol**
- **NSAIDs** should be used cautiously in pregnancy, the elderly, and patients with asthma and liver or renal impairment
- **NSAIDs** should be avoided in patients with current or past peptic ulceration. Refer patients with serious rheumatic disease and peptic ulceration for specialist help.
- **NSAIDs** should be taken with food
- **If dyspeptic symptoms develop in a patient on NSAIDs**, try adding magnesium trisilicate mixture. If dyspepsia persists and NSAID use considered essential antagonist
- Physiotherapy is a useful adjunct treatment in many inflammatory joint conditions

2.1 Rheumatoid Arthritis

It is a chronic multisystem disease of unknown aetiology

Diagnosis

In the majority of patients with RA, the onset is insidious with joint pain, stiffness and symmetrical swelling of a number of peripheral joints

Treatment

A: Acetylsalicylic acid 1.2 g every 6 hours with food.

OR

A: Ibuprofen give 400 – 800 mg every 8 hours. Continue for as long as it is necessary

NOTE: Patients with intractable symptoms may require special treatment at specialists centre

2.2 Gout

Gout is a recurrent acute arthritis of peripheral joints which results from deposition, in and about the joints and tendons, of crystals of monosodium urate from supersaturated hyperuricaemic body fluids. The arthritis may become chronic and forming.

Diagnosis

- The main clinical features are those of an acute gouty arthritis, often nocturnal, throbbing crushing or excruciating
- The signs resemble an acute infection with swelling, hot red and very tender joints.
- The first metatarsophalangeal joint of the big toe is frequently involved

Treatment

General principles

- Termination of acute attack
- Prevention of recurrence
- Prevention of further deposition of urate crystals.

Specific treatment for acute attack

A: Indomethacin 75 mg (O) start then 50 mg every 6 hours until 24 hours after relief of pain.

Reduce dose to 50 mg every 8 hours for 3 doses then 25 mg every 8 hours for three doses

OR

A: Diclofenac sodium 75 mg hourly. Continue as long as necessary.

OR

A: Ibuprofen: give 400 – 800 mg every 8 hours

OR

S: Colchicine give 1 mg stat followed by 0.5 mg every 2 hours orally until patient improves or a maximum of 10 mg is taken or gastrointestinal tract side effects develop. The course should not be repeated within 3 days.

Prevention of recurrence

- Institute prophylactic indomethacin
- In obese patient, reduce weight
- Avoid precipitants e.g. alcohol
- Institute anti-hyperuricaemic therapy e.g. allopurinol give 100 mg every 8 or 12 hours to reduce uric acid synthesis
- Prevention or reversal of deposition of uric acid crystals in males
- Aim is to maintain serum uric acid level below 8 mg/dl (0.48 mmol/l)

2.3 Osteoarthritis

It is a common form of arthritis, characterized by degenerative loss of articular cartilage, subchondral bony sclerosis, and cartilage and bone proliferation subsequent osteophyte formation. Cause is unknown, but genetic, metabolic and biomechanical have been suggested. Gradual onset of one or a few joints involved.

Diagnosis

- Pain is the commonest symptom

- Specific clinical features depend on the joint involved e.g. enlargement of distal interphalangeal joint (Bouchard's nodes)

Treatment guidelines

- Rest the joint . Use crutches or walkers to protect weight bearing joints in severe cases. Crepe bandage or braces also can be worn during the active phase of disease.
- Reduction of weight in obese patients
- Physiotherapy – exercise to the affected joints

Drug therapy

A:Acetylsalicylic acid 600-900 mg orally every 6-8 hours with food

OR

A: Indomethacin 25 mg every 6-8 hours with food

OR

A: Diclofenac sodium (PO)50 mg 8 hourly for 3 – 5 days

NOTE: In severe cases surgery may be indicated e.g. hip joint replacement, knee replacement

3.0 LOW BACK PAIN

Low back pain is a common presenting complaint especially among the elderly. It may be a mild, transient symptom or chronic and disabling complaint. There are many causes of low back pain but a cause can usually be found from a good clinical history and physical examination. In some patients however, no cause will be found and these people are described as having nonspecific back pain. Acute ligamentous (sprain) lesions and muscular strain are usually self-limiting.

Causes

- Acute ligamentous (sprain) lesions
- Muscular strain
- Chronic osteoarthritis

Other causes include:

- Back strain due to poor posture worsened by mechanical factors like overuse, obesity and pregnancy
- A protruding or ruptured intervertebral disk
- Traumatic ligament rupture or muscle tear
- Fracture
- Infection (e.g. tuberculosis or septic discitis)
- Malignancy e.g. metastases, multiple myeloma or spinal tumour, prostatic carcinoma
- Congenital abnormalities e.g. abnormal intervertebral facets, sacralization of L–5 transverse process
- Spondylolisthesis – i.e. Slipping forward of a vertebra upon the one below
- Narrowed spinal canal from spinal stenosis

- Psychogenic pain: The back is a common site of psychogenic pain. Inconsistent historical and physical findings on sequential examination may make one suspicious of this diagnosis
- Fibromyalgia rheumatica, connective tissue diseases (give dexamethasone 0.1mg/kg od)

Table 2: Points of Distinction between Inflammatory and Mechanical Back Pain

	Inflammatory	Mechanical
ONSET	Gradual	Sudden
WORST PAIN	In the morning	In the evening
MORNING STIFFNESS	Present	Absent
EFFECT OF EXERCISE	Relieves pain	Aggravates pain

Features that suggest that back pain may be serious

- Recent onset
- Weight loss
- Symptoms elsewhere e.g. chronic cough, weakness of the lower limbs, incontinence etc
- Localized pain in the dorsal spine
- Fever
- Raised ESR

Investigation

- X-ray is common
- CT scan and/or MRI in case of spinal stenosis
- Full Blood Picture, ESR

Treatment for Acute low back pain

Non-pharmacological Treatment

Treat by relieving muscle spasm with bed rest in a comfortable position with hip and knees flexed; local heat and massage

Pharmacological Treatment

Analgesics:

A: Ibuprofen 400 mg (O) 3 times daily for 3 to 7 days

For severe pain

A: Diclofenac 75 mg (I.M) 12 hourly by deep IM injection

OR

C: Diclofenac 50 mg rectal 8 hourly for 3 days

OR

C: Tramadol 50 mg (O) 8 hourly for 3 days.

Treatment for Chronic low back pain

Non-pharmacological Treatment

Treat the cause, e.g. weight reduction in the obese, improving muscle tone and strength through physiotherapy, improving posture. Depending on the cause, surgical procedures may be necessary, e.g. in disc disease or spinal stenosis.

Pharmacological Treatment

Analgesics are given for pain as above. **AVOID** narcotic analgesics. If symptoms persist, **refer** the patient.

Treatment of Psychogenic pain

- Reassurance is needed
- Explore causes
- Treat depression if appropriate
- Give analgesics but **AVOID** addictive medications, e.g. narcotic analgesics
- Physical therapy may be helpful

At Referral level

Several investigations including X-ray, CT SCAN, MRI, FBP, serum uric acid etc should be performed according to specialist protocol. Treatment may still be non surgical as above or otherwise. For radicular pain in chronic low back pain give:

S: Gabapentin (O) 300mg 12 hourly

C: Vit B1+B6+B12 1 tablets 12 hourly

CHAPTER NINE

KIDNEY AND UROLOGICAL DISORDERS

1.0 KIDNEY DISEASES

1.1 Chronic Kidney Diseases (CKD)

It is structural or functional kidney damage present for > 3 months, with or without a decreased glomerular filtration rate (GFR).

Markers of kidney damage include:

- Abnormalities in urine e.g. proteinuria or haematuria,
- Abnormalities in blood e.g. uraemia,
- Abnormalities in imaging tests e.g. small kidneys on ultrasound,
- Abnormalities on pathological specimens' e.g. glomerular disease on renal biopsy.

The creatinine clearance (CrCl) approximates GFR and may be estimated by the following formula:

Adults

Males:

$$\text{eGFR (mL/minute)} = \frac{(140 - \text{age}) \times \text{weight (kg)}}{\text{serum Cr (micromol/L)}}$$

Females:

Multiply estimated CrCl by 0.85

Children

$$\text{eGFR (mL/minute)} = \frac{K * \times \text{height (cm)}}{\text{Serum Cr (micromol/L)}}$$

Where *K is: infants 0–18 months = 40

Girls 2–16 years = 49

Boys 2–13 years = 49

Boys 13–16 years = 60

Common causes of chronic kidney disease include:

- Hypertension
- Diabetes mellitus
- Glomerular diseases

Note: Chronic kidney disease can be entirely asymptomatic **BUT** early detection and management can improve the outcome of this condition.

Treatment and prevention strategies according to stages

Estimation of the degree of kidney damage and staging is important to guide management and further prevent adverse outcomes of chronic kidney disease.

Note:

Adults with early CKD i.e. stages 0–3 can all be managed at primary care level **once** the cause and plan for care has been established. All children should be referred for investigation and initial management.

Table 1: Staging of kidney disease for adequate management of CKD

Stage/ glomerular filtration rate (mL/minute/1.73)	Description	Action Includes actions from preceding stages
Stage 0 or GFR > 90	Increased risks for CKD e.g. <ul style="list-style-type: none">• Diabetes mellitus• Hypertension• Glomerular disease• and HIV	<ul style="list-style-type: none">• Screening for advanced CKD and CVD disease• CKD risk reduction i. e treat hypertension, diabetes and HIV
Stage 1 or GFR > 90	Kidney damage with normal GFR	<ul style="list-style-type: none">• Diagnose and treat comorbid conditions See for Stage 0
Stage 2 or GFR 60-89	Kidney damage with mild ↓GFR	<ul style="list-style-type: none">• Refer to determine cause and develop care plan• While on the care plan, monitor the GFR in these patients and make sure kidney function is not worsening rapidly and watch for stage 3
Stage 3 or GFR 30-59	Moderate ↓ GFR	Refer
Stage 4 or GFR 15-29	Severe ↓ GFR	Refer
Stage 5 or GFR < 15	Kidney failure requiring renal replacement therapy End stage renal disease	Refer

Note: GFR should be done yearly in all patients at increased risk

General measures

- Reduce salt intake.
- Low protein diet is indicated in the presence of CKD stage 4 and 5.
- Reduce cardiovascular disease risk factors – See section: Prevention of ischaemic heart disease and atherosclerosis.

Drug treatment

- Treat underlying conditions.
- Decrease significant proteinuria, if present.

Significant proteinuria = spot urine protein creatinine ratio of > 0.1 g/mmol or ACR (albumin-creatinine ratio) > 100 g/mol, confirm as positive if raised on at least 2 of 3 occasions, in the absence of infection, cardiac failure and menstruation.

See also section on Diabetic nephropathy

Proteinuria

- In established chronic kidney disease, decrease proteinuria, irrespective of presence or absence of systemic hypertension.
- Monitor renal function and potassium especially with impaired renal function.
- If volume depleted, first rehydrate before commencing ACE-inhibitor.
- ACE-inhibitor are contraindicated in:
 - Hyperkalaemia
 - known allergy to ACE-inhibitor
- Begin with low dosage of ACE-inhibitor and titrate up ensuring blood pressure remains in normal range and no side effects are present, up to the maximum dose or until the proteinuria disappears – whichever comes first.

Adults

- ACE inhibitor, e.g.
D: Enalapril (O) 10–20 mg 12 hourly.

If ACE inhibitor cannot be used, refer.

Hyperlipidaemia

If hyperlipidaemia is a co-existent risk factor manage according to section 4.1: Prevention of ischaemic heart disease and atherosclerosis

Diabetes mellitus

- In diabetics, optimise control according to section 9.6: Diabetes mellitus type 2, in adults
- Avoid oral hypoglycaemics if GFR is < 60 because of the risk of lactic acidosis with metformin and prolonged hypoglycaemia with long acting sulphonylureas.

Hypertension

Treat if present. See Section 4.7: Hypertension

Fluid overload

Treat fluid overload if present and refer.

Adults

C: Furosemide 40–80mg slow I.V or oral, 12 hourly.

If poor response, repeat after 1 hour.

Do not give I.V fluids – use heparin lock or similar I.V access.

Children

- Furosemide, I.V, 1 mg/kg immediately.
 - Do not put up a drip or run in any I.V fluids

Table 2: Treatment of Fluid Overload Using Furosemide Injection

Weight	Dose	Injection 10 mg/mL	Age Months/years
≥ 3.5–5 kg	4 mg	0.4 mL	≥ 1–3 months
≥ 5–7 kg	6 mg	0.6 mL	≥ 3–6 months
≥ 7–9 kg	8 mg	0.8 mL	≥ 6–12 months
≥ 9– 11 kg	10 mg	1 mL	≥ 12–18 months
≥ 11–14 kg	12 mg	1.2 mL	≥ 18 months–3 years
≥ 14–17.5 kg	15 mg	1.5 mL	≥ 3–5 years
≥ 17.5–25 kg	20 mg	2 mL	≥ 5–7 years
≥ 25–35 kg	30 mg	3 mL	≥ 7–11 years
≥ 35 kg and above	40 mg	4 mL	≥ 11 years and adults

Note

Exclude heart failure in patients with persistent pedal oedema.

Referral

- All cases of suspected chronic kidney disease stages 3–5 for assessment and planning
- All children
- All cases of CKD with:
 - – haematuria,
 - – proteinuria
 - – raised blood urea or creatinine initially for assessment and planning
- Uncontrolled hypertension/fluid overload
- CKD associated with hyperlipidaemia
- No resolution of proteinuria with ACE-I therapy

Note: Patients who might qualify for dialysis and transplantation or who have complications should be referred early to ensure improved outcome and survival on dialysis, i.e. as soon as GFR drops below 30 mL/min/1.73 m², or as soon as diagnosis is made/suspected

1.2 Acute renal failure (ARF)

This is (usually) reversible kidney failure, most commonly as a result of:

- dehydration and fluid loss
- drugs/toxins,
- urinary tract obstruction, and
- acute glomerulonephritis in older children

It is often recognised by:

- fluid overload
- decreased or no urine output
- blood result abnormalities of urea, creatinine or electrolytes.
- convulsions in children

General measures

- Give oxygen, and nurse in semi-Fowlers' position if patient has respiratory distress. Early referral is essential.
- If fluid overloaded:
 - stop all fluids oral and give no IV fluids
 - stop intake of all salt and potassium containing foods and fluids
- If not overloaded, dehydrated nor shocked:
 - no IV fluids
 - restrict oral fluid intake to 10 mL/kg/day daily plus visible fluid losses
 - arrange referral in the meantime
- If dehydrated or shocked:
 - treat immediately as in shock section.

Drug treatment

Children

Under 6 years of age: > 120 mmHg systolic BP or 90 mmHg diastolic BP

6–15 years: > 130 mmHg systolic BP or 95 mmHg diastolic BP

C: Nifedipine, oral, 0.25–0.5 mg/kg squirted into mouth.

Withdraw contents of 5 mg capsule with a 1 mL syringe:

10 to 25 kg: 2.5 mg

25 to 50 kg 5 mg

Over 50 kg: 10 mg

If there is respiratory distress (rapid respiration, chest indrawing):

- Furosemide, IV, 1 mg/kg immediately.
 - o Do not put up a drip or run in any IV fluids

Table 3: Dosage Regimen for Furosemide in Treatment of ARF

Weight kg	Dose mg	Injection 10 mg/mL	Age months/years
≥ 3.5–5 kg	4 mg	0.4 mL	≥1–3 months
≥ 5–7 kg	6 mg	0.6 mL	≥ 3–6 months
≥ 7–9 kg	8 mg	0.8 mL	≥ 6–12 months
≥ 9– 11 kg	10 mg	1 mL	≥12–18 months
≥ 11–14 kg	12 mg	1.2 mL	≥18 months–3 years
≥ 14–17.5 kg	15 mg	1.5 mL	≥ 3–5 years
≥ 17.5–25 kg	20 mg	2 mL	≥ 5–7 years
≥ 25–35 kg	30 mg	3 mL	≥ 7–11 years
≥ 35 kg and above	40 mg	4 mL	≥ 11 years and adults

Adults

If diastolic blood pressure is greater than 100 mmHg or systolic blood pressure is above 150 mmHg:

S: Amlodipine (O) 5 mg as a single dose.

If there is respiratory distress (rapid respiration, orthopnoea):

C: Furosemide, as an IV bolus, 80 mg.

Do not put up a drip **and do not** give a fluid infusion.

Referral

- All cases

Where adequate laboratory and clinical resources exists, management according to the hospital level guidelines may be instituted

1.3 Glomerular Diseases (GN)

Glomerular disease may be a result of a primary condition of the kidney, or may be secondary to a systemic disorder. Can present with any, or a combination of the following:

- Proteinuria
- Reduced GFR (and its effects)
- Haematuria
- Hypertension and oedema.

Approach to care is outlined under the syndromes which follow.

Referral

- Unexplained haematuria on two to three consecutive visits
- Proteinuria > 1 g/24 hours or PCR > 0.1 g/mmol or ACR > 100 g/mol

- Nephritic syndrome
- Nephrotic syndrome
- Chronic Kidney Disease

Note:

Where facilities are available investigation should be done e.g. urine and electrolytes calculate the GFR or PCR

1.4 Glomerular disease - Nephritic syndrome

Presents with a varied combination of:

- painless macroscopic turbid, bloody or brownish urine
- peripheral and facial oedema
- pulmonary oedema (circulatory overload)
- hypertension or hypertensive encephalopathy with impaired level of consciousness or convulsions
- little or no urine excretion

In children this is most commonly due to acute post streptococcal glomerulonephritis, but not exclusively so.

General measures

- Give oxygen, and nurse in semi-Fowlers position if patient has respiratory distress.
- Early referral essential especially if patient has had a hypertensive episode or fluid overload.
- If fluid overloaded:
 - stop all fluids oral and give no I.V fluids
 - stop intake of all salt and potassium containing foods and fluids
- If not overloaded, dehydrated nor shocked:
 - no I.V fluids
 - restrict oral fluid intake to 10 mL/kg/day daily plus visible fluid losses
 - arrange referral in the meantime
- If dehydrated or shocked:
 - treat immediately as in shock section.

Drug treatment

Children

Fluid overloads (rapid respiration, chest indrawing)

- Furosemide, I.V, 1mg/kg immediately
 - Do not put up a drip or run in any IV fluids

Table 4: Dosage Regimen for Furosemide in Treatment of Glomerular disease - Nephritic syndrome

Weight kg	Dose mg	Injection 10 mg/mL	Age months/years
≥ 3.5–5 kg	4 mg	0.4 mL	≥1–3 months
≥ 5–7 kg	6 mg	0.6 mL	≥ 3–6 months
≥ 7–9 kg	8 mg	0.8 mL	≥ 6–12 months
≥ 9– 11 kg	10 mg	1 mL	≥12–18 months
≥ 11–14 kg	12 mg	1.2 mL	≥18 months–3 years
≥ 14–17.5 kg	15 mg	1.5 mL	≥ 3–5 years
≥ 17.5–25 kg	20 mg	2 mL	≥ 5–7 years
≥ 25–35 kg	30 mg	3 mL	≥ 7–11 years
≥ 35 kg and above	40 mg	4 mL	≥ 11 years and adults

If hypertension

Under 6 year of age: > 120 mmHg systolic BP or 90 mmHg diastolic BP

6-15 ears: > 130 mmHg systolic BP 95 mmHg diastolic BP

C: Nifedipine, oral, 0.25-0.5 mg/kg squirted into mouth

Withdraw contents of 5 mg capsule with a 1 mL syringe:

10 to 25 kg: 2.5 mg

25 to 50 kg: 5 mg

Over 50 kg: 10 mg

Adults

Fluid overload

C:Furosemide, as an I.V bolus, 80 mg.

- Do not put up a drip **and do not** give a fluid infusion

If hypertension

If diastolic blood pressure is greater than 100 mmHg or systolic blood pressure is above 150 mmHg:

S: Amlodipine, oral, 5 mg as a single dose

Referral

» All cases

The definitive treatment of nephritis depends on the cause – an assumption of acute post streptococcal nephritis or any other disease cannot be made without specific investigation which may include renal biopsy.

1.5 Glomerular disease - Nephrotic syndrome

Glomerular disease characterised by:

- severe proteinuria defined as:
 - Children: $\geq 3+$ proteinuria on dipstick test, or urine protein: creatinine ratio (PCR) ≥ 0.2 g/mmol on spot urine sample
 - Adults: 2.5 g/day, or greater as determined by a spot urine protein measurement, i.e. protein creatinine ratio (PCR)
- and resultant 'classical' clinical picture (not always present) which includes:
 - Oedema and
 - Hypoalbuminaemia and
 - Hyperlipidaemia.

Note: Accurate diagnosis requires a renal biopsy.

Drug treatment

The management of glomerular disease depends on the type/cause of the disease and is individualised guided by a specialist according to the biopsy result.

Referral

- All cases

1.6 Urinary tract infection (UTI)

Urinary tract infections may involve the upper or lower urinary tract. Infections may be complicated or uncomplicated. Uncomplicated cystitis is a lower UTI in a non-pregnant woman of reproductive age and who has a normal urinary tract. All other UTIs should be regarded as complicated.

Note: Differentiation of upper from lower urinary tract infection in young children is not possible on clinical grounds.

Upper UTI is a more serious condition and requires longer and sometimes intravenous treatment. Features of upper UTI (pyelonephritis) that may be detected in adults and adolescents include:

- flank pain/tenderness
- temperature 38°C or higher
- other features of sepsis, i.e.:
 - tachypnoea,
 - tachycardia
 - confusion, and
 - hypotension
- vomiting

In complicated, recurrent or upper UTIs, urine should be sent for microscopy, culture and sensitivity.

Features of urinary tract Infections in children

- Signs and symptoms are related to the age of the child and are often nonspecific.
- Uncomplicated urinary tract infections may cause very few signs and symptoms.
- Complicated infections may present with a wide range of signs and symptoms.

Neonates may present with:

• Fever	• Hypothermia
• Poor feeding	• Sepsis
• Vomiting	• Prolonged jaundice
• Failure to thrive	• Renal failure
Infants/children may present with:	
• failure to thrive fever	• frequency
• persisting fever	• dysuria
• abdominal pain	• enuresis or urgency
• diarrhoea	

Note: In any child with fever of unknown origin, the urine must be examined.

In children the diagnosis must be confirmed.

If a bag specimen reveals the following, a urine specimen must be collected aseptically for culture and sensitivity:

- Positive leukocytes or nitrites on dipsticks in freshly passed urine
- Motile bacilli and increased leukocytes or leukocyte casts on urine microscopy

Urine dipstix should be performed on a fresh urine specimen.

- If leucocytes and nitrites are not present, a urinary tract infection is highly unlikely.
- If leucocytes are present on a second specimen, a urinary tract infection must be suspected.

General measures

- Women with recurrent UTIs, should be advised to:
 - Void bladder after intercourse and before retiring at night
 - Not postpone voiding when urge to micturate occurs
 - Change from use of diaphragm to an alternative type of contraception

Drug treatment

Empirical treatment is indicated only if:

- Positive leucocytes and nitrites on freshly passed urine, or
- Leucocytes or nitrites with symptoms of UTI, or
- Systemic signs and symptoms.

Alkalinising agents are not advised.

Uncomplicated cystitis

Adults:

A: Ciprofloxacin (O) 500 mg as single dose

Complicated cystitis

Adults:

A: Ciprofloxacin (O) 500 mg 12 hourly for 7 days

For pregnant women and adolescents:

A: Amoxicillin/clavulanic acid 500/125 mg(O)12hourly for 7 days

Children who do not meet criteria for urgent referral:

- Amoxicillin/clavulanic acid, oral, 12.5–20 mg/kg of amoxicillin component, 8 hourly for 5 days

Table 5: Dosage Regimen for Treatment of Cystitis in Children Using Amoxicillin/Clavulanic Acid

Weight kg	Dose mg	Use one of the following			Age months/years
		Syrup 125/31.25 mg per 5mL	Syrup 250 62.5 mg per 5 mL	Tablet 500/125 mg	
≥ 3.5–5 kg	75/18.75 mg	3 mL	1.5 mL	-	≥1–3 months
≥ 5–7 kg	100/25 mg	4 mL	2 mL	-	≥ 3–6 months
≥ 7–9 kg	125/31.25 mg	5 mL	2.5 mL	-	≥ 6–12 months
≥ 9– 11 kg	150/37.5 mg	6 mL	3 mL	-	≥12–18 months
≥ 11–14 kg	187.5/46.9 mg	7.5 mL	4 mL	-	≥ 8 months–3 years
≥ 14–17.5 kg	250/62.5 mg	10 mL	mL	-	≥ 3–7 years
≥ kg and above	250/125 mg	-	-	1 tablet	≥ 7 years and adults

Acute pyelonephritis

Outpatient therapy is only indicated for women of reproductive age, who do not have any of the danger signs – see referral criteria. All other patients should be referred.

A: Ciprofloxacin (O) 500 mg 12 hourly for 7–10 days

It is essential to give at least a 7-day course of therapy.

Referral

Urgent

- Acute pyelonephritis with:
 - vomiting
 - sepsis
 - diabetes mellitus
- Acute pyelonephritis in:
 - pregnant women
 - women beyond reproductive age
 - men
- Children over 3 months who appear ill.
- Children less than 3 months of age with any UTI.

Ill patients awaiting transfer

- Ensure adequate hydration with intravenous fluids
 - **Ceftriaxone, IM, 50–80 mg/kg/dose immediately as a single dose**

Table 6: Dosage Regimen for Ceftriaxone

Weight kg	Dose mg	Use one of the following injections mixed with water for injection (WFI)			Age months/ years
		Syrup 250 mg WFI 2mL	500 mg WFI 2 mL	1 000 mg WFI 3.5 mL	
≥ 2–2.5 kg	125 mg	1 mL	0.5 mL	-	
≥ 2.5–3.5 kg	200 mg	1.6 mL	0.8 mL	-	Birth -1 months
≥ 3.5–5.5 kg	250 mg	2 mL	1 mL	-	≥ 1–3 months
≥ 5– 7 kg	375 mg	3 mL	1.5 mL	-	≥ 3–6 months
≥ 7–9 kg	500 mg	4 mL	2 mL	-	≥6–12 months
≥ 9–11 kg	625 mg	5 mL	2.5 mL	-	≥ 12- 18 months
≥ 11–14 kg	750 mg	6 mL	3 mL		≥ 18 months – 3 years
≥ 14–17.5kg	1000 mg	-	4 mL	3.5 mL	≥ 3 – 5 years
≥ 17.5 kg and above	1000 mg	-	4 mL	3.5 mL	≥ 5 years and adults

! CAUTION!

Do not administer calcium containing fluids, e.g. Ringer-lactate, within 48 hours of administering ceftriaxone. Contra-indicated in neonatal jaundice. Annotate dose and route of administration in referral letter.

Non-urgent

- All children for urinary tract investigations after completion of treatment
- No response to treatment.
- UTI more than 3 times within a one-year period in women, and more than 1 time for men
- Recurrent UTI in children for assessment and consideration of prophylaxis

2.0 Urology Disorders**2.1 Haematuria**

It is a bleeding from the urinary tract, which can be from the kidneys, collecting system bladder, prostate and urethra. Glomerular disease is suggested if proteinuria is present as well as casts on routine microscopy. Schistosomiasis (bilharzia) is a common cause of haematuria.

Exclude schistosomiasis.

When haematuria is accompanied by colicky pain a kidney stone should be excluded.

Note:

The presence of blood on urine test strips does not indicate infection and should be investigated as above.

Drug Treatment

If evidence of Schistosomiasis – treat as in Section 10.13: Schistosomiasis

If symptoms of UTI and leucocytes and nitrite positive in urine – treat as UTI

If Haematuria does not resolve rapidly after treatment referral for formal investigation will be required, i.e. next 48 hours.

Referral

- All cases not associated with schistosomiasis or UTI
- All cases not responding to specific drug treatment

2.2 Prostatitis

It is an infection of the prostate caused by urinary or STI pathogens. Clinical features include:

- perineal, sacral or suprapubic pain
- dysuria and frequency
- varying degrees of obstructive symptoms which may lead to urinary retention
- sometimes fever
- acutely tender prostate on rectal examination

The condition may be chronic, bacterial or non-bacterial, the latter usually being assessed when there is failure to respond to antibiotics.

Drug treatment**Acute bacterial prostatitis**

In men < 35 years or if there are features of associated urethritis (STI regimen):

D: Cefixime (O) 400mg as a single dose

Followed by:

A: Doxycycline, oral, 100 mg 12 hourly for 7 days

In men > 35 years or if there is associated cystitis:

B: Ciprofloxacin, oral, 500 mg 12 hourly for 14 days

Referral

- No response to treatment
- Urinary retention
- High fever
- Chronic/relapsing prostatitis

2.3 Benign prostatic hyperplasia

Benign prostatic hyperplasia is a noncancerous (benign) growth of the prostate gland.

May be associated with both obstructive (weak, intermittent stream and urinary hesitancy) and irritative (frequency, nocturia and urgency) voiding symptoms. Digital rectal examination reveals a uniform enlargement of the prostate. Urinary retention with a distended bladder may be present in the absence of severe symptoms, therefore it is important to palpate for an enlarged bladder during examination.

General measures

Annual follow-up with digital rectal examination (DRE) is recommended. For patients presenting with urinary retention, insert a urethral catheter as a temporary measure while patient is transferred to hospital. Remove drugs that prevent urinary outflow e.g. tricyclics and neuroleptics.

Referral

- All patients with suspected BPH

2.4 Prostate cancer

Usually occurs in men over 50 years and is most often asymptomatic. Systemic symptoms, i.e. weight loss, bone pain, etc. occurs in 20% of patients. Obstructive voiding symptoms and urinary retention are uncommon.

The prostate gland is hard and may be nodular on digital rectal examination. As the axial skeleton is the most common site of metastases, patients may present with back pain or pathological fractures. Lymph node metastases can lead to lower limb lymphoedema.

Serum prostate specific antigen (PSA) is generally elevated and may be markedly so in metastatic disease.

Referral

- All patients with suspected cancer (For more detail refer to the Malignant diseases section)

2.5 Enuresis

Enuresis is bedwetting after the age of 5 years. It is a benign condition which mostly resolves spontaneously. It is important, however, to differentiate between nocturnal enuresis and enuresis during daytime with associated bladder dysfunction. Secondary causes of enuresis include:

- diabetes mellitus
- urinary tract infection
- physical or emotional trauma

Note:

Clinical evaluation should attempt to exclude the above conditions. Urine examination should be done on all patients.

General measures

- Motivate, counsel and reassure child and parents
- Advise against punishment and scolding

- Spread fluid intake throughout the day
- Nappies should never be used as this will lower the child's self esteem.

Referral

- Suspected underlying systemic illness or chronic kidney disease
- Persistent enuresis in a child 8 years or older
- Diurnal enuresis

2.6 Erectile dysfunction disorders

It is inability to attain and maintain an erect penis with sufficient rigidity for vaginal penetration. Organic causes include neurogenic, vasculogenic, endocrinological as well as many systemic diseases and medications.

General measures

- Thorough medical and psychosexual history
- Physical examination should rule out gynaecomastia, testicular atrophy or penile abnormalities.
- Consider the removal of drugs that may be associated with the problem.
- A change in lifestyle or medications may resolve the problem, e.g. advise cessation of smoking and alcohol abuse.

Drug treatment

- Treat the underlying condition.
- If persist refer the patient

2.7 Renal calculi

This is a kidney stone or calculus which has formed in the renal tract i.e. pelvis, ureters or bladder as a result of urine which is supersaturated with respect to a stone-forming salt. Clinical features of obstructing urinary stones may include:

- Sudden onset of acute colic, localized to the flank, causing the patient to move constantly.
- nausea and vomiting
- referred pain to the scrotum or labium on the same side as the stone moves down the ureter

Urinalysis usually reveals microscopic or macroscopic haematuria.

General measures

- Ensure adequate hydration.

Drug treatment

Adults:

Analgesia for pain, if needed:

C: Morphine, 10–15 mg, IM/slow IV as a single dose and refer.

Referral

- All patients

CHAPTER TEN

EAR, NOSE AND THROAT DISEASES

1.0 OTITIS (EXTERNA AND MEDIA)

It is an inflammatory condition of the pinna, external auditory meatus and/or the middle ear cavity.

1.1 Otitis externa

Diagnosis

- Itchy, dry and scaly ear canal and painful ear
- There may be a water or purulent discharge and intermittent deafness
- Pain may become extreme when the ear canal becomes completely occluded with edematous skin and debris.

Treatment

- Exclude an underlying chronic otitis media before commencing treatment
- Do a thorough aural toilet at least once a week (ear suctioning under direct vision)
- Instruct the patient to thoroughly clean the ear with a cotton wick regularly and keep it dry
- Give adult and children:
 - A:** Aluminium diacetate drops 3%, instill 3-4 drops every 6 hours after cleaning and drying the ear for 5 days (to reduce edema of the external auditory canal)
 - OR**
 - C:** Gentamycin ear drops 3-4 drops 8 hourly for 7 days or more
 - OR**
 - C:** Ciprofloxacin ear drops 3-4 drops 8 hourly for 7 days or more
 - OR**
 - C:** Boric acid ear drops 3-4 drops 6 hourly for 7 days or more

1.2 Otitis media (acute or chronic)

Diagnosis

- Ear pain, a sensation of fullness in the ear, hearing loss
- If the tympanic membrane has perforated, there is an aural discharge
- Onset usually follows an upper respiratory tract infection.
- Chronic otitis media is nearly always associated with perforation of the eardrum.

Investigation: Examine the pinna; using an otoscope carefully examine the external auditory canal and the tympanic membrane

I. Acute otitis media

It is acute purulent exudates in the middle ear without ear discharge not more than 12 weeks duration

Diagnosis

- Previous common cold
- Painful ears
- Restlessness
- Usually feverish
- Hearing often reduced
- Inflamed bulged tympanic membrane

II. Acute suppurative otitis media

It is acute purulent exudates in the middle ear cavity with an ear discharge (perforated tympanic membrane) of not more than 12 weeks duration

Diagnosis

- Discharge of pus from ear
- Perforated tympanic membrane

Treatment of *Acute otitis media & acute suppurative otitis media*

Acute otitis media should be treated with analgesics, antibiotics and/or paracentesis. Culture of a discharge (if any) could be of a great help to identify the causative bacteria.

Drugs

Adults

A: Phenoxymethylpenicillin 250 – 500 mg every 6 hours for 7 days

Children up to 5 years: 6 mg/kg every 6 hours for 7 days; 6-12 years:
250 mg every 6 hours for 7 days or more

NOTE: Treatment periods shorter than seven days increase the risk of treatment failure

A: Amoxicillin: 500mg 8 hourly for 7 days
Children 40mg/kg daily in 3 divided doses (max. 3g daily)

Erythromycin: Adult and children above 8 years 250 – 500 mg every 6-8 hours for 7 days or more

Symptomatic treatment of acute otitis media

- **Analgesics**
A: Paracetamol 10 mg/kg body weights every 6-8 hours
OR
A: Acetylsalicylic acid
Avoid **Acetylsalicylic acid** if it is a viral infection
- Bed rest

- Decongestive nasal drops or nasal spray e.g.

C:Ephedrine hydrochloride 1-2 drops into each nostril up to 3-4 times daily for not more than 5 days

Referral to ENT specialists

- Children with high fever who are toxic or children with severe ear pain, headache, altered state of consciousness
- A chronically discharging ear that persists in spite of proper treatment.
- Foul smelling ear discharge
- Mastoiditis
- "Ear Children"
- Otitis in the normal (or better hearing) ear combined with permanent hearing loss in the other ear.

III. Mastoiditis with subperiosteal abscess

It is due to infection of the mastoid air cells in the middle ear, a complication of otitis media. It presents as a fluctuant painful swelling on the post auricular area. The overlying skin is inflamed.

Treatment

Aspirate the swelling before incision and drainage, and then refer

IV. Secretory otitis media

It is a multifactorial non-purulent inflammatory condition in the middle ear with serous or mucous discharge. Also there is a residual condition after acute otitis.

Diagnosis

- Little or no pain
- Gradual loss of hearing
- No ear discharge
- often discovered by chance

Treatment

- Close follow-up
- Nasal drops, oral decongestants and antihistamines have no demonstrable effect on this condition
- Secretory otitis with hearing loss that does not improve should be referred to a specialist

2.0 ACUTE RHINITIS AND SINUSITIS

It is inflammation of the mucosal lining of the nose and paranasal sinuses, almost always occurring concurrently, thus also referred as rhinosinusitis, of not more than 12 weeks duration. Rhinitis is caused by a variety of viruses. Acute sinusitis starts with obstruction of the sinus ostium due to mucosal edema from a viral infection, followed by reduced sinus ventilation, retention of mucous in the sinus and bacterial multiplication. If the ostium is blocked for a long

period, sinus empyema occurs. The bacteria most often causing purulent sinusitis are pneumococci and Haemophilus influenzae which in some studies are shown to be equally common. Moraxella catarrhalis and group A streptococci also occur. In sinusitis of dental origin, anaerobic bacteria are often found.

2.1 Acute rhinitis

It is a viral inflammatory condition in the nasal mucous membrane, usually part of a more widespread infection of the upper respiratory tract.

Treatment

- Bed rest
 - C:** Ephedrine hydrochloride (1% for adults and 0.5% for children) 1-2 drops into each nostril up to 3-4 times daily for not more than 5 days
 - OR
 - C:** Beclomethasone nasal spray adult and child over 6 years, 100 micrograms (2 sprays) into each nostril twice daily; max. Total 400 micrograms (8 sprays) daily; when symptoms controlled, dose reduced to 50 micrograms (1 spray) into each nostril twice daily

Oral drugs to reduce swelling of the mucous membrane, antihistamines and antibiotics are not indicated.

2.2 Acute purulent sinusitis

Bacterial infection with pus accumulation in one or more of the sinuses

Diagnosis

Anterior rhinoscopy – watery/purulent nasal discharge occasionally foul smelling; nasal congestion

Plain paranasal sinuses X ray (Water's, Caldwell views)

– mucosal thickening; air fluid levels

Treatment

- **Symptomatic Treatment**
 - Bed rest
 - Nasal drops or spray
 - Oral drugs to reduce swelling of the mucous membrane or anti-histamines are not indicated.
- **Treatment with antibiotics**
 - A:** Phenoxymethylpenicillin: 250 – 500 mg every 6 hours for 14
 - Children up to 5 years** 6 mg/kg every 6 hours for 14; **5 – 12 years** 250 mg every 6 hours for 14 or more days
 - OR
 - A:** Amoxicillin 500 mg every 8 hours for 14 or more days
 - Children Up to 10 years 10 mg/kg every 8 hours for 14 or more days

Alternative:

C: Amoxicillin/Clavulanic acid, children 375mg (250mg amoxicillin, 125 clavulanic acid) 8 hourly for 10 days; Adults 625mg (500mg amoxicillin, 125mg clavulanic acid) 8 hourly for 10 days

OR

A: Doxycycline 200 mg on the first day as a single dose then 100 mg from the following day every 24 hours for 14

NOTE: Doxycycline for adult only and children above 12 years

Children

A: Co-trimoxazole: 6 weeks – 5 years; 0.5 ml/kg every 12 hours for 14 or more days; 6-12 years: 480 mg every 12 hours for 14 or more days

Note: Cephalosporins and macrolides (e.g. Erythromycin etc) are not suitable because of poor effect on Haemophilus influenzae. Treatment duration of less than 2 weeks will result in treatment failure

Referral to specialist

- Children with ethmoiditis presenting as an acute periorbital inflammation or orbital cellulitis must be hospitalized immediately
- Adults with treatment failure and pronounced symptoms
- If sinusitis of dental origin is suspected
- Recurrent sinusitis (>3 attacks in a year) or chronic sinusitis (duration of illness of >12 weeks)

2.3 Allergic rhinitis

It is irritation of the nasal mucosa by an allergen in a previously sensitized individual. Common allergens include house dust (mite's faeces), pollens, cockroach antigen, animal dander, moulds (in-door)

Diagnosis

- Itchy nostrils, throat, eyes
- Watery nasal discharge
- Nasal congestion
- Sneezing

Investigation

- Anterior rhinoscopy – watery nasal discharge, nasal congestion
- Skin allergy test

Treatment

- Avoidance of an allergen (if possible)

- Antihistamines (Cetirizen 10mg daily for adults, 5mg daily for children aged 2-6 years)
- Steroid nasal spray (Beclomethasone one puff each nostril 3 times a day)

3.0 PHARYNGOTONSILLITIS

It is an acute inflammation of the pharynx and/tonsils, characterized by fever and a painful throat. Pharyngotonsillitis is caused by virus or bacteria. Clinical important pathogens are group A beta-haemolytic streptococci (GAS) and Epstein – Barr virus (EBV). In practice GAS is an indication for treatment with antibiotics.

Treatment

- As general rule pharyngotonsillitis caused by GAS should be treated with antibiotics
- If treatment is begun early, duration of the illness can be shortened.
- Antibiotics can hinder the spread of infection and reduce the risk of complications.

A: Phenoxyethylpenicillin: 500 mg every 8 hours for 10 days

OR

A: Amoxicillin 250 – 500mg every 8 hours for 10 days

OR

A: Erythromycin; 250 – 500 mg every 8 hours for 10 days;

Children up to 8 years 10 mg/kg every 8 hours for 10 days

OR

C: Amoxicillin+ Clavulanic acid 625mg 8 hourly for 10 days

Plus

A:Paracetamol 10 mg/kg body weight every 8 hours until fevercontrolled

Children (See under treatment of purulent sinusitis)

Plus

A:Paracetamol 10 mg/kg body weight every 8 hours until fever controlled

NOTE: Duration of treatment is ten days. Shorter treatment involves increased risk of therapy failure

Refer the patient to the specialist with tonsillitis if

- Chronic tonsillitis
- Recurrent tonsillitis (>3 attacks in a year or 5 or more attacks in 2 years)
- Obstructive tonsillitis (causing an upper airway obstruction)

4.0 LARYNGITIS

This is an infectious/non infectious, acute/chronic inflammatory condition of the larynx. Etiological agents include viruses (for acute laryngitis), bacteria, fungi, laryngeal reflux disease, thermal injuries, cigarette smoking, trauma (vocal cord abuse), and granulomatous conditions (for chronic laryngitis).

The picture of the disease is different in children and adults due to the small size of the larynx in children. Acute subglottic laryngitis (pseudocroup) occurs mainly in children under the age of seven, it is a viral infection. Edema of the mucous membrane of the subglottic space causes breathing difficulties, especially on inspiration. Laryngitis in children may require active treatment.

Treatment

● Symptomatic treatment (acute laryngitis)

- Parents should behave calmly and avoid frightening the child
- Bed rest
- Keep the air damp and cold
- Give extra fluid
- Nasal drops or spray may be helpful
- If symptoms persist or worsen, seek medical advice

● Drug treatment in general practice

Epinephrine (Adrenaline) inhalation effectively reduces symptoms, but the effect may be short – lived

Table 1: Dosage of racemic epinephrine preparation

Age	Racemic Epinephrine (20 mg/ml)	0.9% Saline
0-6 months	0.1 ml	2 ml
6-12 months	0.15 ml	2 ml
>12	0.2 ml	2 ml

NOTE: The total fluid volume, is inhaled in 5 minutes with the use of inhalator

● Hospitalization

If severe symptoms persist or worsen or recur after Epinephrine inhalation hospitalization is indicated

● Symptomatic treatment (chronic laryngitis)

- Voice rest
- Stop smoking
- Nasal drops or spray
- Rehydration
- Antireflux/antibiotics/antifungals

Referral to specialist

- Chronic laryngitis

5.0 ACUTE EPIGLOTTITIS (AE)

It is an acute infectious inflammation of the epiglottis, supraglottic and hypopharynx. Epiglottitis is a potentially lethal disease. Edema of the epiglottis may cause acute airway obstruction. Epiglottitis occurs both in children and adults. Haemophilus influenzae is often the cause.

Diagnosis

AE is characterized by throat pain, difficulty swallowing, drooling, husky voice, fever often high and with chills; patients prefer sitting posture with an extended neck, laborious inspiration, cough in some cases and anxiety.

Investigation: Plain X ray of the neck, lateral view characteristically presents with a positive thumb sign (edematous epiglottis)

Treatment

- Immediate hospitalization, preferably in the ICU
- Transportation: sitting, with oxygen supplementation
- Be prepared to treat respiratory failure (intubation or tracheotomy)
- Antibiotics may be given if transport lasts more than one hour.

6.0 RECURRENT RESPIRATORY PAPILLOMATOSIS (LARYNGEAL PAPILLOMAS)

It is the commonest benign laryngeal tumor of the larynx caused by Human papilloma virus (HPV), occurring in both children and adults. It has a higher recurrence rate in children than in adults, among adults it may turn into a malignancy

Diagnosis

- Progressive hoarseness of voice
- Progressive difficulty in breathing
- Progressive inspiratory stridor
- On and off cough

Investigation

- Physical examination
- thorough respiratory system examination
- hoarse voice, audible respiration (inspiratory stridor)
- indirect laryngoscopy – papilloma croups on the larynx
- Chest X ray - ?foreign body inhalation, ?pneumonia (coincidental finding)

Treatment

- Refer the patient for microlaryngeal surgery
- If in distress, perform a tracheostomy first then refer

7.0 EPISTAXIS

It is nose bleeding. May be due to a local cause (in the nasal cavity – trauma, tumor, foreign body, septal varices, septal deviation) or due to a systemic cause (blood disorders, vascular disorders, renal failure, hepatic failure, use of anticoagulants (wafarin, heparin)

Management

Stabilize the patient: put an open intravenous line, blood grouping and cross matching. Put the patient in a sitting position, put on a gown, glasses, and head light, sterile gloves. Advise the patient to pinch the soft part of the nose gently for 3 minutes. Evacuate clots and do a thorough head and neck examination. Remove a foreign body; cauterize septal varices using a silverex stick

If the patient is still bleeding do an anterior nasal packing by introducing as far posterior as possible sterile vaseline gauzes (or iodine soaked gauzes if not available) using a dissecting forcep (if bayonet forcep is not available). Put rolled dry gauze on the nose and plaster it.

If the patient is still bleeding do a posterior nasal packing using a Folley's catheter introduced through the nasal cavity into the oropharynx, balloon it with normal saline up to 10-15cc while pulling it outward to impinge on the posterior nasal coana, then do anterior nasal packing as above. Put dry gauze on the nose to prevent necrosis and fix the catheter on the nose with an umbilical clamp. Almost all of the nasal bleedings will be controlled by this way. Put the patient on oral antibiotics (Amoxycillin 500mg 8 hourly for 5 days), analgesics (Paracetamol 1g 8 hourly for 5 days) and tranexamic acid 500mg 8 hourly for 3 days. Remove the packs after 72 hours. Put an ice cube on the forehead, extending the neck or placing a cotton bud soaked with adrenaline in the vestibule will not help

Referral

- If the patient is still bleeding repack and refer immediately
- Failure to manage the underlying cause, refer the patient

8.0 FOREIGN BODIES

In the ear, nasal cavity – remove using a cerumen hook or a hooked office pin under direct vision using a head light. Refer if the foreign body is in the bronchus, trachea or hypopharynx.

CHAPTER ELEVEN

EYE DISEASE CONDITIONS

1.0 MAJOR BLINDING DISEASES

Blindness is defined as a Visual Acuity of less than 3/60 with the best correction available or central visual field of less than 10° in the better eye by WHO definition. In a simpler way, it is when some one fails to count fingers at a distance of 3 meters in the eye that is considered good with the best available corrective/distance spectacles. The definition is the same to children and infants though there are different methods for testing vision in young children until when they are at pre school age when normal visual acuity chart can be used.

The common causes of blindness are Cataract, Glaucoma, Trachoma, and Vitamin A Deficiency, Diseases of the Retina, uncorrected Refractive Errors and Low Vision.

1.1 Cataract

Diagnosis

- Cloudiness in the lens seen as a white mark behind the pupil and iris
- Conjunctiva and cornea are clear and the whole iris can be seen clearly

Referral

Refer all cases to eye surgeon for cataract surgery. Children should be referred immediately to a Paediatric Eye Tertiary Centre as white pupil may be a tumor in the eye. Late treatment of cataract in children may lead to permanent loss of vision, low vision or squint.

NOTE:

- Cataract may present in all age groups
- Blindness due to cataract is reversible.
- Treatment is only by surgery which takes approximately 20 minutes.
- Early treatment in children is mandatory

1.2 Glaucoma

There are mainly 4 clinical types of glaucoma.

I. Primary Open angle glaucoma

Diagnosis

- Present as painless loss of peripheral vision
- Affects adults of 40 years of age and above
- Cornea and conjunctiva are clear
- Pupil in the affected eye does not react with direct light.
- The optic nerve is always damaged through fundoscopy
- One eye may be affected than the other
- First degree relatives of glaucoma patients are at risk

- All suspected cases of glaucoma should be referred to qualified eye care personnel.

NOTE:

Primary Open Angle Glaucoma does not have symptoms in early stages, hence routine intraocular pressure check up and fundus examinations should be done in all people of 40 years and above

Treatment

Treatment of Primary Open Angle Glaucoma may be surgical or medical. Medical treatment is given to patients with good compliance (targeted intraocular pressure level reached). If medical treatment is given, it should be life long unless there are conditions necessitating other interventions. Surgical treatment is usually preceded by medical treatment.

Medical Treatment

- Topical Beta-blockers
 - D:** Timolol 0.25% or 0.5% Instil one drop in the affected eye 12 hourly.
 - This is a first line treatment and it should be used with caution in patients with Asthma and cardiac diseases.

OR

- Topical Parasympathomimetics
 - C:** Pilocarpine hydrochloride 2 or 4% Instil one drop in the affected eye given at an interval of 6 hourly.

This medicine causes long-standing pupil constriction so it should not be used unless a patient is prepared for glaucoma surgery or as an alternative topical treatment for patients who are contraindicated for Timolol use.

- Topical Prostaglandin analogue:
 - D:** Latanoprost 0.005% Instil one drop once a day

OR

- Systemic Carbonic anhydrase inhibitors:
 - C:** Acetazolamide 250 mg 6 hourly for one or two days or until the intraocular pressure is lower than 40 mmHg.

Surgical Treatment

- It is done in all patients with poor compliance or when prescribed topical medicines are unavailable or unaffordable.
- Surgical treatment is encouraged as a primary treatment in all glaucoma cases in developing countries due to poor compliance to medical treatment.

II. Primary Angle Closure Glaucoma

This is also known as Congestive Glaucoma and commonly affect people aged 40 years and above.

Diagnosis

- Patients present with acute painful red eye in the affected eye
- Severe headache and cloudiness of the cornea
- There is usually dramatic visual impairment and vomiting may be present

NOTE:

- Primary Angle Closure Glaucoma is an Ophthalmological Emergency
- Refer all patients with Congestive Glaucoma to eye specialist after initial treatment

Treatment Guideline*First Line Treatment*

C: Mannitol IV 1-2mg/kg body weight to run slowly over 30 -45 minutes

OR

D: Glycerol syrup (O)1-2 g/kg body weight stat.

These medicines have diuretic effects so they are only used as a single dose. They are also used in emergencies to prepare patients with high intraocular pressure for surgery as they lower intraocular pressure rapidly.

Second Line Treatment

As for Primary Open Glaucoma.

III. Congenital Glaucoma

- Presents from birth to 5 years.
- It is a syndrome where by the intraocular pressure is raised and cause abnormality of the eyeball and visual disturbances even blindness.

Diagnosis

- Patients presents with bigger eyes than normal for age (buphthalmos)
- Photophobia
- Tearing
- Cloudy cornea,
- Red conjunctiva though not severe.

Treatment

Treatment is usually surgery, which is done by pediatric ophthalmologist.

Referral

Refer any child who have the above mentioned signs and you suspect that he/she is having congenital glaucoma to a specialist at a Paediatric Eye Tertiary centre.

IV. Secondary Glaucoma

This presents as a complication of other eye diseases such as uveitis, hypermature cataract, trauma and retinal diseases. It may also be due to prolonged use of steroids

Diagnosis

- Poor vision in the affected eye
- High intraocular pressure
- New vessels on the iris if the cause is retinal diseases

Treatment Guideline

Management of these patients is retrobulbar alcohol injection 99% in the affected eye or laser photocoagulation treatment (Cyclophotocoagulation) in thrombotic glaucoma. Treatment of the pre existing eye disease is highly recommended.

Referral

Refer all patients suspected to have secondary glaucoma to a qualified specialist

1.3 Trachoma

It is a chronic conjunctivitis caused by infection with *Chlamydia trachomatis*. It is one of the commonest causes of blindness worldwide. There is a chronic inflammation of the conjunctiva leading to scarring of the upper eyelid tarsal plate, entropion and in turn of eyelashes.

Note: Trachoma reservoirs are infected children and mothers in hyper endemic areas. The infection is spread by direct contact through Flies, Fomites (kanga, towels) and Fingers, in poorly hand hygienic conditions.

Diagnosis

- Patients presents with photophobia in early stages or re- infection
- Follicles in the upper tarsal plate seen as round and white nodules in active diagnostic.
- In late stages, In-turned eyelashes rub on the cornea leading to corneal ulcers
- Loss of vision due to Corneal Scarring.

Clinical Stages according to World Health Organization

Trachomatous Inflammation Follicular (TF) - Presence of at least 5 follicles on the upper tarsal plate.

Trachomatous Inflammation Intense (TI) – There is intense inflammation, the conjunctival blood vessels cannot be seen.

Trachomatous Scarring (TS) – Presence of white scars in the upper tarsal plate

Trachomatous Trichiasis (TT) – Presence of some eye lashes rubbing against the cornea

Corneal Opacity (CO) – Presence of corneal opacity (scar) affecting the central cornea

Treatment and Prevention

World Health Organization recommended treatment and prevention strategy for Trachoma known as **SAFE**. The components of SAFE strategy are:

- Surgical correction of entropion in TT patients. This procedure can be done at a Dispensary or Health Centre at community level by a trained health worker.
- Antibiotic treatment of individual cases with TF and TI to prevent transmission as follows:-
 - A: Oxytetracycline ointment 3% once a day for 6 weeks
 - OR**
 - C: Azithromycin 1g as a single dose for adults- for preventive chemotherapy in mass treatment campaign.The regimen for children is as shown below:-

Weight (kg)	I-day Regimen
< 15	20mg/kg once daily
15 – 25	400mg (10 ml) once daily
26 - 35	600 mg (15 ml) once daily
36-45	800 mg (20 ml) once daily
> 45	Dose as per adults

F – Face washing and total body hygiene to prevent transmission of disease from one person to the other.

E – Environmental improvement/hygiene

1.4 Vitamin A Deficiency

Vitamin a deficiency is associated with higher infants and childhood mortality rate particularly associated with Measles. The age group at risk of blindness due to Vitamin A deficiency is 6 months to 6 years.

Ocular Manifestations

Xerophthalmia is a term used to describe the ocular symptoms and signs of Vitamin A Deficiency which are:-

- *Night Blindness* - Patients presents/complain of poor vision during the night or in dim light
- *Conjunctival Xerosis* - It is a dry appearance of the conjunctiva
- *Bitot Spots* - This is an advanced stage of Conjunctival xerosis presenting as a localized white foamy appearance most often on the temporal conjunctiva
- *Corneal xerosis* - It is a dry appearance of the cornea
- *Corneal ulceration with Xerosis* – It is an advanced stage of corneal xerosis where you have ulceration of the cornea
- *Corneal Ulceration/Keratomalacia* - It is a corneal melting that is of abrupt onset. It presents in severe Vitamin A Deficiency
- *Corneal Scarring* - It is the end stage of malnutrition in children who survive. Corneal scarring often has a marked effect on vision

Treatment

Give Vitamin A capsules and emphasize on diet containing dark-green-leafy vegetables

Table 2: Vitamin A Dosage for Children

Vitamin A	Dosage
Age up to 1 year	Age above 1 year
100,000 I.U First day	200,000 I.U First day
100,000 I.U Second day	200,000 I.U Second day
100,000 I.U Third dose after 4 weeks	200,000 I.U Third dose after 4 week

Ocular Treatment

Give Tetracycline or Chloramphenicol 1% eye ointment 8 hourly and avoid corneal exposure.

Prevention

- Give mothers Vitamin A 200,000 IU after delivery
- Encourage breastfeeding
- Give Vitamin A supplementation routinely, through Vitamin A campaigns and to children with measles
- Measles Immunization
- Encourage mothers and weaned children to take adequate foods that are rich in Vitamin A
- Weaning foods should be rich in Vitamin A e.g. mangoes, papaya, dark green leafy vegetables.

1.5 Diseases of the Retina

Main diseases of the retina that causes blindness are Diabetic Retinopathy and Age related Macular Degeneration.

I. Diabetic Retinopathy

Diabetic retinopathy is a well recognized complication of diabetes mellitus. It is a chronic progressive sight threatening disease of the retinal blood vessels associated with the prolonged hyperglycemia and other conditions linked to diabetic mellitus such as hypertension.

Diabetic Retinopathy is grouped into three: Background Diabetic Retinopathy, Diabetic maculopathy and Proliferative Diabetic Retinopathy.

Diagnosis: Is reached by doing fundoscopy in a well dilated pupil, Optical Coherence Tomography and or Fluorescence Angiography. Optical Coherence Tomography and Fluorescence Angiography are done in specialized eye clinics.

Treatment

Laser photocoagulation, extent and type of this treatment depending on the stage of the disease. **Dilate the pupils with**

C:Tropicamide eye drops 0.5 or 1 %

Plus

C:Phenylephrine eye drops 2.5%

OR

C:Tropicamide 0.8%/Phenylephrine 5% eye drops.

Medical Treatment: By intravitreal injection of

D: Bevacizumab 1.25 mg per 0.05ml stat

OR

D: Ranibizumab 0.5 mg per 0.05ml.

NOTE:

- Poorly controlled diabetes and diabetic retinopathy can lead to blindness
- There are no warning symptoms in early stages of diabetic Retinopathy
- Diabetic retinopathy may be present without any symptoms
- All patients with Diabetes Mellitus should be screened/examined by an eye specialist
- Dilated eye examination and direct viewing of the retina by an ophthalmologist or qualified eye care personnel is mandatory.
- All diabetic patients with sudden loss of vision should be referred to eye specialist
- Blindness from Diabetic Retinopathy can be prevented in early stages through laser photocoagulation or surgery and intravitreal injection in advanced/ proliferative stage.

Surgical Treatment, this is done in the proliferative stage by eye specialist.

Age Related Macular Degeneration

This is a disease condition, which is characterized by progressive macular changes that are associated with increase in age. It then results in the gradual deterioration of the vision and eventually loss of vision from the center of the field of vision.

Age Related Macular Degeneration is associated with accumulation of abnormal materials in the inner layers of the Retina at the macula. These changes are seen as yellowish excrescence in the retina called drusens. The disease is common in elderly over 60 years. The only symptom in this condition initially is poor central vision, later can lead to blindness. It is diagnosed by fundoscopy through a well-dilated pupil, Optical Coherence Tomography and or Fluorescence Angiography as for Diabetic Retinopathy.

Treatment

Intravitreal injection of Bevacizumab (Avastin) or Ranibizumab (Lucentis) in the affected eye given by vitreoretinal specialist in specialized eye clinics (dosage as in diabetic retinopathy).

1.6 Refractive Errors and Low Vision**Refractive Errors**

This is a condition where one presents with poor vision either at near or distance at any age. There are mainly 4 types of refractive errors namely presbyopia, myopia, astigmatism and hyperopia. A patient may have more than one type of refractive error.

I. Presbyopia: This commonly occurs as people get older. It usually starts after the age of 40 years. The main complain is difficulty in reading/writing or doing near works. Diagnosis is only through Refraction. This is a good opportunity for screening of glaucoma and diabetic retinopathy so it is very important that eyes are examined properly before testing for spectacles.

Treatment: is by provision of plus lens (Convex) spectacles for near vision

II. Myopia (Short Sightedness): This is a condition whereby patient complains of difficulty to see far objects. It is common in young age between 5 to 25 years. The condition persists throughout life. If not treated early it may progress rapidly and lead to retinal complications. It is diagnosed through refraction.

Treatment: Is with minus lens (Concave) spectacles. These spectacles should be worn all the time.

III. Hypermetropia (Long Sightedness): This is a condition where patients have difficulty in seeing near objects. This condition is less manifested in children as they have a high accommodative power. As a person grows older, accommodation decreases and patients may complain of ocular strain. Diagnosis in children should be reached after refraction through a pupil that is dilated.

Treatment: Is by plus lens (Convex) spectacles.

Note:

- Spectacles should be given to children who have only significant hypermetropia (more than +3.00 Diopter of Sphere both eyes)
- To all children who present with squint and have significant hypermetropia and
- To elderly who present with signs of ocular strain.

IV. Astigmatism: This is a condition where the cornea and sometimes the lens have different radius of curvature in all meridians (different focus in different planes). Some myopic and hyperopic patients may have astigmatism. It presents with poor vision at distance, sometimes there is headache. Diagnosis is reached through refraction and **treatment** is with astigmatic cylindrical lenses.

V. Low Vision

A person with low vision is one with irreversible visual loss and reduced ability to perform many daily activities such as recognizing people in the streets, reading black boards, writing at the same speed as peers and playing with friends. These patients have visual impairment even with treatment and or standard refractive correction and have a visual acuity of less than 6/18 to perception of light and a reduced central visual field. Assessment of these patients is thorough eye examination to determine the causes of visual loss by Low vision therapist.

Treatment

- Surgical intervention if indicated e.g if a patient has cataract
- Assessment of the patients' visual function
- Accurate refraction and provision of spectacles if indicated
- Assessment for/and prescription of low vision devices such as optical devices (magnifiers, telescopes) and or non optical devices (reading stands and or reading slits).

Referral

All children with Low Vision should be referred to a Paediatric Tertiary Eye Centre

2.0 RED EYES

The following disease conditions presents with an acute onset of red eyes: ocular trauma, corneal ulcer, uveitis and conjunctivitis

2.1 Ocular Trauma

There are four types of eye injuries and their management depends on the history. The 4 types of ocular injuries are Perforating Injury, Blunt Injury, Foreign Bodies and Burns or chemical injuries. From the history, one will be able to know the type of injury that will guide the management.

I. Perforating eye injury: This is trauma with sharp objects like thorns, needles, iron nails, pens, knives, wire etc.

Diagnosis

- There is a cut on the cornea and or sclera
- A cut behind the globe might not be seen but the eye will be soft and relatively smaller than the fellow eye.
- The pupil may be irregular or not visible
- Part of the intraocular structures like iris or lens may be protruding out with blood into the anterior chamber
- There may be eyelids involvement.

Treatment

- Apply an eye shield

B:Tetanus Toxoid 0.5 ml intramuscular stat as prophylaxis
Plus

A: Oxyteracycline eye ointment 8 hourly for 3 days
Plus

A:Paracetamol 1 gm 8 hourly for 3 days in adults
Children is 10-14 mg/kg

NOTE:

- Eye ointment should be applied very gently and in the lower fornix (behind the lower eyelid).
- Do not apply pressure on the eye in perforating injuries of the eyeball.
- Delay in surgical management of the injury may cause irreversible blindness or may necessitate removal of an eye.

Refer the patient to eye surgeon immediately

Surgery: This is done by a well trained eye specialist within 48 hours of injury.

The injury should be repaired and give **A:** Gentamycin 200µg in 0.1 ml injection stat given in the anterior chamber.

If there are signs of endophthalmitis (pus in the eye) give

D: Vancomycin 1000µg in 0.1 ml

Plus

D: Amikacin 0.4 mg in 0.1 ml

Plus

D: Cefuroxime 1000µg in 0.1 ml injections.

Antibiotics drops used after surgery are

C: Gentamycin 0.3% Instil one drop in the affected eye, hourly or 2 hourly

Plus

C: Chloramphenical 0.5% Instil one drop in the affected eye, hourly or 2 hourly

OR

C: Ciprofloxacin 0.3% Instil one drop in the affected eye, hourly or 2 hourly

Dilating drops give:

C: Cyclopentolate 1% eye drops 12 hourly

OR

C: Atropine 1% eye drops once a day.

II. Blunt injury

This is trauma from objects such as stones, balls or fist.

Diagnosis

- There may be pain and or poor vision
- There may be blood behind the cornea (hyphaema)
- Pupil may be normal or distorted
- There may be raised intraocular pressure

Guideline on Management

Complicated blunt trauma is best managed by eye specialist as surgery may be required in the management. Refer patients with blunt trauma to eye specialist as indicated below:-

Table 3: Management of Complicated Trauma

Findings	Action to be taken
No hyphema, normal vision	Observe
Hyphema, no pain	Refer
No hyphema, normal vision, pain	Paracetamol, Observe for 2 days, Refer if pain persist
Poor vision and pain	Paracetamol, refer urgently
Hyphema, pain, poor vision	Paracetamol, refer urgently

Management by eye specialist

A. Medical Treatment

Steroid eye drops

This treatment is given to all patients with blunt trauma and present with pain and or hyphema:

C: Prednisolone 0.5 to 1% eye drops, 1 to 3 hourly

OR

Steroid + antibiotics eye drops:

C: Dexamethasone/Chloramphenical 0.1% to 0.5 % eye drops, 1 to 3 hourly

Dilating eye drops

This treatment is given to all patients with distorted pupil or hyphema:

C:Cyclopentolate 1% 12 hourly
OR
C:Atropine 1% once per day

Anti-glaucoma medicines

These are given to all patients with high intraocular pressure. Antiglaucoma of choice are

D: Timolol 0.25% to 0.5% eye drops 12 hourly
Plus

C: Acetazolamide tablets

OR

C: Mannitol (Dosage as seen in Angle Closure Glaucoma).

NOTE:

- Do not give Pilocarpine eye drops in patients with hyphema
- Do not give steroids eye drops if there is corneal abrasion

Analgesics

Give Paracetamol, dosage as above.

B. Surgical Treatment

This is indicated in patients with hyphema and persistent high intraocular pressure despite treatment with antiglaucoma medicines (5 days), with or without corneal blood staining. Surgical procedure is washing of the blood clot from the anterior chamber and Observe intraocular pressure post operative.

Refer corneal ulcer treatment

III. Foreign bodies

This is a condition whereby something like piece of metal, vegetable or animal parts entering into any part of the eye.

Diagnosis

- There may be pain, redness, excessive tearing and photophobia if the foreign body is on the corneal or eye lids
- If the foreign body is superficial, it can be seen
- There may be loss of vision

Treatment

For superficial foreign body

- Instill local anaesthetic agents like
B: Amethocaine 0.5% or 1%

Wait for 3 minutes and remove it with a cotton wool bud

- If the foreign body is not vegetable matter.
A: Oxytetracycline eye ointment and pad the eye for 24 hours

If foreign body is vegetative matter give antifungal

D:Natamycin 5% hourly or 2 hourly

OR

D: Econazole 1% eye drops hourly or 2 hourly.

For intraocular foreign body

Apply antibiotic ointment and eye shield

Refer to eye Specialist for surgical management.

NOTE:

- Never use needles when removing foreign bodies in the eye.
- Never attempt to remove a foreign body that is firmly embedded in the cornea,
- Refer to the nearest eye specialist for removal
- Never pad an eye that was injured with a vegetable material, apply antibiotic ointment and refer.

IV. Burns and chemical injuries

This is a condition that occurs when chemicals such as acid or alkali, snake spit, insect bite, traditional eye medicine, cement or lime enter the eye. It may also be caused by open flame burn to eyelids.

Diagnosis

- Diagnosis relies mostly with patients' history
- Patients may present with photophobia
- Excessive tearing
- Cloudiness of cornea
- Loss of conjunctival blood vessels
- Traces of chemical substance such as cement or herbs and blisters or loss of eyelid skin in open flame injuries.

Treatment Guidelines

This is an Ophthalmological emergency. If a patient gives a history of being in contact with the above, the following should be done:

- Irrigate the eye with clean water continually for a minimum of 20 – 30 minutes
- Test the patients' vision and examine the eye
- Apply eye ointment (Chloramphenicol or Tetracycline)
- Refer to eye Specialist for more care.
- For open flame injuries, apply eye ointment if the patient can not open or close the eye or if there are signs of involvement of the eyeball.

2.2 Corneal Ulcer

Corneal ulcer is a raw discontinuity to the corneal epithelium leading to a painful red eye. This may be caused by Infection (bacterial, viral e.g Herpes simplex virus and measles, fungal), Trauma (physical, chemical) and Nutritional (Vitamin A deficiency)

Diagnosis

- Painful and red eye of acute onset
- It may be accompanied by excessive tearing
- Severe photophobia
- Poor vision and gray/white spot on the cornea

- Pupil may be normal

Treatment

Infectious Corneal Ulcers

- These patients are managed by eye specialists
- Apply eye ointment and shield then refer to eye specialist

In specialized eye unit, the following should be done:

- Examination of the eye,
- Use Fluorescein sodium drops or a drop of local anaesthetic on a fluorescein strip to assess the pattern of the ulcer and measure the size of corneal defect
- Corneal scraping for Gram Stain and Potassium Hydroxide staining, while waiting for results, give patient the following medicines as in section 11.3.1
Antibiotics to cover both gram negative and gram positive organisms,

C: Gentamycin 0.3% given hourly or 2 hourly depending on the eye condition for 3-14 days

Plus

B: Chloramphenicol 0.5% given hourly or 2 hourly depending on the eye condition for 3-14 days

OR

C: Ciprofloxacin 0.3% given hourly or 2 hourly depending on the eye condition for 3-14 days

○ **Give antifungal**

D: Natamycin 5% given hourly or 2 hourly depending on the eye condition for 3-14 days

OR

D: Econazole 1% given hourly or 2 hourly depending on the eye condition for 3-14 days.

Treatment can be changed depending on corneal scraping results

- Give antiviral if Viral causes is suspected after the examination of the eye
C: Acyclovir 3% eye ointment 4 hourly. Continue treatment for at least three days after healing
- Give dilating drops all corneal ulcer patients
C: Cyclopentolate 1% 12 hourly
OR
C: Atropine 1% once per day

Traumatic Corneal Ulcers

The management of these patients is outlined in section 11.3.1 above except for corneal abrasion.

Patient with corneal abrasion complains of pain, gritty sensation and excessive tearing.

- Apply antibiotic ointment and pad
- Review after 24 hours. If signs and symptoms persist, refer to the eye specialist

2.3 Uveitis

This is an eye condition where there is Inflammation of the uveal tissue (Iris, choroid, and ciliary body). Majority of the cases are Idiopathic where by other cases are due to autoimmune diseases e.g Rheumatoid Arthritis, Viral and Systemic diseases like Tuberculosis, Leprosy, and Syphilis.

Diagnosis

It has 3 main clinical presentations namely acute, chronic and acute on chronic. In acute type, patients present with painful red eye, Excessive tearing and severe photophobia. Visual Acuity is usually reduced and the pupil is small or it may be irregular due to synechia. With Slitlamp biomicroscopic examination, cells and keratic precipitates and hypopyon may be seen in the anterior chamber.

Treatment

Treatment of uveitis may be multidisciplinary approach as various specialists may be involved. Before starting treatment, investigations such as blood tests and X-Rays should be done to establish the cause of uveitis. Acute uveitis is a serious problem and the patient should be referred urgently for Specialist treatment. Treatment for uveitis is mainly steroids and specific treatment according to the cause. Strong dilating drops are given to break down posterior synechiae

- Topical Steroid:
 - C:** Dexamethasone 1% eye drops 3hrly
 - OR**
 - C:** Prednisolone 0.5% or 1 % eye drops given 3 hourly
- Oral Steroid:
 - C:** Prednisolone tablets 1mg/kg body weight, given in a tapering manner to maximum of 4 - 6 weeks
- Steroid Injections:
 - D:** Triamcinolone 20 mg subtenon start, it can be repeated after 4 week if need arise
 - OR**
 - D:** Methyl prednisolone sodium acetate 20 mg subtenon stat and it can also be repeated after 4 weeks.
- Dilating Drops:
 - C:** Atropine eye Drops or ointment 1% 12 hourly
 - OR**
 - C:** Cyclopentolate 1 % eye drops 8 hourly.

2.4 Conjunctivitis

Conjunctivitis means inflammation of the conjunctiva. It is one of the most common causes of red eyes. The causes may be bacterial, viral or allergy. Clinical features and treatment guideline depends on the type and cause of conjunctivitis as shown in the following sections.

Allergy Conjunctivitis: In this condition patients presents with history of itching of eyes, sand sensation, and sometimes discharge. When examined, the eyes may be white or red, there may also be other pathognomonic signs such as limbal hyperpigmentation and papillae and papillae of the upper tarsal conjunctiva. In very advanced stages, allergic conjunctivitis patients may present with corneal complications.

Treatment

Treatment depends on the severity of the condition.

- Mild cases where the eyes are white, advice the patient to wash the face with clean cool water four times a day.
 - C:** Hydroxypropylmethylcellulose 0.75% (artificial tears) drops 6 hourly for 14 days
 - In moderate cases who presents with papillae on examination, give mast cell stabilizer such as
 - C:** Sodium cromoglycate 2 or 4 % eye drops 6 hourly per day for at least one month.
- OR**
- C:** Iodoxamide tromethazine 0.1%(Alomide) eye drops 6 hourly per day for at least one month.

Plus

- C:** Zinc sulphate 0.25% eye drops 6 hourly per day for at least one month.
 - In severe cases where there is involvement of cornea, apart from mast cell stabilizers, give short term steroid eye drops.
 - C:** Dexamethasone 0.1% 6 hourly may be given for a maximum of 14 days
- OR**
- C:** Prednisolone 0.5% 6 hourly may be given for a maximum of 14 days.
 - In very severe form of allergic conjunctivitis, steroid injection is given
 - D:** Triamcinolone acetonide 20 mg/ml
- OR**
- D:** Methylprednisolone sodium acetate 20mg/ml subtenon stat.

All patients with moderate to severe allergic conjunctivitis should be referred to eye specialist for further specialized care.

Viral conjunctivitis: It presents with painless watery eye discharge, there may be photophobia if the cornea is involved. The disease is bilateral though it may be asymmetrical. If adenovirus is the cause, it appears in epidemics so there will be history of being in contact with patients with similar eye condition. Patients present with haemorrhages of conjunctival vessels. It is usually self-limiting. Apply antibiotic eye ointment or eye drops if there is secondary infection with other organisms

Note:

Viral Conjunctivitis is very contagious so patients and members of the family should be alerted

Bacterial conjunctivitis: Presents with acute onset of painless purulent discharge. The conjunctiva shows a velvety beef red appearance. Sometimes there is ocular discomfort and it is usually bilateral. The diagnosis is mainly clinically. Bacterial conjunctivitis patients who are not responding to treatment should have eye swabs for Gram stain and for culture and sensitivity to tailor down treatment. Bacterial Conjunctivitis is treated with antibiotic eye drops three hourly. If no improvement after two days refer to eye specialist.

Ophthalmia Neonatorum/Neonatal Conjunctivitis; This is a special type of acute bacterial infection of the eyes that affect newborn baby during the first 28 days of life. Causative organisms are Neisseria gonorrhoea, Chlamydia trachomatis and Staphylococcus spp. The infection is acquired from mother's birth canal secretions.

Diagnosis: Patients present with massive oedema and redness of eyelids and with purulent and copious discharge from the eyes. There is usually rapid ulceration and perforation of corneal which eventually leads to blindness if treatment is delayed. It usually presents 3 to 4 days of life. Late presentation may also appear depending on the causative organism.

For Prevention and treatment see the "Neonatal Conjunctivitis (NC) Flow chart under the Sexual Transmitted disease Chapter

Table 4: Summary on Diagnosis Red Eyes

Disease Condition	Visual Acuity	Affected Eye	Cornea	Pupil	Pain	Discharge
Allergic/ viral Conjunctivitis	Good	Both	Clear	Normal	No	Watery/mucoid
Bacterial Conjunctivitis	Good	Both	Clear	Normal	No	Purulent
Ophthalmia neonatorum	Poor +/-	One/both	Cloudy +/-	Normal +/-	Yes	Copious purulent
Cornea ulcer	Poor	One/ both	Gray spot	Normal	Yes	Watery/purulent
Uveitis	Poor	One/ both	Clear or cloudy	Small & Irregular	Yes	watery
Acute glaucoma	Poor	One	Cloudy	Mid dilated	Yes	Watery

3.0 SQUINT

A squint means that the eyes are looking in different directions; one eye appears to be turned in or out. It may occur in children or adults. There are many causes of squint but the most important and common ones in children are refractive errors, amblyopia (lazy eye), retinoblastoma, cataract and syndromic eye diseases that may be of neurologic origin or not. In addition to that, in adults squint may be complication of diabetes mellitus and orbital/head trauma.

Treatment

Treatment of squint depends on the aetiology. Thorough examination of the eyes by a pediatric eye specialist is needed to guide the management of the patients, so refer all children to Paediatric Eye Tertiary Centre.

4.0 OCULAR SURFACE DISEASE

The most common ocular surface diseases are pterygium and Squamous cell carcinoma of the conjunctiva. These affect the exposed area of conjunctiva as a response to chronic dryness and exposure to sunlight.

4.1 Pterygium

This is a triangular sheet of fibrovascular tissue which invades the cornea. Patients present with adherent conjunctival overgrowth on the cornea.

Treatment

Treatment for pterygium is surgical excision in advanced stage where the visual axis is involved. The favoured method is excision that is followed by a free conjunctival graft. Surgery should be done by qualified eye care personnel and antibiotic steroid combination drops should be given postoperative.

4.2 Squamous Cell Carcinoma

This is the most common tumor of the conjunctiva. It is a slow growing tumour which invades the sclera and cornea. It can penetrate the globe in advanced stages. It occurs in increased frequency in patients with Xeroderma Pigmentosum and is one of the ocular manifestations of HIV.

Diagnosis

The tumour is seen as papillary or gelatinous mass associated with feeder vessels. It is located at the limbus and may involve adjacent cornea.

Treatment

If tumour is suspected,

- Excise the mass with wider margin (2 mm)
- Treat the margins with Mitomycin C, 5 Fluorouracil or cryotherapy
- Send the specimen for histological examination
- For advanced tumours where the globe has been infiltrated, removal of the eye is indicated (Enucleation or exenteration)
- Send patients with confirmed diagnosis to Oncologist for radiotherapy

4.3 Retinoblastoma

It is the commonest childhood malignant tumor of the eyes. It is diagnosed between the first 1 to 3years of life.

Diagnosis

The most common initial sign is white pupil reflex (leukocoria), followed by squint, and rarely vitreous haemorrhage, hyphema, ocular/periorbital inflammation, glaucoma and in late stages proptosis and hypopyon. It can be inherited so examine the child and sibs in hereditary for every 4 months until yr 4, then 6 monthly until yr 6 and yearly in over 8yrs.

Management

The goals of treatments are:-

- To save the patient's life
- To save the patient's eye and vision if possible

Choice of treatment depends on Size of tumor, Location and Extent of the tumour.

Treatment is done in Specialized Centres by both Ophthalmologist and Pediatric Oncologist
(Detail in treatment refer to oncology chapter)

Treatment Modalities

- Enucleation of the affected eye and the eye is taken for histology
- Chemotherapy
- External beam radiotherapy
- Plaque radiotherapy
- Cryotherapy and laser photocoagulation

NOTE:

Monitoring is very important due to the following:-

- There is a chance of developing retinoblastoma in the fellow eye.
- The risk is diminished in increase in age
- Also watch for secondary tumors like osteosarcoma

CHAPTER TWELVE

NERVOUS SYSTEM DISEASE CONDITIONS

1.0 INFECTIONS OF THE NERVOUS SYSTEM

Infections of the nervous system can arise secondary to bacteria, fungi, protozoa or viruses. Clinical features will depend on the site of the nervous system involved.

1.1 Bacterial infections

1.1.1 Meningitis

The major features of the disease is Inflammation of the layers (meninges) covering the brain and spinal cord

Diagnosis

- Headache, high fever, chills, backache, nausea and vomiting
- Neck stiffness, convulsions and coma may occur.

Children

In infants under 1 year diagnosis is much more difficult therefore always think of it in a sick child if:

- Refusal to eat and or suckle, drowsiness and weak cry
- Focal or generalized convulsions
- Fever may be absent
- Irritability
- Hypotonia, neck is often not stiff
- Bulging fontanelle

NOTE: A lumbar puncture is essential to confirm diagnosis

General management

- Control of fever and pain with Paracetamol
- If unconscious, insert NGT for feeding and urethral catheter

Treatment

I. Where the organism is not known:

Adults:

B: Chloramphenicol 1 g every 6 hours IV initially and after a good clinical response continue with oral treatment at the same dose for 14 days

Plus

B: Benzyl penicillin 5MU IV every 6 hours initially and after good clinical response give same dose i.m. for 10 days.

Children and Infants < 3 months:

C: Ampicillin 50mg/kg/dose IV 6 hourly for 10 to 14 days

Plus

C: Gentamicin 7.5mg/kg once daily for 10 to 14 days

OR

D: Cefotaxime; 50mg/kg/dose IV 6 hourly for 10 to 14 days

Children 3 months to < 18 years:

B: Chloramphenicol 25mg/kg/dose IV 6 hrly

Plus

C: Ampicillin 50mg/kg/dose 6 hourly

OR

B: Benzyl penicillin 100,000IU(60mg)/kg/dose 6 hourly for 10 to 14 days

Alternative treatment: Third generation cephalosporins:

C: Ceftriaxone 100mg/kg/day in one or two divided doses for 10 to 14 days

Note: For old age, immunosuppression, diabetic or alcoholic patients give, Cefotaxime 2g iv 6hrly or Ceftriaxone 2g I.V 12hrly Plus Ampicillin 2g I.V 4hrly or Cotrimoxazole 50mg/kg I.V daily in two divided doses.

Where the patient has convulsions:

B: Diazepam 0.25-0.5 mg/kg body weight by slow I.V. until control is achieved

In neonates:

C: Phenobarbitone loading dose of 15 mg/kg. If convulsions persist repeat Phenobabitone 15 mg/kg after half an hour, thereafter, 10 mg/kg up to a maximum of 40 mg/kg

II. Where the organism is known the following is advised:

Meningococcal meningitis and pneumococcal meningitis

Adults & children >2yrs

B: Oily Chloramphenicol(IM) 100 mg/kg as a single dose Max. 3g

OR

C: Ceftriaxone(IM) 100mg/kg as a single dose (divide into 2 injections if needed & inject half-dose in each buttock)

Haemophilus influenza meningitis

Adult

C: Ampicillin (IV) 3 g IV every 6 hours initially, then change to oral dose medication as soon as possible

OR

B: Chloramphenicol 50-100mg/kg/day for 10 days

Children

C: Ampicillin 50-100 mg/kg/day for 10 days

OR

B:Chloramphenicol 50 mg/kg body weight every 6 hours for 10 days

NOTE: Neonates require treatment for 3 weeks and the recommended treatment is: Chloramphenicol (IV) 6 mg/kg body weight every 6 hours. But should NOT be used in premature/low-birth weight infants

1.1.2 Tetanus

It is an acute, often fatal disease caused by an exotoxin produced by the anaerobic bacterium *Clostridium tetani*. It is acquired through wounds contaminated with spores of the bacteria and in the case of neonates, through the umbilical stump, resulting in neonatal tetanus.

Diagnosis

- Generalized spasms and rigidity of skeletal muscles
- Patients are usually fully conscious and aware.

General management

- Nurse in dark, quiet room to avoid unnecessary external stimuli which can trigger spasms
- Protect the airway (patient may need to be referred)
- Immediate (preferably after administration of antitetanus immunoglobulin) thorough cleaning of the site of entry (wound/umbilicus), leaving it exposed without dressing
- Pain management with paracetamol (via NGT) as the spasms can be very painful
- Maintenance of fluid balance and nutrition (via NGT)
- Avoid giving medications via IV/IM route as injections can trigger spasms
- Sedation (see below) and care as for unconscious patient
- Prevention:
 - B:** Tetanus (toxoid) vaccine 0.5 ml IM; repeat after 4 weeks and after 6-12 months, then boost every 10 years thereafter

Treatment

Treatment is generally aimed at the following:

For prevention of further absorption of toxin from wound

B: Human tetanus immunoglobulin; Adults & children give 100 – 300 IU/kg IM stat

OR

Horse serum after a test dose

Plus

A: Amoxicillin 500-1000mg via Nasal Gastric Tubes every 8 hours
(Neonates and Infants: ≤3 months: 20-30 mg/kg/day; Infants >3 months and Children: 25-50 mg/kg/day for 14 days)

Plus

A: Metronidazole 500mg every 8 hours
(Neonates I.V. 0-4 weeks, <1200g: 7.5 mg/kg every 48 hours. For postnatal age ≤7 days: 1200-2000 g: 7.5 mg/kg/day given every 24 hours >2000 g: 15 mg/kg/day in divided doses every 12 hours. Postnatal age >7 days: 1200-2000 g: 15 mg/kg/day in divided doses every 12 hours >2000 g: 30 mg/kg/day in divided doses every 12 hours)

For anaerobic infections:

A: Metronidazole Oral, I.V.: 30 mg/kg/day in divided doses every 6 hours; maximum dose: 4 g/day for 14 days

Control of spasms

Give a sedative cocktail of ALL the following VIA NGT:

Adult :

B: Diazepam 10-30 mg every 6 hours
Children 0.5 mg/kg body every 6 hours

Plus

A: Chlorpromazine 100 mg every 8 hours
Children 2 mg/kg body every 6 hours

Plus

A: Phenobarbitone 50 – 100 mg every 12 hours
Children 6 mg/kg every 12 hours

Table 1: Guidelines for Dosage Administration**

Time (hours)	0	3	6	9	12	15	18	21	24
Diazepam	*	*		*		*		*	*
Chlorpromazine		*		*		*			
Phenobarbitone	*		*					*	

** These are general guidelines. Frequency of drug administration should be titrated vs clinical condition

1.1.3 Brain abscess

Brain abscess is a focal collection of pus/ necrotic tissue within the brain parenchyma, which can arise as a complication of a variety of infections, trauma or surgery. The manifestations of brain abscess initially tend to be nonspecific, resulting in a delay in establishing the diagnosis.

Diagnosis

- Headache is the most common symptom, neck stiffness, lethargy progressing to coma, vomiting, and focal neurologic deficit.

General management

- Control of fever and pain with Paracetamol
- If unconscious, insert NGT for feeding and urethral catheter

Table 2: Management of Brain Abscess

Condition	Treatment	Duration
Brain abscess (unspecific bacterial)	Benzyll penicillin (I.V) 5 MU every 6hours(children 125,000 IU/kg/24 hours) <i>Plus</i> Metronidazole (IV) 500mg every 8 hours (children 7.5 mg/kg/day)	4-6weeks 4-6 weeks
Brain abscess (Staph aureus)	Cloxacillin (I.V) 2g every 6 hours(children 5 – 100 mg/kg/day)	6weeks

Note: Where the patient is allergic to penicillin, chloramphenicol 500 mg every 6 hours can be used instead

1.2 Fungal infections

Cryptococcal meningitis

It develops in patients who are immunocompromised e.g. HIV-positive patients with low CD4 cell count.

Diagnosis

- Headache, fever, intolerance to light and sound, neck stiffness, vomiting, seizures, deafness and blindness
- In advanced stages it may present with confusion, altered consciousness and coma.

General management

Refer to section on bacterial meningitis

Treatment

B: Fluconazole 400-800mg (12-15mg/kg/day in children) IV or (O) depending on the patient's condition for 6-10 weeks, then 200mg for the rest of the patient's life

OR

D:Amphotericin B 0.7-1 mg/kg/day by slow infusion IV for 2 weeks

Plus

S: Flucytosine 2-5mg/kg IV four times daily for 14 days.

Followed by maintenance treatment with Fluconazole 200mg for life

Patients started on IV should be switched to oral therapy as soon as patients are clinically stable to reduce the length of hospitalization and lower associated costs. Consider LP as diagnostic and therapeutic tool for cryptococcal meningitis. Cryptococcal antigen test should be done as there are cases of negative Indian ink results with cryptococcal meningitis.

For more details, Refer to Tanzania National Guidelines for the treatment of HIV/AIDS for further details

1.3 Protozoal infections

Toxoplasmosis

Immunocompetent persons with primary infection are usually asymptomatic, but latent infection can persist for the life of the host. In immunosuppressed patients, especially patients with AIDS, the parasite can reactivate and cause disease, usually when the CD4 lymphocyte count falls below 100 cells/mm³.

Diagnosis

- Patients can present with focal paralysis or motor weakness depending on the brain area affected
- Neuro-psychiatric manifestations corresponding to the affected area in the brain, seizures or altered mental status.

Note: Diagnosis is predominantly based on clinical findings after exclusion of other common causes of neurological deficit. If available, a CT scan is very useful for confirmation. Toxoplasma serology has to be done for addition in diagnosis.

General management

Similar to bacterial meningitis

Prophylaxis

Primary prophylaxis therapy

A: Trimethoprim–Sulphamethoxazole (TMP-SMX) 160/800mg orally/day.

Treatment

Acute infection

A: Trimethoprim–Sulphamethoxazole (TMP-SMX) 120mg/kg daily in 2-4 divided dose for 21 days

C: Sulphadiazine 1 gm every 6 hours for 6 weeks

Plus

C: Pyrimethamine 100mg loading dose then 50mg /day for 6 weeks

Plus

C: Folic acid tabs 10mg /day for 6 weeks.

After six weeks of treatment give prophylaxis therapy with Sulphadiazine tabs 500mg 6 hourly + Pyrimethamine tabs 25-50mg /day + Folic acid tabs 10mg /day.

For those allergic to sulphur replace Sulphadiazine tabs with

S: Clindamycin capsules 450mg 6 hourly.

Discontinue maintenance therapy when CD4 count is >200 cells/mm³, initial therapy is completed and patient is asymptomatic.

In case of seizures, *Refer to Tanzania National Guidelines for the treatment of HIV/AIDS for further details*

1.4 Viral infections

In Tanzania, viral infection of the nervous system is mainly caused by *Herpes simplex* virus and HIV.

See section on viral infections and HIV

1.4.1 Rabies

Rabies is an acute viral infection of the central nervous system that affects all mammals and is transmitted to man by animal bites via infected secretions, usually saliva.

Diagnosis

- Early or prodromal clinical features of the disease include apprehensiveness, restlessness, fever, malaise and headache
- The late features of the disease are excessive motor activity and agitation, confusion, hallucinations, excessive salivation, convulsions and hydrophobia

Note: Death is considered as invariable outcome.

General management

- Local wound therapy -wash wound thoroughly with water and soap and repeat process with 10% Povidone iodine to prevent secondary bacterial infection

Prophylactic wound therapy that has lasted less than 8 hours

A: Amoxicillin-clavulanate 500mg/125mg (O) 8hourly for 3-5 days

Infected wounds and wounds older than 24 hours

A: Amoxicillin-clavulanate 500mg/125mg (O) 8hourly for 3-5 days

Plus

S: Clindamycin 150–300 mg every 6 hours for 3-5 days

Plus

B: Ciprofloxacin(adults) 500mg 12 hourly for 3-5 days

OR

A: Trimetoprim/sulphamethoxazole(children) 120-480mg every 12 hours for 3-5 days

Passive immunization

B: Anti-rabies human immunoglobulin 20 IU/kg half the dose given parenterally and the other half injected into and around the wound

Active immunization

B: Human Diploid Cell Vaccine (HDCV) 1ml I.M on days 0, 3, 7, 14, 28. In addition, patients should receive rabies immune globulin with the first dose (day 0)

- Tetanus toxoid vaccine *see section on Tetanus*

1.4.2 Herpes simplex encephalitis

The majority of cases in adults caused by HSV-1, a small number are caused by HSV-2 usually in immuno-suppression or in neonates. It causes inflammation and necrosis in the brain.

Diagnosis

- Early features are fever, headache & altered consciousness which may develop gradually over days or rapidly over hours
- The most common manifestations are personality change, dysphasia, behavioural disturbance and occasional psychotic features
- Focal or generalized seizures can occur
- On lumbar puncture, CSF is under increased pressure and may appear normal or show a mild-moderate lymphocytosis, a mild-moderate increase in protein and normal or mildly decreased glucose.

Note: The disease is easily missed in Tanzanian settings due to lack of diagnostic facilities and should therefore be suspected in patients not responding to antibiotics/other treatment.

General Management

Manage it as for unconscious patients (Control seizures)

Treatment

B: Acyclovir 10–15 mg/kg (O) every 8 hours for 14–21 days

Plus

C: Prednisolone 10-20mg (O) daily preferably taken in the morning. Maximum dose 60mg

2.0 MENTAL CONDITIONS

2.1 Anxiety conditions

It is a group of disorders characterized by a chronic, unrealistic/exaggerated anxiety often punctuated by acute attacks of anxiety or pain. It afflicts 5% of the population and is characteristically a disorder of young adults and affects women twice as often as men. The illness may take many forms. Acute anxiety attacks are characterized by sudden onset of tension, restlessness, tremors, breathlessness, tachycardia and palpitations. Chronic anxiety state presents with persistent diffuse anxiety, motor tension, autonomic hyperactivity, unpleasant anticipation and irritability.

Diagnosis

Refer to ICD – 10 criteria

General management

- Medicines do not resolve the causes of the illness but may reduce anxiety.
- **Education about the nature of anxiety**
- Psychotherapy – **Training in strategies for controlling anxiety and reducing stress**

Treatment

C: Diazepam 5 – 10 mg (O) every 8 hours

OR

A: Chlorpromazine 50 – 75 mg (O) daily, increase gradually to 300 mg daily

OR

C: Amitriptyline give 25 mg every 8 hours

OR

D: Alprazolam 0.25 – 0.5mg every 8-12 hours

OR

D: Lorazepam 1 – 4mg daily in divided doses

Referral

If symptoms persist for longer than 3 months despite above measures refer the patient to the specialists

2.2 Panic disorder

It is characterized by recurrent and sometimes unpredictable attacks of anxiety or panic. Common symptoms include palpitations, sweating, trembling or shaking, shortness of breath, feeling of choking, chest pain, nausea, dizziness, and derealization; fear of losing control, fear of dying, parasthesias, and chills.

Diagnosis

Diagnosed after recurrent (several) panic attacks within a one month period.

General management

- Psycho-education and reassurance.
- Psychotherapy, e.g. cognitive-behaviour therapy.
- Always consider the possibility of an underlying medical condition, e.g. thyrotoxicosis, etc.

Treatment

The initial aim is to control the panic symptoms and exclude an underlying medical cause. Give the patient Benzodiazepines, repeated as necessary to control symptoms, e.g. Diazepam, IV/oral, 5mg daily. Increase to 10 – 15mg daily in divided doses

Note: Do not give the therapy more than two weeks

Referral

If panic disorder is diagnosed, long-term treatment may be required therefore refer the patient to the mental clinic. Most patients can be treated as outpatients, but some may need to be admitted.

Treatment of choice

D: Fluoxetine oral 20 mg once a day for 6 months–1 year

Extended drug treatment over many years and even life-long may be necessary, except where cognitive-behaviour therapy has been successful. Relapses may occur when treatment is discontinued.

2.3 Major depressive disorder

It is a mood disorder characterised by at least 2 weeks of depressed mood as well as diminished interest and pleasure in activities and is associated with:

- Somatic symptoms, e.g. change in appetite and sleep, agitation or retardation and loss of energy
- Psychic symptoms, e.g. feeling of worthlessness, guilt, diminished concentration or indecisiveness, thoughts of death and suicide

General management

Effective psychotherapies include:

- Cognitive Behavioural Therapy
- Interpersonal psychotherapy
- Stress management / coping skills
- Marital and family issues
- Accommodation and vocational issues

Treatment

Adults:

C: Amitriptyline tablets initially 50–75 mg daily at night, increase gradually to a maximum of 150 mg daily. **Elderly:** Initially 25- 50 mg. Max. 75mg

OR

D: Fluoxetine capsules initial dose: 20 mg daily (preferably in the morning), may increase up to 60mg/day

OR

S: Fluvoxamine tablets initially 50 – 100mg daily

OR

S: Citalopram tablets 20mg daily in the morning or evening increase if necessary to a maximum of 60mg daily (Elderly maximum 40mg daily)

If patient has severe psychosis:

C: Haloperidol 5 mg I.M half hourly in 2 hours to a maximum of 20mg/24 hours till acute attack is controlled. Then

B: Haloperidol 3-4.5 mg (O) every 12 hours

Referral

Refer if

- Suicidal ideation
- Major depression with psychotic features
- Bipolar disorder
- Failure to respond to available antidepressants
- Patients with concomitant medical illness, e.g. heart disease, epilepsy
- Poor social support systems
- Pregnancy and lactation
- Children and adolescents

2.4 Bipolar disorder

It is a lifelong illness, which may have an episodic, variable course. The presenting episode may be manic, hypomanic, depressive or mixed. By definition, a diagnosis of bipolar disorder requires either a current or previous episode of mania. An episode of mania is typically characterised by an elevated mood whereby a patient may experience extreme happiness which might also be associated with an underlying irritability. Such mood may be associated with increased energy/activity, talkativeness and a reduction in the need for sleep and features may be accompanied by grandiose and/or religious delusions. Bipolar disorder causes substantial psychosocial morbidity, frequently affecting patients' relationships within the family as well as their occupation and other aspects of their lives.

Diagnosis

Refer to ICD – 10 criteria

General management

- Hospitalisation may be required during acute mania.
- Psychotherapy, usually after the manic episode has been controlled with medication.
- Family therapy and psycho-education of patient and family to increase compliance and knowledge of the condition.
- In severe cases, psychiatrist directed electroconvulsive therapy may be required.

Treatment for Manic or Mixed Episodes

For agitated and acutely disturbed patient:

C: Haloperidol injection IM, 5 mg half hourly for 2 hours to a maximum of 20mg/24 hours.

Plus/OR

A: Diazepam IV, 20 mg 8hourly for 24hours. Maximum dose 120mg. Switch to oral once containment is achieved.

Maintenance therapy

Under specific circumstances such as past or family history of response and rapid cycling, i.e. moving between mood states: Give

C: Sodium valproate 20 mg/kg/day (O) in 2–3 divided doses

OR

C: Carbamazepine 600mg (O) daily, increase by 200mg at three day interval up to a maximum of 2500mg.

OR

S: Lithium carbonate 400-1000mg as a single dose or in 2 divided doses
Elderly 400mg daily

Consider oral haloperidol with adjunctive benzodiazepines in patients who are difficult to manage, i.e. not settling with mood stabiliser monotherapy, and especially where there are features of psychosis.

Treatment for Severe Depressive Episodes in Bipolar Patients

Give antidepressant in combination with mood stabilizer and antipsychotic if there is psychosis:

Drug of choice:

C: Amitriptyline 50mg nocte

Plus

C: Carbamazepine 300mg twice a day.

Plus

B: Haloperidol 3-4.5 mg 12hourly (**if there is psychosis**)

Note: Do not use monotherapy antidepressants in bipolar patients.

Referral

- Mixed or rapid cycling bipolar disorder
- Depressive episodes in bipolar patients not responding to treatment
- Manic episodes not responding to treatment

2.5 Schizophrenia

It is characterized by altered thinking process, emotions, drive, behavior and withdrawal from reality. Symptoms vary from patient to patient and from time to time. These include bizarre appearance, reduced motor activity, withdrawal, flattened affect and mood disturbance, delusions and hallucinations.

Diagnosis

Refer to ICD-10 criteria

General management

Supportive intervention includes:

- Family counselling and psycho-education
- Cognitive Behavioural Therapy (CBT) for stabilised patients
- Supportive group therapy for patients with schizophrenia
- Rehabilitation may be enhanced by:
 - Assertive community programs
 - Work assessment, occupational therapy and bridging programmes Prior return to the community
 - Appropriate placement and supported employment

Treatment

In acute attacks:

C: Haloperidol 5 mg every 30 minutes for 2 hours

Plus

A: Diazepam 20mg 8 hourly for 24 hours.

For maintenance:

A: Chlorpromazine 100 – 600 mg (O) daily in divided doses

OR

C: Haloperidol 3-4.5 mg (O) 12hourly

OR

S: Olanzapine 5-10mg 12 hourly. Maximum dose 25mg/day

OR

S: Risperidone 1mg bid then increase by 1mg every 2-3 days to 2 – 3mg twelve hourly. Maximum dose 16mg/day

Note:

- The above medicines cannot be given in combination.
- The atypical antipsychotics are drug of choice for patients with negative symptoms

Long-term therapy

For patients who have poor compliance

C: Fluphenazine decanoate 12.5–50 mg IM every 4 weeks.

Adjunct treatment

Antiparkinsonian drugs should only be used if **extrapyramidal side effects** occur or at higher doses of antipsychotics likely to cause extrapyramidal side effects. Any of the following can be used:

C: Trihexyphenidyl (Benzhexol 5mg once to two times a day (O) last dose before 1400 hours

S: Procyclidine 10mg two times a day last dose before 1400 hours

Referral

- First psychotic episode
- Poor social support
- High suicidal risk or risk of harm to others
- Children and adolescents
- The elderly
- Pregnant and lactating women
- No response to treatment
- Intolerance to medicine treatment
- Concurrent medical or other psychiatric illness
- Epilepsy with psychosis

2.6 Parkinsonism

Is a syndrome characterised by tremor, rigidity, bradykinesia and postural disturbances. It may be primary, i.e. Parkinson's disease, or secondary, i.e. drug-induced.

Treatment Objectives

- Minimise disabling symptoms.
- Prevent complications and avoid serious drug-induced side effects.
- To exclude secondary forms

Management

- Educate the patient.
- General supportive therapy and advice about lifestyle modification, physiotherapy and occupational therapy.

Treatment

Note: Set therapeutic targets so that the patient is not overtreated.

For Predominant tremors

Give Anticholinergics, e.g.:

B: Trihexyphenidyl (Benzhexol) 1–2 mg (O) daily increasing gradually. Maximum dose: 15 mg/day in 3–4 divided doses.

For Bradykinesia, rigidity and postural disturbance

S: Carbidopa/levodopa 25/100 mg (O) 8 hourly. Increase by 25mg as levodopa every 1–2 days until the desired response is achieved. Maximum dose 800mg as levodopa.

OR

Dopamine agonists, e.g.:

S: Bromocriptine 5 – 10mg (O) daily for 1 week. Increase according to response: Week 2: 2.5 mg daily; Week 3: 2.5 mg twice daily; Week 4: 2.5 mg 3 times daily; Week 5: 5 mg 3 times daily

For Drug-induced extrapyramidal syndrome

Give Anticholinergic agent, e.g.:

B: Trihexyphenidyl (Benzhexol) 5mg (O) daily, and increase to 10 mg daily.

For Acute dystonic reaction

Usually follows administration of dopamine-antagonistic drug, e.g. metoclopramide and phenothiazines. Anticholinergic agent, e.g.:

B: Trihexyphenidyl (Benzhexol) 5mg (O) daily, and increase to 10 mg daily.

OR

A: Diazepam IV 5 – 10mg

Referral

- If no improvement with treatment
- If increasing on/off phenomenon

2.7 Epilepsies

Are disorders of the central nervous system (CNS) which are characterized by chronic spontaneous recurring seizures

Management

- Make sure that all other causes (alcohol, eclampsia, meningitis, hypoglycaemia etc) are excluded
- Patients with more than one fit should be considered for treatment
- Treatment should be started with phenobarbitone alone. Full effect can be experienced usually after two weeks.
- Phenobarbitone can be increased to maximum if seizures persist (refer to a table below)
- When no improvement is obtained change to phenytoin, tapering phenobarbitone by reducing the dose by 30 mg every week. If seizures persist, increase phenytoin by 50 mg increment to a maximum dose of 600 mg daily
- If no appreciable improvement, change to carbamazepine, stopping phenytoin by reducing dose by 50 mg per week. Increase the dose to maximum
- If possible the combination of these drugs should be avoided

- Patients still having seizures despite of having the above drugs should be referred to a higher level of treatment.

Note: Phenytoin has a lot of side effects therefore prefer Carbamazepine if available

Treatment

Table 3: Dosages for epilepsy treatment

	DRUG / INITIAL DAILY	DAILY MAXIMUM DOSE
	Phenobarbitone (O) once daily at night	
Children	3mg/kg/24 hours	8 mg/kg/24 hours
Adult	60 to 90 mg	240 mg
	Phenytoin (O) once daily at night or twice daily when required	
Children	5mg/kg/24 hours	8mg/kg/24hrs(2 divided doses)
Adult	200mg	600mg (2 divided doses)
	Carbamazepine (O) as 2 divided doses	
Children	10mg/kg/24 hours	20 mg/kg/24 hours
Adult	300 mg 12 hourly	2000 mg (3divided doses)
	Sodium valproate(O)	
Adult	600mg daily in divided doses	3000mg
Children	20mg/kg in divided doses	40mg/kg

Referral

- All new patients, for diagnosis and initiation of therapy by a doctor
- Patients with seizures other than generalised tonic clonic seizures, including absence seizures
- Increased number of seizures or changes in the seizure type
- Patients who have been seizure free on therapy for 2 years or more (to review therapy)
- Pregnancy or planned pregnancy
- Development of neurological signs and symptoms
- Adverse drug reactions
- Suspected toxicity

2.8 Status Epilepticus

It is persistent seizures, without regaining consciousness. It is a medical emergency therefore treat it promptly.

Management

Adults:

- Protect airway, give oxygen
- Give dextrose 5%, 80 ml as bolus
- Give anticonvulsant

Treatment

A: Diazepam (IV) 10 - 20 mg at a rate of 5mg per minute. Repeat in 30 -60 minutes if necessary to a maximum of 200 mg in 24 hours; monitor respiration
OR

S: Clonazepam (O) 0.5mg to 2mg.

Once the status epilepticus has been controlled the patient should be maintained on other antiepileptics.

Second choice

C: Phenobarbitone 200mg (IV) slowly. Repeat after 10 minutes, thereafter it may be repeated every 30 minutes to a maximum of 15mg/kg/24 hours

Third choice

S: Phenytoin (IV) 150-250 mg at a rate not exceeding 50 mg/minute. Continue with 100 mg every 6 hours, but do not exceed 15mg/kg/24 hours

Note: These drugs when given together may cause serious respiratory depression

Children:

- Protect airway, give oxygen
- Give dextrose 50% (I.V) 15 ml (1ml/min) as a bolus
- Give anticonvulsant:

A: Diazepam 5 mg/minute (slow I.V). Maximum dose 0.25 mg/kg body weight

2.10 Serial Epilepsy

Patient gets frequent seizures but regains consciousness between attacks.

Treatment:

C: Phenobarbitone (I.M)400mg (maximum 15 mg/kg/24hours),
Children 5 mg/kg/24 hours as loading dose

For febrile Convulsions in Children aged 1-5 years

Do not give anticonvulsant except to known non-febrile convulsion cases or neurological abnormalities. Sponge the child and give antipyretics. For prolonged or recurrent febrile convulsions, **Diazepam** should be administered rectally by using a syringe.

Note: Phenytoin, phenobarbital and carbamazepine are potent enzyme inducing agents and should be used with caution with other drugs metabolised by the liver, especially warfarin, ARVs and oral contraceptives.

2.11 Substance Abuse

It is a non-medical use of drugs, i.e. any use of drugs for other than recognized therapeutic purposes, commonly abused drugs include, marijuana, diazepam, **heroin, cocaine**, alcohol etc.

Diagnosis

Refer to ICD 10 criteria

Treatment

- Supportive therapy e.g. I.V fluids, chlorpromazine for acute confusional state
- Management of acute problems depends on the substance of abuse being identified.
- Rehabilitation

Selected drugs management

I. HEROIN

Withdrawal is generally poorly tolerated, but not dangerous, except in very frail debilitated patients or during the first trimester of pregnancy.

Mild Withdrawal: May be done on an outpatient basis

Symptomatic treatment

C: Diazepam 5–20 mg (O) once daily or in divided doses only as inpatient, taper off over 5–7 days

OR

A: Promethazine 50mg once daily at sleeping time

OR

A: Chlorpromazine 50 -100mg once daily at sleeping time

For abdominal cramps

A: Hyoscine butylbromide 20 mg (O) up to 3 times daily as required

OR

A: Diclofenac tablets 50mg (O) 8hourly

For diarrhea

B : Loperamide 4 mg (O) immediately, then 2 mg after each loose stool

Moderate to Severe Withdrawal: Refer to specialized clinic **for**

S: Methadone maintenance therapy

II. COCAINE

Non-Drug Treatment

- These patients usually do not require admission

- Beware of depression and assess suicide risk

Drug Treatment

- No substitute drug available for detoxification.
- For severe anxiety, irritability and insomnia, short-term benzodiazepines, e.g.

C: Diazepam 5–10mg (O) 3 times daily for 5–7 days

Referral

Refer patients to specialized clinic

III. Alcohol Dependence Syndrome

Alcoholism is a syndrome consisting of two phases: problem drinking and alcohol addiction. Problem-drinking is the repetitive use of alcohol, often to alleviate tension or solve other emotional problems. Alcohol addiction is a true addiction similar to that which occurs following the repeated use of barbiturates or similar drugs.

Diagnosis

- Painless hepatomegally and palmar erythema
- Signs of more advanced disease secondary to liver cirrhosis are jaundice, ascites, testicular atrophy and gynaecomastia.
- Refer to ICD 10 criteria for Diagnosis
- Also use C.A.G.E questionnaire which determine extent of use
C- Cut down alcohol use
A-Annoyed by people criticising you're drinking
G-Guilty about drinking
E-Eye opener

General management

- Support group that encourage abstinence
- Alcoholic anonymous (AA)
- Inpatient rehabilitation programme exists

Treatment

a) Alcohol-related withdrawal syndrome

S: Thiamine 50 – 100 mg I.M every 24 hours.

For the CNS symptoms

B: Diazepam (O) 10 mg every 4-6 hours on the first 24 and reduce by 20% over 3-5 days **(only in inpatient care)**

OR

S: Chlordiazepoxide tablets 20 – 60mg daily in divided doses and taper over month

For severe agitation and restlessness

B: Haloperidol 5 mg I.M Repeat after 4–8 hours as required to a maximum of 20 mg. Once patient has responded and is able to take oral haloperidol: 1.5–3 mg 12 hourly

S: Naltrexone 50mg daily decreases the craving for alcohol

b) Delirium Tremens (DT)

It is an acute episode of delirium that is usually caused by withdrawal from alcohol. Although the typical delirium occurs 2–3 days following cessation of prolonged alcohol intake, reaching a peak at around 5 days, some withdrawal symptoms such as tremor may start within 12 hours.

Diagnosis

- Predominantly visual hallucinations
- Disorientation
- Agitation
- Tachycardia
- Hypertension
- A low-grade fever may be present
- Withdrawal tonic-clonic seizures may occur between 24 and 48 hours following cessation of alcohol intake

Note: It is important to consider alternative causes, when making the diagnosis. This is especially true for cases with an atypical presentation.

General measures

- Secure airway
- Ensure Breathing
- Circulation
Give IV fluid (Dextrose Normal Saline) to prevent hypoglycaemia and hypotension
- Monitor for respiratory depression

Drug treatment

A: Diazepam IV, 10 mg

OR

S: Lorazepam, IM/IV, 2 mg for immediate sedative or hypnotic action.

If no response give a second dose.

Note: Do not administer at a rate over 5 mg/minute

Switch to oral once containment is achieved

OR

S: Chlordiazepoxide 20 -60mg taper over one month

Plus

C: Thiamine IM 100mg daily

OR

C: Vitamin B Complex 1 ampoule in half litre of 5% Dextrose

IV. Dementia

It is a progressive loss of cognitive function usually of insidious onset. Initial presentation may be with mild personality or memory changes, before more pronounced defects become more evident. Patients need to be investigated for treatable (reversible) systemic, neurological and psychiatric illnesses. Transient worsening of condition may be due to metabolic disorders, infections and drug side effects.

General management

- Appropriate care and support, according to level of impairment.

- Ambulatory care is preferred to hospitalization if feasible.
- Family counselling and support.

Treatment

Management is mainly symptomatic. To control the restless patient: give

D: Thioridazine tablets 25 – 50mg two times a day.

AIDS Related Dementia

- It may be treatable with ARVs
- Exclude opportunistic diseases of CNS

Referral

Patients, in whom a treatable underlying condition is suspected, refer for specialized investigations including a CT scan

CHAPTER THIRTEEN

METABOLIC AND ENDOCRINE DISEASE CONDITIONS

1.0 Diabetes Mellitus

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia (persistently higher blood glucose values than the normal range) due to deficiency or diminished effectiveness of insulin. There are three main types of diabetes:

- **Type 1 diabetes (T1DM)**: results from the body's failure to produce insulin and requires the person to inject insulin [*insulin-dependent diabetes mellitus (IDDM)*].
- **Type 2 diabetes (T2DM)**: results from [insulin resistance](#), a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency. [*Formerly referred to as non-insulin-dependent diabetes mellitus (NIDDM)*].
- **Gestational diabetes**: is when a pregnant woman, who has never had diabetes, has a high blood glucose level during pregnancy. It may precede development of T2DM.

Diagnosis

- Main clinical features of diabetes are thirst, polydipsia, polyuria, tiredness, loss of weight, blurring of vision, white marks on clothing, pruritus vulvae, balanitis, paraesthesia or pain in the limbs and recurrent bacterial infection
- Fasting plasma glucose level ≥ 7.0 mmol/L (126 mg/dL)
- [Plasma glucose](#) ≥ 11.1 mmol/L (200 mg/dL) two hours after a 75 g oral glucose load as in a [glucose tolerance test](#)
- Symptoms of hyperglycemia and casual plasma glucose ≥ 11.1 mmol/L (200 mg/dL)
- [Glycosyated hemoglobin](#) (Hb A1C) $\geq 6.5\%$.

Non pharmacological Management

- Healthy lifestyles
 - Encourage weight loss if the patient is obese or has body mass index (BMI) of more than 25
 - Reduce intake of fatty foods
 - Avoid the intake of refined sugar
 - Increase in fibers intake $> 15\text{g}/100\text{kcal}$ (traditional African diets are rich in fibers)
 - Increase physical activeness levels e.g. brisk walking 30 minutes at least three times a week
 - Encourage reduction and stoppage of alcohol intake
 - Encourage to stop smoking

Note: Regular ongoing blood glucose level monitoring is recommended

Diabetic Diet

Ideally a dietician should calculate dietary requirements for individual patient. The aim of diet control is to reduce the blood sugar to normal and maintain a constant blood sugar level. 45-

50% of energy intake should be in the form of carbohydrates. Complex carbohydrates are preferable to simple sugars. Carbohydrates and calories should be evenly distributed through the day. Meals must not be missed. An insulin dependent diabetic may have snack between meals. Sugar and sugar-containing food/drinks should be avoided. It is only recommended when a patient feels faint, or ill and cannot eat normally. It is also recommended that, for diabetics a snack should be taken before and after playing sport.

Treatment with Oral Hypoglycemic

If dietary control on its own fails or the blood glucose levels are persistently high initiate

C: Glibenclamide 2.5- 15mg (O) *once daily* for non obese patients

OR

C: Metformin 500-2000mg (O) *in 2-3 divided doses with or after meals* for obese patients.

Review the blood glucose at diabetic clinic and adjust medicines as needed until blood glucose is controlled.

Alternatively

C: Chlorpropamide 125-500mg (O) 1-2 divided doses

OR

D: Gliclazide 40-320mg (O) in 2 divided doses

OR

D: Glipizide 2.5 – 5mg (O) daily shortly before breakfast or lunch, adjust according to response; maximum 20mg daily; up to 15mg may be given as a single dose).

Note

- The lower dosage are appropriate when initiating treatment in elderly patients with uncertain meals schedules, or in patients with mild hyperglycemia
- Activity of sulphonylurea is prolonged in both hepatic and renal failure
- Sulphonylurea are best taken 15 to 30 minutes before meals
- Chlorpropamide should not used in elderly since it has long half life

Treatment with Insulin Injection

Treatment with insulin injection is indicated in Type I Diabetes Mellitus or in uncontrolled Type II Diabetes Mellitus, Hyperglycemic emergencies, Pancreatitis, Pregnancy and trauma or Surgery.

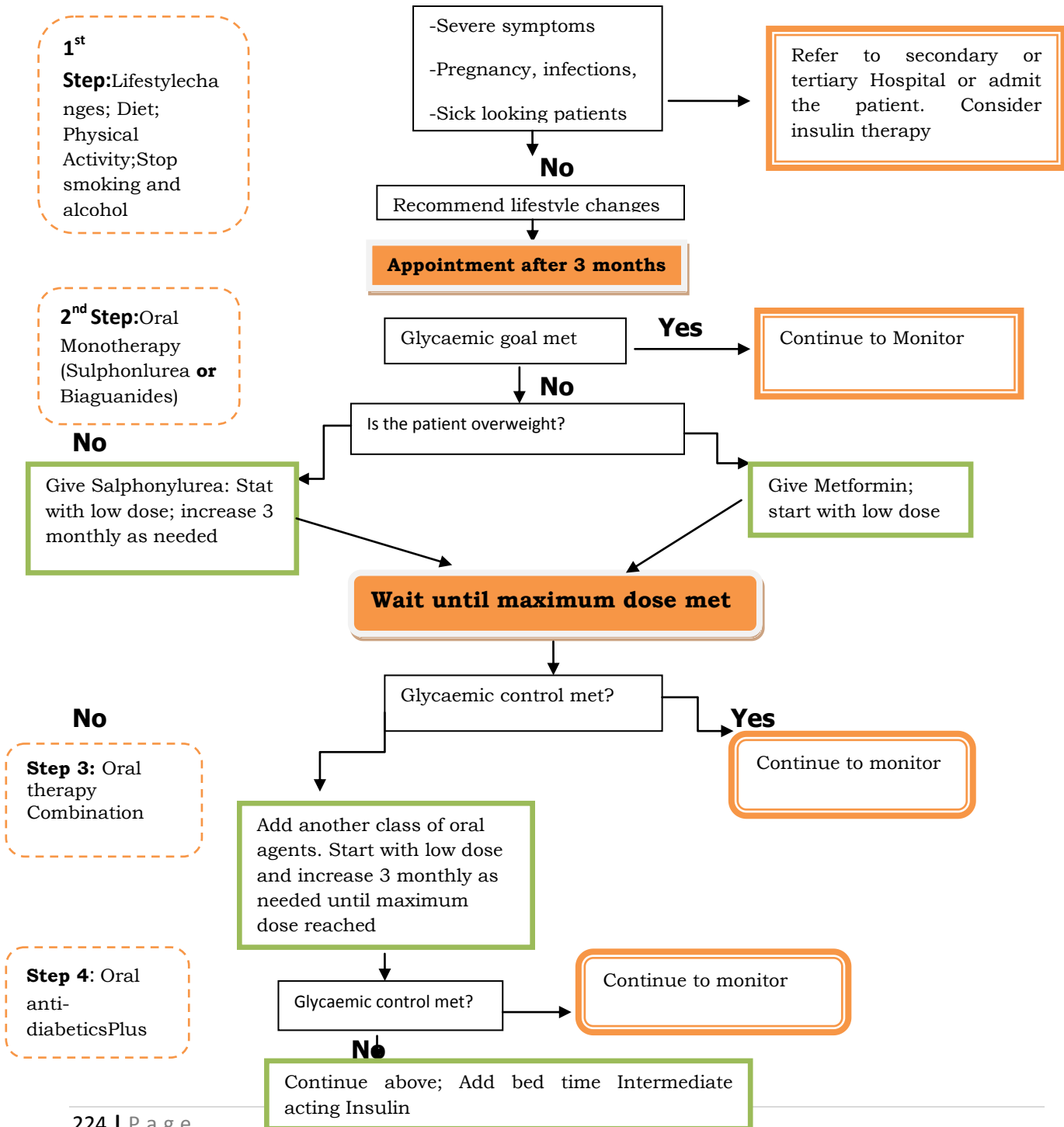
For T1DM use insulin such as ultra short, short, intermediate, long acting and mixed insulin. The dose of insulin is 0.5 to 0.7Unit/kg/day as initial dose; adjust accordingly (Increase or decrease) depending on the response

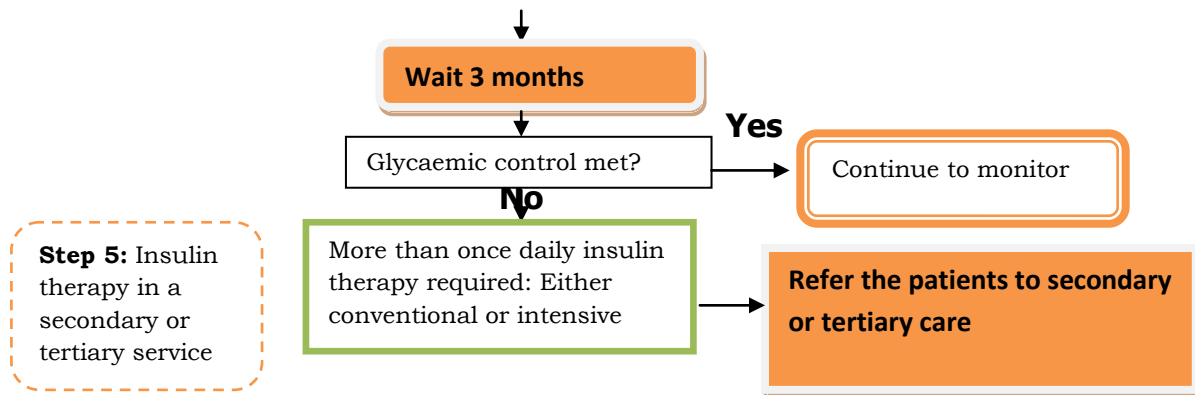
For the poorly controlled T2DM, use 0.5unit/kg/day as initial dose; adjust accordingly (Increase or decrease) depending on the response

Note

- One unit of insulin covers 10-15 grams of carbohydrates
- It is recommended that short acting insulin should be withdrawn first then intermediate insulin
- Insulin injection is given immediately after loading the syringe

MANAGEMENT OF TYPE 2 DIABETES MELLITUS FLOW CHART





2.0 HYPOGLYCAEMIA

Hypoglycaemia is an acute metabolic complication of diabetes

Symptoms: Hunger, sweating, anxiety, palpitation, headache, confusion, convulsions, weakness and coma

Commonest causes;

- Doing more exercise than normal
- Omission or delay of snacks or main meal
- Insulin overdose
- Eating insufficient carbohydrate
- Overuse of alcohol
- Overdosage of sulphonylurea

Test: Blood sugar < 3.0 mmol/L

Management

For conscious patients

Quickly take a glass of a sugary rich drink or eat one table spoon of sugar or honey and have a meal. If symptoms persist after 5 minutes repeat the above.

For unconscious patient, give:

- IV 50% glucose bolus (40 – 50mls) or 100 – 150 mls of 20% dextrose followed by 8 – 10 % glucose if necessary
- If Glucagon injection available administered 1mg IM or SC
- On recovery give long acting carbohydrate snack
- Prolonged IV dextrose infusion (5 – 10% for 12 - 24 hrs) may be necessary if hypoglycaemia is a result of long acting sulphonylureas/ long and intermediate acting insulin or alcohol
- If IV access is impossible, consider nasogastric tube or rectal glucose or if available glucagon 1mg IM
- On recovery, attempt if you can identify the cause of hypoglycaemia and correct it
- Assess the type of insulin used, injection site (Lipohypertrophy can alter the rate of absorption) and injection techniques

- Inquire into correct and inappropriate eating habits, exercise and alcohol consumption
- Review of other drugs therapy and renal function

Table 1: Organization on Diabetic Care

Initial visit	3 Month visit	Annual visit
History and diagnosis Physical examination <ul style="list-style-type: none"> • Height and weight • Waist/Hip circumference • Blood pressure • Detailed foot examination • Tooth inspection • Eye examination <ul style="list-style-type: none"> ➢ Visual acuity+ ➢ Fundoscopy • Biochemistry <ul style="list-style-type: none"> ➢ Blood sugar ➢ Glycosylated Haemoglobin (HbA1C) ➢ Lipid profil (TC,HDC,LDLC,TG) ➢ Creatinine Sodium, Potasium ➢ Urine: glucose, ketones, protein • Education • Nutritional advice • Medication if needed 	<ul style="list-style-type: none"> • Relevant history • Weight • Blood pressure • Foot inspection • Biochemistry <ul style="list-style-type: none"> ○ Blood Glucose ○ HbA1C levels • Urine protein • Education advice • Nutrition advice • Review therapy 	History and examination - as at initial visit Biochemistry as at initial visit
TC=Total cholesterol, HDLC=high density lipoprotein, LDLC= low density lipoprotein, TG=Tryglycerides		

3.0 DIABETIC KETOACIDOSIS

It is an acute metabolic complication of diabetes mellitus may present with a decreased level of consciousness due to hyperglycaemia

Symptoms

- Nausea/Vomiting
- Thirst/polyuria
- Altered mental fuction
- Abdominal pain
- Shortness of breathing
- Dehydartion
- Drowness, Confusion and coma

- Acetone/fruity smelling breath
- Fever
- Lethargy/Obtundation/Cerebral Oedema/possible **COMA**

!!CAUTION!! Diabetic Ketoacidosis (DKA) is a medical emergency. All patients should be admitted in Intensive care unit (ICU), kept under care of *registrar or consultant*.

Investigation: Timely diagnosis is crucial

- Check blood glucose
- Urine for ketones
- Arterial blood pressure
- Urea, creatinine, and electrolyte
- Use DKA chart to guide treatment and monitor the patient

Management

- Admit to ICU
- IV line
- Insert Naso gastric tube for feeding
- Fluid and electrolytes replacement
 - A:** 1 Litre of NS + 2g KCL (when available) hourly
OR
 - A:** 1 Litre of Ringer’s Solution (when KCL is not available)
 - When blood glucose falls to 14 mmol/L or bellow START 5% Dextrose 500mls 4hrly (1000mls 8 hourly)
 - Isotonic dextrose saline may be used in place of dextrose 5%
 - If a patient still dehydrated Continue Normal saline or Ringer’s solution as well.
- Insulin Therapy
 - C:** Soluble insulin 8 Units IM and 8 Units IV at a time. Then give 8 Units IM soluble insulin bolus hourly
 - When blood glucose falls to 14 mmol/L or bellow give soluble Insulin 4Units S.C. 4 hourly **OR** IM 2 hourly and continue until the patient is able to eat again then change to B.D. or T.I.D Insulin
- If blood glucose is fluctuating widely, then use the following guide:

Table 2: Treatment of Diabetic Ketoacidosis in Case Of Blood Glucose Fluctuations

Blood glucose		Insulin 4 hourly S.C. OR 2 hourly I.M	4hourly Dextrose 5%
mmol/L	Mg/dl		
>14.0	>250	12	500ml
7.2 – 14.0	130 – 250	8	500ml
2.5 – 7.2	45 – 350	4	500ml
<25	<45	4	100ml

Acidosis correction

- With severe acidosis NaHCO₃ 50mmol should be given under Doctor’s instruction

Monitoring

- Assess CVS for volume overload (Input output chart, oedema (lungs, peripheral))
- Check blood glucose 2hrly if using I.M. route or 4 hrly if S.C. route.

4.0 NON-KETOTIC HYPEROSMOLAR STATE (NKHS)

Most common elderly in T2DM

Symptoms

- Polyuria,
- Ortostatic hypotension
- Altered mental state Lethargy, obtundation, seizures, possible coma
- Weight loss
- Diminished oral intake of fluids
- Mental confusion
- Profound dehydration
- Lethargy or comatous
- Tachycardia
- Hypotension
- Differentiated from DKA (No nausea and vomiting, no abdominal pain, and Kussmaul breathing)

Note: Try to identify precipitating factors;

- Poor oral fluid intake
- MI, stroke, sepsis, pneumonia, and other serious infection must be sought
- Drugs: Thiazides diuretic, glucocorticoids, phenytoin

Laboratory investigation and diagnosis:

- Check blood glucose (May be > 55.5mmol/L (1000mg/dl))
- Check electrolytes (K^+ , Na^+ , Cl^-)
- Check Renal function (Urea and Creatinine)
- Check osmolarity (usually >330 mosmol/L)
- *Serum osmolarity = $2(Na^+ + K^+) + glucose + Urea$ (Glucose and Urea in mmol/L)*
(Normal is < 310 as calculated)
- For Hyperosmolar Non Ketotic Coma (HNC/HONC) osmolarity is usually over 330mosmol/L

Note: A patient may be acidotic due to lactic acidosis or shock/sepsis

- In this case principle management as in case of DKA
 - IV fluids should be replaced as half-normal saline (0.45%) if hypernatremia,
 - Normal saline if serum sodium is normal
- There is a frequent intercurrent illness usually sepsis, CVA, or cardiac and these must be diagnosed and treated. PROPHYLACTIC HEPARIN MAY BE USED (Monitor bleeding indices-PT, PTT, platelets count).

5.0 DIABETES AND OTHER CARDIOVASCULAR DISEASES

Diabetic patients are 2 – 4 times likely to develop cardiovascular disease than people without, due to two major processes: Atherosclerosis and hypertension. The clinical spectrum of cardiovascular diseases is:

- Coronary heart disease:
 - Angina (which may be silent)
 - Acute coronary artery syndrome
 - Congestive cardiac failure
 - Sudden death
- Cerebral vascular accident;
 - Stroke
 - Transient ischaemic Attacks
 - Dementia
- Peripheral vascular diseases
 - Intermittent claudication
 - Foot ulcer
 - Gangrene

Assessment

- Do annual assessment
- Refer to secondary and tertiary health institution
- Evaluation will include; ECG, Chest X-Ray, if with symptoms/signs of heart failure need echocardiogram, stress test, coronary angiography, and carotid Doppler in case of cerebral vascular diseases
- Peripheral vascular disease evaluation include Doppler and angiography of lower limbs.

Treatment

- Initiate aspirin

Use of Aspirin in Type 2 DM reduces risk of cardiovascular events. It is indicated for secondary prevention for coronary and cerebrovascular diseases; primary prevention for people with Type 2 DM over the age of 40 years having: Family history of ischaemic heart disease (IHD), cigarette smoking, obesity, proteinuria and dyslipidemia

C: Soluble Aspirin 75 – 150 mg

Note: Aspirin is contraindication in peptic/duodenal ulcer, dyspepsia, hurt burn. Malignant hypertension, haemorrhagic stroke. Consider beta-blockers, ACE inhibitor, angiotensin receptor blocker (ARBs) and tight glycemic control.

Caution: The patient should be monitored in the cardiac clinic

6.0 GESTATIONAL DIABETES MELLITUS

Gestational Diabetes Mellitus (GDM) is any degree of glucose intolerance first recognized in pregnancy.

Screening

Perform screening for GDM between 24 – 28 weeks of gestation. The most risk women are those with: BMI > 25 kg/M²; Previous history of GDM; Glycosuria; Previous big baby; Poor obstetric history; Family history of DM; Known impaired Glucose Tolerance/Impaired fasting Glucose and Grand multipara

Management

Target Glycaemia; Preprandial blood glucose 3.5 – 5.5mmol/L and postprandial blood glucose 5 – 7.5mmol/L

A combined health-care team (Obstetrician, diabetologist or internist, diabetes educator, pediatrician/neonatologist) is required.

7.0 SURGERY AND THE DIABETIC PATIENT

Correct pre-operative management depends on:

- Type of surgery: Major or minor
- Type 1 or Type 2 DM
- Recent diabetic control

Note:

- Diabetic patient should be first on the operation list
- Minor surgery: does not involve general anesthesia or starvation
- Major Surgery: Involve a general anesthesia and therefore a period of fasting.

Type 1 DM and Surgery

- Once snack is missed it is better to start an I.V. regimen irrespective of the size of the procedure
- Maintain interrupted insulin administration (hourly) to prevent DKA
- Administer 5% dextrose in maintenance IV fluids to avoid lipolysis and ketoacidosis in patients with restricted oral intake.
- Blood glucose monitoring 1 -4 hourly (Aim reading 6 – 10 mmol/L)
- Measure electrolyte and urine for ketones hourly
- Patients using conventional therapy may be given a dose of intermediate-acting Insulin(at least half of the usual dose)
- Hyperglycemia may be managed with regular insulin, given 4 -6 hrs and continued until oral intake is resumed. Mixed insulin may be given
- Patients receiving MDIT (Multiple Daily Insulin Therapy) should receive preoperative basal insulin dose without interruption in the perioperative period. When oral intake is restricted, regular Insulin may be given 4-6hrs to control hyperglycemia. When a diet is tolerated, the MDIT regimen should be resumed
- Post operatively –give IV 1 Litre of 5 – 10% dextrose + 20mlKCl + 2/3 of total daily dose of Insulin over 8Hrs and repeat until able to take orally
- Check Na⁺ levels (Caution-Hyponatraemia)

Table 3: Insulin Dosage after Surgery

Blood glucose	Actrapid (short acting Insulin)
0 – 6	0
6 – 8	2
8 – 10	4
10 – 14	5
14 – 18	6
18 – 20	8
>20	10 +

NOTE: Insulin Infusion Pump

An intravenous infusion pump is essential in the management of DKA, major illness or major surgery in the patient with DM. The advantages are: 1. Ability to tightly control the blood glucose levels. 2. Separation of Insulin and fluid regimen. Use 50Units of short acting insulin in 50mls of normal saline (0.9%). (Thus unit/hr=ml/hr)

Table 4:

Blood glucose(mmol/L)	Short acting (Actrapid)Units S.C
0 -4	0.5
4-6	1.0
6-8	1.5
8-10	2.0
10-12	4.0
12-14	5.0
14-16	6.0
16-18	8.0
18-20	10.0 or more

Check blood glucose hourly initially and 2hourly when stable. Continue I.V regimen until patient is taking normal diet postoperatively. Calculate s.c. dose from i.v insulin requirement in previous 24 hours. The first dose of s.c. insulin is given thirty minutes (unless a short-acting analogue) prior to stopping the I.V. insulin infusion. The patient then eats a normal diet.

T2DM and Surgery*Preoperatively*

Delay surgery if possible if glycaemic control is poor;

- HbA1C >9%
- FBG >10mmol/L
- RBG >13mmol/L

Note: Optimize the glycaemic control if Surgery is elective. Screen for complication that may affect surgical risk: Nephropathy, cardiac disease, proliferative retinopathy. Inform surgical team of the complication.

If on diet and/or oral antidiabetics and well controlled and surgery is minor:

- Omit dose on the mornig of surgery
- Resume therapy when eating normally

If on insulin therapy or poor glycaemic control or major surgery;

- Use continuous IV insulin infusion
- Monitor blood glucose before, during and after surgery.
- Target blood glucose levels 6 – 10mmol/L

For Major surgery (glucose-insulin Potassium Regimen)

- Add 16 Units of short acting Insilin and 10mmol/L KCl to 500mls of 10% dextrose.
 - Infuse at 80ml/hr IV using volumetric pump (infusion pump)
- If obese or Initial blood glucose is high consider higher dose (20Units)
- If very thin or usual insulin dose is very low consider lower dose (12Units)
- Monitor blood glucose levels hourly
 - If blood glucose is low or falling reduce dose by 4 Units
 - If blood glucose is high or raising increase dose by 4 Units
- Continue the infusion until 60minutes after the first meal.
- Resume usual therapy after first meal
- Check daily electrolytes (**Caution-** Dilution hyponatremia)

8.0 DIABETES IN CHILDREN

Is a chronic lifelong disease caused by insufficient or no insulin production causing raised blood glucose concentration

Classification

- Type 1-4
 - The common type in pediatrics is Type 1 Diabetes Mellitus with few children having Type 2 Diabetes mellitus

Diagnostic criteria

- Polyuria (may cause nocturnal enuresis-for children who had the controlled bladder child)
- Polydipsia
- Polyphagia
- Weight loss
- Weakness- easy fatigability
- Fasting blood glucose > 6.5mmol/l
- Two hour post prandial blood glucose> 11.1mmol/l

Differential Diagnosis

- Any child presenting with impaired consciousness and acidosis
- Pneumonia-Tachypnoea and Hyperventilation
- Acute abdomen-Abdominal pain and tenderness
- Secondary Nocturnal enuresis

Investigation

- Blood Glucose concentration
- Venous blood gas measurements if in Diabetes Keto Acidosis (DKA),
- Serum electrolytes, urea and creatinine concentration
- FBP (leucocytosis in DKA)
- For children with signs of infection (Blood and urine cultures, radiography)

Treatment

- Hospitalization
 - Those in DKA, with electrolyte imbalance and dehydration (Below)
- Outpatient
 - C:** Insulin 0.5IU/kg/day s/c; 60% of the dose given during the day and 40% at night as baseline. Then dose adjusted according to blood glucose control, stage of growth-(e.g. Puberty). Regular (Soluble) insulin given pre-meals for the main meals (Breakfast, Lunch and supper), long acting insulin at bedtime

Table 5: Insulin Regimens

Regimen 1		
Breakfast	Intermediate/long acting(2/3) + Short acting (1/3)	2/3 of daily dose
Supper	Intermediate/Long acting (2/3) + Short acting (1/3)	1/3 of daily dose

Regimen 2		
Breakfast	Intermediate/long acting + Short acting	2/3 of total daily dose
Supper	Short acting	1/3 of total daily dose
Bedtime	Intermediate/long actin + short acting	

Regimen 3		
Breakfast	Short acting	20% of daily dose
Lunch	Short acting	20% of daily dose
Supper	Short acting	20% of daily dose
Bedtime	Intermediate/long acting	40% of daily dose

Table 6: Insulin adjustment (how to adjust insulin)

	Blood glucose-High/Low	Insulin dose to adjust-↑/↓
Twice daily injection regimen	Before breakfast or overnight Before lunch Before dinner Before bed	Evening intermediate-acting Morning short acting Morning intermediate Evening short acting
Three-times daily injection regimen	Before breakfast or overnight Before lunch Before dinner	Evening intermediate- acting Morning short-acting Morning intermediate-acting Evening short-acting

	Before bed	
Basal-bolus (multiple injection) regimen	Before breakfast or overnight Before Lunch Before Dinner Before Bed	Evening intermediate acting Morning short acting Lunchtime short acting Evening short acting

- Give education on
 - What is diabetes?
 - Insulin
 - Diet
 - Complications : hypoglycemia, Cerebral oedema

Complications

- Acute- DKA, hyperglycemia and Hypoglycaemia
- Chronic- Retinopathy Nephropathy, Neuropathy, cataract
 - Growth failure, delayed puberty

10.0 DIABETES KETOACIDOSIS (DKA)

Definition

- A state of coma or pre-coma with severe metabolic de-compensation as the result of relative or absolute insulin deficiency combined with counter regulatory/stress hormone excess.

Categories of DKA

- Mild pH <7.3, bicornated <15mmol/l
- Moderate pH < 7.2, bicarbonate <10mmol/L
- Severe pH < 7.1, bicarbonate < 5mmol/l

Causes of DKA

- Onset of Diabetes
- Missed insulin doses
- Growth spurt (Puberty)
- Increased needs of insulin (with stress or illness)

Risk factors

- At initial presentation
- Young age <5 years
- Low social economic status
- In established Diabetes
- Higher glycosylated haemoglobin (HbA1c)
- Adolescents in particular , females
- Psychiatric disorders
- Long duration of Diabetes

Precipitating factors

- Poor metabolic control and frequently missed insulin doses
- Infections

— Medication

Diagnostic criteria

Clinical

- Hyperglycaemia
- Dehydration and thirst
- Precoma or coma

Investigation

- Acidosis
- Ketonemia
- Ketonuria
- Electrolyte disturbances (Hyponatraemia, Hypokalaemia)

Treatment of DKA

Goals of treatment

- Correct dehydration
- Restore blood glucose to near normal
- Correct acidosis and reverse ketosis
- Avoid complications of treatment
- Identify and treat any underlying event

Table 7: Management of DKA

Item	Requirement	Action
Assessment	History	History and physical examination Polyuria, polydipsia despite dehydration
	Physical Examination- Laboratory	Assume 10% dehydration Hyperventilation-Acidosis Level of consciousness- GCS/ Blantyre Determine weight if possible/use recent weight Determine -glucose and urine ketone at bed side Venous/arterial blood gases RBG, Urea and electrolytes, Haemoglobin (Hb) and WBCs ± HbA1c Appropriate Microbial culture- urine, throat swab, skin and blood, chest X-ray.
Resuscitation	Ensure appropriate Airway Breathing Circulation-	-Check airway Insert NGT, if there is vomiting to avoid aspiration -Give 100% O ₂ by mask, if in shock Give O ₂ Insert IV line In shock -Give NS /RL 10mls/kg over 30min, repeat boluses of 10ml/kg to max of 30mls/kg
Fluid replacement Types of fluid to use	Rehydrate child with normal saline Reassess Hydration hourly resuscitation (1 st -1-2hrs) When RBG ≤ 15mmol/l	<u>10% Deficity + Maintenance for 48hrs- boluses =</u> 48hr Mls/Hr Normal saline 0.45% Saline, or 0.9% saline with 5% dextrose

Oral fluids	In severe dehydration/shock When there is substantial clinical improvement , no vomiting	Ringer's lactate or Ringer's acetate 10% dextrose mixed with sodium chloride 70mmol/l sips of cold water or ice to suck ORS, fruit juices Oral fluid volume should be subtracted from IV fluids calculations
Insulin Therapy	Start insulin when the patients circulation has been restored (1-2hrs after rehydration) Type of insulin When to change to subcutaneous insulin How to change to subcutaneous insulin	Infusion 0.1 I.U/kg/hr When pump not available- separate low dose insulin infusion should be used [Soluble Insulin 50 units in Normal Saline 500ml (ie 1 unit Insulin per 10ml Saline)] may be given at a rate of 0.1 units/kg/hour (this is in addition to the Saline infusion). When insulin infusion methods are not available - use 0.1iu IM 2 hourly Regular/ Soluble insulin Clinical improvement has occurred (mild acidosis / ketosis may still be present) When oral fluids are tolerated Do not stop the IV insulin infusion until 60 minutes after the first subcutaneous injection of short or rapid acting insulin
Potassium replacement	Ideally, start replacement when the serum potassium is known or urine output has been documented Where Possible-ECG does not show elevated T wave and the insulin is about to start	Add Potassium chloride 40 mmol in each litre of Saline infusion
Correction of acidosis	No bicarbonate	Fluid and insulin usually will correct the acidosis
Treat infection		Give antibiotics accordingly
Monitoring	Vital signs Record fluid intake, insulin therapy and urine output	Half hourly- HR, T, RR, Level of consciousness, Hourly- RBG, rehydration, urine ketones ,BP, fluid input and output 4 Hourly- Urea, electrolyte once urine ketones are absent consider making transition to subcutaneous insulin

- **Fluid calculation**

- Requirements = DEFICIT + MAINTENANCE

- Calculate DEFICIT = estimated % dehydration x body weight (kg)

- o Assume weight loss is 10% for all children in DKA

- Calculate MAINTENANCE
- Then add **DEFICIT** to **48 HOURS MAINTENANCE** and replace this volume evenly over 48 hours as **Normal Saline 0.9% initially (OR a balanced salt solution such as Ringer’s lactate or acetate).**

Table 8: Fluid Calculations

Approximate Age (years)	Weight (kg)	MAINTENANCE FLUID (ml/kg/24hrs)
< 1	3 – 9	80
1 – 5	10 – 19	70
6 – 9	20 – 29	60
10 – 14	30 – 50	50
> 15	> 50	30

Example: A 6 year old 20kg boy who is 10% dehydrated has already been given 20ml/kg to improve his circulation now requires –

- $10/100 (10\%) \times 20\text{kg} = 2\text{kg}$
- *ie. 1kg=1000mls: therefore 2kg= 2000mls DEFICIT*
- $60\text{ml} \times 20\text{kg} = 1200\text{ml}$ as MAINTENANCE each 24 hours
- *Deficit + Maintenance*

∴ 2000+2400= 4400ml over 48 hours = 91ml/hour

NB: This calculation will usually cover ongoing urinary losses which in most cases do not need additional replacement but excessive continuing fluid losses such as severe vomiting might need replacing if the severity of dehydration is not improving.

Insulin Dilutions

A solution of Soluble Insulin 1 unit / ml made up in Normal Saline. Dilute 50 units soluble (regular) insulin in 50ml normal saline-1unit=1ml)

When syringe pumps are not available a separate low dose insulin infusion [Soluble Insulin 50

units in Normal Saline 500ml (ie 1 unit Insulin per 10ml Saline)] may be given at a rate of 0.1

units/kg/hour (this is in addition to the Saline infusion).

[The bag or bottle should be changed every 24 hours to avoid inactivation of insulin]

- If BG rises again above 15mmol/l., increase the insulin infusion by 25%
- If BG falls to < 8 mmol/l or falls too rapidly, change the infusion to Glucose 10% (or more if necessary) and add normal saline 75 mmol per litre.

Monitoring

Note: After resuscitation the rate of fall of BG should not be more than 4 –5 mmol / hour

Do not stop insulin infusion or decrease below 0.05 units/kg/hour because a continuous supply of both insulin and glucose substrate is needed to promote anabolism and reduce ketosis.

Note: To prevent rebound hyperglycemia do not stop the IV insulin infusion until 60 minutes after the first subcutaneous injection of short or rapid acting insulin

Complications of DKA and its management

Cerebral oedema

- Approximately 0.4-1% of children with DKA develop cerebral oedema with a high mortality/morbidity
- Cerebral oedema most commonly occurs in the first 24 hours after starting rehydration when the general condition of the child might seem to be improving. Vigilant observations throughout the 24 hours must not diminish
- In many cases warning signs/symptoms occur which should prompt the emergency administration of Mannitol

Warning signs/symptoms of cerebral edema

- Headache
- Slow heart rate
- Change in neurological status (restlessness, irritability, increased drowsiness, incontinence, specific neurological signs (eg. cranial nerve palsies)
- Rising BP, decreased O₂ saturation

Action

- Exclude hypoglycemia
 - C:** Mannitol 1 g/kg IV over 20 minutes. Repeat if there is no response in 30 – 60 minutes
- Halve rehydration infusion rate until clinical state has improved
- Nurse with child's head elevated
- Alert anesthetic and senior pediatric staff (if assisted ventilation is required maintain pCO₂ above 3.5 kPa)
- Consider continuation of Mannitol infusion 0.25 g/kg/hour to prevent rebound increase in intracranial pressure (or repeat bolus doses every 4-6 hours)
- Intracranial events other than edema may occur eg. haemorrhage, thrombosis, infarction
- If Mannitol not available or no response to Mannitol try hypertonic saline (3%), 5-10 ml/kg over 30 minutes
- **Hypoglycemia** – avoid by careful monitoring and adjustment of glucose/insulin infusion rates.
- **Hypokalaemia**
 - Avoid by infusing sufficient KCl and monitoring of Potassium levels.

- When oral intake is being established include fluids/foods rich in potassium (e.g. milk, fruits such as banana).
- **Aspiration pneumonia –**
 - Avoid by nasogastric tube in vomiting child with impaired consciousness
- **Other associations with DKA**
 - These require specific management e.g. continuing abdominal pain (due to liver swelling/gastritis/bladder retention but beware appendicitis), pneumothorax ± pneumomediastinum, interstitial pulmonary edema, unusual infections (eg TB, fungal infections), hyperosmolar hyperglycaemic non - ketotic coma, ketosis in type 2 diabetes.

Monitoring and follow up

- Inpatient
 - Vital signs-neurological deterioration, Temperature, Respiratory rate
Blood glucose 2hourly, urine ketones 4 hourly
- Outpatient
 - Blood glucose-personal glucometers,
 - Hyperglycemia-shown by frequent micturation at night ,
 - Urine glucose,
 - Glycaemic control –glycosylated Haemoglobin-(Ranges)(HbA1c),
 - Growth (Height and weight) every visit ,
 - Complications,
 - Hypoglycaemia-management ,
 - Continuous diabetes education-every visit

Surgery

Minor surgery(duration < 3h.

- Insulin: in the morning intermediate-acting insulin, 1/2 to 2/3 of total daily dose if blood glucose is above 20 mmol/l supply with a small dose short-acting insulin in the evening give intermediate-acting insulin, 1/3 of daily dose.
- Fluid: glucose 5% intravenously, volume according to age.
- Blood glucose monitoring: every 1–2 hours values between 10–14 mmol/l

Major surgery> 3hours.

- Insulin and fluid: infusion solution containing 5% glucose and 20 mmol/l sodium chloride (maintenance volume)
- Insulin infusion 0.05 IU/kg/hour.
- Blood glucose monitoring: every 1–2 hours ;values between 6–14 mmol/l,if < 5 mmol/l reduce infusion rate, continue infusion therapy until food intake is re-established

Table:

Parameter	Optimal	Acceptable	Additional action suggested
Capillary Blood Glucose			
Fasting (mmol)	4-6	6-8	>8
2-Hours post prandial	4-8	8-10	>10
Glycosylated Haemoglobin (%)	< 7	7-8	>8

11.0 HYPOTHYROIDISM

It is a clinical state that results from a decreased production or secretion of thyroid hormone.

Primary Causes:

- Iodine deficiency
- Congenital
- Drugs; Iodine excess (contrasts media containing iodine), lithium, antithyroid drugs, p-aminosalicylic acid, interferon alfa and other cytokines, aminoglutethimide.
- Autoimmune disease (Hashimoto's thyroiditis), atrophic thyroiditis
- Infiltrative disorders: amyloidosis, sarcoidosis, hemochromatosis, scleroderma,
- Iatrogenic (unknown) cause Iodine 131 treatment, total thyroidectomy, radiation treatment

Secondary Causes:

- Hypopituitarism: tumour, pituitary surgery, Sheehan's syndrome, trauma, genetic pituitary hormones deficiencies.
- Autoimmune hypothyroidism: May be associated with goitre (Hashimoto's or goitrous thyroiditis) or at minimal residual thyroid tissue (Atrophic thyroiditis)

Symptoms/Signs

Tiredness, Weakness, dry coarse skin, feeling cold, difficult in concentration and poor memory, constipation, weight gain with poor appetite, dyspnoea and hoarseness of voice, menorrhagea (late oligomenorrhoea or amenorrhoea), paresthesias, impaired hearing. Others are cool peripheral extremities, puffy face, hands and feet (Myxedema), diffused alopecia (hair loss), bradycardia, peripheral odema, delayed tendon reflex relaxation, carpal tunnel syndrome and serous cavity effusions.

Treatment

Target is to maintain normal TSH levels

D: Levothyroxine 1.5µg/kg. Maximum dose 100 -150µg daily.

In case of Hypothyroidism after Treatment of Grave disease:

D: Levothyroxine 75µg to 125µg/day

Note: Measure TSH after 2 months of Levothyroxine or if dose change

- Symptoms relief at 3 to 6 months after normal TSH
- Follow up TSH at 2nd and 3rd year once TSH levels are normal
- For subclinical hypothyroidism: (Long treatment is avoided, Low dose of 25 to 50 µg/day with goal to normalizing TSH)
- Women in reproductive period should be euthyroid before conceiving, as the hypothyroidism is associated with neural development. Dose may be doubled during pregnancy and returned to normal dose after delivery.

12.0 THYROTOXICOSIS

It is a state of excess thyroid hormone. Diseases of the thyroid gland are manifested by qualitative or quantitative alterations in hormone secretion or enlargement of the thyroid gland or both. Enlargement of the thyroid gland may result in normal increased, or decreased hormone secretion.

Treatment

- **Iodised salt** may not provide sufficient iodine and should therefore not be prescribed alone
- **Lugol's solution** is too concentrated for daily use, and should be diluted by a factor of 30 to give 4.2 mg/ml (**Schiller's iodine**).

Treatment Age less than 45 years

- **First choice**
B: Schiller's iodine 2 drops (460 micrograms) once daily for one year.
Response may be obtained within 6 months
- **Second choice**
B: Lugol's solution 3 drops (21mg) once each month for up to one year.

Post thyroidectomy

- Iodine should be given daily indefinitely to prevent recurrence, following dosing schedule given above
- Physiological doses of iodine can be given even in pregnancy. It is actually necessary to provide the therapy to avoid iodine deficiency to the foetus
- Patients should continue taking iodized salt indefinitely (Ref. National Policy on Nutrition) after the completion of treatment or begin giving 1 drop (7mg) of Lugol's sol per month.
- All salts in Tanzania should be iodized (Government law)

13.0 HYPERTHYROIDISM

Hyperthyroidism (thyrotoxicosis) results from an excess of circulating thyroxine or liothyronine or both. It is usually due to diffuse hyperplasia and hypertrophy of the thyroid gland (Graves' disease). Hyperthyroidism is characterized by an increased metabolic rate, which causes weight loss, increased appetite, fatigue, emotional disturbances, heat intolerance, sweating, muscle weakness and diarrhea.

Treatment

Graves' disease:

C: Carbimazole 40mg (O) once daily for 3 weeks then 20mg daily for 3 weeks.
Maintenance dose 5mg for up to one year

Toxic Nodular Goitre

- Can be treated with antithyroid drugs and surgery or radio-iodine
C: Carbimazole 40mg (O) once daily for 3 weeks then 20mg daily for 3 weeks.
Maintenance dose 5mg for up to one year

CAUTION: Carbimazole may induce bone marrow suppression. Patients should be told to report any type of infection. The drug should be stopped immediately if neutropenic. Check iodine function at 5-6 weeks.

CHAPTER FOURTEEN

HAEMATOLOGICAL DISEASE CONDITIONS

1.0 ANAEMIAS DUE TO RED CELL DISORDERS (NUTRITIONAL DISORDERS)

1.1 Iron deficiency anaemia

Clinical features: Clinical presentation in patient with iron deficiency anaemia includes fatigue, palpitation, dizziness, koilonychia and pica. Iron deficiency is mainly due to blood loss secondary to haemorrhage, malabsorption and hookworm infections.

Diagnostic criteria include Low MCV and MCH with microcytic/ hypochromic red cell.

Treatment guidelines

General

- Treat the cause of blood loss, for example upper GI bleeding due to peptic ulcer and lower GI bleeding secondary to hookworm infections and malignancy.
- Oral Iron supplementation
- Blood transfusion is only indicated if it is life threatening.

Iron deficiency anaemia

A: Ferrous sulphate 200 mg (O) every 8 hours
Children 5 mg/kg body weight every 8 hours.

Continue for 3 months after the normal haemoglobin has been achieved.

1.2 Megaloblastic anemia

Clinical features of megaloblastic anaemia includes: fatigue, palpitation, numbness of lower limbs, glossitis, Progressive neuropathy affecting peripheral sensory nerves and posterior and lateral column and mildly jaundiced (Lemon yellow tint)

- Megaloblastic anaemias is due to inadequate intake, malabsorption due to Gastric causes: Pernicious anaemia, congenital lack or abnormality of intrinsic factor, Total or partial gastrectomy
- Treatment of megaloblastic anaemias include supplementation of folic acid and injection Vitamin B12

Folic Acid deficiency

A: Folic acid 5 mg (O) once daily for a least 2 months

Vitamin B 12 deficiency anaemia

C: Hydroxocobalamin 1 mg daily parenterally for one week and thereafter 1 mg every 2-3 months for life if it's due to pernicious anaemia.

1.3 Haemolytic anaemias

Haemolytic anaemia are anaemias which result from an increase in the rate of red cell destruction and this take place intravascular in some pathological disorders

Classification of haemolytic anaemia

I. Acquired haemolytic anaemias:

Immune:

- Autoimmune (Warm antibody type, cold antibody)
- Alloimmune:
 - Haemolytic transfusion reactions
 - Hemolytic Disease of the Newborn
 - Allograft esp marrow transplantation

Red cell fragmentation syndromes:

- Arterial grafts, cardiac valve
- Microangiopathic haemolytic anaemias

Others

March haemoglobinuria

Infections (Malaria, Clostridia)

Chemicals and Physical agents

Paroxysmal nocturnal haemoglobinuria

II. Hereditary haemolytic anaemia

a) Membrane

1. Hereditary spherocytosis
2. Hereditary elliptocytosis

b) Metabolism

1. G6PD deficiency
2. Pyruvate kinase deficiency

c) Haemoglobin

-Abnormal haemoglobin such as HbS, C, Unstable Hb

Clinical features

- The disease may occur at any age and sex
- Patient may present with symptom and features of Anaemia
- Symptoms are usually slow in onset however rapidly developing anaemia can occur
- Splenomegaly is common but no always observed
- Jaundice

Treatment

- i. Treat the underlying cause
- ii. Corticosteroids (prednisolone is the usual first line treatment 1mg/kg/day)
- iii. Splenectomy in those who fail to respond
- iv. Immunosuppressive drugs for the patients who fail to respond to corticosteroids and splenectomy.

S: Cyclophosphamide (60mg/m²) IV daily
OR
S: Azathioprine (80mg/m²) daily
OR
S: Immunoglobulin IgG 400mg/kg (IV) daily for 5 days

- v. Folic acid is given to severe cases
- vi. Blood transfusion if anaemia is severe

- vii. Plasmapheresis

1.4 Sickle Cell Anaemia

Clinical features: Sickle cell disease is a spectrum of disorders resulting from inherited haemoglobin S due to substitution of Valine for glutamic acid. In the homozygous state there may be sickle cell anaemia. Onset of symptoms is usually after 6 months of life. Symptoms may include anaemia, dactylitis, recurrent infections, impaired growth and development.

Crises

Three distinct types of crises develop in patients with sickle cell disease

- Vaso-occlusive or painful crises are more common occurring with a frequency from almost daily to yearly. It is important to distinguish between painful crises and pain caused by another process
- Aplastic crises occurs when erythropoiesis is suppressed
- Sequestration crises occurs in children or occasional in adult with an enlarged spleen due to massive pooling of red cells in the spleen

Treatment Guidelines

Nonspecific measures

A: Folic acid 5mg once daily

Specific measures

S: Hydroxyurea 15mg/kg/day. Maximum dose: 35mg/kg

Management of Complication

- Patients undergoing vascular crises should be kept warm and given adequate hydration and pain control (Inj pethidine 100mg 6hrly, Oral morphine 5mg/kg) and oxygen
- Acute chest syndrome is a life threatening complication and empiric antibiotics should be given.
- Stroke in children are the occurring complication, vigorous therapy is recommended (A regular transfusion program is recommended to reduced haemoglobin S, Exchange transfusion program is recommended)
- Priapism should be treated with exchange transfusion or possible surgical decompression.
- Bed rest, elevation and zinc sulphate dressings are should be used to treat leg ulcers
- A transfusion program or skin grafting can enhance healing

1.5 G6PD deficiency

Clinical features: G6PD is an inherited X-linked recessive genetic disorder. Usually asymptomatic but liable to haemolysis if incriminated drugs or foods are taken (e.g. sulphonamides, fava beans, tabs chloroquine or proguanil).

Treatment Guidelines

- Avoid incriminated agents/foods or drugs
- Transfusion of packed red blood cells in severe anaemia. Give 10ml/kg body weight over a period of 8 hours. Then assess the level of haemoglobin.

1.6 Aplastic anaemia (Bone marrow failure)

Aplastic anaemia is defined as pancytopenia resulting from aplasia of the bone marrow
Pancytopenia – a reduction in the blood count of all the major cell lines

Table 1: Causes of Aplastic anaemia

Primary	Secondary
Congenital (Fanconi and non-fanconi)	Ionozing radiation: Accidental exposure (radiotherapy, radioactive isotopes, nuclear power stations)
Idiopathic acquired	Chemicals: Benzene, DDT, insecticides T lymphocyte mediated autoimmune suppression of haemopoietic stem cell
	Drugs esp chloramphenical Infections esp viral hepatitis (A or non-A) Connective tissue diseases, pregnancy

1.7 Fanconi anaemia

- Autosomal recessive pattern of inheritance and often associated with growth retardation and congenital defect of the skeleton
- Any of 8 gene mutations FANCA through FANCL are associated
- The majority of the patients have mutations of FANCA, C or G
- Marrow hypocellularity and pancytopenia may appear gradually after age 5yrs
- Abnormal skin pigmentation (café-au-lait spots)
- The underlying problem appear to be defective DNA repair

Clinical features

- Fatigue
- Pallor and dyspnoe on exertion
- Bleeding
- Infection as a consequence of cytopenia
- Growth retardation result in short stature especially dysplastic radii and thumbs

- Microcephaly and mental retardation may be a feature
- Hypogonadism

The median survival of untreated severe aplastic anaemia is 3-6 months (~20% survive longer than 1 year)

Treatment

Supportive

- Blood transfusion (irradiated, leucodepleted) when Hb <7
- Platelet transfusion if bleeding (Using single donor)
- Antibiotic esp broad spectrum to prevent infections
- Netropenic measure possible isolation of the patient, use of mask

Immunosuppressive therapy

S:Antithymocyte globulin (ATG) or antilymphocyte globulin (ALG) 15-40mg/kg/daily IV 4-10 days

OR

S:Cyclosporine 3-7mg/kg daily 4-6 month

OR

S:Methylprednisolone 5-10mg/kg for 3 to 14 days

OR

S: Cyclophosphamide 45mg/kg per day for 4 doses

OR

S:Danazol 5mg/kg/day for 6 months

CAUTION: Give supportive therapy and refer patient to tertiary hospital for diagnosis and treatment.

2.0 BLEEDING DISORDERS

2.1 Hereditary bleeding disorders

Hereditary bleeding disorders includes haemophilia A and B, Von Willebrand disease

2.1.1 Haemophilia

Haemophilia is an inherited, X-linked lifelong bleeding disorder which affects males almost exclusively.

Most frequent haemorrhage involves joints or muscles and bleeding patterns differ with age: Infants usually bleed into soft tissues or from the mouth but as the boy grows, characteristic joint bleeding becomes more common.

Haemophilia A (Factor VIII deficiency)

- Is the most common of the hereditary clotting factor deficiencies and are caused by deficiency of factor VIII

- The inheritance is sex linked but up to 33% of patient have no family history and result from spontaneous mutation

Clinical presentation includes: spontaneous joint bleeding without injury, posttrauma prolonged bleeding after injury, spontaneous muscle bleeding, retroperitoneal bleeding, epistaxis and easy bruising. Complication includes arthropathy and disability.

Haemophilia B (Factor IX deficiency)

- Is due to deficiency of clotting factor IX

Precaution and Management of Haemophilia

- Avoid I.M injections and use small gauge needles if necessary
- Avoid use of aspirin, instead use paracetamol
- Inform the patient and parents thoroughly on the problem, and provide means of alerting other medical/pharmaceutical personnel
- Genetic counselling
- For haemarthrosis – AVOID incising or aspiration of the affected joint. Treat by replacing the specific factor e.g factor 8 or 9 concentrate if available or FFP (10ml/kg), joint support and tabs diclofenac for pain.

CLASSIFICATION OF HAEMOPHILIA

Haemophilia is classified as mild, moderate or severe according to the levels of circulating factor VIII or IX and indicates the expected frequency of bleeding.

Table 2: Classification of Hemophilia

Classification	Haemophilia A Factor VIII level	Haemophilia B Factor IX level	Clinical features
Severe	<2% of normal ≤ 0.01 U/ml	≤ 1% of normal ≤ 0.01U/ml	1.Spontaneous haemorrhage 2.Frequent spontaneous haemarthrosis factor is needed several times
Moderate	2-5%of normal 0.01-0.05 U/ml		1Haemorrhage secondary to trauma or surgery 2.Occasional spontaneous haemarthrosis
Mild	5-25%of normal	5-25% of normal	1.Haemorrhage post trauma or surgery 2. Rare spontaneous

Amount of factor VIII and IX is given depending on assessment of severity of bleeding.

Treatment of bleeding episodes

Haemophilia A (Factor VIII deficiency) no inhibitor

- Dose depends on bleeding severity

Minor bleed:

S: Factor VIII 15-25IU/kg.

Major bleed:

S: Factor VIII 40 IU/kg

Expected response: 1IU/kg = 2% rise in factor VIII level

Half life Factor VIII: 8-12 hrs

For serious bleeding factor VIII assay may be required to monitor the response to infusion

CAUTION: If there is no response to appropriate replacement therapy test for inhibitors

Haemophilia B (Factor IX deficiency) no inhibitor

- Dose depends on bleeding severity

Minor bleed:

S: Factor IX 15-20IU/kg

Major bleed:

S: Factor IX 40IU/kg

Expected response: 1IU/kg = 1.5 rise in the factor IX level

Half life Factor IX: 16-24 hrs

OR

S: Fresh frozen plasma (FFP) can be used where factor concentrate is unavailable. Average dose 10-15mls/kg

CAUTION: If there is no response to appropriate replacement therapy tests for inhibitors.

Factor VIII Inhibitor management Options

- Acute Bleeding episodes: - Ice/cold pack – 5 minutes on, 10 min off
- Immobilise joint with a splint

S: Factor VIII at 2-3 times the normal dose

- Low Responder (<5BU): High responder >5-10BU:

S: Activated Prothrombin Complex Concentrate (APCC) 50-100IU/kg every 12-24hrs

OR

S: Recombinant factor VIIa 90 microgram per kg every 2-3 hrs or by continuous infusion (at 20µg/kg/hr)

CAUTION: All patients suspected with haemophilia A or B refer to the haemophilia treatment centre or consult haematology Unit

2.1.2 Von Willebrand Disease (VWD)

Von Willebrand Disease is inherited disease due to deficiency of vW factor. Patients present with a history of easy bruising, menorrhagea, gum bleeding and spontaneous joint bleeding in severe form.

Treatment

D: Tranexamic acid 500mg (O) 8 hourly until bleeding is stopped.

If no response

S: Desmopresin (DDVAP) infusion 0.3µg/kg IV. Max. dose 20µg.

Note: Patient unresponsive to DDVAP may be treated with virus-inactivated vWF containing FVIII concentrate.

2.2 Acquired Bleeding Disorders/Platelet Disorders

2.2.1 Disseminated Intravascular Coagulation (DIC)

Disseminated intravascular coagulopathy is caused by procoagulants that are introduced into or produced in the blood and overcome the natural anticoagulant mechanisms. In the acute form massive activation of coagulation does not allow time for compensatory increase in production of coagulant and anticoagulant factors.

Clinical features are related to the underlying disorder leading to DIC. Patients present with bleeding manifestation, extensive organ dysfunction, shock, renal cortex ischemia, coma, delirium and focal neurological symptoms.

Treatment of the underlying disorder is of utmost importance including

- Antibiotics for infection
- Surgical debridement of necrotic tissues
- Chemotherapy for acute leukemia,
- Evacuation of dead fetus;
- Transfusion with platelets support for thrombocytopenia, fresh frozen plasma (FFP) for coagulation factor depletion and cryoprecipitate for hypofibrinogenemia.

Multifactor deficiency, Liver disease gives Fresh Frozen Plasma 10-15mls/kg until bleeding is stopped

- Monitor prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin (APTT), platelet count and fibrinogen.

CAUTION: If patient is not bleeding Platelets concentrate is contraindicated. If DIC is severe enough to cause multiorgan dysfunction, management in an intensive care unit is required.

2.2.2 Idiopathic thrombocytopenic Purpura (ITP)

Idiopathic thrombocytopenic purpura is an acquired disease of children and adults and defined as isolated thrombocytopenia with no clinically apparent associated condition or other causes of thrombocytopenia. The diagnosis relies on exclusion of other causes of thrombocytopenia.

Clinical feature for adult thrombocytopenia appears to be more common in young women than in young men but among older patients, the sex incidence may be equal. Most adult patient presents with a long history of purpura, menorrhagia, epistaxis and gingival haemorrhage. Intracerebral haemorrhage occurs infrequently but is the most cause of death.

Note: A palpable spleen strongly suggests that ITP is *not* the cause for thrombocytopenia.

Treatment

Patients who are incidentally discovered to have asymptomatic mild or moderate ITP can safely be followed with no treatment. Patients with platelet counts over 50,000/ μ l usually do not have spontaneous bleeding and may undergo invasive procedure

Emergency treatment of acute bleeding caused by severe thrombocytopenia

- Immediate platelet transfusion is indicated in patient with haemorrhagic emergencies
C: Prednisolone 1mg/kg/day orally

IV immunoglobulin may be given as a single dose infusion of 0.4-1.0g/kg followed immediately platelets transfusion

Splenectomy is indicated in patient with refractory to prednisolone.

3.0 COAGULATION DISORDERS

- Venous thromboembolism is a common disorder with annual incidence of 117 per 100,000 persons;
- VTE comprise deep vein thrombosis (DVT) and pulmonary embolism (PE);
- Most clinically important pulmonary embolism arise from proximal deep vein thrombosis i.e. popliteal, femoral or iliac veins in at least 90%;
- Other less common sources are deep pelvic veins, renal veins, inferior vena cava, axillary veins and Rt side of the heart.

3.1 Deep Vein Thrombosis (DVT) Propagative

Clinical features of Deep Vein Thrombosis includes

- Leg pain, tenderness and swelling.
- A palpable cord representing thrombosed vessels.
- Discoloration, venous distention and prominence of superficial veins and cyanosis.
- The clinical diagnosis of DVT is highly nonspecific.

- In most patients the symptoms and signs are nonspecific.

3.2 Pulmonary embolism (PE)

Clinical features of PE includes

- Transient dyspnea and tachypnea in the absence of other clinical features
- Pleuritic chest pain, cough, haemoptysis, pleural effusion, and pulmonary infiltrate
- Severe dyspnea and tachypnea and right side heart failure
- Cardiovascular collapse with hypotension, syncope, and coma
- Several less common and nonspecific presentation including unexplained tachycardia or arrhythmia, resistant cardiac failure, wheezing, cough, fever, apprehension and confusion.

Treatment of Venous Thromboembolism

Long term anticoagulation is required to prevent a frequency of symptomatic extension of thrombosis and/or recurrent venous thromboembolic events. Warfarin is started with initial heparin or clexane therapy and then overlapped for 4-5 days.

D: Warfarin 5mg PO for 4-5 days

OR

S: Low Molecular weight Heparin 1mg/kg subcutaneous for 4-5 days

D: Unfractionated Heparin by IV 75units/kg followed by continuous infusion of 18units/kg/hrs.

For small adult or child lower loading dose then 15-25Units /kg/hr by IV infusion or 250units/kg every 12hrs by subcutaneous injection.

Pregnant woman

S: Clexane 1mg/kg and should be monitored by anti-Xa levels.

NOTE

Warfarin therapy should be monitored by INR after 5-7 days of treatment. Heparin should be monitored by aPTT before treatment is initiated and monitor aPTT hourly until aPTT is twice of the initial.

CHAPTER FIFTEEN

TRAUMA & INJURIES

1.0 GENERAL MANAGEMENT OF TRAUMA

Major trauma is associated with fractures, multiple lacerations and other major injuries. Major trauma may occur as a result of motor vehicle accidents or fights. The aim in handling major trauma is to look for life threatening complications which if missed may endanger the patient's life.

Diagnosis

- There is usually a history of trauma or accident
- If the patient is conscious he/she may complain of pain at specific places on his/her body
- Some patients may present with confusion, some semi-conscious and others may be in coma and/or shock

General Treatment

Community/Dispensary Level Interventions

- Clear airway
- Minimise bleeding and dress wounds
- Assess cardiac function – (arterial pulse, BP, capillary refill)
- Administer analgesics for pain control
 - A:** Diclofenac 75mg inj 8 hourly
- Splint long bone fractures
- If unconscious put in coma position and protect the spine.
- Refer

Health Centre Level Interventions

- Manage as above, capitalizing on ABCDE Trauma Protocol
- Catheterize bladder in unconscious patient.
- Set up IV line normal saline or ringer's lactate
- Do not feed patient
- If there are open wounds clean and dress and give

C: Ampicillin 500 mg IV 6 hourly

OR

B: Chloramphenicol 500 mg IV 6 hourly

- Refer

Hospital Level Interventions

- Manage as above
- Search systematically according to ABCDE Trauma Protocol for any signs of major injury such as:-
 - Head injury
 - Eye injury
 - Dental trauma
 - Fractured spine
 - Chest injuries
 - Internal Abdominal/Pelvic injuries
- Manage accordingly. Emergency/Casualty room set up is mandatory.
- Refer if specialist intervention is required

Table 1: ABCDE Trauma Protocol

	Assess	Intervention
A (airway)	Is it patent? Any secretions? Tongue fall? Any mouth/nose bleeding? Did patient drowned? Vomited? Aspirated?	Position him/her in semiquater prone. Place an oral airway. Raise the chin of mandible Suctioning if required Endotracheal intubation - ETT
B (breathing)	Record the respiratory rate (normal 10-20/min adults; 30-60/min children) Assess for chest asymmetry, abnormal movements or chest in-drawing Locate the trachea centrality Ensure air entry into both lungs by auscultation	Assist breathing by mouth to mouth, ambu bag or nasal prongs If fails do ETT and mechanical ventilation Place the chest tube in case of hemothorax, pneumothorax or tension types Plaster the open chest wound
C (circulation)	Assess arterial pulse, BP and heart sounds for signs of shock	Treat shock accordingly Set an I.V. line with isotonic fluids
D (Disability)	Assess level of consciousness using GCS scale	Treat the head injury accordingly
E (exposure)	Un-dress the patient to observe for signs of soft tissue injuries. Blunt injuries to the chest, abdomen or the dorsal spine may indicate the life threatening ailment underneath.	Catheterize NGT insertion Treat accordingly. Surgery may be indicated based on specialist requirement

2.0 TRAUMATIC BRAIN INJURIES

It is any episode of trauma to the head. We will exclude maxillo-facial injuries and eye injuries from this discussion (Ref this to eye section). Mortality is increased if hypotension or airway/breathing problem is not adequately solved.

Diagnosis

- Head injury may be associated with ophthalmic, ENT and dental injuries which are discussed separately.
- It is classified into two:
 - Involving scalp only;
 - Traumatic brain injury

Table 2: Illustration of Traumatic Brain Injuries

Mild Traumatic Brain injury	-Glasgow coma scale 13-14 -Involves a "brief" period of loss of consciousness -Good progress with minimal or no long term sequel
Moderate Traumatic Brain Injury	- Glasgow coma scale 9-12 -Confused patient with focal neurological deficits but able to follow simple commands -Some mild long-term sequel -Good prognosis
Severe Traumatic Brain injury	- Glasgow coma scale <8 (This is the definition of coma) -Unable to follow commands initially - Significant long-term disability

Treatment

Community/Dispensary level Interventions

- Clean and dress any wound
- If unconscious, ensure airway is patent
- Keep patient warm
- Put in coma position
- Prevent spinal injury by stabilizing the neck with collar
- Refer immediately

Health Centre Interventions

- Take full history from patient, relatives or whoever has brought patient where indicated
- Ensure adequate oxygenation
- Clean and suture wound as appropriate
- Record and monitor vital signs including pupil size and symmetry
- Inset IV line Normal saline or Ringer's lactate
- Treat seizures by Inj Phenytoin 100mg 8 hourly
- Catheterize
- Refer if moderate or severe TBI, pupil asymmetry or can not perform brain CT scan

Hospital Level Interventions

- History as above
- Examine patient thoroughly, note the level of consciousness, pupils' asymmetry and any lateralizing signs
- Brain CT scan if GCS score is 9 or below
- Admit to ICU if GCS score is 8 and below, or refer if required
- Craniotomy is indicated for specialist cases e.g. intracranial hematomas, depressed skull fractures based on pupil asymmetry, lateralizing signs and brain CT scan

- Refer or Consult the specialist if indicated especially moderate and severe traumatic brain injury Refer if pupil asymmetry is noted

Table 3: Use GLASGOW Coma Scale

SCORE	MOTOR RESPONSE	SCORE	VERBAL	SCORE	EYE
6	obeys verbal command	5	Oriented and Converses	4	Eye open spontaneously command
5	Localises painful stimulus	4	Disoriented and converses	3	Eye open to verbal command
4	Flexes limb to painful stimulars	3	Inappraiate words	3	Eye open to pain
3	Abnormal flexion painful stimulare	2	Inappropriate sound	2	Eye open to pain
2	. Extension to painful stimulus	1	No response		
1	No response				

Severe Traumatic Brain Injury

It is the most disabling condition that is associated with great mortality if not treated optimally. It is invariably followed by permanent disabilities. Multidisciplinary approach is of paramount importance. Long-term admission is advised.

Treatment

- ICU admission
- Craniotomy if indicated based on brain CT scan findings
- Rehabilitation upon discharge from hospital

3.0. INJURIES

3.1 Soft tissue injuries

Diagnosis

- Pain only, traumatic swelling, bruises with intact skin, cuts, abrasions, puncture wounds or open wounds of varying size and severity
- Injury to internal organs must be recognized and referred, including subtle signs of organ damage, e.g.:
 - blood in the urine – kidney or bladder damage
 - shock – internal bleeding
 - blood or serous drainage from the ear or nose – skull base fracture
- An injury causing a sprain or strain may be initially overlooked. Exclude fractures by performing appropriate X-rays

Note

- Referral must not be delayed by waiting for a diagnosis if treatment is logistically impossible
- Closed injuries and fractures of long bones may be serious and damage blood vessels
- Contamination with dirt and soil complicates the outcome of treatment

I. Emergency management

- Immobilize injured limb by POP cast or splint
- Monitor vital signs
- Monitor the arterial pulse and capillary refill below an injury on the limb with swelling.

II. Wound care

- Clean the wound
- Suture or splint when needed
- Avoid primary suture if the wound is infected:
 - Dirty or contaminated
 - Crushed
 - In need of debridement
 - Projectile inflicted
 - Caused by bites

III. Treatment

A: Paracetamol 15 mg/kg (O) 4–6 hourly when required. Maximum of 4 doses per 24 hours

Plus

S: Cloxacillin 500mg 6 hourly for 7 days

Plus

B: Tetanus prophylaxis: 0.5 mL Tetanus toxoid and 1 mL Tetanus immunoglobulin (Depending on the immunization protocol)

Table 4: Protocol in Provision of Tetanus Prophylaxis

Patient Category	Non-tetanus Prone	Tetanus Prone
Immunized and booster within 5 years	Nil	Nil
Immunized and 5 to 10 years since booster	Nil	TT
Immunized and >10 years	TT	TT
Incomplete immunization or unknown	TT and TIG	TT and TIG

TT = T. toxoid; TIG = Tetanus Immunoglobulin

3.2 Sprains and Strains

Diagnosis

- Pain, especially on movement
- Tenderness on touch
- Limited movement
- History of trauma

These may be caused by:

- Sport injuries
- Slips and twists
- Overuse of muscles
- Abnormal posture

Note: In children always bear non-accidental injuries (assault) in mind.

1 Emergency treatment

- Immobilize with firm bandage and/or temporary splinting e.g. triangular sling, back slab etc
- Children over 12 years and adults:
 - A:** Ibuprofen 200–400mg (O) 8 hourly with or after a meal
 - OR/Plus**
 - A:** Paracetamol, oral, 15 mg/kg 4–6 hourly when required. Maximum of 4 doses per 24 hours. In children less than 6 months calculate dose by weight
- Perform X-ray to rule out dislocations or subluxations

2 Referral

- If Severe progressive pain. Do X-ray to exclude bone fractures or joint dislocation.
- Progressive swelling
- Extensive bruising
- Deformity
- Joint tenderness on bone
- No response to treatment
- Severe limitation of movement

3.3 Extremity Fractures

Fractures of long bones of upper and lower limbs are quite common. If not properly treated they often lead to long-term deformities. Osteomyelitis is always the complication of open fractures. Hemorrhagic shock may ensue in situations involving multiple fractures or pelvic ring fractures.

Diagnosis

- Pain
- Swelling
- Loss of limb function
- Deformity and abnormal movement
- X-ray is mandatory to confirm the deformity

Management

Community / dispensary level

- Immobilize injured limb by POP cast or splint.
- Monitor vital signs.
- Monitor the arterial pulse and capillary refill below an injury on the limb with swelling
- Refer the patient

Health Centre

- Immobilize injured limb by POP cast or splint
- Monitor vital signs
- Monitor the arterial pulse and capillary refill below an injury on the limb with swelling
- Give tetanus toxoid to non immune cases
- Refer the patient if open fracture or if specialist service not available

Hospital level

- Immobilize injured limb by POP cast or splint
- Monitor vital signs
- Monitor the arterial pulse and capillary refill below an injury on the limb with swelling
- Treat open fractures by proper surgical debridement and ORIF as per specialist guideline.

3.4 Spine fractures

Motor traffic injuries and falls constitute the burden of most spine injuries. Paralysis may be associated, often been brought by improper transfer of the patient to the hospital. C-spine injury is always accompanied by traumatic brain injury.

Diagnosis

- History of trauma
- Pain
- Neurological deficit
- X-ray, CT scan and MRI are mandatory.

Treatment

- Immobilize the neck by collar or pillows/sand bags
- Patient should lie flat in bed, preferably the flat bed or air mattress
- Treat shock as per the guideline
- Catheterize if urine retention
- Immediate transfer to the hospital that handles spine surgeries

4.0 BITES

4.1 Animal Bites

Animals that bite man include both wild and domesticated ones. Thus lion, tiger, leopard, hyena, bear, elephant, hippopotamus, buffalo, wolf and wild pig are examples of the wild animals that have bitten man. Others are fish, crocodiles and dogs. Clinical features of these bites arise from the pathology inflicted by teeth, tusks, claws and horns. They produce lacerations, penetrating and crushing injuries. Severe facial and eye injuries are common and pneumothorax, hemothorax, bowel perforation and compound fractures have occurred.

Treatment

- Emergency surgery is often needed
- Replace any blood lost
- Treat complications of injury e.g. resultant rabies, tetanus, pneumothorax
- Treat infection with relevant antibiotics.

Give **Tetanus Toxoid** 0.5ml start. Repeat after 4 weeks and then 6-12 months later

4.2 Insects Bites

Important insects' bites are those from scorpions.

Symptoms: Most bites and stings result in [pain](#), swelling, redness, and [itching](#) to the affected area

Treatment and Management

Treatment depends on the type of reaction

- Cleanse the area with soap and water to remove contaminated particles left behind by some insects
- Refrain from scratching because this may cause the skin to break down and an infection to form
- Treat itching at the site of the bite with [antihistamine](#)
- Give appropriate analgesics
- Where there is an anaphylactic reaction treat according to guideline.

4.3 Snake Bites

Refer to the poisoning chapter.

5.0 BURNS

It is thermal trauma to the skin, mucosae and deeper tissues. Classification depends on depth and extent. If area burnt is larger than 10% of body surface then this is extensive because of fluid loss, catabolism, anaemia and risk of secondary infection.

The 'rule of 9' to calculate % of body surface burned, can be used.

Table 5: Rule of Nine for calculatin % of Body surface burned

Body Areas	Adult (%)	Child %
Entire head	9	18
Upper limb	9	18
Anterior or posterior surface of trunk	18	18
Lower limb	18	14
Perineum	1	1

Treatment

Ensure that there is an adequate airway, adequate breathing and adequate circulation

- Immerse burnt area in cold water for 10 minutes
- Clean with Normal saline or Chlorhexidine – cetrimide solution
- Apply Gentian Violet solution
- Do not cover
- Calculate fluid requirement per 24 hours: weight x % of surface burnt x 2 = quantity of fluid
- Give 75% of fluid requirement as sodium lactate compound solution and 25% as 6% Dextran 70 as blood/plasma expanders. Give first half in 8 hours and the rest within 24 hours.
- Give paracetamol 1000 mg every 8 hours and Diazepam 10 mg IM start
- Give tetanus toxoid 0.5 ml. stat
- Immobilize in position of function and leave any dressing undisturbed for 5-7 days
- Debridement where indicated
- Give Procaine Penicillin 1.2 MU IM every 24 hours where indicated but not antibiotic ointment
- In full thickness burns, skin grafting may be indicated to speed wound healing. In such cases refer to secondary or tertiary level health care centre
- Children give
 - A:** Paracetamol 10 mg/kg every 8 hours
 - Plus**
 - C:** Procaine Penicillin 0.4 – 1.2 MU IM every 24 hours.

6.0 FOREIGN BODIES

Foreign bodies may be introduced into any of the body orifices nose, ears, vagina and urethra. Foreign bodies introduced through the mouth (or nose) may be arrested in the larynx, bronchial tree, oesophagus or stomach.

Diagnosis

It depends on the affected site. The symptoms may be due to obstruction or inflammation around the foreign body.

Treatment Guidelines

Foreign bodies into the ears, nose, urethra, vagina, larynx and bronchial tree invariably should be removed. Foreign bodies in the stomach rarely produce symptoms and active treatment is usually not required.

CHAPTER SIXTEEN

MALIGNANT DISEASE CONDITIONS

Cancer is a word covering a wide range of malignant diseases which contribute significantly to the overall morbidity and mortality of people world-wide. The true magnitude of the cancer situation in Tanzania is unknown, however more than 3000 new cases are recorded in ORCI-based registry; and that is estimated to be only 10% of cancer incidence in the country.

1.0 GYNAECOLOGICAL MALIGNANCIES

1.1 Carcinoma of the cervix (Cacx)

Clinical features:

- Asymptomatic if early
- Later predominant symptoms are: Postcoital, intermenstrual or postmenopausal vaginal bleeding.
- Pain and incontinence are rather late symptoms.

Investigations:

- Laboratory tests: FBC, LFTs, creatinine, urea,
- Radiological investigations: CXR, abdominal/pelvic ultrasonography, IVU.
- Biopsy of cervix or abnormal Papanicolaou smear or VIA/Vili during screening confirm the diagnosis.
- Bimanual examination under anaesthesia (EUA), recto-vagina examination is mandatory for proper disease staging.

Histopathology: Squamous cell carcinoma (SCC)-90%,
Adenocarcinoma -10%,
Rarely – others – clear cell, small cell, sarcoma, etc

Staging: FIGO: IA, IB, IIA, IIB, IIIA, IIIB, IVA and IVB

- Early cancer stages IB, IIA, and selected IIB)
- Late cancer stages IIB bulky, IIIA, and IIIB)

Referral

All patients must be referred to a gynecologist for evaluation and decision on mode of treatment. Decision of treatment for carcinoma of the cervix is best done in hospital under specialist care.

Treatment

Surgery:

- Early stage disease: Conservative surgery or TAH
- Advanced stages: Chemoradiation: Cisplatin 50 mg infusion weekly x 6 together with concomitant radiotherapy – External beam therapy (EBRT) and intracavitary (ICT) as indicated.

EBRT: 50Gy/25F/5 wks plus ICT 6.7Gy wkly x 3.

Chemotherapy regimen

A: D0:Pre-medication: 0.9% saline 3000mls i/v over 24 hours

S: D1: Cisplatinum 50mg/m² synchronous with radiotherapy.

Primary prevention (screening) and early detection:

- Vaccination is now available
- Avoid early sex.
- Visual inspection method using acetic acid and lugols solution, PAP smear.

1.2 Carcinoma of the endometrium

Clinical features: Usually postmenopausal PV bleeding in an elderly.

Investigations:

- **Laboratory** :FBC, LFTs, urea, creatinine
- **Radiological:** IVU, CXR, Abdominal/pelvic USS.
- **Cytology:** Endometrial curettings confirm the diagnosis.
- Both inspection and bimanual examination under anaesthesia (EUA) recto-vagina are mandatory.

Histology: Usually Adenocarcinoma

Others: Clear cell, small cell carcinomas, sarcomas.

Histological grade bears the prognosis: GI better than GIII.

Staging: FIGO: IA, IB, IC, IIA, IIB, IIIA, IIIB, IVA, and IVB

Early cancer stages: IAG1, IBG1, II and most II

Late cancer stages: III and IV

Referral: All patients must be referred to a gynecologist for evaluation and decision on mode of treatment. Decision of treatment for the uterine carcinoma is best done in hospital under specialist care.

Treatment:

Surgery: TAHBSO with generous vaginal cuff removal suffices for early stages.

Radiotherapy: Post-operative radiotherapy is indicated for high risk patients (node positive, higher grades II, III and positive margins). ICT is indicated in case of parametrial and vaginal involvement. EBRT/ICT alone is indicated for patients who have contraindication for surgery or too advanced disease. Chemotherapy with RT is indicated for uterine sarcoma sequentially. RT 50Gy/25F/5wks.

Chemotherapy regimen for leiomyosarcoma:

S: Adriamycin 40mg/m² single agent every 3 wks x 6. Then RT 50Gy/25F/5wks is indicated post-operatively.

1.3 Cancer of the vagina and vulva

Clinical features:

- Presence of Leukoplakia and other dystrophic changes in the vagina and/or vulva
- Itching is a big problem and may become ulcerative (“non-healing ulcers”)
- Pain from superinfection

- Usually Bartholin’s gland, labia majora, labia minora and clitoris can be primary sites and Lymphadenopathy of the groin is involved

Investigations:

- Laboratory: FBC, LFTs, Urea, creatinine
- Radiological: IVU, CXR, Ultrasonography of abdomen and pelvis.
- Both inspection and bimanual examination under anaesthesia (EUA) recto-vagina are mandatory to exclude primary disease or extension from other sites such as cacx.
- Biopsy from the vulval or vaginal lesion is mandatory to confirm the diagnosis

Histology: Usually squamous cell carcinoma
Rarely – others – KS, clear cell, small cell, sarcoma.

Histological grade bears the prognosis: GI better than GIII.

Staging: FIGO: IA, IB, IIA, IIB, IIIA, IIIB, IVA, and IVB
Early cancer stages IAG1, IBG1, II and most III
Late cancer stages: III and IV.

Referral: All patients must be referred to a gynecologist for evaluation and decision on mode of treatment. Decision of treatment for the vulvo-vaginal carcinoma is best done in hospital under specialist care. Regional/zonal or tertiary depending on treatment expertise

Treatment: Predominantly surgical. Aim: Cure.

Surgery: Wide excision of the primary site and lymphadenectomy for early stages.
Radiotherapy: Post-operative radiotherapy is indicated for high risk recurrence (positive

margins and nodal involvement). ICT is indicated in case of vaginal involvement. EBRT/ICT/Chemoradiation is indicated for patients who have contraindication for surgery or advanced stages not amenable for surgery.

Chemotherapy regimen: See chemotherapy for cervical cancer.

1.4 Malignant Trophoblastic disease

Clinical features: Rare solid tumour. Usually follows pregnancy which has resulted in a hydatidiform mole. This may accompany normal, ectopic or even termination of pregnancy.

Patient presents with abnormal vaginal bleeding during or after pregnancy associated with a "large-for-date" uterus. Other findings include: marked symptoms of pregnancy or pre-eclampsia.

Investigations:

- Laboratory: FBC, LFTs, urea, creatinine, Levels of β HCG, 24-hour urinary HCG
- Radiological: CXR, CT scans according to symptoms to confirm metastases, abdominal/pelvic USS
- Cytology: Currettings from D&C.

Histology: Choriocarcinoma, Hydatidiform mole, chorioadenoma destruens

Staging: Low risk, moderate risk and High risk groups are defined by prognostic variables such as: Interval between antecedent pregnancy and the start of chemotherapy, height of initial HCG levels, number, size and site of metastases (the brain is a particularly adverse site), age of the patient (older patients do worse), parity, previous administration of chemotherapy if any.

Referral: All patients must be referred to a gynecologist for evaluation and decision on mode of treatment. Decision of treatment for malignant trophoblastic tumours is best done in hospital under specialist care. Regional/zonal or tertiary depending on treatment expertise.

Treatment

Surgery: Trial of D & C and if symptoms continue – Hysterectomy.

Chemotherapy: Choriocarcinoma is extremely chemosensitive. It can be cured even when metastatic.

Low risk patient (Patient with above minimal prognostic indicators): - Methotrexate (MTX) 50mg i/m Day 1, 3, 5, 7 then Folinic acid 6mg i/m Day 2, 4, 6, and 8. **Methotrexate single agent and higher doses of MTX with folinic acid rescue are indicated for early disease (low risk).**

Repeat cycles every 6 days, continue 8 weeks after HCG has become undetectable.

Moderate and high risk patients (Those with worse above prognostic indicators): Combination chemotherapy:

MTX + Actinomycin- D + Cyclophosphamide + Etoposide:

D1: MTX 50mg i/v in 200mls N/S over 3 hrs; Etoposide 100mg i/v in 200mls N/S over 30 minutes; Actinomycin-D 0.5mg i/v

D2: : MTX 50mg i/v in 200mls N/S over 3 hrs; Actinomycin-D 0.5mg i/v; Folinic acid 6mg i/m or i/v.

D3: Folinic acid 6mg i/m or i/v

Repeat cycle after 6 days. Cycles are continued for 8 wks after HCG in serum has become undetectable *wkly assay).

Note: Response is assessed by serial β HCG measurement, and treatment repeated until the marker has been undetectable in the serum for 6 – 8 weeks.

1.5 Cancer of the Ovary

Epithelial tumours: Comprise 90% of ovarian malignancies.

Clinical features: Minimal or no symptoms early on. However increasing abdominal distension, palpable mass in the abdomen, pain and presence of ascites are all late signs.

Investigations:

- Laboratory: FBC, U&Es, LFTs;
- Radiological: CXR, CT scans, Pelvic and abdominal ultrasound, etc according to complaints.
- Histology of oophorectomy specimen or biopsy obtained at laparotomy.

Histologies of epithelial tumours: Serous (cyst) adenoma, mucinous (cyst) adenoma, endometrioid adenocarcinoma, clear cell adenocarcinoma, granulosa cell tumour, theca cell tumour, sertoli-Leydig cell tumour, mixed tumours.

Staging: Is surgical (laparotomy): FIGO: IA, IB, IC, IIA, IIB, IIC, III, and IV.

Referral: All patients must be referred to a gynecologist for evaluation and decision on mode of treatment. Decision of treatment for malignant trophoblastic tumours is best done in hospital under specialist care. Regional/zonal or tertiary depending on treatment expertise.

Treatment:

Surgery: TAHBSO with omentectomy. If total tumour removal is not possible, then maximum debulking (cyto-reductive) surgery should be done. Unilateral salpingo-oophorectomy is only justified for stage IA tumour with favourable histology.

Chemotherapy

Adjuvant chemotherapy: Is indicated for all unfavourable histologies as well as advanced stages.

Chemotherapy regimen

A:D0: Prehydration: 0.9 NS 3000mls/24 hrs

Plus

S: D1: Paclitaxel 175mg/sq m

Plus

S: Cisplatin 100mg/sq m

Cycle: Every 21 days x 6 cycles with FBC, Electrolytes, Urea, Creatinine and 24-hr creatinine clearance check prior to each treatment.

Radiotherapy

It is recommended for residual or recurrent disease. Whole abdominal radiation 30GY/20F/4wks with pelvic boost to 50Gy can produce long remissions. However contraindication to RT: Massive ascites and marked weight loss.

2.0 CANCERS OF THE SKIN

2.1 Non-melanotic skin cancers

Clinical features: Chronic sun exposure, old burns, non-healing ulcer and nodal involvement. The most common warning sign of skin cancer is a change in the appearance on exposed areas of the skin, such as a new growth or a sore that will not heal. Occasionally, such changes may appear on an old burn area.

Investigation:

- None if lesion is small.
- Local x-ray if bone involvement is suspected. CXR if undifferentiated tumour,
- Biopsy – preferably excisional biopsy where possible.

Histologies: Basal cell (BCC) and Squamous cell carcinomas (SCC).

Staging: TNM staging classification

Referral only where indicated in case expertise is required.

Surgery: The aim of surgery is total local excision where possible; wide local excision and graft; amputation sometimes is required. Locally destructive methods such as curetting, desiccating or cryotherapy may be employed.

Radiotherapy: Indication: Positive margin, high grade disease or inoperable tumour.

Chemotherapy:

S: Topical 5- fluorouracil for very superficial lesions or carcinoma in situ.

- Systemic chemotherapy: for advanced stages as radiosensitizer in conjunction with cisplatin:

S: Cisplatin 50mg infusion wkly x 6 concomitantly with RT.

Prehydration is mandatory, FBC & blood chemistry is mandatory before every cycle (see cacx).

Detection/Prevention: Frequent self-check or screening exercise and prompt treatment of early keratotic changes. For light skinned people-avoid U/V light.

2.2 Malignant melanoma

Clinical features: History of a pre-existing naevus which has changed recently – itching, colour change, increase in size, satellite lesions, elevated surface, ulceration and/or oozing.

Investigation:

- None or minimal if lesion is small
- Radiological: Chest x-ray in case of clinically suspected lung involvement or abdominal ultrasound in case of suspected liver metastases.
- Excisional biopsy of suspicious lesion and finding of malignant melanocytes within the lesion.

Staging: Clark's or Breslow classifications are used. Tumour size closely correlates with prognosis.

Detection/Prevention: Frequent self-check or screening exercise and prompt treatment of naevus.

Referral where indicated. Aim: Cure for early localized lesion.

Treatment

Surgery:

- The aim of surgery is total local excision where possible.
- Wide local excision and graft
- Amputation sometimes for advanced useless limb.

Chemotherapy: Not effective. Temozolamide and DTIC can be tried.

Radiation: Not first choice; but can be performed if:

- Lesion is inoperable. May use large fractions: 30Gy/6F/1 wk
- Excision margins are involved or very close
- Palliative intent (brain mets, fungation or profuse bleeding, bone pain, etc)

2.3 Kaposi Sarcoma (KS)

Definition: Kaposi's sarcoma is a malignant tumour of angio-formative cells usually starting from the skin but occasionally involving many other organs of the body. There are three epidemiological variants-sporadic, endemic and epidemic forms which may or may not be associated with infection of human immunodeficiency virus (HIV). These two types are commonly referred to as: Non AIDS related (endemic) KS and AIDS related (epidemic) KS.

Clinical features:

- KS presents as a firm, dark brown nodules or plaque in the skin. Usually more on the limbs.
- In young children and those with immunodeficiency it presents as wide spread lymphadenopathy with or without skin lesions.

- Presence of B symptoms (fever, sweating and weight loss) is commonly associated with epidemic type.
- Clinical course can be indolent or aggressive.

Investigations:

- **Laboratory:** FBC, LFTs, Urea & creatinine, Elisa test with confirmation
- **Radiological:** CXR in case of symptoms
- **Skin biopsy** followed by histological confirmation

Histological appearance for both endemic and epidemic types is the same.

Referral

Patients with AIDS related KS are referred to CTC clinic. Uses of ARVs are mandatory for patients with epidemic disease.

Treatment:

Chemotherapy:

- Adults:
 - S:** Adriamycin 40mg/sq m i/v D1
 - Plus**
 - S:** Vincristine 1.4mg/sq m i/v D1
 - Plus**
 - S:** Bleomycin 7.5mg/sq m i/v D1.

Repeat ABV every 3 wks x 6. FBC check is mandatory before each treatment.

Note: Pegylated liposomal Doxorubicine (PLD) is superior.

- Children under 12 years:
 - S:** Actinomycin-D 15microgram/kg i/v D1 – D5
 - Plus**
 - S:** Vincristine 1.5mg/m sq i/v D1, D8.

Repeat every 3 wks until remission then give further 2 courses.

Radiotherapy:

Indication: Palliation of pain, bleeding, oozing and fungation.

Note:

- Sequential hemibody irradiation is sometimes necessary for aggressive disease. 6Gy /SF followed by 6wks interval before other half is treated.
- Check FBC for guidance before treatment. 24-hr observation is mandatory when treating upper hemibody (UHB), Vital signs should be monitored.

3.0 HEAD AND NECK CANCERS

Definition: Carcinomas of the head and neck constitute an important group of tumours. They may interfere with vital functions such as: Respiratory, swallowing, sight, speech and mastication. Important aetiological factors include excessive intake of tobacco either by smoking or chewing and alcohol intake (particularly spirits).

Clinical features: Presence of premalignant lesions in buccal mucosa and tongue. Other features include: Non-healing ulcers, lymphadenopathy, hoarseness, pain and difficult in swallowing.

Investigations:

- **Laboratory:** FBC, LFTs, Urea & creatinine
- **Radiological:**, CXR or CT scan of the region as indicated.
- **Direct/indirect laryngoscopy/panendoscopy/bronchoscopy plus biopsy**

Histologies: Squamous cell carcinoma is the most common histology, though the frequency of other histological types and the degree of differentiation varies markedly with site. Other histologies include: Adenocarcinoma, mucoepidermoid, acinic, adenoid cystic carcinomas, basal cell carcinoma, KS, lymphomas, plasmacytoma, sarcoma, melanoma, verrucous carcinoma , rhabdomyosarcoma.

Staging: TNM

Referral: All patients must be referred to tertiary hospitals for evaluation and decision on mode of treatment. Decisions of treatment for head and neck tumours are best discussed at Tumour board.

Treatments

- The treatment plan for an individual patient depends on a number of factors: in the exact location of the [tumor](#), the disease [stage](#), the person's age and general health.
- **Radiotherapy:**
 - Is standard treatment for nasopharyngeal carcinoma and other inoperable tumours of head and neck.
40 – 60Gy/20-30F/4-6 wks.
 - Palliative RT: To relieve pain, reduce swelling, ulceration and bleeding.
30Gy/10F/2wks.
- **Chemoradiation** is a superior treatment of choice for all stages though mainly an early stage because this mode of treatment preserves anatomical functions including voice.
Regimen: Cisplatinium 100mg infusion D1+ 625mg 5FU D1 – D4

A: D0: 0.9 %NS 3000 mls i/v over 24 hrs prehydration

Plus

S: D1: Cisplatinum 80mg/sq m in 1 litre over 6 hrs

Plus

S: D1: D4 5FU 625mg i/v in 1 litre over 2 hrs

- Synchronous Cisplatin with radiotherapy 60Gy/30F/6wks

OR

S: Cisplatin + Mitomycin- C regime:

D0, D1 as above only substitute Mitomycin C 10mg/sq m for 5-FU.

- CHOP x 6 is standard treatment for lymphomas.

Surgery:

- Partial or total laryngectomy is for advanced stages only where voice is compromised.
- Verrucous carcinoma is best treated surgically
- Leukoplakia should be excised totally

3.1 Thyroid carcinoma

Definition: This group of diseases is exceptional in many ways. Some thyroid cancers are very indolent with long natural history. Tumour present as "goiter" and can remain silent for decades without any discomfort.

Clinical features: Presence of a thyroid mass or scar, laryngeal nerve palsy, hoarseness, dyspnoea, dysphagia.

Investigations:

- Laboratory: Thyroid function tests (T3, T4, TSH), FBC, LFTs, Urea & creatinine, serum calcitonin, serum thyroglobulin levels.
- Radiological: Thyroid scan, CXR, isotope bone scan, CT scan of the neck
- FNAC of a thyroid lesion

Histology: Four histological types: Papillary, Follicular, Medullary and Anaplastic

Staging: TNM

Referral: All patients must be referred to a specialized hospital for evaluation and decision on mode of treatment. Decisions of treatment for thyroid tumours are best discussed at Tumour board.

Treatment

- Radioactive iodine ablation
- Further thyroxine replacement therapy (for life).

Surgery: Total/near total thyroidectomy as indicated.

Radiotherapy: Is indicated in all cases of anaplastic carcinoma.

60Gy/30F/6wks

Chemotherapy: is still experimental.

Radioactive: Iodine ablation is indicated in all patients after surgery.

100mCi (3,500 MBq) is given 3-4 wks after surgery. NOTE: Stop T3 or T4 three wks before ablative treatment.

4.0 GASTROINTESTINAL MALIGNANCIES

4.1 Esophageal cancer

It can develop as a result of long standing achalasia or chronic irritation. Smoking and alcohol are both contributory.

Symptoms:

Difficult in swallowing ([dysphagia](#)) is the commonest symptom which is associated with weight loss and poor performance status.

Investigations:

Laboratory: FBC, LFTs, urea , creatinine

Radiological: Barium swallow and meal, CT scan, Esophagoscopy, Abdominal USS,

Biopsy: Rigid oesophagoscopy or Oesophagoduodenoscopy (OGD) with a biopsy.

Histology: Majority are SCC, ADC (lower third of oesophagus)

Staging: TNM

Treatment: Palliation in most cases. Cure rate with any modality is 5-10%.

Chemoradiation: Is the treatment of choice and few patients can be cured.

- Cisplatin & 4 day 5FU

A: D0: 0.9 %NS 3000 mls i/v over 24 hrs prehydration

Plus

S: D1: Cisplatin 80mg/sq m in 1 litre over 6 hrs

Plus

S: D1- D4 5FU 625mg i/v in 1 litre over 2 hrs

- Synchronous Cisplatin with radiotherapy

Surgery: Only for very selective patients with curative intent. Often possible for lower third.

Paliative surgery: Had once been indicated for patients unable to swallow, feeding gastrostomy has been recommended but does not improve quality of life or survival benefit. Dilatation with or without intubation should always be considered to ensure continued ability to swallow.

Radiotherapy alone: Any inoperable patient should be considered for RT on palliative basis.
30Gy/10F/2wks; 40Gy/20F/4wks
Intraluminal RT using HDR insertions in some centres.

4.2 Stomach Cancer

Clinical features: Epigastric pain worsened by food intake, early satiety, anorexia, weight loss, weakness, and obstructive symptoms may be present with distal tumours. Bleeding occult or manifest may be a feature. Look for pallor, weight loss, supraclavicular foss nodes, abdominal and rectal examination, epigastric mass, hepatomegally, periumbilical nodes.

Investigations:

- **Laboratory:** FBC, LFTs, stool for occult blood
- **Radiological:** Gastroscopy, CXR, Ba meal (double contrast), abdominal USS.
- **Biopsy at surgery.**

Histology: Adenocarcinoma – 95%, NHL – 4%
Others: Leiomyosarcoma, KS – 1%

Staging: Surgical: I, II, III and IV.

Treatment: Palliation in most cases.

Surgery: Total or partial gastrectomy, bypass with or without tumour removal eg gastrojejunostomy.

Chemotherapy: Combination therapy is the best approach: 5- FU, cisplatin, capecitabin, leucovorin, levamisole, CHOP in case of NHL.

RT: Has a minimal role in chronic bleeding and relief of pain.

Screening and early detection: Has a role in endemic regions like in Japan.

4.3 Hepatocellular carcinoma

Clinical features: This is a malignant neoplasma of the liver which may occur either with or without accompanying hepatic cirrhosis. There is a strong association of this cancer and hepatitis B infection and/or alcohol consumption. An arterial bruit and ascites may be present. Right upper abdominal swelling and pain often associated with weight loss, fever, jaundice.

Investigations:

- Laboratory: FBC, LFTs,, biochemistry, serum alpha feto protein, HBsAg, HB core antibody, PTT,
- Radiological: CXR, Abd/pelvic USS, Liver FNAC, angiography if surgery is an option
- Biopsy or FNAC of the liver.

Histology: Hepatocellular carcinoma 90%, Cholangiocarcinoma 7%, Hepatoblastoma, angiosarcoma, sarcomas 3%.

Staging: TNM. Anatomic extent of involvement: A: One lobe only; B: Two lobes; C: Metastatic disease; D: Cirrhosis.

Treatment: Palliation in the majority of patients.

Surgery: Lobectomy where feasible

Chemotherapy is not effective; However single agent Doxorubicin is used.

Radiotherapy: Is of very little value.

Prevention: Vaccination Hepatitis B

4.4 Colo-rectal cancer

Clinical features: Family history for polyposis, ulcerative colitis or any other genetic predisposition. Patient with history of passing melena. Abdominal mass with or without obstructive symptoms. Frequent episodes of blood transfusion, disturbed bowel habits.

Investigations:

- Laboratory: FBC, ESR, LFTs, Stool for occult blood,
- Radiological: CXR, Barium enema (double contrast), Abdominal and pelvic USS, colonoscopy.
- DRE under EUA and biopsy
- Biopsy at colonoscopy or laparotomy.

Histology: Usually adenocarcinoma 95%. Others: Lymphoma, carcinoid, sarcoma, KS 5%.

Staging: TNM

Treatment: Aim: Cure for early stages.

Surgery: Aim is cure for early stages. Surgical resection is the mainstay of treatment. Total or partial obstruction may require defunctioning colostomy.

Chemotherapy:Combination: FOLFOX, Irinotecan + 5-FU/Cisplatin

Radiotherapy: Preoperative: May render surgery easier and may reduce local recurrence or distal metastases.40Gy/20F/4wks

Post-operative RT: Is indicated for positive or very close margins and where lymphnodes are involved. 40Gy/20F/4wks.

Chemoradiation is indicated for rectal tumours (Oxaliplatin & 5FU). Early stages may be superior to surgery in the sense that sphincter function is preserved.

Palliative RT: For recurrence or metastases to relieve pain or obstruction or reduce discharge, bleeding or ulceration. Dose same as above.

Screening and early detection: Annual digital rectal examination (DRE) and foecal occult blood test (FOBT) is advocated.

5.0 LUNG CANCER

5.1 Non small cell lung cancer (NSCLC)

Clinical features:

- Chronic chest symptoms in a smoker
- Haemoptysis may be part of it
- May present with superior vena cava obstruction (SVCO) syndrome
- Occupation with asbestos exposure
- Findings of chest symptoms, weight loss, poor KPS

Investigations:

Laboratory: FBC, LFTs, urea, creatinine,

Radiological: CXR PA & lateral views or CT scan of thorax, abdominal USS,
Abdominal USS.

Biopsy or cytology of sputum or bronchial aspirate examination.

Histology: SCC, Adenocarcinoma, large cell carcinoma.

Staging: TNM

Treatment: Aim: Cure for stages I and some stage II

Surgery: Aim: cure for stages I and some II (pneumonectomy. Significant survival rates follow pneumonectomy or lobectomy.

Chemotherapy: Indicated for all other stages – palliation only.

Use Cisplatin & Etoposide:

- D0: 0.9 %NS 3000 mls i/v over 24 hrs prehydration
- D1: Cisplatin 80mg/sq m in 1 litre over 6 hrs
- D1- D3 100mg od daily
- Repeat cycle every 21 days with FBC check and renal clearance.

Use Gemcitabine 1000md i/v bolus every 3 wks x 7 cycles

Palliative RT: Local control of haemoptysis, SVCO, bone pains, atelectasis, obstructive pneumonitis and fungating masses, etc.
30Gy/10F/2wks.

5.2 Small cell lung cancer (SCLC)

Clinical features: Virtually always is a systemic disease at presentation.

Investigations:

- As in NSCLC however brain scan and bone marrow aspirate are necessary.
- Biopsy

Staging: Limited disease vs Extensive disease.

Treatment: Aim: Local control and palliation. Cure rate is low.

Chemotherapy:

S: Vincristine (VCR) 1.4/m sq i/v D1

Plus

S: Adriamycin (ADM) 50mg/m sq i/v D1

Plus

S: Cyclophosphamide 750mg/m sq i/v D1.

Repeat every 3 wks x 6

Alternatively

S: Etoposide 100mg/m sq i/v D1

Plus

S: ADM 50mg/m sq i/v D1

Plus

S: Cyclophosphamide 750mg/m sq i/v D1

Plus

S: Etoposide 200mg/m sq PO D2, D3. Repeat every 3 wks x 6.

RT: - Consolidation to primary site and mediastinum: 50Gy/25F/5wks

- Prophylactic brain irradiation in complete responders.

- Temporary relief of respiratory, bone or CNS symptoms: 30Gy/10F/2wks.

Surgery: Is of a minimal value in SCLC.

6.0 BREAST CANCER

It is a malignant tumour of the glandular or lobular tissue of the breast.

Symptoms:

- A solitary lump in the breast
- Hardness, attachment to skin or deeper tissues, skin ulceration,
- Nipple retraction
- Presence of axillary lymphadenopathy or elsewhere

Investigations:

- **Laboratory: FBC, LFTs, urea, creatinine**
- **Radiological:** Mammography of the contralateral breast, CXR, abdominal USS, bone scan in case of complaints.
- FNAC of the lump or open biopsy.

Histology: Ductal or lobular carcinoma

Staging: TNM

Referral: All patients must be referred to tertiary hospital for evaluation and decision on mode of treatment. Decisions of treatment for breast cancer are best discussed at Tumour board.

Treatment

Surgery

- Modified radical mastectomy
- Lumpectomy
- Simple mastectomy with axillary node dissection
- Toilet mastectomy to improve patient's quality of life.

Chemotherapy:

CMF regimen:

S: Cyclophosphamide 750mg/sq m i/v D1

Plus

S: Methotrexate 40mg/sq m i/v D1

Plus

S: 5FU 750mg/sq m i/v D1

Treatment interval 3 wks x 6

CAF regimen:

S: Adriamycin 50mg/sq m i/v D1

Plus

S: Cyclophosphamide 750mg/sq m i/v D1

Plus

S: 5FU 750mg/sq m i/v D1

Treatment interval 3 wks x 6

CEF regimen:

S: Epirubicin 50mg/sq m i/v D1

Plus

S: Cyclophosphamide 750mg/sq m i/v D1

Plus

S: 5FU 750mg/sq m i/v D1

Treatment interval 3 wks x 6

CA regimen:

S: Adriamycin 50mg/sq m i/v D1

Plus

S: Cyclophosphamide 750mg/sq m i/v D1

Treatment interval 3 wks x 6

Bezacizumab plus Capecitabine or 5-FU regimen:

S: Bezacizumab 300mg in 100ml N/S every 2 wks x 8

Plus

S: Capecitabine) 1500mg every 12 hours x 14 days then rest 1 wk. Recheck FBC; if ok continue x 6 cycles.

***** Herceptin for Triple negative breast cancers: 300mg in 200 mls N/S to run for 1 hr every 2 wks x 12 cycles**

***** Very important drug for ER/RP Negative and Her 2 neu Positive group of patients.**

Radiotherapy: Is indicated to all patients with high risk of local recurrence.

50Gy/25F/5wks; 45Gy/20F/4wks

Hormonal therapy:

Anti-estrogen

D: Tamoxifen 20mg PO daily x 5 years

Aromatase inhibitors:

S: Anastrozole 1mg PO daily x 5 years.

Detection/Prevention

- Any woman particularly at the age of 50 years should undergo mammography annually
- Anyone with familial risk ought to start earlier

Self breast examination on monthly basis

7.0 GENITO-URINARY MALIGNANCY

7.1 Bladder cancer

Clinical features: Bladder cancer characteristically presents with haematuria. This may be visible to the naked eye gross [hematuria](#) or detectable only by microscope. Other possible symptoms include: Dysuria or increased frequency and bilharzia exposure, weight loss and anaemia.

Investigations:

- **Laboratory:** FBC, LFTs, urea, creatinine, urinalysis, culture and sensitivity, urine for cytology
- **Radiological:** Bimanual examination under anaesthesia at time of cystoscopy, CXR, IVU, abdominal and pelvic USS or CT scan of abdomen and pelvis..
- Biopsy is mandatory during cystoscopy.

Histology: SCC or Transitional cell carcinoma (TCC).

Staging: TNM

Referral: All patients must be referred to tertiary hospital for evaluation and decision on mode of treatment. Decisions of treatment for urinary bladder tumour are best discussed at Tumour board.

Treatment:

- **Surgery:** Total cystectomy is mutilating and causes poor quality of life. This has been used for early bladder cancers.
- **Chemoradiation** yields better results and preserves bladder function.
- **Palliative RT aims** at reducing pain and massive bleeding.

7.2 Carcinoma of the Prostate

Carcinoma of prostate is among the commonest of all cancers in men and is the third largest cause of death from cancer in males. Prostate cancer is associated with circulating testosterone and family history is significant in a very small percentage of patients.

Clinical features:

- Early stages of this cancer is asymptomatic, meaning that it can run an indolent course
- May present incidentally following examination for benign prostatic hypertrophy or elevated serum prostatic specific antigen (PSA).
- Prostatic symptoms are associated with advanced stages of the disease, which include: reduced potency, urinary frequency and nocturia, poor stream, hesitancy and terminal dribbling. However, very often patient may present with bone pain – backache or pathological fracture.
- DRE typically reveals a hard, irregular prostate. TURP is carried out to both confirm the diagnosis and also as part of the treatment (to relieve obstruction).

Investigations:

- **Laboratory:** FBC, LFTs, urea, creatinine, serum PSA, acid phosphatase,
- **Radiological:** X-rays of the painful bone or spine, IVU, abdominal USS or transrectal ultrasound, bone scan.
- **TURP** for histological confirmation

Histology: All are adenocarcinomas. Histological grading is a good predictor of survival. Gleason score provides prognostic information in addition to clinical stage.

Staging: TNM

Treatment

- Treatment is based on the stage of the disease, patient's age and functional performance of that individual
- Surveillance: Non-intervention may be appropriate particularly in an elderly man with limited life expectancy
- **Surgery:** Early stages can be treated with either radical prostatectomy with intention of Cure. However, surgery may cause postoperative impotence and impaired urinary control. Bilateral orchidectomy is a surgical procedure which aims at surgical castration
- **Hormonal therapy:** May be given as the sole treatment for patients deemed unfit for surgery. Alternatively hormonal therapy is used as adjunct to other treatments with the intention of reducing the chance of local recurrence or metastatic disease.
Androgen ablation/medical castration: inj. Zoladex 3.6md s/c every 28 days + Tab Casodex 50 mg od PO daily until PSA normalizes.
- **Radiotherapy:** Early stages can also be treated with radical RT. Palliative radiotherapy is valuable to bone metastases, massive haematuria, spinal cord compression, pathological fracture, etc as indicated.
64Gy/32F/6.5wks
- **Chemotherapy:** Some chemo drugs have shown effect on refractory prostatic disease:
S:Docetaxel 135mg/sq m i/v infusion D1 after premedication cover x 6 cycles.
- **Cancer of prostate medical emergency:** Spinal cord compression: Steroids-

S:Dexamethasone 8mg 8hrly x 72 hrs then orally x 10 days

Plus

S: Radiotherapy to the affected area

Plus

S: Intensive physiotherapy.

Detection/Prevention: Prostate cancer is among the cancers in human beings which could be prevented by screening procedures. Annual check up for a man 50 years and above is mandatory. Digital rectal examination (DRE) coupled with PSA check is enough to control incidence of this killer disease in men.

8.0 LYMPHOMAS

8.1 Non Hodgkin's Lymphoma (NHL)

It is a cancer that starts in cells called lymphocytes, which are part of the body's immune system. Lymphocytes are in the lymph nodes and other lymphoid tissues (such as the spleen and bone marrow).

Clinical features:

- Peripheral lymph node enlargement (commonest site- neck)

- Hepatomegally and/or splenomegally in advanced stages.
 - B-symptoms: Unexplained [weight loss](#), [fever](#), night sweats
 - Coughing, trouble breathing, or [chest pain](#) in case of SVCO.
 - Weakness and tiredness that don't go away (advanced disease)
 - [Pain](#), swelling, or a feeling of fullness in the abdomen (advanced disease)
- NOTE: 1. Presence of B symptoms signifies disease aggressiveness.

2. NHL is an AIDS-defining malignancy

Investigations:

- **Laboratory:** FBC, ESR,, Urea & creatinine, LDH, serum immunoglobulins, LFTs
- **Radiological:** CXR, Bone marrow aspirate/trephine, abdominal USS, CT of thorax/abdomen/pelvis,
- **Other:** baseline ECG.
- **Biopsy** for histological confirmation

Histology

- Low grade malignancy
- Intermediate grade
- High grade
- Other: Mycosis fungoides, extramedullary plasmacytoma

Staging: Ann Arbor classification

Referral: All patients must be referred to tertiary hospital for evaluation and decision on treatment. Decisions of treatment for NHL are best discussed at Tumour board.

Treatment: Depends on the disease stage.

Curative:

- **RT is directed to genuinely stage IA and IIA disease.**
 - Mantle or inverted Y: 40Gy/20F/4weeks with shielding of the critical organs.
 - Involved field RT (IFRT): 45Gy/23F/4.5wks
- **Chemotherapy:** for cure Stages IIB- IV disease + RT to bulk sites especially mediastinum
 - R-CHOP is the 1st line management for NHL.
 - Chemotherapy regimen for R-CHOP:
 - S:** Rituximab 500mg i/v infusion in 100mls N/S to run for 1 hr. (Repeat every 2 wks x 12 cycles)
 - Plus**
 - S:** Adriamycin 40mg/ m sq i/v D1
 - Plus**
 - S:** Cyclophosphamide 750mg/m aq i/v D1

Plus

S: VCR 1.4mg/m² sq i/v D1

Plus

A: Prednisolone 100mg/sq m po D1- D5 (25mg qid D1- D5)

- **Repeat cycle every 21 days x 6**

Note: The treatment should be given in hospital in order to combat unforeseen incidents like allergic (anaphylactic) reactions.

Oncological emergency: Superior vena cava obstruction (SVCO), profuse bleeding, increased intracranial pressure (ICP). Prompt action is mandatory.

8.2 Hodgkin's disease (HD)

The incidence of HD steeply rises from the age of 10 – 20 years. Then there is a slight fall in the middle age, following by a rise after 50 years.

Clinical features:

Enlarged, painless l/nodes in the neck or elsewhere.

B symptoms (weight loss, night sweats, and fever), pruritus, alcohol induced pain, general condition, throat, lymphnodes (site, number, size, consistency, mobility, matting), respiratory system, abdomen (liver, spleen, other masses), bone tenderness.

Investigations:

- **Laboratory:** FBC, ESR,, U & Es, LDH, serum immunoglobulins, bone marrow trephine and biopsy, LFTs,
- **Radiological:** CXR, abdominal USS, CT of thorax/abdomen/pelvis,
- **Biopsy** for histological diagnosis
- **Other:** baseline ECG.

Histology: There are 4 types histology in HD:

- Lymphocyte predominant
- Nodular sclerosing
- Mixed cellularity
- Lymphocyte depleted

Staging: Ann Arbor classification

Each stage is denoted either with: A= No B symptoms; B= Presence of B symptoms

Referral: All patients must be referred to tertiary hospital for evaluation and decision on treatment. Decisions of treatment for HD are best discussed at Tumour board.

Treatment:

- **Chemotherapy:** Aim: Cure for any stage of the disease.
Indication for chemotherapy: Stages II- IV.

Chemotherapy regimen: ABVD:

S: Adriamycin 40mg/sq m i/v D1

Plus

S: Bleomycin 10mg/sq m i/v D1

Plus

S: Vincristine 1.4mg/sq m i/v D1

Plus

S: Dacarbazine 450mg/sq m i/v D1

- **Repeat cycle every 21 days x 6**

Note: The treatment should be given in hospital in order to combat unforeseen incidents like allergic reactions.

- **Radiotherapy:** Can be the 1st line treatment for early stage I disease – Treating involved field RT only. RT radiotherapy for stages I and IIA – mantle or inverted Y: 40Gy/20F/4wks.

9.0 COMMONEST PAEDIATRIC MALIGNANCY

91 Burkitts Lymphoma (BL)

It is a cancer of lymphatic system (in particular B lymphocytes). Burkitt's tumour is an undifferentiated lymphoblastic lymphoma. It shows close association with the Epstein Barr virus infection. Peak onset age: 6 – 10 years.

Clinical feature:

May first be noticed as a painless swelling of the facial bone or jaw which is typical presentation in equatorial Africa setting. This tumour can grow very rapidly. Paraplegia and/or cranial nerve palsy is a result of disease spread to the CNS.

Investigations:

- **Laboratory:** FBC, biochemical profile, serum acid phosphatase,
- **Radiological:** x-rays of the jaw, abdominal USS,
- FNAC. This is typically a B cell lymphoma

Staging: A, B, C and D staging system; where A and B represent early disease stage and C and D – advanced disease stage.

Referral: Early detection and urgent referral to specialized centre).

Treatment:**Treatment of choice is chemother**

- Curative treatment comprises of combination chemotherapy- COMP:
- Palliation is good but with temporary benefit only. Palliation may include some form of CNS prophylaxis in case of symptoms.
- Surgery and radiotherapy has no place in the management of this childhood tumour.

Oncological emergency: Tumour lysis syndrome: This must be prevented by issuing enough fluids before and during treatment PLUS allopurinol 100mg od PO daily in the days of treatment.

9.2 Wilm's tumour

This is a common primary malignant renal tumour of childhood younger than 5 years of age. Children with this disease may have some associated anomalies such as: Aniridia, hemihypertrophy, cryptorchidism and hypospadias.

Clinical feature: Abdominal swelling/mass. Rarely abdominal pain or gross haematuria. 25% of cases may be hypertensive as a result of associated rennin secretion.

Investigations:

- **Laboratory:** FBC, LFTs, urea, creatinine, uric acid,
- **Radiological:** radiographic and bone scan where symptomatic, IVU to assess renal calyces and/or filling defects,
- **Other:** Cystoscopy.
- **FNAC** to confirm diagnosis.

Staging: Surgery plays a major role in tumour removal, tumour staging and confirmation of diagnosis as well as visualization of whole abdomen.

Referral: Urgent referral to a specialized centre).

Treatment: Multimodality approach

- RT is used to control microscopic disease after surgery or to treat distant metastases.
- Chemotherapy is used for advanced disease.

9.3 Neuroblastoma

Is a childhood malignancy, majority of cases occurring below 4 years of age
It arises from neural crest tissue in adrenal medulla and sympathetic ganglia.

Clinical features: Manifest according to the site: Abdominal swelling/mass, neurological deficit in case of paravertebral tumours, orbital swelling, and skin lesions.

Investigations:

Laboratory: FBC, LFTs, blood chemistry, ESR, urinalysis, 24-hr urine sample for quantitation of excretion of catecholamines, bone marrow trephine and biopsy

Radiological: CXR, abd/pelvic USS, CT scan of abdomen, bone scan in case of symptoms.

Histopathology: Is by surgical plus CT findings

Staging: Is by International neuroblastoma staging system (INSS). Stage I & II- early disease stage; stage III & IV- advanced disease.

Referral: Urgent referral to a specialized centre

Treatment: Combined modality approach:

Surgery: Is for early disease or organ preservation.

Chemotherapy: Pre-operative chemotherapy to down-stage the tumour
Stage IV treatment is individualized.

9.4 Retinoblastoma (RB)

Is a most common childhood malignancy at ORCI. Average age younger than 5 years – 90%. Majority acquire RB sporadically; however 10% of the cases are hereditary. RB is a neuro-ectodermal tumour in the retina.

Clinical feature: “Cat’s eye reflex” or white pupil; rarely orbital inflammation or proptosis

Investigations:

- **Laboratory:** FBC, LFTs, urea, creatinine, serum CEA & alpha feto protein, lumbarpuncture for CSF evaluation,
- **Radiological:** MRI/CT scan of brain and orbit, CXR and/or x-ray of spine in case of symptoms.
- **Ophthalmic EUA** is mandatory PLUS complete neurological evaluation.

Staging: Localised in the retina vs brain involvement (through optic nerve)

Referral: Urgent referral to a specialized centre

Treatment:

Surgery: Enucleation plus as long a segment of the optic nerve as possible.

RT: Is indicated to the tumour bed/residual disease.

Photocoagulation, cryotherapy, plaque RT are for selected cases only.

Chemotherapy: Is indicated for advanced disease only.

9.5 Ewing sarcoma

It is primarily a bone tumour in childhood. Peak incidence 10 – 20 years of age.

M:F ratio 5:1

Clinical features: Local pain, tender warm and swollen area over the region of the affected bone (usually midshaft – diaphysis of the long tubular bones (femur). Symptoms mimic infection such as osteomyelitis. As such constitutional symptoms are frequent. Chest signs particularly in case of pulmonary involvement.

Investigations:

- **Laboratory:** FBC, LFTs, urea, creatinine, ESR, ALP,
- **Radiological:** Plain x-ray of whole bone, MRI/CT of the entire limb.
- **Biopsy** of suitable soft tissue mass is preferred to avoid bone complications.
- Biopsy is the only pathognomonic for Ewing sarcoma. Is usually undifferentiated sarcoma.

Staging: No established staging system.

Referral: Urgent referral to a specialized centre.

Treatment: Aim: Cure

Surgery: Lesions amenable to wide excision without causing severe functional disabilities are resected.

Chemotherapy: Is indicated in all cases.

Chemotherapy regimen: VAC

S: Vincristine 1.5mg/m sq D1

Plus

S: Adriamycin 30mg/m sq D1, D2

Plus

S: Cyclophosphamide 1 g/m sq D1

Repeat every 3 wks x 12 cycles

- When total dose of Adriamycin reaches 400 mg/m sq, substitute with Actinomycin D 15 microgram/kg/day D1 – D3 is used.
- During RT give only VCR and cyclophosphamide 3 wkly, omitting ACT-D or ADM.

Radiotherapy:

- Bulky lesions may be treated with chemoradiation.
- Shrinking field technique is used. Whole done is irradiated to 45Gy/25F/5wks, then reduced field to bulk site. A further 20GY/10F/2wks.

- In case of distant metastases, individual chemotherapy and local RT may be given. Further treatment depends on response and clinical status.

CHAPTER SEVENTEEN

MALARIA

1.0 Overview of Malaria

Malaria is a parasitic infectious disease presenting with fever, chills and profuse sweating. However, patient with malaria infection may be completely asymptomatic.

Diagnosis

The clinical features of malaria vary from mild to severe. The disease presentation will vary according to patient's state of immunity, the intensity of the infection and the presence of accompany conditions such as malnutrition, anaemia and other diseases.

Signs and Symptoms includes:-

malaise, fever, fatigue, muscle pain, nausea, anorexia, chill, rigors, sweats, headache, cough, vomiting and diarrhea etc.

The above signs and symptoms are not specific for malaria and can be found in other disease conditions. Therefore it is necessary to investigate for other causes of febrile illness.

Laboratory investigation is mandatory and urgent for all patients admitted with severe malaria. Parasite-based diagnosis by microscopy is important while rapid diagnostic tests (RDTs) may be an alternative. Laboratory tests should be interpreted in conjunction with clinical findings.

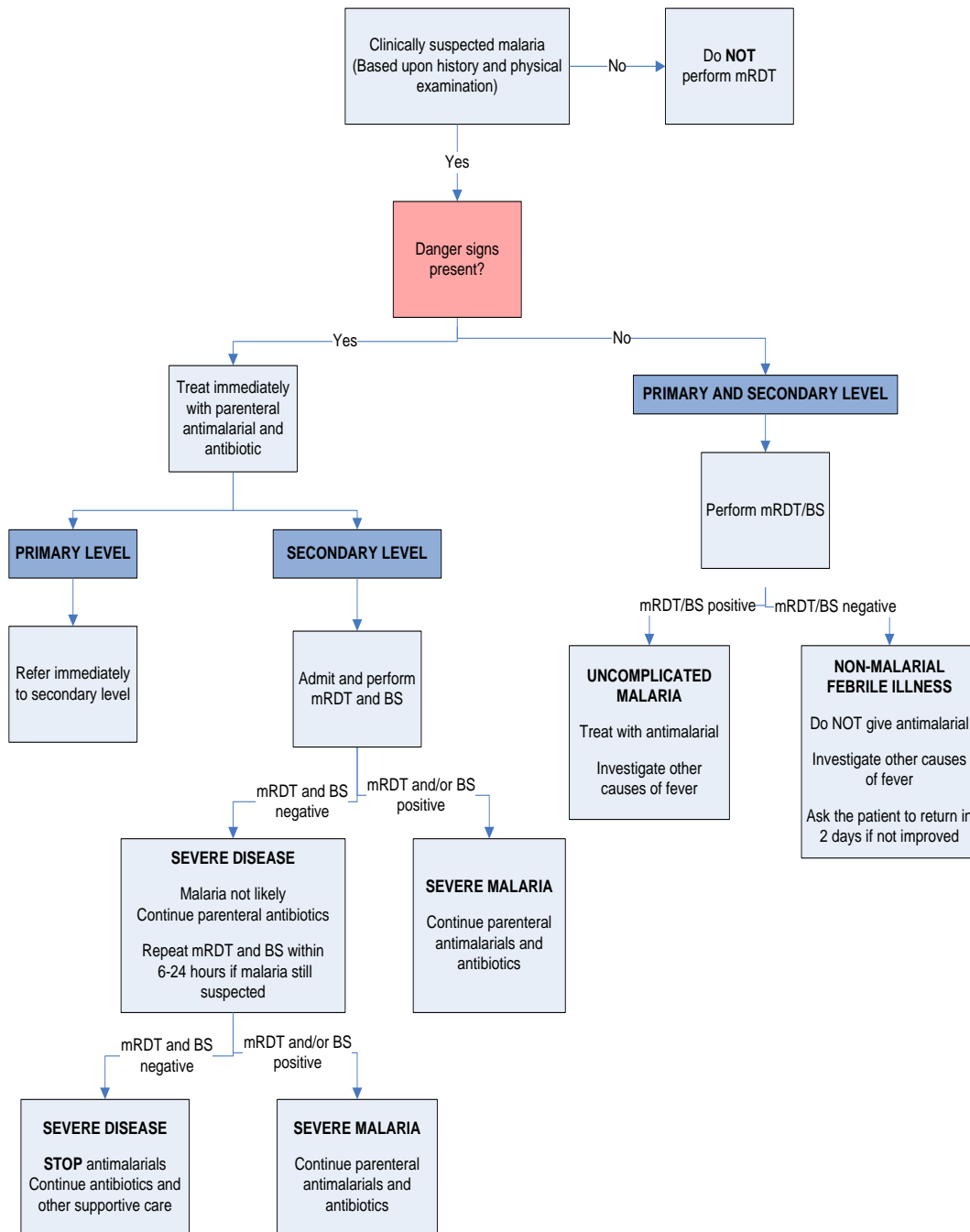
Management

The management and referral for patient with malaria will be determined by the clinical presentation and the diagnosis of either uncomplicated or severe disease, as well as results of RDT and/or microscopy (see flow chart -[Figure 1](#)).

In children under five years of age, IMCI practical algorithms for management of sick child with fever should be used to ensure full assessment and appropriate case management of children, in particular at the primary level health.

In the case of negative blood slide/RDT without signs or symptoms of severe disease, look for other causes, manage and follow up accordingly, and ask the patient to come back if condition does not improve. The exception is in children under 5 years living in high malaria transmission areas, if unable to return for follow up or in case the condition worsens, treat as for uncomplicated malaria.

Figure1: Management of suspected malaria based on both clinical presentation and laboratory investigations



2.0 Treatment of Uncomplicated Malaria

Definition: Uncomplicated malaria is defined as symptomatic malaria without signs of severity or evidence (clinical or laboratory) of vital organ dysfunction.

Give antimalarial medicines only to those who test positive for parasites.

Treatment on the basis of clinical suspicion alone should only be considered if parasitological diagnosis is not accessible.

The objectives of treatment of uncomplicated malaria are:

- To provide rapid and long lasting clinical and parasitological cure
- To reduce morbidity including malaria related anaemia
- To halt the progression of simple disease into severe and potentially fatal disease

Since the progression towards severe and fatal disease is rapid, especially in children under five years of age, it is recommended that diagnosis and initiation of treatment of uncomplicated malaria should be within 24 hours from the onset of symptoms.

First line:

Artemether Lumefantrine (ALu).

- Standard tablet: fixed formulation Artemether 20mg, Lumefantrine 120mg
- Dispersible tablet: fixed formulation for children, Artemether 20 mg, Lumefantrine 120mg

Dosage regimen

Table 1: Dosage of Artemether 20mg & Lumefantrine 120mg (ALu) tablets

Kg		Day 1		Day 2		Day 3		Colour Code	
		Dose	1st	2nd	3rd	4th	5th		6 th
		Hours	0 (*)	8	24	36	48		60
up to 15	0 to 3 years	1	1	1	1	1	1	Yellow	
15 up to 25	3 years up to 8 years	2	2	2	2	2	2	Blue	
25 up to 35	8 years up to 12 years	3	3	3	3	3	3	Red	
35 and above	12 years and above	4	4	4	4	4	4	Green	

The first dose should be given as direct observed treatment (DOT); the second dose should strictly be given after 8 hours; subsequent doses could be given twice daily (morning-evening)

in the second and third day of treatment until completion of 6 doses.

Note: Artemether-Lumefantrine is not recommended for:

- **Infants below 5kg body weight:**

Malaria is quite uncommon in infants below 2 months of age (approximately below 5 kg). Rarely, congenital and neonatal malaria does occur. ALu is currently not recommended for infant below 5kg body weight because the dosing and safety profile of the partner component lumefantrine is not well studied. Therefore, an artemisinin alone is the drug of choice as 1st line treatment in the category of neonates and infants below 5Kg, treating as for severe malaria. Injectable quinine remains a suitable alternative where artesunate is not available. See section on Treatment of Severe Malaria for dosage of parenteral artesunate.

- **First trimester of pregnancy:** See section on Malaria in pregnancy

During the second and third trimesters of pregnancy Artemether-Lumefantrine should be used as drug of choice for treatment of uncomplicated malaria

As far as possible malaria cases should be followed up on the third day if symptoms persist or immediately if the condition worsens. Failure to respond to the recommended drug regimen indicates the need for further investigations and appropriate management, with referral if needed.

Where a patient returns between 4 to 14 days after treatment with ALU complaining of continued symptoms of malaria, non-response should be considered and the following recommendations followed after a full history and examination:

- Where laboratory facilities are not available and malaria is still suspected, second line treatment should be started immediately with strict follow up
- Where laboratory facilities are available, a blood smear (and not RDT) should be examined. If parasites are found second line treatment should be started and treatment failure recorded. If parasites are not found other causes for the symptoms should be sought and treated accordingly

Second line for uncomplicated malaria:

Dihydroartemisinin plus Piperaquine (DPQ)

- Fixed-dose combination with tablets containing

C: Dihydroartemisinin (D) and Piperaquine (PQ).

40 g D + 320 mg PQ

20 mg D + 160 mg PQ

Dosage regimen

Table 2: Dosage of DPQ for defined categories by body weight

Body Weight (kg)	Daily dose (mg)		Tablet strength and number of tablets per dose
	Piperaquine	Dihydroartemisinin	
5 to <7	80	10	½ x 160mg / 20mg tablet
7 to <13	160	20	1 x 160mg / 20mg tablet
13 to <24	320	40	1 x 320mg / 40mg tablet
24 to <36	640	80	2 x 320mg / 40mg tablets
36 to <75	960	120	3 x 320mg / 40mg tablets
75 to 100	1,280	160	4 x 320mg / 40mg tablets

Based on 4 mg/kg/day Dihydroartemisinin and 18 mg/kg/day Piperaquine once a day for 3 days

3.0 Treatment of Severe Malaria

Severe *Plasmodium falciparum* malaria is a medical emergency. Delay in diagnosis and provision of appropriate treatment may lead to serious complications and even death. In Tanzania the commonest presentations of severe malaria are severe anaemia and coma (cerebral Malaria). Complications include hyperpyrexia, convulsions, shock, hypoglycaemia, metabolic acidosis, acute renal failure or pulmonary oedema

Early diagnosis of severe malaria based upon a complete history, physical examination and where possible, blood smear or rapid diagnostic test (RDT) examination for malaria parasites. Taking and reporting of blood smear must not be allowed to delay treatment unduly.

Definition: In a patient with *P.falciparum* asexual parasitaemia and no other obvious cause of symptoms the presence of one or more of features listed below classify the patient as suffering from severe malaria.

Table 3: Features of severe malaria

Clinical features	Description/criteria
Prostration/extreme weakness	Unable to stand or sit up without support
Impaired consciousness	Altered level of consciousness Acute confusional state, coma
Change of behaviour	Hallucinations, delusions, agitation
Convulsions	Repetitive abnormal muscular movements
Respiratory distress (due to lactic acidosis and/or pulmonary oedema)	Acidotic breathing: deep and laboured breathing Pulmonary oedema: laboured breathing, restlessness, blood stained frothy sputum especially in adults
Bleeding tendency/DIC	Easy/prolonged bleeding
Jaundice	Yellow colouration of mucus membranes
Circulatory collapse/shock	Low systolic BP and fast pulse rate
Vomiting everything	Throwing up after every feed/drink
Inability to drink or breast feed	Not able to swallow

NOTE: If effective management of severe malaria and supportive care for complications is not possible, patients should be given pre-referral treatment and referred immediately to an appropriate facility for continued treatment.

Pre-referral treatment options:

B: Artesunate IM/rectal

OR

B: Quinine IM

Rectal artesunate is the recommended pre-referral treatment at the community level. At a health facility the pre-referral dose of parenteral therapy should be initiated without delay.

Pre-referral rectal artesunate:

- Available as suppository containing 50mg or 100mg or 400mg

Dosage regimen:

Single dose of 10 mg/kg body weight artesunate should be administered rectally. In the event that an artesunate suppository is expelled from the rectum within 30 min of insertion, a second suppository should be inserted and, especially in young children, the buttocks should be held together for 10 min to ensure retention of the rectal dose of artesunate.

Table 4: Dosage for initial (pre-referral) treatment using rectal artesunate

Weight (Kg)	Age	Artesunate dose (mg)	Regimen (single dose)
5-8.9	0-12 months	50	One 50 mg suppository
9-19	13-42 months	100	One 100 mg suppository
20-29	43-60 months	200	Two 100 mg suppository
30-39	5-13 years	300	Three 100 mg suppository
40-59	>14 years	400	One 400 mg suppository
60-80	>14 years	800	Two 400 mg suppository
>80	>14 years	1200	Three 400 mg suppository

Pre-referral artesunate IM:

- Artesunate is provided as a powder vial of artesunic acid with a 1ml ampoule of diluent sodium bicarbonate solution 5%. Reconstitute for IM injection:
 - The vial of artesunate powder is mixed with 1ml of diluent sodium bicarbonate solution to form sodium artesunate. Shake for 2-3 minutes until completely dissolved and solution is clear. The solution is 60mg/ml artesunate
 - Dilute with **2ml** of 5% dextrose or dextrose/saline. The concentration is now 20mg/ml artesunate.

One vial makes 3ml solution (20mg/ml) for **IM** injection. Use immediately; discard any solution not used within 1 hour.

Dosage regimen:

Single dose of 2.4 mg/kg body weight administered by intramuscular injection to the anterior thigh after reconstituted and diluted as directed.

Table 5: Dosage for initial (pre-referral) treatment using artesunate IM (20mg/ml solution)

Weight (Kg)	Age	Artesunate dose in ml (Solution 20mg/ml)
<5	0-xx months	0.5 ml
5-8	xx-xx months	1 ml
9-12	xx-xx months	1.5 ml
13-16	xx-xx months	2 ml
17-20	xx-xx months	2.5 ml
21-25	xx-xx months	3 ml
26-29	xx-60 months	3.5 ml
30-33	5-xx years	4 ml
34-37	x-xx years	4.5 ml
38-41	x-xx years	5 ml
42-45	>14 years	5.5 ml
46-50	>14 years	6 ml
51-54	>14 years	6.5 ml
55-58	>14 years	7 ml
59-62	>14 years	7.5 ml
63-66	>14 years	8 ml
67-70	>14 years	8.5 ml
71-75	>14 years	9 ml
76-79	>14 years	9.5 ml
80-83	>14 years	10 ml
84-87	>14 years	10.5 ml
88-91	>14 years	11 ml
92-95	>14 years	11.5 ml
96-100	>14 years	12 ml

Pre-referral Quinine IM:

- Dilution of Quinine Dihydrochloride injection (300 mg/ml) for intra-muscular use:

One part of Quinine solution should be diluted with four parts water for injection to a concentration of 60 mg/ml. This dilution will minimize the risk of sterile abscess formation.

Dosage regimen:

Give single dose of 10mg of quinine salt per kg bodyweight (not exceeding a maximum dose of 600mg). The calculated dose should be divided into two halves and then administered by deep intra-muscular injection preferably into the mid anterolateral aspect of the thigh (one injection on each side).

Table 6: Dosage for initial (pre-referral) treatment using intramuscular quinine (IM)

Age (years)	Weight (Kg)	Volume of undiluted Quinine (300 mg/ml)	Volume of diluents (to add to each dose)	Total volume of diluted Quinine (60 mg/ml)
2 up to 4 months	4 up to 6	0.2 ml	0.8 ml	1.0 ml
4 up to 9 months	6 up to 8	0.3 ml	1.2 ml	1.5 ml
9 up to 12 months	8 up to 10	0.4 ml	1.6 ml	2.0 ml
12 months up to 3yrs	10 up to 14	0.5 ml	2.0 ml	2.5 ml
3 up to 5	15 up to 19	0.6 ml	2.4 ml	3.0 ml
5 up to 8	19 up to 25	0.7 ml	2.8 ml	3.5 ml
8 up to 12	25 up to 35	1.0 ml	4.0 ml	5.0 ml
12 up to 14	35 up to 50	1.4 ml	5.6 ml	7.0 ml
14 up to 16	50 up to 60	1.8 ml	7.2 ml	9.0 ml
16 and above	60 and above	2.0 ml	8.0 ml	10.0 ml

Refer with clinical summary to the nearest hospital when clinical need dictates (e.g. blood transfusion or intensive care)

Treatment of Severe Malaria

Treatment in both children and adults where facilities for admission and effective management of severe malaria are available:

First choice:

Artesunate 2.4 mg/kg body weight IV or IM given on admission (time = 0), then at 12 hours and 24 hours, then once a day.

Artesunate (i.v. infusion)

- Artesunate is provided as a powder vial of artesunic acid with a 1ml ampoule of diluent sodium bicarbonate solution 5%. Reconstitute for **IV** injection:
 - The vial of artesunate powder is mixed with 1ml of diluent sodium bicarbonate solution to form sodium artesunate. Shake for 2-3 minutes until completely dissolved and solution is clear. The solution is 60mg/ml artesunate
 - Dilute with **5ml** of 5% dextrose or dextrose/saline. The concentration is now **10mg/ml** artesunate.

One vial makes 6ml solution (10mg/ml) for **IV** injection. Use immediately; discard any solution not used within 1 hour.

Table 7: Dosage for treatment using intravenous artesunate (IV; 10mg/ml solution)

Weight (Kg)	Age	Artesunate dose in ml (Solution 10mg/ml)
<5	0-xx months	1 ml
5-8	xx-xx months	2 ml
9-12	xx-xx months	3 ml
13-16	xx-xx months	4 ml
17-20	xx-xx months	5 ml
21-25	xx-xx months	6 ml
26-29	xx-60 months	7 ml
30-33	5-xx years	8 ml
34-37	x-xx years	9 ml
38-41	x-xx years	10 ml
42-45	>14 years	11 ml
46-50	>14 years	12 ml
51-54	>14 years	13 ml
55-58	>14 years	14 ml
59-62	>14 years	15 ml
63-66	>14 years	16 ml
67-70	>14 years	17 ml
71-75	>14 years	18 ml
76-79	>14 years	19 ml
80-83	>14 years	20 ml
84-87	>14 years	21 ml
88-91	>14 years	22 ml
92-95	>14 years	23 ml
96-100	>14 years	24 ml

Alternative if parenteral artesunate is not available:

Quinine 20 mg salt/kg body weight (BW) on admission (IV infusion or divided IM injection), then 10 mg/kg BW every 8 hours; infusion rate should not exceed 5 mg salt/kg BW per hour.

Quinine (i.v. infusion)

Quinine dose: 10 mg/kg body weight of salt, to be diluted in 5-10 ml/kg body weight of 5% Dextrose or dextrose-saline and infused over 4 hours and repeated every 8 hours. Infusions should be discontinued as soon as the patient is able to take oral medication. Patients should be properly instructed to complete the 7-day treatment with quinine tablets or, alternatively, a full course of ALu may be administered to complete treatment

The **drop rate** for quinine IV infusion is calculated as follows:

Drop rate per minute = amount of fluid to be infused (in ml) x 20 (drop factor) / time period to be infused (in minutes)

The table below is given for easier calculation.

Table 8: Dilution schedule and drop rate for intravenous Quinine administration

Age (years)	Weight(kg)	Quinine dose	Volume of undiluted quinine solution (300mg/ml)	Amount of fluid to be infused (in 4 hours)	Drop rate per minute
2 up to 4 months	4 up to 6	60 mg	0.2 ml	50 ml	4 drops
4 up to 9 months	6 up to 8	90 mg	0.3 ml	100 ml	8 drops
9 up to 12 months	8 up to 10	120 mg	0.4 ml	100 ml	8 drops
12 up to 3yrs	10 up to 14	150 mg	0.5 ml	100 ml	8 drops
3 up to 5	15 up to 19	180 mg	0.6 ml	150 ml	13 drops
5 up to 8	19 up to 25	210 mg	0.7 ml	200 ml	17 drops
8 up to 12	25 up to 36	300 mg	1.0 ml	250 ml	21 drops
12 up to 14	36 up to 50	420 mg	1.4 ml	350 ml	30 drops
14 up to 16	50 up to 60	540 mg	1.8 ml	500 ml	42 drops
16 and above	60 and above	600 mg	2.0 ml	500 ml	42 drops

Give parenteral antimalarials in the treatment of severe malaria for a minimum of 24 h, once started (irrespective of the patient's ability to tolerate oral medication earlier), and, thereafter, complete treatment by giving a complete course (3 days) of:

A: Artemether plus lumefantrine (ALu),

OR

C: Dihydroartemisinin plus piperazine (DPQ),

General measures for severe malaria

- Treatment of hypoglycaemia. Hypoglycaemia remains a major problem in the management of severe malaria especially in young children and pregnant women. It should be deliberately looked for and treated accordingly.
- **Coma** (cerebral malaria): maintain airway, nurse on side, and exclude other causes of coma (e.g. hypoglycaemia, bacteria meningitis); avoid giving corticosteroids
- **Hyperpyrexia**: fanning, paracetamol (preferred over NSAIDs)
- **Convulsions**: maintain airways; treat with rectal or IV diazepam.
- **Hypoglycaemia**: urgent and repeated blood glucose screening;
- In children: give 5 mls/kg of 10% dextrose OR 2.5 mls/kg of 25% dextrose as bolus; if 50% dextrose solution is available, it should be diluted to make 25% by adding an equal volume of water for injection or normal saline
- In adults: give 125 mls of 10% dextrose OR 50 mls of 25% dextrose dextrose as bolus
- Where dextrose is not available, sugar water should be prepared by mixing 20 gm of sugar (4-level tea spoons) with 200 ml of clean water. 50 ml of this solution is given ORALLY or by naso-gastric tube if unconscious
- **Severe anaemia**: transfusion of packed cells if Hb equal or less than 4 g/dl and/or signs of heart failure and/or signs of respiratory distress
- **Acute pulmonary oedema**: Prop patient up to 45 degree angle; review fluid balance and run patient on "dry side"; give diuretic but avoiding inadequate perfusion of kidneys; set up Central Venous pressure (CVP) line, give oxygen. Intubation/ventilation may be necessary

- **Acute renal failure:** exclude pre-renal causes, check fluid balance and urinary sodium. If adequately hydrated (CVP>5cm) try diuretics. Haemodialysis /haemofiltration (or if available peritoneal dialysis) should be started early in established renal failure.

4.0 Management of malaria in pregnancy

Malaria is an important cause of morbidity and mortality for the pregnant woman, the foetus and the newborn. The effects of malaria in pregnancy are related to the malaria endemicity, with abortion more common in areas of low endemicity and intrauterine growth retardation more common in areas of high endemicity. Early diagnosis and effective case management of malaria illness in pregnant women is crucial in preventing the progression of uncomplicated malaria to severe disease and death.

4.1 Management of uncomplicated malaria in the first trimester

If a laboratory blood slide is negative, it does not rule out malaria. RDTs have an added value, as they can be positive even if parasites are hidden in the placenta.

Note: During the second and third trimesters of pregnancy Artemether-Lumefantrine is the drug of choice for treatment of uncomplicated malaria

First trimester:

During the first trimester of pregnancy, treat with quinine plus clindamycin for seven days or quinine alone if clindamycin is not available or unaffordable.

Quinine is safe in pregnancy. In therapeutic doses it does not induce labour. Uterine contractions and foetal distress with the use of quinine may be attributable to fever and effects of malaria disease. Clindamycin is considered safe in the first trimester of pregnancy. At present, artemisinin derivatives cannot be recommended in the first trimester of pregnancy. However, they should not be withheld if treatment is considered life saving for the mother, and other suitable antimalarials are not available. For dosage of quinine, see section on treatment of severe malaria.

S: Clindamycin dosage: 10mg/Kg (O) twice daily for 7 days.

Note: Lactating women should receive the recommended antimalarial treatment (including ALu)

4.2 Management of severe malaria in pregnancy

Pregnant women infected with malaria are more susceptible to develop severe malaria. They commonly present with one or more of the following signs/symptoms: high fever, hyperparasitemia, low blood sugar, severe haemolytic anaemia, cerebral malaria, pulmonary oedema.

The management of severe malaria in pregnant women does not differ from the management of severe malaria in other adult patients, except pregnant women in the first trimester. (See section on Treatment of Severe Malaria).

The risk of quinine induced hypoglycaemia is greater in pregnant than non-pregnant women. Blood sugar should be monitored regularly and if falls below 2.5 mmol/L (< 45 mg/dl) give IV 10% or 25% dextrose. While the patient is on IV Quinine treatment, pay

particular attention to the feeding of the patient.

4.3 Intermittent preventive treatment in pregnancy (IPTp)

The drug of choice for IPTp is **Sulfadoxine/Pyrimethamine (SP)**. SP remains the drug of choice for IPTp even though it is no longer the first line drug for malaria treatment. This is because the aim of IPTp is to prevent the worst effects of infection, rather than to cure a potentially life threatening illness. As such, lower efficacy antimalaria is acceptable for IPTp than for curative purposes. It is particularly important that drugs used in pregnancy are known to be safe. It is also likely that drugs with a long half-life are the most effective when used as IPTp.

The first IPTp dose is administered between 20-24 weeks of gestational age. The second IPTp dose should be administered at 28 – 32 weeks.

NOTE:

- IPTp should be administered as direct observed treatment (DOT) during an antenatal care visit
- Treatment or intermittent preventive treatment with sulfadoxine-pyrimethamine should not be given to HIV-infected patients receiving cotrimoxazole (trimethoprim plus sulfamethoxazole) prophylaxis.

CHAPTER EIGHTEEN

TUBERCULOSIS AND LEPROSY

1.0 TUBERCULOSIS

Tuberculosis is chronic airborne infection caused by mycobacterium tuberculosis also known acid fast bacilli; less frequent it can be caused by mycobacterium avium and mycobacterium africanus. It is a public health problem and all cases must be notified to the MoHSW.

Sings and symptoms

- Cough of more than two weeks
- Fever
- Excessive Night sweats
- Haemoptysis(sputum mixed with blood stains)
- Loss of weight

Types of Tuberculosis

- Pulmonary TB : Its most common and infectious affecting the lungs
- Extra pulmonary TB: occurs when bacteria spread outside the lung to cause damage in any organ such as meninges, lymphnodes, kidneys, spine, intestinal and ostearticular. it common among people with HIV/AIDS

Control of Tuberculosis

Important key points are:

- Treatment should be short, effective and provided free of charge
- TB services should reach all areas, integrated in PHC system and ensure widespread use of BCG vaccination and case finding (especially sputum positive patients)
- Priority should be on identifying and treat all infectious TB cases

Prevention

BCG vaccination is given at birth or at first contact with the child after birth. It is given intradermally on the right upper arm, above the insertion of the deltoid muscle.

NOTE: The batch number of the vaccine and the date has to be recorded on the antenatal card, dosages are recommended by EPI programme. BCG should be give to all babies having clinical signs of HIV infection

Non-healing ulcers after vaccination with BCG (up to 8 weeks) or regional lymphadenopathy can be treated with:

Isoniazid (O) 10 mg/kg once daily for two months

Case Management

Diagnosis

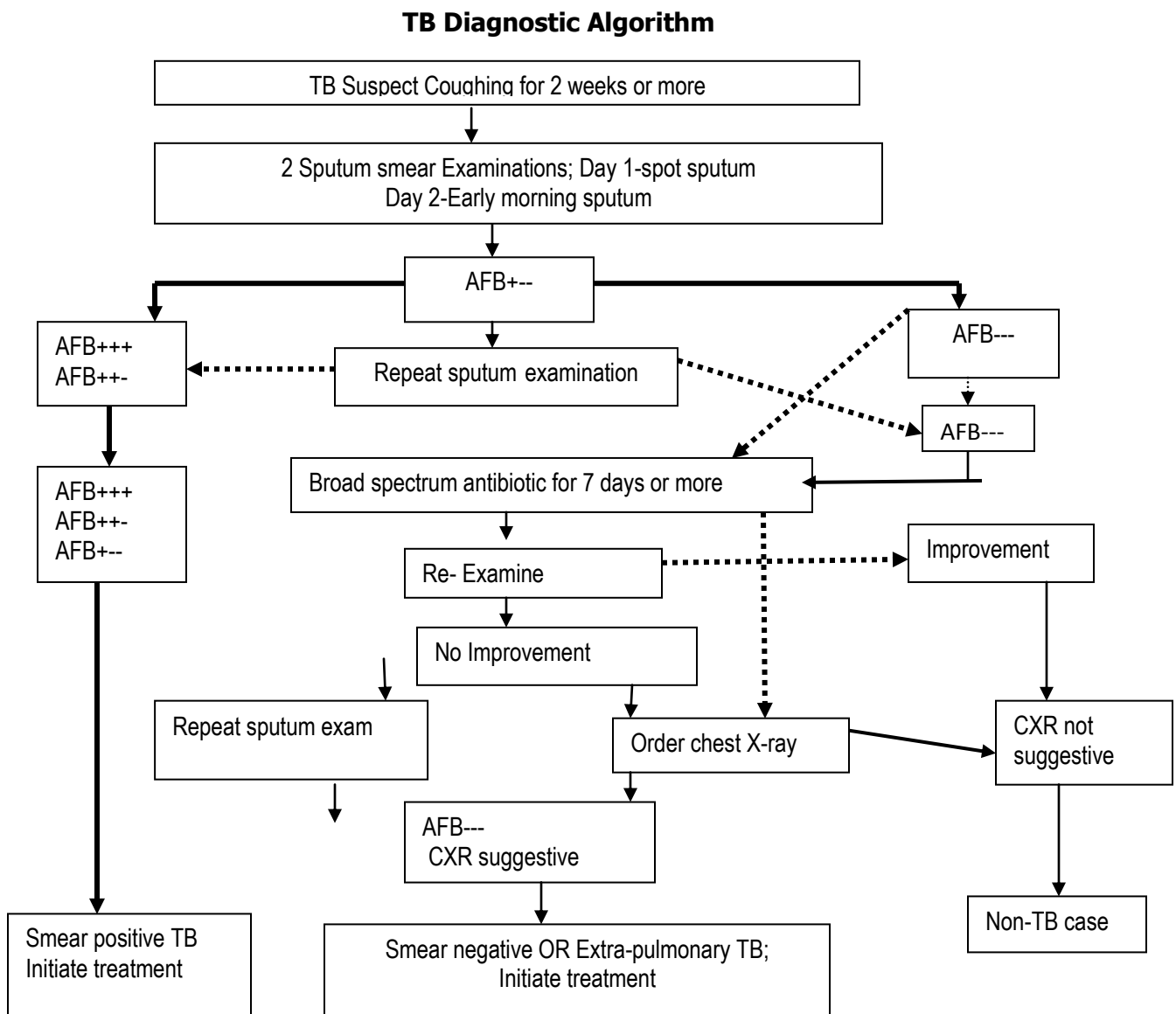
Sputum: Each patient should have direct smear microscopy (DSM) on two sputum specimens for diagnosis. DSM should be repeated at the end of the intensive phase to confirm sputum conversion. Sputum of TB patients MUST be sent or taken to the TB Reference Laboratory when:

- Sputum conversion to negative has not taken place
- There is concern that the patient has developed drug resistance
- All re-treatment and treatment failures
- Culture and sensitivities are required.

Chest X-rays: This has to be done upon

- Smear negative TB cases
- Completion of outpatient treatment

Figure:....



The diagnosis of TB in children can be very difficult owing to the wide range of symptoms. Sputum cannot often be obtained from children and in any case it is often negative even on culture. Symptoms in children are not typical. The diagnosis should therefore be based on clinical findings, family history of contact with a smear positive case, X-ray examination and tuberculin testing, culture (if available) and non-response to broad spectrum antibiotic treatment. A score chart below can help to reach the diagnosis of tuberculosis. Older children who are able to cough up sputum should go through the same assessment as adults using smear microscopy as the "gold standard".

Chart for the Diagnosis of TB in Children

SCORE IF SIGN OR SYMPTOM IS PRESENT						
	0	1	2	3	4	SCORE
GENERAL FEATURES						
Duration of illness	Less than 2 weeks	2-4 weeks		More than 4 weeks		
Failure to thrive or weight loss	Weight gain		No weight gain		Weight loss	
TB contact	None	Reported not proven		Proven smear+/EP	Proven smear +	
Tuberculin test				Positive		
Malnutrition				Not improved after 4 weeks		
Chronic infant disease				Not improved after 4 weeks		
Duration of illness		Recurrent		No response to antibiotics		

Chart for the Diagnosis of TB in Children (2)

	0	1	2	3	4	SCORE
LOCAL FEATURES						
Chest X-ray				TB suggestive features like infiltration, cavity or lymphnodes		
Lymph nodes				Cervical submandibular		
Swelling of bone or joint				Suggestive features on X-ray		
Ascites			Without abdominal mass	With abdominal mass		
Meningitis				Chronic CNS signs		
Angle deformity of the spine					X-ray features	
TOTAL SCORE						

Tuberculin Testing

The tuberculin skin test is valuable as a diagnostic tool in children, in child who did not receive a BCG vaccine an induration of 10mm or more interpreted as positive, if a child did receive a BCG, the induration should be at least 15mm to be positive

These results may indicate:

- Active infection (especially when strongly positive)
- Previous infection or
- Previous BCG

NOTE: Absence of a response does not exclude TB because individuals with HIV may not have sufficient immunity for a positive Mantoux test despite active TB

TB Treatment Categories

These guidelines are given according to the National TB and Leprosy programme (NTLP) which are;

Category	Patients
I	New sputum smear PTB (positive pulmonary TB) and new patients with severe forms of EPTB
II	Relapse Treatment failure and Sputum smear positive return after default
III	New sputum smear negative and EPTB (less severe forms)

TB patients are divided into three categories, treatment regimen, new smear positive tuberculosis other than smear forms of TB and children

Table: Treatment regimen category I & III

DURATION	DRUG	CHILD Pre-treatment weight			ADULTS Pre-treatment weight	
		5-10 kg	11 – 20 kg	21 – 30 kg	< 50kg	> 50kg
Two months of intensive phase, daily observed treatment	{RHZE} [150/75/400/275]	½	1	2	3	4
Four months of continuation phase, daily observed	{RH} [150/75]	½	1	2	3	4

R = Rifampicin *H* = Isoniazid *Z* = Pyrazinamide
E = Ethambutol *S* = Streptomycin

Note: The numbers indicate number of tablets to be taken daily for treatment according to body weight and content of tablets.

These recommendations are based upon the following dosages by body weight: rifampicin 10mg/kg; isoniazid 5mg/kg; Pyrazinamide 25 mg/kg; ethambutol 25 mg/kg, If Ethambutol is given for any reason for more than 8 weeks, the daily dose must be reduced to 15 mg/kg body weight.

Some Important Notes

- The oral drugs should preferably be given on an empty stomach in a fixed dose combination
- The oral drugs must be swallowed under observation from health facility staff or treatment supporter of his/her choice at home

Table: Treatment guidelines Category II

DURATION	DRUG	CHILD Pre-treatment weight			ADULTS Pre-treatment weight	
		5-10 kg	11 – 20 kg	21–30 kg	< 50kg	> 50kg
Two months of intensive phase, daily observed treatment	Streptomycin Inj. i..m	15mg/kg	15mg/kg	500mg	750mg	1gm*
	{RHZE} [150/75/400/275] Tablets	½	1	2	3	4
One month, intensive phase daily observed	{RHZE} [150/75/275/400] Tablets	½	1	2	3	4
Five months of continuation phase, three weekly observation	{RHE} [150/75/400]	½	1	2	3	4
	{ RH}** [150/75]	½	1	2	3	4
	{E} [400]	¼	1	2**	3**	4**

* Patients older than 50 years of age should not exceed a dose of 750 mg streptomycin

** Notice the higher dose – formulation of RH and increase in dosage of Ethambutol in the three weekly regimen

NOTE

- If Ethambutol is to be given for more than 8 weeks, reduce to 15 mg/kg body weight
- Ethambutol should not be given to children

2.0 TREATMENT OF TUBERCULOSIS IN SPECIAL CASES

2.1 Pregnancy: Always ask a woman if she is pregnant before commencing treatment, most of anti-TB is safe during pregnancies except streptomycin, which causes permanent deafness in the foetus therefore it should be avoided during pregnancy

2.2 Breastfeeding: Full TB treatments are safe and are best way to prevent tuberculosis in the baby mother and child can stay together for the entire duration of treatment. In the mothers with pulmonary tuberculosis, the baby should receive INH preventive (5mg/kg) for 6months followed BCG vaccination

2.3 Oral contraceptives: Rifampicin interacts with oral contraceptives and reduces the efficacy of this contraception. Women using contraceptive should be adviced to use pills with higher dose of oestrogen (50mcg) or change to another method

2.4 Liver disease: Most of anti-TB can cause liver damage. In case a patient develops jaundice, treatment should be stopped and restarted as soon as the jaundice resolves. In severely ill patients start streptomycin and ethambutol only. If the patient improves follow with a gradual step up introduction of isoniazid followed by rifampicin until full dose. Monitor liver functions and clinical picture. If the condition deteriorates stop the drug which was added. Patients with established chronic liver disease should not receive pyrazinamide.

2.5 Renal failure; Isoniazid, Rifampicin and Pyrazinamide are almost entirely excreted by the liver and therefore safe to use. Streptomycin and Ethambutol are excreted by the kidneys and should either be avoided or given in a reduced dose. The safest regimen for patients with renal failure is 2 RHZ/4 RH combined with pyridoxine to prevent Isoniazide induced peripheral neuropathy.

2.6 HIV/AIDS: There is a danger of interaction between Rifampicin and protease inhibitors in HIV positive patients receiving antiretroviral (ARV) treatments. Rifampicin stimulates the activity of the liver enzyme system, which metabolises protease inhibitors (PI) and Nucleoside Reverse Transcriptase Inhibitors (NRTIs). This can lead to decreased blood levels of PIs and NsRTIs. Of the NsRTIs the concentration of Nevirapine is significantly reduced and hence Nevirapine and Rifampicin should not be used concomitantly. On other hand PIs enhance the liver enzyme system which influences the blood levels of rifampicin resulting in ineffective TB treatment or drug toxicity. NRTIs can cause peripheral neuropathy, which can result in an added toxicity caused by Isoniazid.

2.7 The role of adjuvant steroid therapy

Steroid therapy given in addition to anti-TB treatment is beneficial in tuberculosis meningitis, pleural TB with large effusion and TB pericarditis. The recommended dosage in TB meningitis and TB pericarditis is 40-60mg/daily for 1 – 4 weeks, gradually decreasing the dosage over several weeks. Other less frequent conditions, which can benefit from steroid treatment, are:

- TB laryngitis with airway obstruction
- Massive lymphadenopathy with signs of obstruction of e.g airway
- TB of renal tract to prevent ureteric scarring
- TB of adrenal glands causing hypo-adrenalism
- Severe hypersensitivity reaction to anti-TB drugs

Although steroids are immunosuppressant they can be used in HIV positive patients as the overall benefit of steroids, in the context of above conditions, outweighs the risk of other opportunistic infections.

2.8 Multi drug resistance Tuberculosis

MDR TB is a laboratory diagnosis confirmed after culturing *Mycobacterium tuberculosis* strains and performing drug susceptibility tests (DST). Resistant strains will be identified because they will be able to survive exposure to anti TB drugs which were previously toxic to them. Four different categories of drug resistance have been identified:

- Mono-resistance: Resistance to one anti-tuberculosis drug
- Poly-resistance: Resistance to more than one anti-tuberculosis drug, other than both isoniazid and Rifampicin (e.g. against both pyrazinamide and isoniazid)
- Multidrug-resistance: Resistance to at least isoniazid and rifampicin

- Extensive drug resistance TB (XDR-TB): Multidrugresistance with additional resistance to any fluoroquinolone, and at least one of three injectable second-line drugs (capreomycin, kanamycin and amikacin).

Diagnosis of MDR -TB

The required baseline investigations of any DR TB suspect include:

- Comprehensive medical history including outcomes of prior TB treatment
- Physical examination
- Collection of 2 sputum samples (spot – morning) for smear microscopy, culture and DST
- Provider Initiated Testing and Counseling (PITC) for HIV
- Education on cough hygiene
- Chest X-ray examination

DST confirmed MDR TB patients shall be referred and transported by a special ambulance to the MDR TB Hospital where they will be admitted.

Treatment of MDR –TB

Standardized treatment: Regimens are designed according to representative Drug Resistance Well-defined patient populations. All patients in a patient group or category receive the same regimen. Suspected MDR TB should be confirmed by DST whenever possible.

Standardized MDR TB treatment regimen for Tanzania:

6Z Amk (5) Ofx Eto Cs±E /12Z Ofx Eto Cs±E

Intensive Phase (minimum 6 months, or 6 months postculture conversion)

- Amikacin or Kanamycin
- Ofloxacin or Levofloxacin
- Pyrazinamide
- Ethionamide
- Cycloserine
- Ethambutol

Continuation Phase (minimum 12 months or 18 months post culture conversion)

- Ofloxacin or Levofloxacin
- Ethionamide
- Pyrazinamide
- Cycloserine
- Ethambutol

2.9 Management of TB/HIV co –infections

- HIV is the highest known risk factor for developing TB
- HIV promotes progression to active TB in people with both recently acquired and latent *M. Tuberculosis*
- All patients with TB should be screened and managed for HIV

Reducing the burden of HIV in TB patients

- All patients with TB infection should be counselled and tested for HIV
- All TB patients should be given health education HIV prevention methods

- All TB patients co – infected with HIV should be given cotrimoxazole preventive therapy(CPT)
- All TB/HIV patients with CD 4 less than 200 cells treat TB first for at least two months
- TB/HIV patients with CD 4 less than 50cells/ul treat anti -TB for atleast two weeks before initiation of ARVs

3.0 LEPROSY

It is a chronic granulomatous disease caused by mycobacterium leprae, an acid and alcohol fast bacillus that has a very slow multiplication. It mainly affects the skin, the peripheral nerves and the mucous membranes. It is a disease mainly of human beings, which affects people of all races, all ages and both sexes. Leprosy is the commonest cause of peripheral neuritis in the world.

Patients harboring many bacilli in their bodies, the multi bacillary patients, are the main sources of infection. If not treated, they spread the disease in the community and infect others through coughing and sneezing (droplet infection). These infectious patients represent only about 25% of the registered leprosy patients in Tanzania. The other 75% of patients with few leprosy bacilli, the paucibacillary patients are less infectious. Skin contact with leprosy patients is no longer considered to be an important means of transmission. The different manifestation of leprosy is due to differences in the degree of resistance (immunity) of the human body and not due to different kinds of bacilli.

The majority of people (about 85%) have a strong resistance to M. Leprae that even when infected they do not develop the disease. About 75% of children who get infected with leprosy bacilli have such a high resistance that they overcome the disease themselves, without treatment, at very early stage. People who have a fairly high but incomplete immunity to leprosy bacilli will develop paucibacillary leprosy. There are only very few people in the community (5-10%) whose immunity to M. Leprae is naturally very low. When somebody from this group of people is infected by M. Leprae, the bacilli may multiply freely and attain large numbers causing multi-bacillary leprosy.

Diagnosis

The major clinical features therefore include hypopigmented anaesthetic macula or nodular and erythematous skin lesions and nerve thickening. Patients should be suspected of having leprosy when they show one or more of the following signs of symptoms:

- Burning sensations in the skin
- Pale patches on the skin with loss of feeling
- Numbness and tingling of the feet and/or hands
- Weakness of eyelids, hands or feet
- Tender nerves
- Painless swellings or lumps in the face and earlobes
- Painless wounds or burns on the hands or feet

Note:

- Patients presenting the above symptoms need to be examined by the District TB and Leprosy Coordinator at the earliest possible time. The DTLC will examine the patient and will decide whether or not to put him on treatment.
- The diagnosis of leprosy must be based on the history of the symptoms and careful clinical examination of the person for signs of the leprosy. Only rare instances a

laboratory and other investigation may be needed to confirm diagnosis of leprosy, if one is not sure, the suspect should be seen by the DTLC or other person trained in leprosy

- The three cardinal signs of leprosy are:
 - Skin patch with loss of sensation
 - One or more enlarged peripheral nerves
 - The presence of leprosy bacilli

History taking

Proper history taking and collection of certain information on the patient are very important for understanding the patient's situation and for tracing a lost patient. The following must be obtained:

- General information: all three names, sex, year of birth, full address from home to clinic, occupation
- Contact information: other leprosy cases in the patient's household
- Main complaints, including date of onset, site of first lesions, subsequent changes and development received.

Physical examination

- Physical examination should always be carried out with adequate light available and with enough privacy for the person to feel at ease.
- The patient is asked to undress. To ensure that no important sign is missed, a patient must be examined systematically. A well tried system is to examine the patient as follows:
 - Start with examination of the skin, first head, then neck, shoulders, arms, trunk, buttocks and legs
 - Then palpation of the nerves; starting with the head and gradually going to the feet
 - Then the examination of other organs
 - Examination of the skin smear
 - Finally the examination of eyes, hands and feet for disabilities.

Complications due to nerve damage

Patients should be examined for the following complications which result from nerve damage:

- Injury to cornea and loss of vision due to incomplete blink and/or eye closure
- Skin cracks and wounds on palms and soles with sensation loss
- Clawed fingers and toes
- Dropfoot
- Wrist drop
- Shortening and scarring in fingers and toes with sensation loss. Mark and draw also wounds, clawing and absorption levels on the maps using the appropriate marks.

Note: A diagnosis of leprosy should be made if ONE of the following CARDINAL SIGNS is presents

- *Skin lesion with the loss of sensation*
- *One or more enlarged peripheral nerves*
- *A skin smear positive for leprosy bacilli*

Classification of Leprosy

The main purpose of classification is to decide on the treatment regimen to be given to the patient. Leprosy is classified into two groups depending on the number of bacilli present in the body. Patients considered to harbor many bacilli belong to multibacillary (MB) group, with few bacilli form the paucibacillary (PB) group. Classification is also important as it may indicate the degree of infectiouness and the possible problems of leprosy reactions and further complications.

There are two methods of classifying leprosy, based on:

- The number of leprosy skin lesions
- The presence of bacilli in the skin smear

Skin smear is recommended for all new doubtful leprosy suspects and relapse or return to control cases.

Classify the patients as follows:

4.0 MULTIBACILLARY (MB) LEPROSY

- Patients with six or more leprosy skin lesions
- Positive skin smear

Paucibacillary (PB) Leprosy

- Patients with one to five leprosy skin lesions
- Negative skin smear

If there is any doubt regarding the classification, the patient should be classified and treated as a multibacillary case. This certainly applies to patients who have been treated in the past and of who insufficient information is available on the treatment previous used.

Treatment

Multiple drug treatment (MDT) is recommended treatment for leprosy. MDT is the combination of minimum two anti- leprosy drugs. Treatment of leprosy with only one drug monotherapy will result in development of drug- resistance, therefore it should be avoided. Patient having multibacillary leprosy are given a combination of Rifampicin, Dapsone and clofazimine while those having paucibacillary leprosy are given a combination of Rifampicin and Dapsone. Both regimens are given in the form of blister pack on a four weekly basis. A patient takes a first dose under direct observation of health worker. For the following 27 days, the patient takes the medicines at home under observation of treatment supporter.

Dosage (Adult MB)

Monthly Treatment: Day 1

S:Rifampicin 600mg (2x 300mg)

Plus

S:Clofazimine 300mg (3 x 100mg)

Plus

S:Dapsone 100mg

Daily Treatment: Days 2 – 28,

S:Clofazimine 50mg

Plus

S:Dapsone 100mg

Duration of treatment: 12 blister packs to be taken within a period of between 12-18 months

Dosage (Child MB 10 – 14 years)**Monthly Treatment:** Day 1;**S:**Rifampicin 450mg (3 x 150mg)**Plus****S:**Clofazemin 150mg (3 x 50mg)**Plus****S:**Dapsone 50mg**Daily Treatment:** Days 2 – 28**S:**Clofazemine 50mg every other day**Plus****S:**Dapsone 50mg daily**Duration of treatment:** 12 blister packs to be taken within a period of between 12-18 months**Dosage (Adult PB)****Monthly Treatment:** Day 1**S:** Rifampicin 600mg (2 x 300mg)**Plus****S:**Dapsone 100mg**Daily Treatment:** Days 2 – 28;**S:** Dapsone 100mg**Duration of treatment:** 6 blister packs to be taken within a period of between 6-9 months**Dosage (Child PB 10 – 14 years)****Monthly Treatment:** Day 1**S:** Rifampicin 450mg (3 x 150mg)**Plus****S:**Dapsone 50mg**Daily Treatment:** Days 2 – 28**S:**Dapsone 50mg daily**Duration of treatment:** 6 blister packs to be taken within a period of between 6-9 months**Duration of MDT*****Paucibacillary leprosy***

- Patients should receive 6 doses to be taken within a maximum period of nine months. When collecting the 6th dose the patient should be released from treatment (treatment Completed)
- Every effort should be made to enable patients to complete chemotherapy. A patient whose treatment is cumulatively interrupted for more than three 'months' or patient who has missed three doses of MDT in a total and hence cannot complete the 6 doses within 9 months, should be recommended as defaulter
- If a defaulter returns later to the clinic, s/he should be given ONE- second course of

Multibacillary leprosy

- MB patients should receive 12 doses to be completed within a maximum period of 18 months. When collecting the 12th dose of MDT the patient should be released from treatment (treatment completed)

- Patient who fail to collect the 12 doses of MDT within 18 months should given ONE second chance to complete a full course of Blister Pack. The procedures for a second course fo MB

Note

- A patient whose treatment is cumulatively interpted for more than six 'months' or A patient who has missed 8 doses of MDT in total and hence cannot complete the 12 doses within 18 months, should be recorded as defaulter
- When a defaulter report at a clinic, a second course of MDT should be started after the importance of regular treatment has been discussed with the patient
- Patients who restart the treatment must be registerd into the unit register District
- Leprosy Register again with a new number as return after default and thus should be included in another treatment cohort for assessing completion of treatment
- Every effort should be made to ensure that patients complete the second course of MDT as recommended
- After completion of the second course of MDT the patient should be recorded as treatment completed

Treatment in special cases

Pregnancy: The standard MDT regimens are considered safe, both for mother and child and should therefore be continued during pregnancy.

Tuberculosis: Patients suffering from both tuberculosis and leprosy require appropriate anti-tuberculosis therapy in addition to the MDT. Rifampicin must be given in the dose required for the treatment of tuberculosis. Once the intensive phase of anti TB treatment is completed, the patient should continue with his/her monthly rifampicin for leprosy treatment.

HIV/AIDS: The management of a leprosy patient infected with HIV is the same as that for any other patient. The response and cure rate of HIV positive patient is the same as in other patients. The management, including treatment reactions, does not require any modifications.

Leprosy Reactions and Relapse

Leprosy reaction is sudden appearance of acute inflammation in the lesions (skinpatches, nerves, other organs) of a patient with leprosy. This is due to an alteration in the immunological status of the patient. Reactions are the major cause of nerve damage and disability in leprosy. Therefore should be detected early and treated. Leprosy reactions are of natural cause of the disease and can occur at any time. Reaction commonly occurs during the early stage of disease. Sometimes patients report for first time to a health facility because of leprosy reaction. Some reactions are seen after completion of the treatment.

There are two types of reactions

- Reverse Reaction(RR) or type I reaction
- Erythema Nodosum Leprosum (ENL) or type II reaction (For detail refer Manual for management of Leprosy for Health Workers)

Treatment of Reversal Reaction or Type I Reaction

Depending on severity, treatment of RR is by giving anti- inflammatory drugs or corticosteroids usually prednisolone for a prolonged period.

Standard treatment of Severe Reversal Reaction with Prednisolone

40 mg daily (8 tablet of 5mg or 1 tablet of 40mg Prednic pack) 2 weeks
30 mg daily (6 tablet of 5mg or 1 tablet of 30mg Prednic pack) 2 weeks
20 mg daily (4 tablet of 5mg or 1 tablet of 20mg Prednic pack) 2 weeks
15 mg daily (3 tablet of 5mg or 1 tablet of 15mg Prednic pack) 2 weeks
10 mg daily (2 tablet of 5mg or 1 tablet of 10mg Prednic pack) 2 weeks
5 mg daily (1 tablet of 5mg or 1 tablet of 5mg Prednic pack) 2 weeks

Treatment of severe Reversal Reaction with Prednisolone at Hospital level

60 mg daily (12 tablets of 5 mg prednisolone) 1 week
50 mg daily (10 tablet of 5 mg prednisolone) 1 week
40 mg daily (8 tablets of 5 mg prednisolone) 2 weeks
30 mg daily (6 tablets of 5 mg prednisolone) 2 weeks
20 mg daily (4 tablets of 5 mg prednisolone) 10 weeks
15 mg daily (3 tablets of 5 mg prednisolone) 2 weeks
10 mg daily (2 tablets of 5 mg prednisolone) 2 weeks
5 mg daily (1 tablet of 5 mg prednisolone) 2 weeks

Treatment for Erythema Nodosum Leprosum (ENL) or Type II reaction

Erythema Nodosum Leprosum occurs only in multibacillary leprosy patients. An estimated 5 to 10% of MB patients develop ENL reaction. It is caused by an interaction between dead M. leprae and substances accumulating in the blood and tissues. The reaction is often triggered by special circumstances like emotional stress, pregnancy or childbirth, infectious diseases (malaria, TB), etc

Mild Erythema Nodosum Leprosum: Advise the patient to rest and provide analgesics such as aspirin (600mg three times a day) and chloroquine if available (150 mg two times daily), for one week duration. Re-examine the patient for improvement after six weeks with analgesics or signs of a more severe ENL reaction occur, use prednisolone.

Severe Erythema Nodosum Leprosum: Refer the patient to the nearest hospital for appropriate examinations and treatment. Prednisolone is given for three weeks as per schedule shown below.

The standard treatment schedule of severe ENL at Hospital level

Daily dose prednisolone (mg)
Days
Weeks 1 2 3 4 5 6 7
1st week 50 50 50 50 40 40 40
2nd week 40 30 30 30 30 20 20
3rd week 20 10 10 10 5 5 5

Recurrent Erythema Nodosum Leprosum

A few patients get regular episodes of ENL as soon as the dose of prednisolone comes below 20 or 15 mg per day. This is called chronic or recurrent ENL. Patients with recurrent ENL should be referred to hospital.

CHAPTER NINETEEN

SEXUALLY TRANSMITTED INFECTIONS (STI)

1.0 General guidelines

Accurate laboratory-proven diagnosis of sexually transmitted infections (STI) is not always possible except in a few health facilities with well equipped functional laboratory services. For health facilities without laboratory services, one must treat on clinical grounds i.e treat a disease based on suspected causative agents diagnosed clinically or by syndromic approach. In syndromic approach clinical syndromes are identified followed by syndrome specific treatment targeting all causative agents which can cause the syndrome. Contact tracing is encouraged as an important means of preventing further spread. Appropriate health education should be given at every opportunity.

First line therapy is recommended when the patient makes his/her first contact with the health care facility

Second line therapy is administered when first line therapy has failed and reinfection has been excluded.

Third line Therapy should only be used when expert attention and adequate laboratory facilities are available, and where results of treatment can be monitored.

In order to ensure complete cure, doses LESS than those recommended must NOT be administered. The use of inadequate doses of antibiotics encourages the growth of resistant organisms which will then be very difficult to treat.

Accurate laboratory-proven diagnosis of sexually transmitted infections (STI) is not always possible. The treatment recommended in this section is based on diagnosis of STI associated syndromes. Contact tracing is encouraged as an important means of preventing further spread. Appropriate health education should be given at every opportunity.

2.0 Reasoning in Choosing STI Drug Treatment Regimens

In choosing STI treatments, high efficacy (cure rate >95%) is most important. There is increasing evidence (clinical and now laboratory confirmation) that some of the first line drugs in these treatment protocols are below acceptable levels of effectiveness. This is particularly the case for chancroid and gonorrhoea. New drugs have been introduced for these conditions, but are currently advised as second line and third line.

3.0 The syndromic treatment of STI

Refer also to specific disease flow chart (**section 12 below**)

3.1 The syndromic treatment of STI in men

Possible symptoms or signs

Genital ulcers/erosion. Genital ulcer-**Yes** Disease (GUD)

B: Benzathine penicillin 2.4 MU, half into each buttock

Plus

A: Co-trimaxazole 8 x 480 mg tablets in one dose

Plus

A: Gentian violet 0.5-1.0% to ulcers.

Check for improvement, in 7 days, if none, **REFER**

If **NO** Genital ulcers; Swelling and inflammation in scrotum, - **Yes** – with possible urethral discharge: Give

A: Doxycycline (O) 100mg every 12 hours for 10 days.

Support Scrotal to take weight off spermatic cord, worn for a month, except when in bed. Check for improvement in 5 days, if none **REFER.**

If **NO** Urethral discharge alone Urethritis – **Yes;** Give

A: Co-trimaxazole (O)8 x 480 mg tablets once a day for 2 days,

Plus

A: Doxycycline (O) 100mg every 12 hours for 7 days.

Check for improvement, at the end of treatment, if non **REFER.**

If **NO** Bubo; Swollen tender lymph-glands-Yes (nodes) in the groin; Give

A: Doxycycline(O) 100mg every 12 hours for 14 days

Check for improvement, at least of tenderness after 7 days. If none, **REFER.**

If NO Ulcer Swelling and inflammation-Yes

- Cleans with salty water. Dry under fore skin and on the glans penis
- Paint with

A: Gentian Violet 0.5-1% every other day x 3 if not better in 7 days Change to:

B: Nystatin cream, 0.5-10 cm behind the glans 12 hourly for 7 days, cleansing before reapplication

- Check for improvement, at the end of treatment. If none, **REFER.**

If NO

Non-itchy rashes on the body or non-Yes

Treat for secondary syphilis with

B: Benzathine penicillin2 - 4MU deep IM half into each buttock.

If no improvement in 7 days or if tender swollen lymph glands at several sites, **REFER.**

3.2 The syndromic treatment of STI in women

Possible symptoms or signs

Lower abdominal pain with possible vaginal/cervical discharge-**Yes:**

A:Co-trimoxazole (O) 8 x 480 mg tablets once a day for 2 days

Plus

A: Doxycycline (O)100mg 12 hourly for 14 days

Plus

A: Metronidazole (O) 400 - 500 mg 12 hourly for the first 7 days. Check for improvement at the end of 7 days. **REFER** if none

If Vaginal discharge WITHOUT ANY LOWER ABDOMINAL PAIN **Yes:**

A: Co-trimoxazole (O) 8 x 480 mg tablets once a day for 2 days

Plus

A: Doxycycline (O) 100mg 12 hourly for 7 days

Plus

A: Metronidazole (O)400-500 mg 12 hourly for the first 7 days. Check for improvement at the end of 7 days **REFER** if none

If **NO** Vaginal discharge WITHOUT ANY LOWER ABDOMINAL PAIN-**Yes:**

A: Co-trimaxazole (O)8x 480 mg tablets once a day for 2 days

Plus

A: Doxycycline (O)100mg 12 hourly for 7 days

Plus

A: Metronidazole (O)2g in one dose.

Check for improvement at the end of treatment. **REFER** if none.

If **NO** Genital ulcers/erosions Genital Ulcer Disease, (GUD):

B:Benzathine penicillin 2.4 MU deep IM, half into each buttock

Plus

A: Co-trimoxazole (O)8 x of 480 mg tablets in one dose. Check for improvement after 7 days **REFER** if none

If **NO;** Check for **Candida infection:** Swelling and itchy soreness of the labia, possibly with some thick discharge – **Yes:**

A: Clotrimazole ointment 1% on lower vaginal, labia and skin daily

Plus

A: Clotrimazole pessary 500mg. One pessary inserted deep into the vagina at bed time.

Check for improvement, after 7 days **REFER** if none.

Genital Warts:

Carefully apply either

C:Podophyllin 10-25% to the warts, and wash off in 6 hours, drying thoroughly. Treat every 2-3 days until warts are gone.

OR

S:Trichloroacetic acid 80% to the warts, and wash off in 6 hours, drying thoroughly. Treat every 2-3 days until warts are gone.

Non-itchy rashes on the body or non-tender swollen lymph glands at several sites-**Yes**; treat for secondary syphilis with **Benzathine penicillin** 2.4 MU deep IM half into each buttock. If no improvement in 7 days **REFER**.

4.0 Gonorrhoea

Gonococcal and chlamydial infections frequently co-exist. Therefore combined therapy should be given. Treatment guidelines: see under "The Syndromic Treatment of STI".

- All gonococcal infections are likely to be resistant to common drugs such as Penicillins, Tetracyclines, Co-trimoxazole and erythromycin and Doxycycline
- Other causes of treatment failure should be considered;
- Gonococcal and chlamydial infections frequently co-exist. Therefore combined therapy should be given
- For general treatment guideline see under "The Syndrome Treatment of STI".

Note:The tradition of norfloxacin (a quinolone antibiotic) is specifically for the second line treatment of gonorrhoea. Norfloxacin is contraindicated in pregnancy and age less than 16 years (damage caused to the joints in animal studies) unless advised by a specialist for compelling situations. Reported adverse effects include rashes, photosensitivity and anaphylaxis in patients with AIDS.

5.0 Chancroid

Diagnosis

- Presence of painful genital ulcers with undermined ragged edges
- The base is covered with dirty purulent exudates and easily bleeds on touch.

Treatment

First line

A: Co-trimoxazole (O) 960 mg twice daily for 10 days

Second line

A: Erythromycin (O) 500 mg 6 hourly for 10 days

Third line

A: Ciprofloxacin (O) 250 mg 8 hourly for 7 days

6.0 Epidymo-Orchitis

It is an acute severe inflammation of the epididymis, testis and spermatic cord. The main clinical features include swollen and tender epididymis, severe pain of one or both testes and reddened oedematous scrotum. Causative organisms include filarial worms, *Chlamydia trachomatis*, *Neisseria gonorrhoea*, *E.coli* as well as viruses such as which cause mumps.

Note: Exclude other pathology such as torsion of testis.

Treatment

First line

A: Doxycycline (O) 100mg 12 hourly for 7 -10 days

Plus

A: Co-trimoxazole 960 mg every 12 hourly for 5 days

Plus

A: Diclofenac 50-100mg 2-3 times per day

Second line

A: Erythromycin (O) 500mg every 6 hours for 10 days

OR

C: Azithromycin 500mg daily for 6 days

Third line

D: Kanamycin (IM) 2g, 1g in each buttock, as a single dose

OR

S: Clindamycin 1.2 g (IM/IV) 12hrly for 3 days

PLUS

A: Doxycycline (O) 100 mg every 12 hours for 10 days

Note: Patient may need to wear a scrotal support

7.0 Chlamydia infections

Presence of scanty to moderate white mucoid or serous discharge and is often seen 1- 3 weeks after sexual intercourse

Treatment

First line

A: Ciprofloxacin (O) 500mg 12hrly for 3 days.

Doxycycline is added to the first line treatment for urethral discharge in men and women (See Syndromic treatment flow chart).

8.0 Syphilis

Syphilis is a chronic infectious disease caused by the spirochete treponema pallidum. It can be acquired mainly through sexual intercourse or congenitally when the mother transfers it to the fetus. The main classification of syphilis is shown below.

Table: Classification of Syphilis

Type	Stage	Clinical features/presentation
Congenital	Early	Rhinitis
	Late	Mucocutaneous lesions e.g. bullae, stigmata of osteochondritis, osteitis (or scars)
Acquired	Primary and secondary syphilis	<ul style="list-style-type: none">• A painless chancre• Rash, Non-tender lymphadenopathy, condylomata accumulata
	Tertiary (benign gummatous)	Interstitis, photophobia, corneal infection, 8th cranial nerve deafness, bilateral knee effusion, recurrent arthropathy
	Quarterly (cardiovascular and neurosyphilis)	Cardiovascular syphilis and neurosyphilis will give clinical features associated with that system. Also seen are gumma and osteitis

Treatment guidelines

For primary and secondary syphilis:

B: Benzathine penicillin 2.4 MU deep i.m as a single dose given as two injections in different buttocks.

If there is penicillin allergy

A: Erythromycin 500 mg 6 hourly for 14 days

OR

A: Doxycycline 100mg 12 hourly for 14 days

CAUTION: Doxycycline should not be given to pregnant and breast feeding women and children under 12 years of age

Late Syphilis

Give Benzathine penicillin give 2.4 MU IM weekly for 3 weeks.

Congenital syphilis

Up to 2 years of age

A: Benzyl Penicillin 15,000MU/kg body weight IM/IV 6 hourly for 10days

OR

A: Procaine benzylpenicillin 50,000 MU/kg body weight every 24 hours for 10 days

Over 2 years of age

A: Benzyl penicillin 50,000-75,000MU/kg body weight IV or IM every 6 hours for 10-14 days

OR

A: Erythromycin 10mg/kg body weight every 6 hours for 30 days.

For pregnant women allergic to penicillin

A: Erythromycin 500mg (O) 8hrly for 14 days

OR

C: Azithromycin 500mg (O) once a day for 6 days

9.0 WARTS

9.1 Genital Warts

These are usually caused by papilloma group of viruses infecting the skin or mucous membrane. The common sites affected by warts include genital region (condylomata acuminata) hands and legs. The lesions are usually asymptomatic fleshygrowths. In the genital region, lesions are often finger like and increase in number and size with time. When extensive they may interfere with sexual intercourse and child birth. The removal of the lesion does not mean cure of the infection.

Treatment

C: Podophyllin10-25% to the warts, and wash off in 6 hours, drying thoroughly.

OR

C: Silver Nitrate to the warts, and wash off in 6 hours, drying thoroughly.

OR

C: Salicylic acid to the warts, and wash off in 6 hours, drying thoroughly.

Treat every 2-3 days until warts are gone.

Alternatively

S:5% Imiquimod cream with a finger at bedtime, left on overnight, 3 times a week for as long as 16 weeks.

The treatment area should be washed with soap and water 6-10 hours after application. Surgery may be useful in selected cases to remove the warts.

Note: Do not apply on healthy surrounding skin.

CAUTION: It is contraindicated in pregnancy and lactation.

Cervical warts

This case should be referred to specialist /expert. Most expert advice against the use of podophyllin for cervical warts; therefore apply imiquimod cream as above.

Meatal and urethral warts

Accessible meatal warts may be treated with podophyllin or povidone-iodine solution. Great care is needed to ensure that the treated area is dried before contact with normal, opposing epithelial surface is allowed.

10.0 TRICHOMONIASIS

It is caused by a flagellate protozoa *Trichomonas vaginalis*. It causes inflammation of vagina and cervix in females and inflammation of urethra and prostate gland in males. Patient may be asymptomatic or may present with a frothy green/yellowish discharge, itchness, erosion of cervix.

Treatment

Adult:

A:Metronidazole 400mg 8hrly for 5 days
Children: 5mg/kg body weight every 8 hours for 7 days

OR

C:Tinidazole 2gm stat
Children: 50-75 mg/kg single dose

OR

D:Secnidazole 2gm stat.

Give the same treatment to partner. In pregnancy treatment with metronidazole should be delayed until after first trimester.

11.0 VAGINAL CANDIDIASIS

It is caused mainly by candida albicans. Vulvae-vaginal Candidiasis is common in women on the pill, in pregnancy and diabetics and in people on prolonged antibiotic courses. Vulvae vaginal candidiasis is characterized by pruritic, curd-like vaginal discharge, dysuria and dyspareunia. Disseminated Candidiasis; resulted from complications of the above, presents with fever and toxicity.

Medicine of choice

B: Nystatin Pessaries insert 1 at night for 14 days

OR

A: Clotrimazole pessaries/vaginal creaminsert/apply 1 at night for 6 days

OR

C: Miconazole Pessaries/vaginal cream insert/apply once at night for 3 days

OR

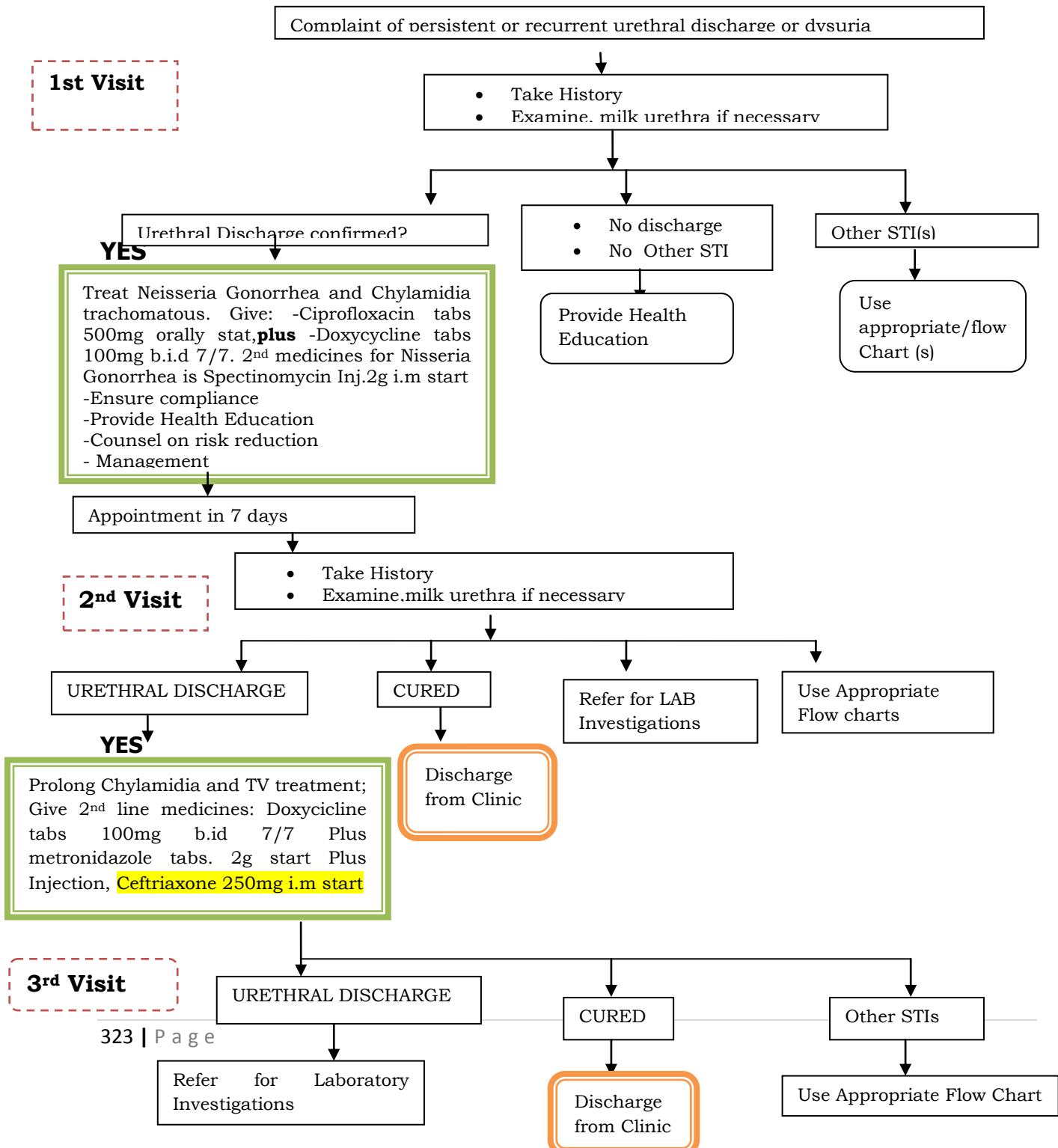
C: Ketoconazole200-600mg (O) every 24 hours for 10 days

OR

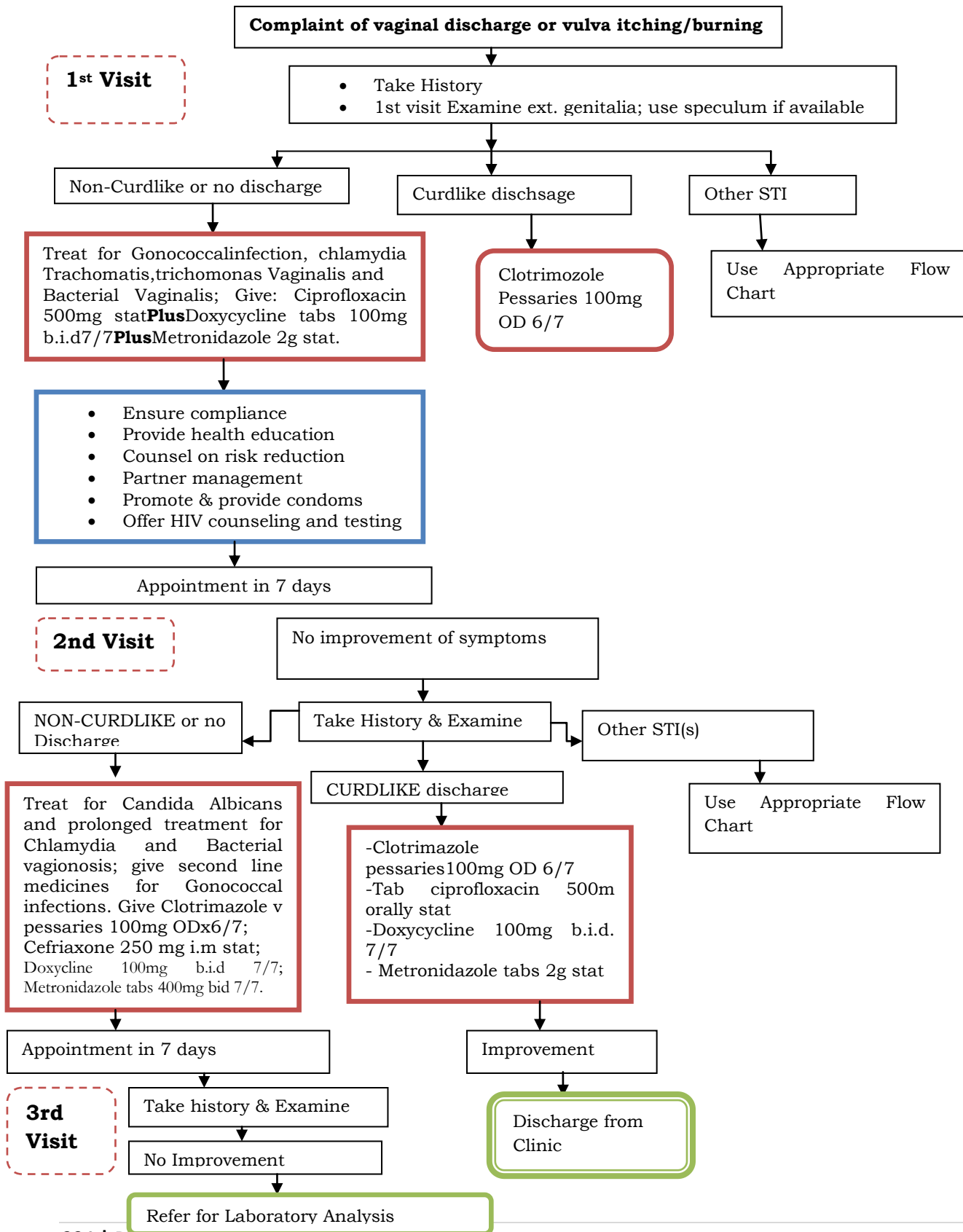
B: Fluconazole 200mg once daily for 14 days

12.0 CHARTS ON SYNDROMIC TREATMENT OF STI

12.1 Urethral Discharge Syndrome (UDS) Management Flow Chart

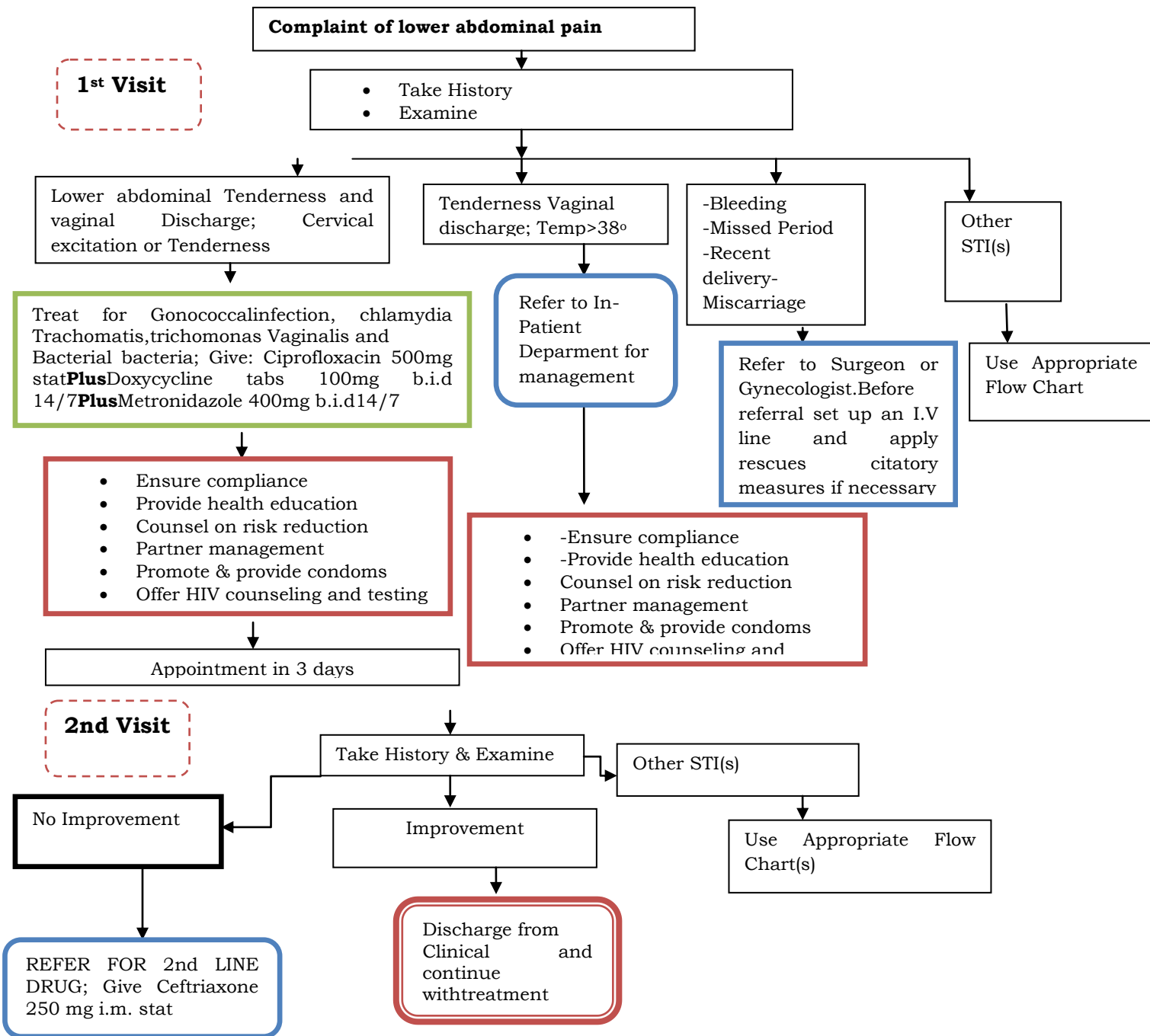


12.2 VAGINAL DISCHARGE SYNDROME (VDS) MANAGEMENT FLOW CHART



12.3 LOWER ABDOMINAL PAIN (PID) MANAGEMENT FLOW CHART

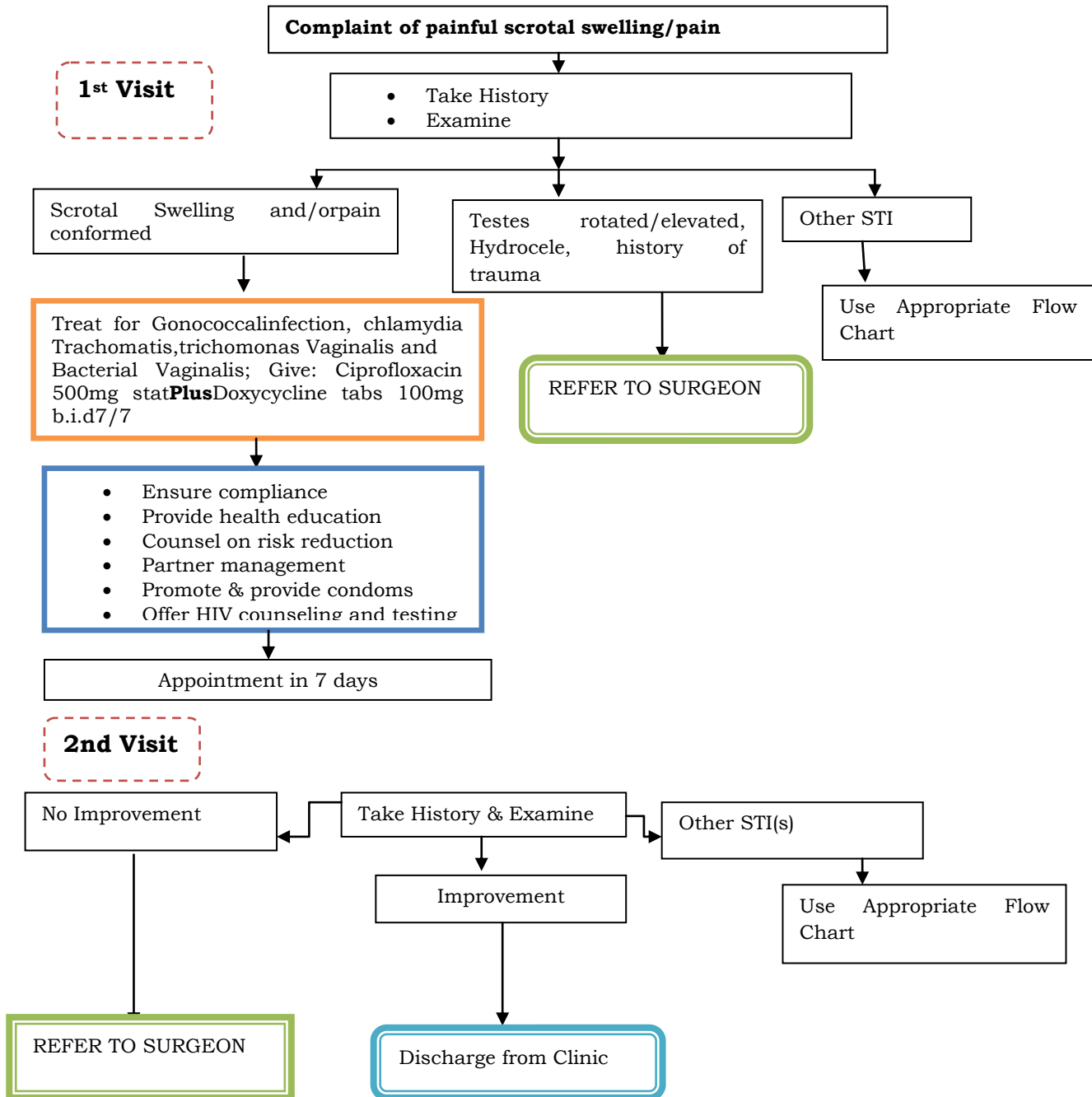
Other STI



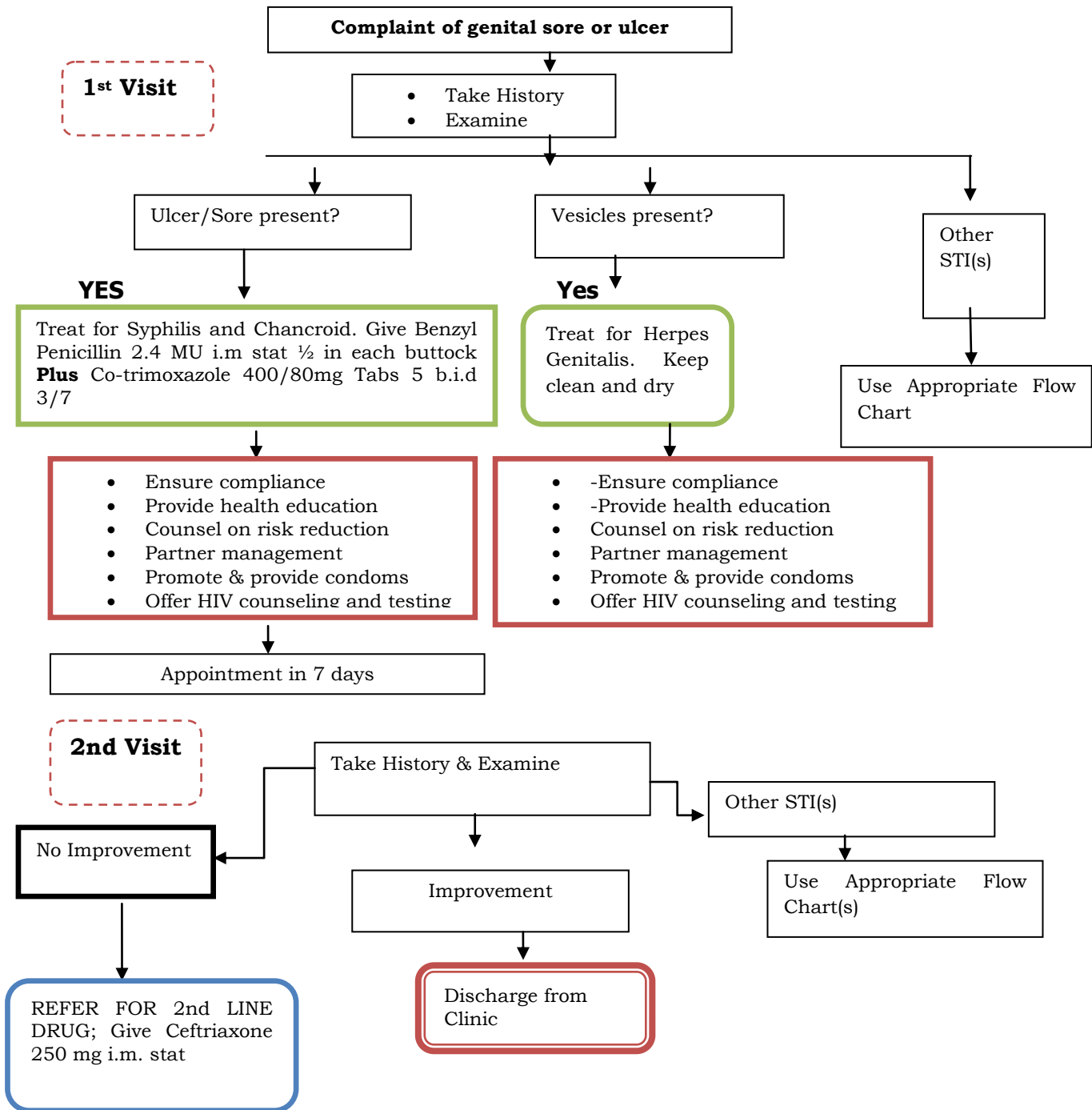
Note

- Do not give Metronidazole in 1st trimester of pregnancy: Do not give Doxycycline or Ciprofloxacin in pregnancy or to lactating mother. Substitute with Erythromycin 500mg t.i.d 7/7 or Ceftriaxone 250 mg i.m. stat
- Even with no tenderness the risk for infection in someone complaining of lower abdominal pain should be considered

12.4 PAINFUL SCROTAL SWELLING (PSS) MANAGEMENT FLOW CHART



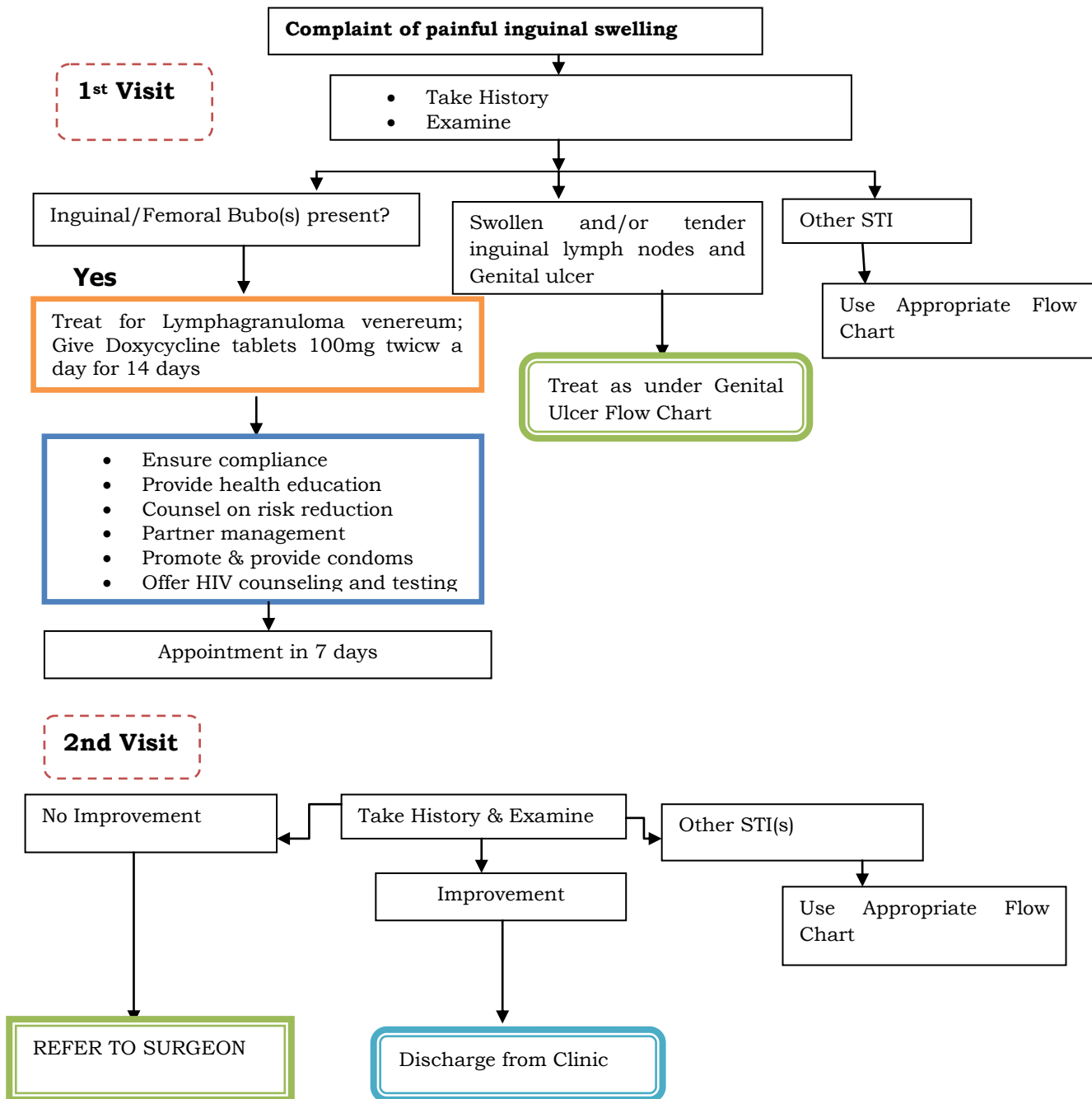
12.5 GENITAL ULCER DISEASE (GUT) MANAGEMENT CHART



Note

- Do not give Co-trimoxazole during pregnancy; substitute with Erythromycin tablets 500mg QID 7/7
- Patient allergic to penicillin substitute with Erythromycin tablets 500mg QID for 15 days
- Other option to treatment of chancroids is Tablets Ciprofloxacin 50mg Orally twice daily for 3 days or Azithromycin 1g orally single dose

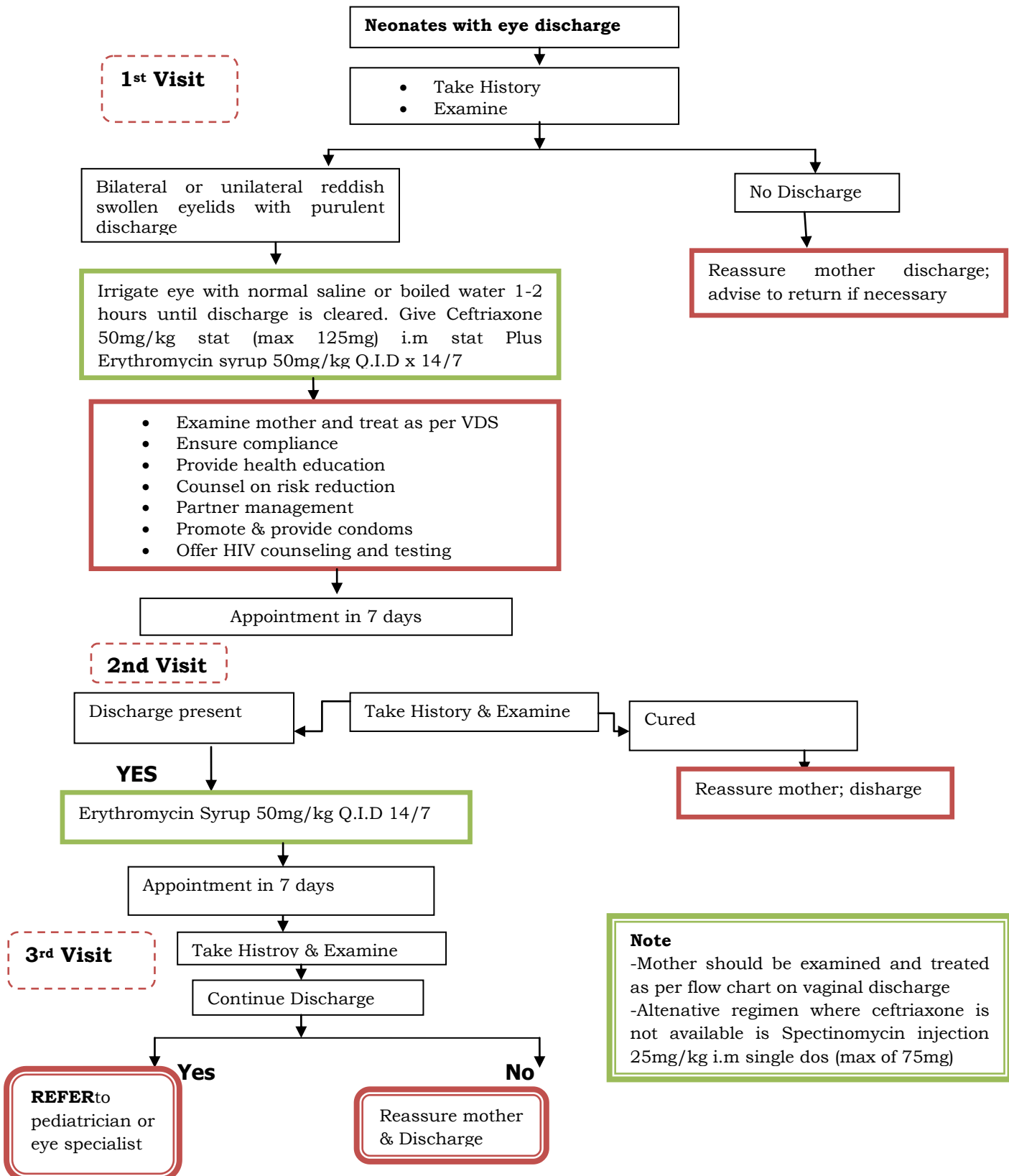
12.6 INGUINAL BUBOS (IB) MANAGEMENT FLOW CHART



Note

- Alternative treatment for Chancroids is Ciprofloxacin 500mg orally twice daily for 3 days
- Replace Erythromycin in pregnant women

12.7 NEONATAL CONJUNCTIVITIS (NC) MANAGEMENT FLOW CHART



CHAPTER TWENTY

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

The spectrum of disease due to HIV infection ranges from mild, non-specific conditions (e.g. persistent generalized lymphadenopathy, herpes zoster, and seborrheic eczema) to its severe form i.e. Acquired Immunodeficiency Syndrome (AIDS). Infection by the human immunodeficiency virus leads to gradual and progressive destruction of the cell mediated immune system. The clinical features may be due to HIV per se or as a result of immune system destruction.

Diagnosis

- Fever, diarrhoea, weight loss, skin rashes, sores, generalized pruritis, altered mental status, persistent severe headache, oral thrush or Kaposi's sarcoma may be found in patients with advanced disease
- Most patients, however, present with symptoms due to opportunistic infections e.g. tuberculosis, candidiasis or pyogenic infections.

1.0 TREATMENT OF HIV/AIDS IN ADULTS AND ADOLESCENTS

HIV positive patients should be referred to Care and Treatment Clinics. The initial management involves signing of the informed consent by the patient. Followed by a complete blood count, renal and hepatic chemical function tests, urine pregnancy test and viral load where applicable should be done at baseline. Initiation of treatment should be based on the extent of clinical disease progression.

CD4+ T lymphocytes counts remain the standard for evaluating immune function.

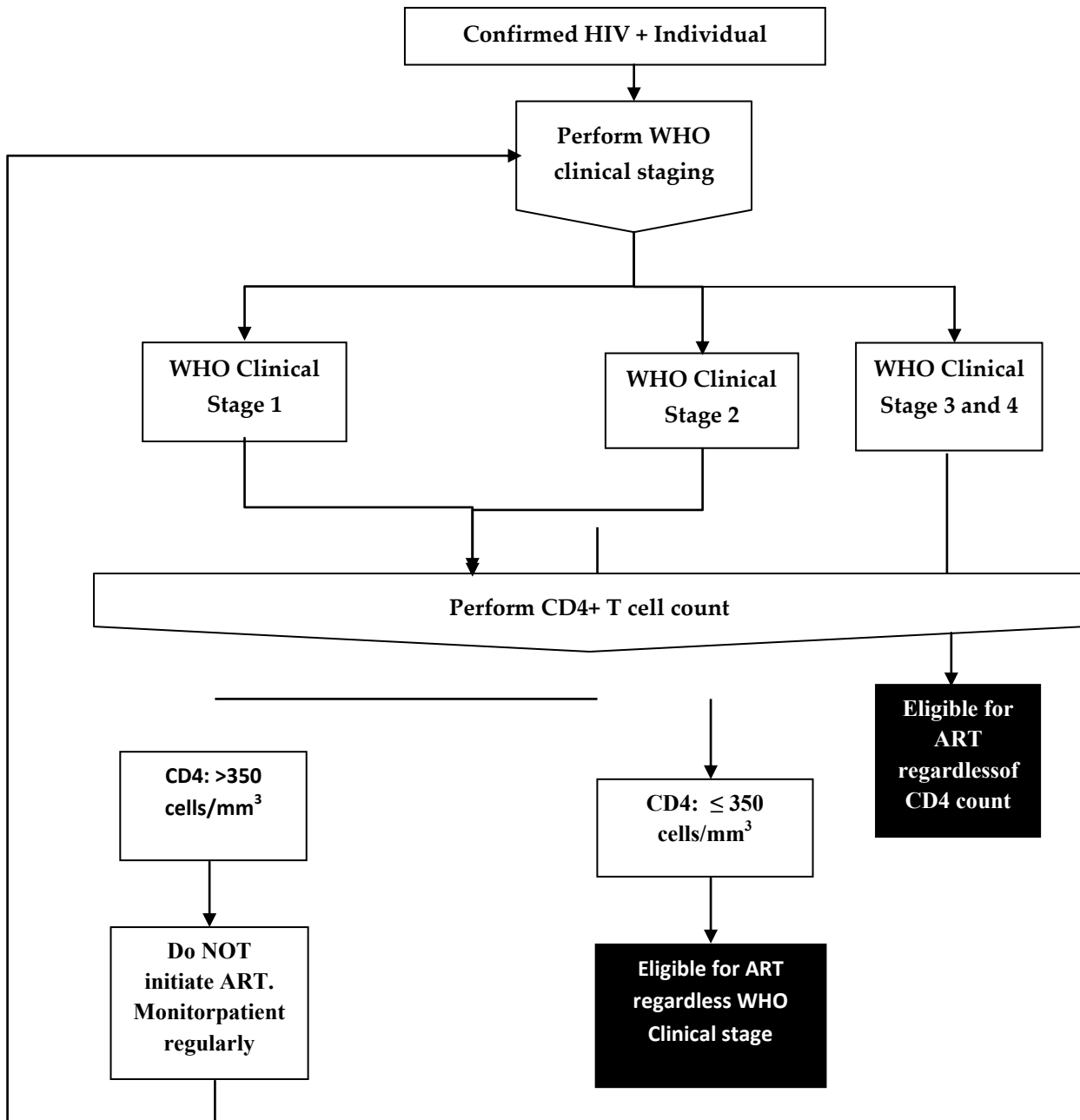
1.1 Criteria of initiation of ART in Adults and Adolescents Patients

Based on experience and available evidence, use of ART improves quality of life and survival for PLHIV. Optimal time of ART initiation is important for desirable health outcome in terms of reducing risk of death, disease progression including tuberculosis and occurrence of serious adverse events. WHO recommends that HIV infected patients to be initiated based on WHO clinical stage and CD4 cells count level.

There are therefore 3 classes of patients that are eligible to begin treatment:

- All patients in WHO stage 3 and 4 clinical criteria, regardless of CD4 cell count
- All adolescences and adults including pregnant women with a CD4 count \leq 350cells/mm³, regardless of clinical symptoms
- Special population including TB-HIV co-infection patients and those with chronic active hepatitis based on clinical and available laboratory findings, regardless of CD4 count

Figure 1: Criteria for initiation of ART in Adults and Adolescents



Note

Before initiating therapy in any patient, apart from clinical eligibility, it is important to assess the patient’s willingness and readiness to be on ART adherently.

1.2 Evaluation to be done before initiating therapy

Before initiating therapy in any patient, a good history of the patient must be taken and a head-to-toe physical examination conducted. In addition the TB screening questionnaire should be

administered followed by screening. Thereafter, the following baseline laboratory tests are recommended:

- Urinalysis
- Renal Function Tests (Creatinine, Blood Urea Nitrogen (BUN))
- A complete blood count (If not available do Hgb)
- Chemistry profile for liver (serum alanine aminotransferase, ALT)
- Tests to rule out active TB where indicated (sputum AFB, CXR) in case of indication from the screening questionnaire
- CD 4 count (if it was not done in the past 6 months)
- Urine for pregnancy (To women of reproductive age)
- VDRL (when necessary)

The following could be done if available:

- Serum creatinine and lipids
- Hepatitis B and C serology
- Viral load

The patient and other family members (with patients' consent) should then be educated on HIV/AIDS and the need to adhere to the agreed treatment plan.

General orientation of the patient and family members should include:

- Who to call and where to get refills
- Who to call and where to go when clinical problems arise
- Who to call/where to go for assistance on social, spiritual and legal problems that might interfere with adherence to treatment

1.3 First Line Treatment

Antiretroviral therapy both in naïve patients and those who have received treatment before involves the use of a combination of drugs. Triple therapy consisting of 2 NRTI + 1 NNRTI or 2 NRTI + 1 PI or 3 NRTI's is recommended. It is important to remember that there is no single combination that is best for every patient and/or that can be tolerated by all patients. Regimens should be recommended on the basis of a patient's clinical condition, lifestyle, and ability to tolerate the regimen.

CAUTION!!!: The use of monotherapy in the treatment of HIV infection is prohibited.

The ARVs drug combinations should be used according to indications and contraindications that govern the use of ARVs to minimize side effects and drug-drug interactions. The default first line regimen in Tanzania is:

Zidovudine (AZT) 300 mg/Lamivudine (3TC) 150 mg twice daily and Efavirenz (EFV) 600 mg once daily at night.

- For women in the child bearing age, Nevirapine (NVP) 200mg twice a day is given instead of Efavirenz.

Note

- For adolescents the dose of AZT is 200 mg BD for a body weight of between 20-40 kgs.
- For patients with <40kg the dose of EFV should be <600mg.

- Efavirenz has been reported to be associated with teratogenicity in early pregnancy and liver toxicity in children below three years. In these cases, Nevirapine should be prescribed instead.
- In women for whom effective contraception can be assured, EFV remains a viable option for the NNRTI component of the regimen.

Under certain circumstances however, the following regimens can be used as first line:

Zidovudine (AZT)+Lamivudine(3TC)+Nevirapine (NVP)

This regimen can be prescribed when Efavirenz is contraindicated, e.g. in Neuropsychiatric complications of Efavirenz, in pregnancy and in children less than three years, and when Tenofovir cannot be used such as in the presence of renal disorder and when haemoglobin is stable.

Note: Nevirapine challenge dosing is required during the beginning of treatment. In the first two weeks of treatment only half of the required daily dose of Nevirapine should be given, and a full dose if there are no side effects such as skin rash or hepatic toxicity. In summary, this means:

Zidovudine 300 mg/Lamivudine 150 mg/Nevirapine 200 mg in the morning + Zidovudine 300 mg/Lamivudine 150 mg OD in the evening for the first 2 weeks. And if there are no problems, THEN Zidovudine 300 mg/Lamivudine 150 mg/Nevirapine 200 mg twice daily.

Tenofovir 300mg / Lamivudine 300mg / Efavirenz 600mg

A triple FDC is available for use and the treatment of HIV/HBV co-infection.

Tenofovir (TDF) + Lamivudine (3TC) + Nevirapine (NVP)

Tenofovir (TDF) + Emtricitabine (FTC) + Efavirenz (EFV)

Tenofovir (TDF) + Emtricitabine (FTC) + Nevirapine (NVP)

The major concern with Tenofovir-based treatment is renal safety. Tenofovir-associated nephrotoxicity is especially likely in patients with pre-existing renal dysfunction or those receiving other concomitant nephrotoxic medications, low birth weight, advanced age and lower CD4 cell counts. Otherwise the overall rate of discontinuation for renal events is extremely low. Renal function should be monitored through routine urine testing for the occurrence of proteinuria and if available serum creatinine.

Note: For based Regimen

+ Lamivudine (3TC) + Efavirenz (EFV)

OR

+Lamivudine (3TC) + Nevirapine (NVP)

Initiation

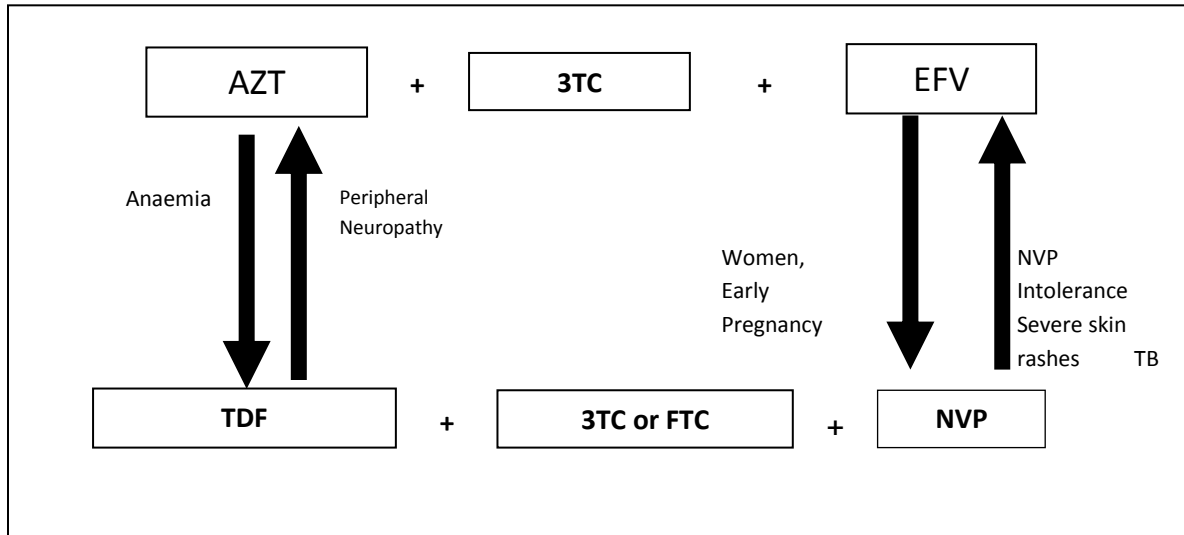
- New patients should not be started on Stavudine based regimen.
NB: Stavudine can only be used when Zidovudine or Tenofovir is contraindicated

Continuation

- Stavudine can also be used for continuing patients, who are stable on stavudine regimen without any signs of side effects.

In cases where Nevirapine or Efavirenz cannot be used as a first line drug, a single drug from the second line drugs can be used; for example LPV/r or ABC.

Figure 2: First line drug regimen flow chart



1.4 Changing Antiretroviral Therapy

There are multiple reasons which may prompt the need to change antiretroviral therapy. These can be grouped into two major categories:

Drug adverse events –Toxicities, including

- Intolerable side effects
- Drug interactions
- During pregnancy if the patient is on EFV.

Treatment failure or type of treatment failure

- Clinical failure – occurrence or persistence of HIV related OIs
- Immunological failure
- Virological failure

Changing antiretroviral therapy due to toxicity

From a clinical perspective, it is generally recommended that when changing a patient's regimen due to toxicity, only the toxic drug(s) should be replaced, if possible. Table 8.3 below provides guidance on ARV drug combinations with some common toxicity switches. It is based on the first line drugs in the latest National HIV/AIDS treatment guideline.

Table 1: Common toxicity switches for first line drugs

First Line	Problem	Substitution
AZT + 3TC + EFV	Anaemia due to AZT	TDF*** + 3TC + EFV TDF*** + FTC + EFV d4T + 3TC + NVP or EFV*
AZT + 3TC + NVP	Anaemia due to AZT	TDF*** + 3TC + NVP TDF*** + FTC + NVP d4T + 3TC + NVP or EFV*
	Hypersensitivity due to NVP	AZT + 3TC + EFV TDF*** + 3TC + EFV d4T + 3TC + EFV*
TDF + 3TC + EFV or NVP (TDF containing regimen)	Nephrotoxicity due to TDF	AZT + 3TC + NVP or EFV* d4T + 3TC + NVP or EFV*
d4t + 3TC + NVP or EFV*	Peripheral neuropathy due to d4T	AZT + 3TC + NVP or EFV* TDF*** + 3TC + NVP or EFV TDF*** + FTC + NVP or EFV
	Lipodystrophy due to d4T	TDF*** + 3TC + NVP or EFV TDF*** + FTC + NVP or EFV

- *Only if the patient is older than 3 years of age and weight \geq 10kg or in a woman in reproductive age.
- ** Follow liver function tests (LFTs) closely.
- ***Follow renal functions closely

1.5 Severity of adverse events due to ARVs

Side effects or toxicities caused by ARVs can be classified into three broad categories:

First category: Symptoms are mild and transient and often require patient assurance that these symptoms are common and will decrease over time. These can be mild headaches, mild gastric upset, nausea, fatigue and the CNS disturbances particularly with EFV. ARV interruption is seldom indicated in this situation.

Second category: Symptoms are somewhat more severe and often respond to some medical intervention. They include more severe gastric upset with nausea and vomiting, more severe headaches and mild peripheral neuropathy that does not incapacitate or interfere with a patient's lifestyle. These symptoms can often be successfully treated with anti-emetics, anti-diarrhoea medicines, analgesics, neuroleptics (e.g. Amitriptyline) and other medicines. ARV interruption is usually not indicated in this situation and often symptomatic treatment is only temporary. The mild rash associated with NVP (dealt with under a separate paragraph below) can often be treated with medical intervention.

Third category: Symptoms are severe such that ARV drugs must be stopped and replaced by an alternative drug. These include anaemia (haemoglobin $<$ 7.5 gm/dl or a falling haemoglobin, that often drops by 2 gm/dl) as can occur with the use of AZT. Severe symptoms noted in the first two categories can sometimes lead to the stopping of ARV due to severe toxicities such as nausea with severe discomfort and minimal intake for 3 or more days, vomiting all in take in 24 hours or dehydration due to vomiting, severe headache not responsive to non-narcotic analgesics, or fatigue reducing activity by more than 50%. In these situations, one or more ARVs should be replaced by another. It also includes the hypersensitivity reaction to NVP which

can include a severe rash or liver function test (LFT) elevations to grade III or >5 times the upper limit of normal range.

Nevirapine hypersensitivity reactions

NVP hypersensitivity reactions can manifest as a rash and/or elevated LFTs. The rash can occur in up to 20 % of patients and usually occurs in the first 6-8 weeks of therapy. NVP will be initiated at a lower dose for the first 2 weeks when only one NVP dose is given per day for 14 days. If there are no clinical signs or symptoms of a NVP hypersensitivity or allergy, the LFT (ALAT) will be checked and the NVP dose will be escalated to 2 doses per day starting at the second week.

Note:

- If a mild drug-reaction type rash occurs, patients will continue treatment with caution and careful monitoring. LFTs that are less than grade III (<5 times the upper limit of normal) can usually be followed until it is resolved. This rash will be treated with patient assurance, antihistamines and close follow up until resolved. NVP dose escalation will be delayed for up to one week until symptoms disappear. If symptoms worsen, this may indicate that the patient has severe hypersensitivity reaction and NVP will have to be stopped immediately and other medical interventions considered.
- If a severe drug-reaction type rash occurs e.g. severe erythema, urticaria, moistening of skin (desquamation), skin blistering, sloughing of skin, exfoliative dermatitis, erythema multiforme (when severe and involving the mucous membranes known as SJS), anaphylaxis, involvement of mucous membranes, angioedema, cracked/fissured lips, or systemic signs (body aches, arthalgias, myalgias, fevers, lymphadenopathy or significantly elevated LFTs); patients will discontinue NVP treatment, begin high dose prednisolone, antihistamines, analgesics, and be admitted to the hospital for IV fluids and careful monitoring.. NVP will be stopped immediately and not re-introduced. Continue with remaining two drugs for one week then stop all. Once the patient recovers, 3 ARV drugs will be started that do not include NVP. The remaining 2 ARVs will be paired with a replacement ARV such as EFV, if not contraindicated

Abacavir (ABC) hypersensitivity reactions

ABC hypersensitivity occurs in up to 5% of patients and can be fatal. Hypersensitivity symptoms include: flu symptoms, shortness of breath, cough, fever, aches and pains, a general ill feeling, fatigue/tiredness, swelling, abdominal pain, diarrhoea, nausea, muscle or joint aches, numbness, sore throat or rash. ABC will be stopped immediately and not re-started if this occurs.

Note: If there is a history of ABC hypersensitivity, then ABC is contraindicated.

Efavirenz (EFV) Side effects

EFV can cause CNS side effects such as vivid dreams, nightmares, vertigo, or confusion. These symptoms are often mild and transient. Patients may benefit from assurance that these symptoms are common and will decrease over time.

Stavudine (d4T) Side effects

Peripheral neuropathy is a common side effect with the use of Stavudine and occurrence of lactic acidosis has been reported. Cumulative exposure to d4T has the potential to cause disfiguring, painful and lifethreatening side-effects, such as lipodystrophy and lactic acidosis; for

patients who are still on d4T; prescribe 30 mg every 12 hours for all individuals, irrespective of body weight. New patients should be started on AZT or TDF based regimen.

1.6 Changing antiretroviral therapy due to treatment failure

Treatment failure can be virologic, immunologic and/or clinical. It results from failure to suppress viral replication with the development of viral resistance.

Virological Failure is defined if:

- There a less than 10 fold drop in viral load after 6-8 weeks of therapy, or when the viral load is detectable after 6months of therapy or when the viral load (VL) is persistently above 5,000 copies/ml..

Immunologic failure if defined as a:

- 50% drop in CD4 count from peak value, or
- Return to pre-ART baseline CD4 count or lower.

Clinical failure results in disease progression which clinically may present with the development of opportunistic infections or malignancy occurring 3 months or more after initiation of ART.

In Tanzania, immunological and clinical parameters are used to identify treatment failure. However, in light of declining costs of performing viral load measurements, along with the simplification of processes, where available, viral load parameters should also be applied. Where available, Viral Load should be used to confirm immunological failure. Furthermore, clinical failure must be distinguished from the Immune Reconstitution Inflammatory Syndrome (IRIS), in that, while clinical failure is associated with failing CD4 counts, IRIS is associated with improvements in immune response, i.e. CD4 counts.

1.7 Second-Line ARV Regimen

Before treatment failure is presumed and a particular regimen discarded, every effort should be made to rule out causes other than drug resistance. Patients should be evaluated for correctable factors, such as:

- Inappropriate dosing schedules
- Drug interactions that may reduce the efficacy of some of the ARV
- Non adherence due to side effects
- Evidence of malabsorption.

Each of the above scenarios could result in sub-therapeutic drug levels and poor clinical response. In such cases, the regimen in question may be salvaged with palliative medication and/or patient education. If clinical assessment indicates the presence of treatment failure due to confirmed drug resistance, the best approach is to switch to an entirely new regimen, choosing two or more drugs to which the patient is naive as the second line drug regimen. Before changing to the second line drug regimen, the patient needs to go through the treatment readiness and education process again. This needs to be carefully monitored as some patients might hide their non-adherence.

1.8 Second-line antiretroviral therapy in adults and adolescents

Drugs used as the second line drugs in Tanzania include:

NRTIs

- Tenofovir (TDF)
- Abacavir (ABC)

PIus

- Lopinavir boosted by Ritonavir (LPV/r)
- Atazanavir boosted by Ritonavir (ATV/r)

The second line NRTI choice for adults and adolescents depends on the first line regimen. For patients on AZT or d4T in first line, the default second line option is to use TDF plus 3TC or FTC combined with a ritonavir-boosted PI, either LPV/r or ATV/r. (TDF+3TC or FTC +LPV/r or ATV/r)

If patients were started on TDF and had never used AZT regimen, the default second line option will be AZT based regimen.

For patients who were initiated on TDF in first line because of intolerance to AZT and d4T, the default second line option is to use ABC plus 3TC combined with a ritonavir-boosted PI, either LPV/r or ATV/r. (ABC + 3TC + LPV/r or ATV/r)

Doses for these drugs are given in Appendix 4.

Note that LPV/r, TDF/3TC and TDF/FTC are currently available as FDC formulations which simplify dosing and administration.

1.9 ART in Women of Childbearing Potential or Pregnant Women

The guiding principle for the treatment of women of childbearing potential or pregnant women is that therapeutic decisions should be based solely on their need and eligibility for ART. The recommended first-line regimen for this patient subgroup is: **AZT + 3TC + NVP**. However, special circumstances of pregnancy or breast-feeding raise additional issues concerning toxicity to mothers and children, the choice of ARV drugs, and the prevention of HIV transmission from mothers to infants.

Women who are receiving ART and become pregnant should continue their treatment unless they are in the first trimester of pregnancy and EFV has been part of the regimen, in which case, EFV should be discontinued and replaced by NVP.

Note: ARV drugs have the potential to either decrease or increase the bioavailability of steroid hormones in hormonal contraceptives. Thus, if a woman on ARV treatment decides to initiate or continue hormonal contraceptive use, the consistent use of condoms must be recommended for preventing HIV transmission. This may also compensate for any possible reduction in the effectiveness of the hormonal contraceptive.

1.10 Antiretroviral drugs for non-ART naive patients

Treatment for patients who have been previously exposed to antiretroviral therapy should be discussed with an authorized medical personnel before they are enrolled in the CTC and (re)started on treatment.

Generally:

- Patients that are controlled on their antiretroviral medication at appropriate doses should continue on the same regimen if possible.
- Those who stopped for reasons other than treatment failure and for whom failure is not suspected, can restart the original regimen.
- Those known or suspected to have failed a previous regimen should be started on drugs they have not been exposed to before as appropriate.

1.11 Antiretroviral drugs for Intravenous Drug users (IDU) on Methadone Assisted Therapy

Drug use and addiction do not preclude successful ARV treatment. HAART is as effective for HIV positive IDUs as it is for other people with HIV/AIDS. Given appropriate support, former and active IDUs can adhere just as well as others and should have equal access to HAART. Special attention should be paid to the particular needs of former and active IDUs when administering ART, including those related to substance dependence, co-morbidities and co-infections. ART might be started not earlier than 2 -3 months after starting Methadone assisted therapy. There is an increased risk of interactions through cytochrome CYP450 3A between Nevirapine, Efavirenz, Ritonavir and Methadone.

Give once daily regimen:

- Efavirenz(EFV) 600mg+Tenofovir (TDF) 300mg+Emtricitabine (FTC)200mg or
- Efavirenz (EFV) 600mg+Abacavir (ABC) 600mg+Lamivudine (3TC) 300mg
- Nevirapine(NVP) 400mg+TDF 300mg+FTC 300mg
- Nevirapine 400mg+ABC 600mg+Lamivudine 300mg.

Note:

- Efavirenz decrease Methadone plasma concentration up to 50% it requires constant methadone dose correction
- Nevirapine decrease methadone plasma concentration by up to 80% in addition increased propensity to liver toxicity and skin rash

Combination for second line

Give once daily regimen:

Lopinavir 800mg/Ritonavir 200mg+TDF 300mg+FTC 200mg

OR

Lopinavir 800mg/Ritonavir 200mg+Abacavir 600mg+Lamivudine 300mg.

OR

Atazanavir 300mg/Ritonavir 100mg+TDF 300mg+FTC 200mg

OR

Atazanavir 300mg/Ritonavir 100mg+Abacavir 600mg+Lamivudine 300mg.

Note: Boosted Atazanavir has no interaction with Methadone, is well tolerated and has high genetic barrier to resistance development. It's contraindicated in liver failure.

2.0 ANTIRETROVIRAL REGIMENS FOR HIV INFECTED INFANTS AND CHILDREN

Most antiretroviral drugs approved for treatment of HIV infection can be used for children. However, there may be limitations for young children requiring syrup or liquid formulations as there some ART drugs that are not available in these formulations. Moreover, pharmacokinetic parameters in children vary with age and therefore are more complicated than in adults. There are some Paediatric FDCs now available. The use of tablets that require cutting in order to use a portion of the drug should be discouraged as it can lead to under dosing or overdosing of the drug. This in turn can lead to an increased risk of resistance or toxicity. Dosing in children is usually based on either body surface area or weight. Drug doses must be adjusted as the child grows in order to avoid risk of under dosage, resistance to drugs and sub optimal response. Standardization is also important so that non-expert personnel can safely dispense correct

doses. It is therefore preferred to provide health care workers with job aids such as dosing charts or dosing wheel that can be administered according to weight bands.

Criteria for Initiating Antiretroviral Therapy in Children (eligibility for ART) – *Refer current WHO clinical staging of HIV/AIDS*)

2.1 Initiation of ART for Infants and children under 24 months

Initiation of ART is recommended for all children below 24 months of age who have a confirmed diagnosis of HIV irrespective of WHO Paediatric Staging and irrespective of CD4 percentage or CD4 count. For children less than 18 months old HIV–infection needs to be virologically proven (using HIV DNA PCR, HIV RNA PCR). For children 18 months of age or older two positive antibody test confirm HIV infection. HIV exposed and serological test positive children aged less than 18 months with neither virological confirmation nor CD4 count or % available but who meet WHO criteria for severe HIV disease (see presumptive diagnosis of HIV page 114) should be initiated on ART. In such cases, HIV antibody testing must be repeated at age 18 months to definitely confirm that the child is HIV infected. Only children with confirmed infection should continue with ARV therapy.

2.2 Initiation of ART for Children 24 months or older

For children over 18 months of age, a positive antibody test is an indication of HIV infection since any acquired antibodies from the mother would have degenerated, but needs to be confirmed by a second serological test.. All children older than 2 years in WHO Paediatric Stage 3 or 4 HIV diseases should start ART irrespective of CD4 % or count and all children in Stage 1 or 2 with:

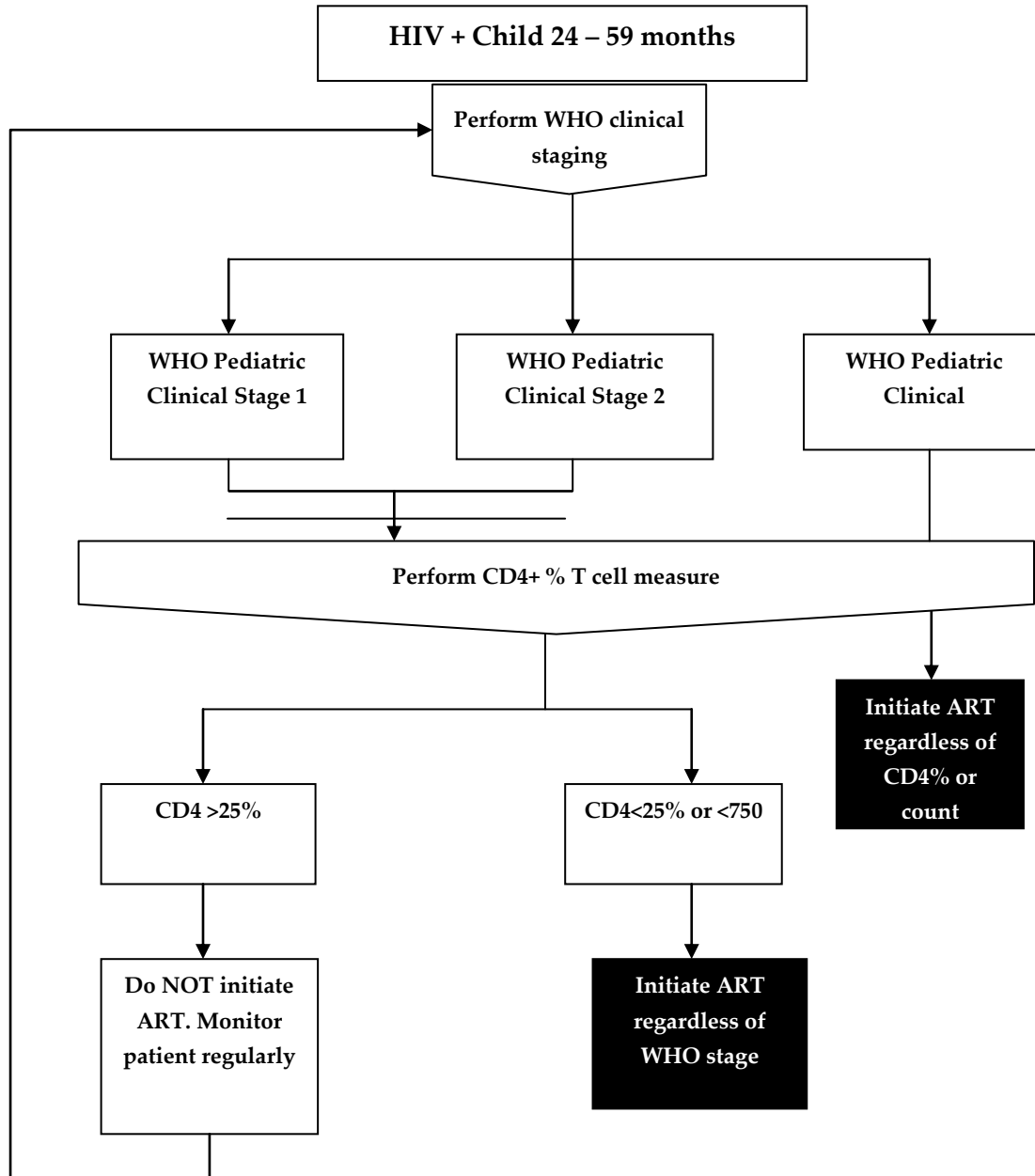
Table 3: CD4 Age-adjusted thresholds for ART initiation in children

Age	Infants < 24 months	24 – 59 months	5 years or over
CD4 percentage	All	≤ 25%	N/A
Absolute CD4		≤ 750 cells/mm ³	≤ 350 cells/mm ³

Table 4: Criteria for ART initiation in HIV infected children

Age	Clinical stage	Immunological status
< 24 months	<i>Treat all</i>	
> 24 months	Stage 4*	<i>Treat all</i>
	Stage 3*	<i>Treat all</i>
	Stage 2*	Treat if CD4 is below age-adjusted threshold (see table below) Don't treat if no CD4 is available
	Stage 1*	

Figure 3: Clinical Eligibility Criteria for ART in Children 24 to 59 months



Breastfeeding and ART

The penetration of ARVs into human breast milk in lactating women has not been quantified for most ARVs. Although some ARVs, such as Nevirapine, are known to be present in breast milk, the concentration and quantity of drug that would be ingested by the infant would be less than needed to achieve therapeutic levels. As a matter of fact infected breastfeeding infants whose mothers are receiving ARV therapy may end-up with sub-therapeutic levels of some ARVs and this could lead to development of drug resistance in the infant's virus. Thus, if a breastfeeding infant requires ARV treatment, ARVs at standard pediatric doses should be initiated regardless of whether the mother is receiving ARV therapy or not. There is no risk of ARV overdose or toxicity in a breast feeding baby with a mother who is on ART.

Evaluation to be done before initiating therapy in children

A good history of the patient should be taken together with a thorough physical examination.

The following baseline clinical assessment should be done:

- Weight, height, head circumference and other measures of growth
- Clinical staging of HIV disease
- Developmental status
- Screening for malaria, TB disease, and exposure to TB
- Identification of concomitant medical conditions (e.g. hepatitis B or C infection, TB, other Co-infections or OIs, pregnancy in adolescent girls)
- Details of concomitant medications, including Cotrimoxazole and traditional or herbal therapies
- Nutritional status, including assessment of the quality and quantity of intake
- For those eligible for ART, assessment of the child's and caregiver's preparedness for therapy

Baseline laboratory tests

Laboratory tests that should be done as shown in table 12 below

Treatment Using ARV Drugs in children

The guiding principles for antiretroviral treatment apply for children are the same as for adolescents and adults. Any child irrespective of the age, diagnosed to be HIV infected should immediately be referred to CTC. The initial management should include a complete physical assessment and staging using WHO staging system as well as complete history including possible exposure to ARV (i.e. for PMTCT or treatment). One objective is the evaluation for presence of active Opportunistic infections.

Children under 2 years should be initiated on ART as soon as possible and waiting for results of laboratory tests should not delay treatment initiation. The first line treatment options for children are as follows in preferential order:

Less than 36 months of age:

- Zidovudine (AZT)+Lamivudine (3TC)+Nevirapine (NVP)
- Abacavir (ABC)+Lamivudine (3TC)+ Niverapine (NVP)
- Stavudine (d4T) + Lamuvidine (3TC) + Nevirapine (NVP)

36 months or older and bodyweight 10kg or higher:

- Zidovudine (AZT)+Lamivudine (3TC)+ Efavirenz (EFV) or Nevirapine (NVP)
- Abacavir (ABC)+Lamivudine (3TC)+Efavirenz (EFV) or Niverapine (NVP)
- Stavudine (d4T) + Lamuvidine (3TC) + Nevirapine (NVP)

Note: Stavudine (d4T) is an alternate for AZT in cases of anaemia (i.e. haemoglobin of <7.5g/dl). However, it should be noted that d4T in liquid formulation needs refrigeration. Side effects of Stavudine such as peripheral neuropathy are less common than in adults but this may be because they are difficult to recognise in children. From age of 12 years onwards Tenofovir (TDF) is the alternative drug for AZT and d4T.

Antiretroviral Drugs for ARV exposed children

If the mother received ARVs during pregnancy, either for her own treatment and /or to prevent mother to child HIV transmission (PMTCT) , there is a possibility that she may transmit a resistant virus to her baby. Resistance could also develop in the infant who has used ARV for prophylaxis. This is particularly the case if NVP, either alone or as a component of a two-drug regimen for PMTCT.

Children who require ARV therapy and who have previously received either single-dose NVP or 3TC or daily NVP while breastfeeding as MTCT prophylaxis should be given a PI based regimen. If PI based regime is unavailable these children should be given the first line regimen available. For dosing of ARV regimens see Annex 5, Paediatric Antiretroviral Dosing.

Recommended Second-Line ARV Therapy for Infants and Children

The recommended second line regimen for infants and children are as follows:

- After failure on a first-line NNRTI-based regimen, a boosted PI(LPV/r) plus 2 NRTIs are recommended for second-line ART
- After a failure of first line of LPV/r + 2 NRTIs; NNRTI + 2 NRTIs is the recommended choice
- After failure on a first-line regimen of AZT or d4T + 3TC then ABC + 3TC is the preferred NRTI backbone option for second-line ART.

3.0 USE OF ARVS IN SPECIAL CIRCUMSTANCES

ART Eligibility for patient with TB/HIV CO-INFECTIONS?

ART should be initiated for *all* people living with HIV with active TB disease irrespective of CD4 cell count. TB treatment should be started first, followed by ART as soon as possible, within the first 8 weeks of starting TB treatment. The recommended first-line ART regimens for TB patients are those that contain Efavirenz (EFV), since interactions with anti-TB drugs are minimal.

For those who are unable to tolerate or have contraindications to an EFV-based regimen, **AZT +3TC + NVP** or **TDF +3TC** or **FTC + NVP** or a triple NRTI regimen e.g **AZT+3TC+TDF** is recommended.

When using Nevirapine based regimen, the patient should be started on a normal dose (200mg bd). **Note:** A loading dose is not required.

In individuals who need TB treatment and who require an ART regimen containing a boosted protease inhibitor (PI), it is recommended to use Rifampicin and a boosted antiretroviral regimen containing Lopinavir with additional Ritonavir dosing (LPV/r 400mg/ 400mg BID). This

regimen is associated with high levels of toxicity, and requires close clinical and laboratory monitoring.

Treatment of TB for HIV Infected Children

In principle, TB treatment in children does not differ from that in adults. Nearly all pulmonary TB in children is sputum smear negative (in most cases smear is “not done”) or extra-pulmonary tuberculosis and thus fall into category III. However, severe forms of TB such as meningitis, miliary TB or TB of the spine should be defined as category I. Treatment can be provided with adult formulation following the dose-body weight relationship presented. For children with severe forms of TB, Ethambutol is recommended at a dose of 15 mg/kg (2RHZE/4RH). The feared side effect of retro-bulbar neuritis is rarely seen in children taking higher dosages exceeding 20 mg/kg for a long period of time. Nevertheless, if there is any doubt, an alternative regimen (2RHZ/4RH) for young children can be applied.

BCG vaccination

In HIV positive neonates, BCG rarely causes disseminated infection of *M. bovis* and if it occurs it should be treated with 2{RH}E/4RH. The WHO recommends that in countries with a high prevalence of tuberculosis like Tanzania, BCG should be given to all neonates immediately after birth, regardless of HIV status. The possible benefits of BCG outweigh the possible disadvantages. However, BCG should not be given to children who present with clear signs and symptoms of HIV-disease or AIDS.

4.0 HIV PREVENTION METHODS

The close association between TB and HIV infection necessitates that specific policies will be needed to guide the nation in introducing and implementing HIV preventive services for all TB patients.

Provision of Cotrimoxazole Preventive Therapy

TB patients who are co-infected with HIV are eligible to receive Cotrimoxazole prevention therapy. Cotrimoxazole therapy is effective in preventing secondary bacterial and parasitic infections.

A: Cotrimoxazole 960mg (O) once a day

Provision of Antiretroviral Therapy

Antiretroviral therapy improves the quality of life and greatly improves survival rates for PLHA. High levels of adherence is required in order to achieve long-term benefits and minimise the risk of developing drug resistance

Reduce the burden of TB in PLHIVs (3Is Strategies)

Since TB is a leading opportunistic infection among the causes of deaths in people living with HIV, regular screening for TB to all PLHIV is crucial for success in reducing morbidity and mortality of those living with HIV. According to the WHO 2004 the interim policy on collaborative TB/HIV activities, strategies for controlling TB in persons with HIV infection should include:

Establish Intensified TB case finding (ICF)

Intensified TB case finding involves screening for symptoms and signs of TB (TB screening tool) in settings where HIV-infected people are concentrated. Early identification of signs and symptoms of TB, followed by diagnosis and prompt treatment in people living with HIV/AIDS, their household contacts, groups at high risk for HIV, and those in congregate settings (e.g., prisons, police quarters, military barracks, refugee camps, mining camps, schools, and living quarters for workers, especially labour-intensive agricultural areas), increases the chances of survival, improves quality of life, and reduces transmission of TB in the community

Isoniazid Preventive Therapy (IPT)

TB disease develops in only 10% of all the individuals infected with *M.tuberculosis*. However, in HIV infected individuals this can be up to 50%.TB preventive therapy is an intervention that should be part of the package of care for people living with HIV. IPT is given to individuals with latent infection of *M. tuberculosis* in order to prevent progression to active disease. In these patients, the risk of developing tuberculosis is reduced by about 60% and their survival is also prolonged. Isoniazid is given daily for six to nine months and the protective effect is expected to last for 18 months. This therapy requires several steps to be taken, including identification of HIV-positive clients, screening to exclude active TB and monitoring of client' s adherence to treatment.

Eligibility for TB Preventive Therapy among PLHAs

For patients with no history of TB treatment:

- All HIV positive individuals with no signs or symptoms suggestive of active TB and with positive tuberculin skin test are eligible for TB preventive therapy.
- A Tuberculin skin test should be offered to all HIV infected individuals where possible.

For patients with history of TB treatment:

- Patients who had active tuberculosis in the past 2 years should not be considered for preventive therapy.
- Patients who were treated for tuberculosis more than 2 years earlier may be considered because they may have already been re-infected with TB.
- Patients who receive TB preventive therapy and who are eligible for antiretroviral therapy can complete their TB preventive therapy even if ART is started as there is no interaction between Isoniazid and the current ART regimen used.

IPT should only be offered in the following situations:

- Where quality supportive counselling is available
- After effective screening for active TB
- Where there is capacity for follow up and monitoring of patients to encourage adherence to preventive therapy
- Where there is capacity to manage side effects and exclude active TB during IPT

CHAPTER TWENTY ONE

OTHER VIRAL INFECTIONS

1.0 Measles

Measles is caused by a paramyxovirus which is spread by droplet infection. The main clinical features include fever and generalized maculopaular (Red rash appearing first behind the ears and spreading to rest of body) plus any of the following: Cough, runny nose or conjunctivitis. Others include lacrimation, photophobia, and copius nasal discharge, koplik spots, tearing and eyelid oedema. It is rare at the age of less than 6 months. It is recommended that all children should be vaccinated at the age of 9 months. .

Treatment

Adults:

A: Paracetamol tablets 1g every 8 hours for 5 days

Plus

A: Vitamin A 200,000 IU orally

Plus

A: Tetracycline eye ointment 1% apply once daily for 7 days.

Children: Give **Paracetamol** 10-15mg/kg body weight every 8 hours for 5 days Plus **Vitamin A** if less than 1 year give 100,000 IU stat and if over 1 year give 200,000 IU

Note: Give extra fluid and food

2.0 Poliomyelitis

It is a rare cause of hypotonia with abrupt onset of weakness (often asymmetrical) in association with a febrile illness. It is caused by one of the three related polio viruses, types 1, 2 and 3 which comprise a subdivision of the groups of enteroviruses. Clinical features of the disease can be divided into three group's i.e.

- Non-specific febrile illness of 2-3 days duration without CNS involvement
- Aseptic meningitis include features mentioned above
- Paralytic poliomyelitis – which is the major possible outcome of the infection but occurs in less than 10% of those infected.

Treatment guidelines

Give supportive therapy

Prevention

- This disease is preventable by immunization with polio vaccine starting at birth. Give 4 doses at intervals of 4 weeks.
- Parents should be told about the World program to eliminate Polio and the importance of actively participating.

3.0 Viral Hepatitis

Viral hepatitis is a systemic infection predominantly affecting the liver. It is almost always caused by one or another of the hepatitis viruses; A, B, C, and delta viruses. The clinical spectrum of the disease due to viral hepatitis is variable. These ranges from asymptomatic and inapparent to fulminant and fatally acute infections. Subclinical persistent infections with hepatitis virus B and C may progress to chronic liver disease, cirrhosis and possible hepatocellular carcinoma.

Treatment guidelines

Treatment is mainly supportive; the condition can be self-limiting (healing on its own) or can progress to fibrosis (scarring) and cirrhosis.

Prevention

Hepatitis types A,B and C are preventable by immunization. Vaccines for other types may become available.

Table 1: Viral hepatitis nomenclature (Hepatotropic viruses Antigens and Identified Antibodies)

Type	Antigen	Antibody
Hepatitis A virus (HAV)	HAV	anti-HAV*
		IgM anti-HAV
Hepatitis B virus (HBV)	HBsAg*	anti-HBsAg*
		IgM anti-HBsAg*
	HBcAg	anti-HBcAg*
	HBeAg*	anti-HBeAg*
Hepatitis C Virus (HCV)	HCV	anti-HCV*
Hepatitis D Virus (HDV)	HDVAg	anti-HDV*
Hepatitis E Virus (HEV)	HEV	anti HEV8*
		IgM anti-HEV
Hepatitis G Virus (HGV)	HGV	anti-HGV

3.1 Hepatitis A

Essentials of diagnosis

Infection occurs in both epidemic and sporadic. Typical feature are:-

- GI upset (anorexia, vomiting, diarrhoea).

- Jaundice, tender and enlarged liver.
 - Abnormal liver function tests.
 - Anti-HAV IgM elevated.
 - RNA virus.
 - Low social economical status (poor hygiene)
- Mode of transmission:** Mainly fecal - oral route.

Clinical presentation

- History of direct exposure to a previously jaundiced individual.
- Consumption of seafood or contaminated water.
- Initial non-specific symptoms usually precede the development of jaundice by 5-10 days.
- Fever, anorexia and epigastric pain are the usual symptoms.
- Darkening of the urine precede jaundice, which peaks in 1-2 weeks and then begins to subside.
- Tender hepatomegaly and jaundice are typically present; splenomegaly is variable.

Differential diagnosis

Before jaundice appears, the symptoms are those of non-specific enteroviral diseases

Note: Hepatitis mainly resolves spontaneously (95%) but rarely complicates into fulminant Hepatitis that is fatal.

Lab investigations and findings

- A positive anti-HAV IgM indicates acute disease, where as IgG anti-HAV persist after recovery or chronic disease.
- The initial lab evaluation should include biochemical tests for hepatic inflammation and tests of liver function (ALAT,ASAT Bilurubin total and direct bilirubin Alkaline phosphatase),
- ALAT and ASAT level are elevated and roughly reflect the degree of parenchymal inflammation. Elevated alkaline phosphatase, gamma glutamic acid and total and direct (conjugated) bilirubin levels are indicators of the degree of cholestasis, which may be a result of hepatocellular and bile duct damage.
- FBP, Leukocyte count is normal or low
- Hypoalbuminaemia, hypoglycaemia, and marked prolongation of prothrombin time are serious prognostic findings.
- Stool for macroscopic (consistence varies) and microscopic examination. (Parasitic ova, RBC's).

Treatment

Supportive treatment: For pain give paracetamol 15mg/kg /dose).

Prevention

General measures: Sanitation and hygiene that includes hand washing, proper disposal of infectious materials.

3.2 Hepatitis B

Essentials of Diagnosis

- History of parenteral, sexual, or house hold exposure, maternal HBsAg carriage
- GI upsets,

- Jaundice,
- Tender hepatomegaly.
- Presence of HBs Antibody used to document recovery and/or immunity to HBV infection.

Mode of transmission

Mainly through parenteral, sexual and vertical transmission 5%

Clinical presentation

- The symptoms are non-specific, consisting only of slight fever (which may be absent) and mild gastrointestinal upset
- Visible jaundice is usually the first significant finding
- Dark urine and pale or clay-coloured stools
- Hepatomegaly is present
- Occasionally a symptom complex (caused by antigen-antibody complexes) of macular rash, urticarial lesion, and arthritis antedates the appearance of icterus.

Lab investigation and findings

- To diagnose acute HBV infection, the HBsAg, and anti-HBs,
- Other investigations are like above with HAV infection plus alpha -1- ant trypsin, PTT
- Abdominal ultrasound when complication suspected.

Treatment

- Supportive
 - Low fat diet, oral fluids,
 - Give paracetamol (dose as above) if pain present
- Specific treatment
 - The use of interferon alfa in children has not yet established.
 - Lamivudine
 - In children 2-11years-3mg/kg/once daily
 - In children 12-17 years and adults-100mg daily

Note: Patient Receiving lamivudine for concomitant HIV infection should continue to receive lamivudine in appropriate dose for HIV infection

- Hepatitis B Vaccination and Prevention

There are two components for **preventing** hepatitis B:

- Prevention of transmission of the virus
- Immunisation

Immunization recommendations

Hepatitis B vaccine is safe and effective, but should not be seen as an alternative to a strategy of prevention of transmission.

3.3 Chronic hepatitis C infection

It is another cause of cirrhosis and hepatocellular carcinoma and is transmitted by parenteral routes. Acute infection is often milder than Hepatitis A with moderately raised transaminases. Anti-HCV is positive.

Treatment

Interferon alfa: Usual dose: SC, 5–10 million units 3 times weekly for 4–6 months.

Category	Management
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3.4 Hepatitis D virus (delta virus) infection occurs only in children coinfecting with HB virus.

3.5 Hepatitis E disease is enterically transmitted, resembles infection with hepatitis A, but most commonly affects young adults.

4. Rabies

Rabies is a zoonotic (transmitted from animals) viral neuroinvasive disease caused by a virus that belongs to genus lyssavirus in the family Rhabdoviridae. It causes acute encephalitis (inflammation of the brain) in warm-blooded animals. It is transmitted most commonly to human by a bite from an infected animal but occasionally by other forms of contact. Rabies is almost invariably fatal if post-exposure prophylaxis is not administered prior to the onset of severe symptoms.

The incubation period of the disease depends on how far the virus must travel to reach the central nervous system, may take one week to six months. Once the infection reaches the central nervous system and symptoms begin to show, the infection is practically untreatable and usually fatal within days.

Early-stage symptoms of rabies are malaise, headache and fever, later progressing to more serious ones, including acute pain, violent movements, uncontrolled excitement, depression and inability to swallow water. Finally, the patient may experience periods of mania and lethargy, followed by coma. The primary cause of death is usually respiratory insufficiency.

In unvaccinated humans, rabies is almost always fatal after neurological symptoms have developed, but prompt post-exposure vaccination may prevent the virus from progressing.

Management: Post Exposure treatment consists of local treatment of the wound, followed by antirabies vaccine therapy (with or without rabies immunoglobulin) for contacts of category II and III.

A summary of management of rabies exposed individuals is as indicated below:

<p>CATEGORY I</p> <p>touching or feeding animals, licks on the skin</p>	<p>No treatment</p>
<p>CATEGORY II</p> <p>nibbling of uncovered skin, minor scratches or abrasions without bleeding, licks on broken skin</p>	<p>Wash wound with running water and soap for 15 minutes.</p> <ul style="list-style-type: none"> • Administer antirabies vaccines: <ul style="list-style-type: none"> - 0.2ml (ID) in divided doses of 0.1 ml on deltoid on one hand and another 0.1ml on the deltoid of the second hand on days 0, 3, 14 and 28 OR - 1 ml (IM) on deltoid muscle for days 0, 3,7,14, and 28 <p>Note: Children are given the same doses but vaccine should be administered on the lateral part of the thigh.</p>
<p>CATEGORY III</p> <p>single or multiple transdermal bites or scratches with bleeding, contamination of mucous membrane with saliva from licks; exposure to bat bites or scratches</p>	<p>Wash wound with running water and soap for 15 minutes.</p> <ul style="list-style-type: none"> • Administer Rabies Immunoglobulin (RIG) on day 0 • 40 IU/kg body weight for Equine (ERIG) • 20 IU/kg body weight for Human (HRIG) • Administer antirabies vaccines <ul style="list-style-type: none"> - 0.2ml (ID) in divided doses of 0.1 ml on deltoid on one hand and another 0.1ml on the deltoid of the second hand on days 0, 3, 14 and 28 OR - 1 ml (IM) on deltoid muscle for days 0, 3,7,14, and 28 • Note 1: Children are given the same doses but vaccine should be administered on the lateral part of the thigh. • Note 2: The World Health Organization recommends ID route of vaccination administration because it is cost effective.

For rabies-exposed patients who have previously undergone complete pre-exposure vaccination or post-exposure treatment with cell-derived rabies vaccines, antirabies vaccines are given at days 0 and 3 regardless of route of administration i.e ID or IM. Rabies immune globulin treatment is not necessary in such cases. The same rules apply to persons vaccinated against rabies who have demonstrated neutralizing antibody titres of at least 0.5 IU/ml.

5.0 VIRAL HEMORRHAGIC FEVERS

5.1 EBOLA

Ebola Hemorrhagic fever (Ebola HF) is a severe often fatal disease in humans and non-humans (monkeys, gorillas and chimpanzees). It caused by ebola virus of the family Filoviridae.

Transmission

The natural reservoir of the virus is unknown, the manner in which the virus first appears in a human at the start of an outbreak has not been determined. Researchers have hypothesized that the first patient becomes infected through contact with an infected animal. After the first case-patient in an outbreak setting is infected, the virus can be transmitted in several ways:

- Direct contact with blood or other secretions of an infected person (blood, secretions, organs or other bodily fluids)
- Exposure to Ebola virus through contact with objects, such as needles, that has been contaminated with infected secretions.
- Nosocomial transmission i.e. exposure to the virus has occurred when health care workers treated individuals with Ebola HF without wearing PPE
- Burial ceremonies where mourners have direct contact with the body of the deceased person.
- Through handling of infected chimpanzees, gorillas, and forest antelopes- both dead and alive

Incubation period

Incubation period is between 2 to 21 days. Infections with Ebola virus are acute. All age groups are susceptible to infection. There is no carrier state.

Signs and symptoms start with sudden onset of fever, intense weakness, muscle pain, Headache and Sore throat. These symptoms are followed by vomiting, diarrhea, rash, impaired kidney and liver functions.

In some cases; rash, red eyes, hiccups, both internal and external bleeding can occur.

Treatment

There is no specific treatment, cure, or vaccine for Marburg Hemorrhagic fever. However, supportive hospital therapy should be utilized. These include:

- Fluid and Electrolyte balancing
- Maintaining oxygen status
- Blood transfusion and clotting factors
- Treat for any complicating infections.

5.2 Marburg Hemorrhagic Fever

Marburg is severe type of hemorrhagic fever which affects both animals and humans. It is related to Ebola virus and a parent type belongs to Viral Hemorrhagic fevers of Filoviridae family.

Mode of transmission

How the animal host first transmits Marburg virus to humans is unknown. However, humans who become ill with Marburg hemorrhagic fever virus may spread virus to other people. For example, persons who have handled infected monkeys and have come in direct contact with their fluids or cell cultures have become infected. Spread of the virus between humans has occurred in a setting of close contact, often in a hospital. Droplets of body fluids, or direct contact with persons, equipment, or other objects contaminated with infectious blood or tissues are all highly suspect as sources of disease. Transmission through infected semen can occur up to seven weeks after clinical recovery.

Transmission does not occur during the incubation period.

Incubation Period

Incubation period is between 3-9 days. All age groups are susceptible but most case to adults. Signs and symptoms are into two phases:

Phase One: Sudden onset of fever, chills, headache and myalgia.

Phase Two: Maculopapular rashes, Trunk rash, Nausea, Vomiting, Sore throat, Abdominal pain, Diarrhea, Jaundice, Pancreas inflammation, Severe weight loss
Liver failure, Massive hemorrhage (all orifices), Multi-organ dysfunction, Delirium, Shock, and Death.

Prognosis

Case fatality rate of Marburg is between 23-25% (But the Angola situation the CFR was >90%)

Treatment

There is no specific treatment, cure, or vaccine for Marburg Hemorrhagic fever. However, supportive hospital therapy should be utilized. These include:

- Fluid and Electrolyte balancing
- Maintaining oxygen status
- Blood transfusion and clotting factors
- Treat for any complicating infections.

5.3 Rift Valley Fevers

Rift Valley Fever is a viral zoonosis that primarily spread amongst animals by the bite of infected mosquitoes. Rift Valley virus is a member of Phlebovirus genus in the family Bunyaviridae. A wide variety of mosquitoes may act as vector transmission in different regions. *Aedes* mosquitoes are the main vector biting animals. Transmission to human is mainly through direct or indirect contact with blood or organs of infected animals. The virus can be transmitted to human through the handling of animal tissue during slaughtering or butchering, assisting with animal births, conducting veterinary procedures. The virus infects human through inoculation e.g via wound from infected knife or through contact with broken skin or through inhalation of aerosols produced during the slaughter of an infected animals. Human can also get infection through infected mosquito.

Human become viraemic; capable of infecting mosquitoes shortly before onset of fever and for the first 3–5 days of illness. Once infected, mosquitoes remain so for life.

Incubation period is between 2-6 days. Signs and symptoms are Influenza like illnesses: sudden onset of fevers, headache, myalgia, backache neck stiffness photophobia and vomiting. Meningoencephalitis and haemorrhagic fever syndrome follow thereafter.

Most human cases are relatively mild small proportion develop a much more severe disease. The total case fatality rate is less than 1%. Symptoms last from 4-7 days after which the immune response to infection becomes detectable with appearance of IgM and IgG. And disappearance of circulating virus from blood stream.

Treatment

There is no any established course of treatment of this disease.

Most of human cases are relatively mild and of short duration so will not require any specific treatment.

Studies in monkeys and other animals have shown promise for ribavirin. Interferon, immune modulators and convalescent phase plasma can also help.

5.4 Yellow Fever

Yellow fever is an acute viral infection that is transmitted to human through a bite of infected mosquito-predominantly *Aedes* mosquitoes. It is caused by a virus that belongs to Flavivirus. Though many cases of yellow fever are mild and self-limiting, the disease can also be a life threatening causing hemorrhagic fever and hepatitis. It is endemic in equatorial Africa and South America, with estimated 200,000 cases and 30,000 deaths annually. Overall case-fatality rate in Africa 23%

Incubation period of 2-6 days and human become viremic - capable of infecting mosquitoes, shortly before onset of fever and for the first 3–5 days of illness. Once infected, mosquitoes remain so for life

Treatment, prevention and control

No specific anti-viral treatment, supportive therapies are recommended.

Prevention and Control involve mosquito control and provision of yellow fever vaccine.

Indication for Yellow fever vaccine:

- persons \geq 9 months of age
 - Planning travel to or residence in an endemic area
 - Planning travel to a country with an entry requirement
- Needs to be given \geq 10 days prior to arrival in endemic area
- Revaccination at 10 year intervals

6.0 Expanded Program on Immunization

The childhood diseases which are targeted by Expanded Programme on Immunization (EPI) in Tanzania are: Tuberculosis, Poliomyelitis, Diphtheria, Whooping cough, Tetanus, Hepatitis B, Measles and *Haemophilus influenza* type B infections.

Table 2: The schedule for immunization for children is as follow:

Age	Vaccine	Type of vaccine/state	Disease prevented	Remarks (dose, site and route)	Protection
Birth	1.BCG	Live attenuated/Freeze dried	Tuberculosis	0.05ml Intradermally (Right shoulder)	Life long

				2 drops orally	
	2.OPV 0*	Live attenuated/ Liquid	Poliomyelitis		
1 Month	1. OPV	Live attenuated / Liquid	Poliomyelitis	2 drops orally	
	2.DTPHepB Hib 1 (Pentavalent 1)	Killed bacteria, toxins and genetically modified vaccines /Liquid	Diphtheria Tetanus Pertusis Hepatitis B Haemophilus influenza type b infections	0.5 ml Intramuscular (Left thigh)	
2 months	1.OPV 2	Live attenuated /Liquid	Diphtheria Tetanus Pertusis Hepatitis B Haemophilus influenza type b infections	2 drops orally	
	2.Pentavalent	Liquid		0.5 ml Intramuscular (Left thigh)	
3 Months	1.OPV 3	Live attenuated /Liquid	Poliomyelitis	2 drops orally	Full dose life long
	2.Pentavalent	Liquid		0.5 ml Intramuscular (Left thigh)	Full dose 10 years
9 Months	Measles	Live attenuated / Freeze dried	Measles	0.5ml Deep SC or IM (Right thigh)	Life long

*Do not give after 14 days

CHAPTER TWENTY TWO

OTHER PROTOZOA INFECTIONS

1.0 Leishmaniasis

This group of diseases is caused by protozoa of the genus *Leishmania*. It can take two forms i.e. generalized visceral infection (kala-azar) or a purely cutaneous infection (oriental sore). Onset of kala-azar is shown by low grade fever, splenomegaly, enlarged liver and lymphadenopathy. In the cutaneous form, single or multiple lesions are found on exposed parts, from where *Leishmania* Donovan bodies can be demonstrated.

Treatment

Visceral/cutaneous leishmaniasis

First choice

C: Sodium stibogluconate 20mg IM/slow IVper kg body weight per day for 30 days. Maximum dose 850 mg per day.

If parasites persist, treatment may be repeated, two to three times with a ten day interval in between.

Second choice

C: Pentamidine Isethionate I.M at 2 to 4 mg/kg body weight every 48 hours for a total of 10 injections.

Since an immediate hypotensive reaction may occur, patients should lie down during the injection and adrenaline should be at hand. Pentamidine like Suramin is contraindicated in renal disease. Further, due to possible nephrotoxicity, urine must be examined for albumin and/or casts. The presence of either contraindicates continued use of **pentamidine**.

Children The same dosage as above

CAUTION: Close medical supervision is necessary during treatment

2.0 Trypanosomiasis

The causative organisms are the parasitic protozoa of *Trypanosoma brucei gambianse* and *T. brucei rhodesiense*. Clinical features include fever, lymphadenopathy and CNS involvement like headache, mental confusion, tremors and pyresis. However for relevance in treatment, two clinical divisions are noted, that is, there are patients with no CNS involvement and those with CNS signs/symptoms.

Treatment Medicine of choice

Suramin is the medicine of choice for the early stages of African trypanosomiasis (T.b.g.) before there is CNS involvement.

C: Suramin 20mg/Kg (to a max. of 1g in adults) (IV) given every week for 5 – 6 weeks

Second choice

C: Melarsoprol 100mg (children 20 mg) I.V as a test dose then if there is no reaction give 20mg/kg body weight single dose, freshly prepared (maximum 1 g) every 5 – 7 days.

NOTE:

- Usual course is 5 doses (do not exceed 7 doses or a total of 6 g)
- Suramin may cause renal toxicity therefore it is contraindicated in renal diseases
- Further, due to possible nephrotoxicity, urine must be examined for albumin and/or casts the presence of either contraindicates continued use of Suramin.

In Trypanosomiasis due to T.b gambianse without CNS involvement the recommended drug is

C: Pentamidine isethionate freshly prepared 4 mg/kg I.M every 24 hours for 7 days (Max. 300 mg/dose).

CAUTION:

In patients with CNS involvement:

Start treatment with Suramin (day 1 and 2) for a total of two doses to clear blood of trypanosomes in order to avoid a Jarisch-Herxheimer reaction which will be precipitated by destroying both CNS and peripheral trypanosomes by melarsoprol. Then give melarsoprol 3.6 mg/kg body weight in IV infusion dissolved in 200 ml of dextrose 5% given over a 2 hour period for 3 consecutive days. The patient should lie supine during injection and for five hours afterwards. The patient is then rested for 5-7 days and then the above regime of melarsoprol is repeated. This is done once again after a further rest of 5-7 days, thus completing 3 courses of melarsoprol. Blood film and CSF are then examined for trypanosomes.

CHAPTER TWENTY THREE

OTHER BACTERIAL INFECTIONS

1.0 Anthrax

Anthrax is a disease of animals. However, man is infected directly through contact with infected hides or inhalation of spores in the lungs or ingestion of infected meat. Hence it can be cutaneous, pulmonary and/or intestinal. The main clinical features are itching, a malignant pustule, pyrexia and rarely pulmonary and gastrointestinal signs.

Treatment

Medicine of choice

A: Benzylpenicillin. Adult 0.6 MU I.V every 6 hours until local oedema subsides then continue with

A: Phenoxymethylpenicillin 250 mg 6 hourly for 7 days.

Children

Premature infant and neonate

A: Benzylpenicillin 6mg/kg body weight every 6 hours until local oedema subsides then continues with

A: Phenoxymethylpenicillin 62.5 mg 6 hourly for 7 days.

Infants (1-12 months)

A: Benzylpenicillin 75 mg/kg body weight daily 8 hourly until local oedema subsides then continue with

A: Phenoxymethylpenicillin 62.5 mg 6 hourly for 7 days.

Children (1-12 years)

A: Benzylpenicillin 100 mg/kg body weight daily 6 hourly until local oedema subsides. Then give

A: Phenoxymethylpenicillin 125-250mg 6 hourly for 7 days

Second choice

A: Erythromycin (O) 500 mg 8 hourly orally for 10 days

Children: 10 mg/kg body weight 8 hourly for 10 days

2.0 Mastitis (Breast Abscess)

Mastitis is an inflammation of the breast. The common causative organisms of the disease are either staphylococcus or streptococcal bacteria. The breast becomes red, swollen and painful. In breast abscess, there is a collection of pus in the breast. Clinical features of a breast abscess are tenderness, swelling, red, warm, fever and painful lymph nodes. General: In mastitis stage the treatment is antibiotics and antiflogistics. In abscess stage treatment is both surgical and antibiotics.

Treatment

A: Erythromycin 500 mg orally on the first day then 100mg daily for further 6 days

OR

C: Flucloxacillin 500 mg orally every 6 hours for 7 days in an empty stomach

Plus

A: Acetylsalicylic acid 600 mg orally, after food, give every 6 hours (as needed).

Instruct the patient to apply hot compresses and a constriction bandage to relieve pain in the affected breast, and to express milk if applicable to reduce engorgement.

3.0 Plague

Plague is a zoonotic systemic bacterial infection caused by *Yersinia pestis* (*Y. pestis*, plague bacillus) usually transmitted to humans by rodent fleas. The main disease forms are bubonic, septicaemic and pneumonic with the former being the commonest. The incubation period is within 7 days and case fatality rate may exceed 50 to 60% in untreated bubonic plague and approaches 100% in untreated pneumonic or septicaemic plague.

Treatment

When preliminary diagnosis of human plague is made on clinical and epidemiological grounds:

- Subject the patient to appropriate antimicrobial therapy without waiting for definitive results from the laboratory.
- Use protective gears (gloves, face mask, and gowns) when managing a suspected plague case.

Specific treatment

a) Bubonic plague:

The drugs of choice are: - Doxycycline and Gentamycin

(i) Doxycycline:

Adult and children aged 12 years and above:

Give 100mg every twelve hours for 7 days

Do not use Doxycycline in children below 12 years and pregnant mothers

(ii) Gentamycin:

(a) Adults: Give 3mg/kg IM or IV every 12 hours for 7 days.

(b) Children: Give 7mg/kg/day IM or IV every 12 hours for 7 days

Gentamycin is a drug of choice for pregnant women

(iii) Chloramphenical (as alternative drug)

Adults and children 1 year and above: Give 50mg/kg/day orally or IV every 6 hours for 7 days

(iv) Provide suitable analgesics

(v) Use suitable antiseptics to dress wound in case of bursting of the bubo

CHEMOPROPHYLAXIS OF CONTACTS

Persons in close contact with pneumonic plague patients, or persons likely to have been exposed to *Y. pestis*-infected fleas, to have had direct contact with body fluids or tissues of a *Y. pestis*-infected mammal, or exposed during a laboratory accident to known infectious materials must receive antibiotic preventive therapy.

Combination of drugs Co-trimoxazole (Septrin) is the most effective measure.

(a) Adults: 2 tablets b.id for 7 days

(b) Children: 1 tablet b.i.d for 7 days.

Other sensitive drugs to *Y. pestis* and those patients allergic to sulphadimidine should use **(Chloramphenical and tetracycline)**

4.0 TICK BORNE RELAPSING FEVERS

Tick Borne relapsing fever is a bacterial infection characterized by recurring febrile episodes that last for 3 days and are separated by afebrile periods of 7 days duration. Along with fever, patients may experience a wide range of nonspecific symptoms. Each febrile episode ends with a sequence of symptoms collectively known as a "crisis." During the "chill phase" of the crisis, patients develop very high fever (up to 106.7°F or 41.5°C) and may become delirious, agitated, tachycardic and tachypneic. Duration is 10 to 30 minutes. This phase is followed by the "flush phase", characterized by drenching sweats and a rapid decrease in body temperature. During the flush phase, patients may become transiently hypotensive. Overall, patients who are not treated will experience 1 to 4 episodes of fever before illness resolves.

It is caused by spirochetes known as *Borrelia duttoni*. It is transmitted to humans by a bite of soft tick infected by spirochetes known as *ornithodoros moubata*. The incubation period is within 2 weeks.

Treatment

Treatment involves antibiotics often tetracycline, doxycycline erythromycin and penicillin. Procaine penicillin G should be used when oral therapy is not tolerated.

- Chloramphenicol is administered at 500mg every 6 hrs for 7-10 days.
- Procaine Penicillin G is administered at 600,000 IU daily for 7 days
- In children younger than 8 years and in pregnant or nursing women erythromycin is preferred.

CHAPTER TWENTY FOUR

NUTRITIONAL DISORDERS

Nutritional disorders can be caused by an insufficient intake of food or of certain nutrients; by inability of the body to absorb and use nutrients, or by over-consumption of certain foods. The major nutritional disorders in Tanzania, in ranking order, are:

- Protein-energy malnutrition (deficiency of carbohydrates, fats, protein)
- Nutritional anaemia (deficiency of nutrients that are essential for the synthesis of red blood cells i. e iron, folic acid and vitamin B₁₂)
- Iodine deficiency disorders (deficiency of iodine which is important for the synthesis of the thyroid hormones), and
- Vitamin A deficiency.

Other disorders do exist, though are of less public health significance. These include:

- Overweight/obesity
- Disorders associated with various vitamin deficiencies
- Disorders associated with deficiency of some trace minerals

1.0 PROTEIN-ENERGY MALNUTRITION (PEM)

This develops as a result of inadequate intake of carbohydrates, fats and protein. Deficiency of some micronutrients, particularly iron and vitamin A, become partly responsible for the signs of PEM. Infection also plays a role in the development of the features of PEM. The population group most affected by PEM is children aged below five years. With regard to manifestation, clinical and anthropometric features are distinguished:

1.1 Clinical forms of PEM

- **Underweight** – is moderate malnutrition. Casually the child may appear normal, but on close examination, the child looks thinner and smaller than other children of the same age. Oedema is absent.
- **Marasmus** – is severe malnutrition. The child shows remarkable failure of growth. He has very severe muscle wasting with flaccid, wrinkled skin and bony prominence. The child looks awake and hungry and displays what is referred to as 'old person's face'. Oedema is absent.
- **Kwashiorkor** – is also severe malnutrition. There is failure of growth but the child is not as severely wasted as in marasmus. The abdomen is swollen (hepatomegaly due to fatty infiltration). The child shows hair changes (having turned brown, straight and soft) and rashes on the skin (flaky paint dermatitis). He is inactive, apathetic, irritable and difficult to feed. The child has bilateral oedema.
- **Marasmic-kwashiorkor** – is a condition combining severe wasting (marasmus) and oedema (kwashiorkor). The child has other clinical features characteristic of marasmus and kwashiorkor.

[NB: Presence of oedema (of any grade) is considered severe malnutrition, regardless of the weight of the child].

1.2 Anthropometric features of PEM

PEM can be detected by use of anthropometry (body measurements). The following are the anthropometric indicators commonly used in describing PEM: Stunting, wasting, underweight, small body mass index (BMI) and small mid-upper arm circumference (MUAC).

- **Stunting** – is low height for age. It reflects failure to receive adequate nutrition over a long period of time and is also affected by recurrent and chronic illness.
- **Wasting** – is low weight for height. It reflects a rapid decline of weight while height has remained unchanged. Therefore wasting is acute malnutrition – a result of inadequate food intake or a recent episode of illness causing loss of weight and onset of malnutrition.
- **Underweight** – is low weight for age. This is a composite indicator which takes into account both chronic and acute malnutrition. That is, underweight is caused by either chronic malnutrition (e.g. long period of illness or not having enough to eat) or acute malnutrition (due to diarrhoea, infection etc).
- **Low birth weight** – is a reflection of intrauterine growth retardation. The WHO defines low birth weight as less than 2.5 kg. Causes include inadequate maternal food intake during pregnancy, short maternal stature and infection such as malaria. Cigarette smoking on the part of the mother also is associated with low birth weight.

Table 1: Anthropometric features of PEM

Malnutrition condition	Z – score (SD from median of the reference value)	Diagnosis
Stunting, Wasting or Underweight	Below -3 SD	Severe
	-3 SD to below – 2 SD	Moderate
	Below – 2 SD	Total malnutrition

- **Low BMI:** BMI relates weight to the body's surface area and is derived as follows: **weight (in kg) ÷ height² (in meters)**. BMI thus provides a measure of the body mass, ranging from thinness to obesity. Categorization of BMI is as follows:

Table 2:

BMI (kg/m²)	Diagnosis
Below 16.0	Severe under-nutrition (<i>thinness grade 3</i>)
16.0 – 16.9	Moderate under-nutrition (<i>thinness grade 2</i>)
17.0 – 18.4	Mild under-nutrition (<i>thinness grade 1</i>)
18.5 – 24.9	Good nutritional status
25.0 – 29.9	Overweight (<i>overweight grade 1</i>)
30.0 – 39.9	Obesity (<i>overweight grade 2</i>)
40 or above	Severe obesity (<i>overweight grade 3</i>)

Small MUAC: MUAC is the circumference of the left upper arm, measured at the mid-point between the tip of the shoulder (acromium) and the tip of the elbow olecranon process). MUAC is measured in cm; cut-off points are different for different population groups, as follows:

Table 3:

Population group	Severe under-nutrition	Moderate under-nutrition	Total under-nutrition
Children below 5 years	Below 11.5 cm	11.5 to 12.4 cm	Below 12.5 cm
Children 5 to 9 years	Below 13.5 cm	13.5 to 14.4 cm	Below 14.5 cm
Children 10 to 14 years	Below 16.0 cm	16.0 to 18.4 cm	Below 18.5 cm
Adolescents 15+ years, non-pregnant women, non-lactating women, adult men.	Below 19.0 cm	19.0 to 21.9 cm	Below 22.0 cm
Pregnant women, lactating women from 0 to 6 months	Below 19.0 cm	19.0 to 22.9 cm	Below 23.0 cm

1.3 Management of PEM (under-nutrition)

Management of PEM varies with the form of malnutrition, severity of the condition and presence or absence of medical complications. Most common medical complications in severely malnourished children include generalized oedema, hypothermia, hypoglycaemia, dehydration, anaemia, septicemia/infections and cardiac failure. Management focuses on appropriate feeding practices, nutritional supplements and treatment of any accompanying medical complications, as follows:

- Acute malnutrition (wasting as well as underweight):
 - Severe acute malnutrition (SAM): If no medical complications, the patient should be managed at home using ready to use therapeutic food (RUTF).
 - SAM accompanied by medical complications, the patient to be admitted for in-patient care. Treat complications eg dehydration, shock, anemia, infections, hypothermia, hypoglycemia and electrolyte imbalance. Give F75, F100 and ReSomal, managed according to the standard guidelines - National Guidelines for Management of Acute Malnutrition (issued by the MOHSW).
 - Patients aged five years or above, pregnant and lactating women – to be managed at home using RUTF, also according to the existing standard guidelines.
- Chronic malnutrition (Stunting): nutrition counseling emphasizing on adequate balanced diet and increased frequency of feeding. Accompanying diseases to be managed at health facility.

1.4 Overweight/Obesity

This is an increase of body weight as a result of excessive accumulation of fat in the body. In some cases obesity occurs secondary to other disorders or conditions such as hypothyroidism, Cushing's disease and others. Obesity may also occur due to prolonged use of medicines such as corticosteroids. Body fat can range from 2 to 70 percent of the body weight. In this regard men with over 24 percent body fat and women with over 35 percent body fat are considered obese. Desirable amounts are 8 to 24 percent body fat for men and 21 to 35 percent for women. NB: Women need more body fat because some sex-specific fat is associated with reproductive functions. This fat is normal and is factored into the above calculations. Obesity is associated with increased incidences of cardiovascular disease, hypertension, type II diabetes; some types of cancer, certain bone and joint disorders and some digestive disorders.

Anthropometric features of overweight/obesity

For children under five years of age:

- Z score - +2SD - < +3SD = overweight**
- +3SD and above = Obesity**

Dietary and lifestyle measures

Nutrition counseling focusing on:

- Diet modification (less carbohydrates and fats, more fruits and vegetables).
- Less alcohol consumption
- More active life to increase energy expenditure (physical work, physical activities, exercises such as sports and gym)

2.0 ANAEMIA

Anaemia is a pathological condition arising as a result of low level of haemoglobin in the body. Reduction of haemoglobin impairs oxygen transport to the tissues – the basis of the clinical features of anaemia. Anaemia can be classified according to cause and mechanism of development. Four major groups are distinguished:

- ***Haemorrhagic anaemia*** develops due to various forms of bleeding (trauma, excessive menses, bleeding associated with pregnancy and birth giving, and parasitic infestations such as hookworms and scistosomiasis).
- ***Haemolytic anaemia*** – due to massive destruction of red blood cells as occurs in malaria and sickle cell disease.
- ***Hypoplastic/Aplastic anaemia*** – due to failure of bone marrow to produce sufficient red blood cells. Bone marrow depression can be caused by diseases (autoimmune, viral infection), radiation and chemotherapy and intake of some drugs (anti-inflammatory, antibiotics).
- ***Nutritional anaemia*** – due to deficiency of the nutrients needed for the synthesis of red blood cells: iron, folic acid and vitamin B₁₂. Nutritional anaemias are
 - *Iron deficiency anaemia*
 - *Folic acid deficiency anaemia*
 - *Vitamin B₁₂ deficiency anaemia*

Anaemia affects all population groups but children aged below five years and pregnant women are the most vulnerable. Detection of anaemia is by determining the concentration Hb and the cut-off points at sea level are as follows:

Table 4:

Population group	Hb levels indicating anaemia (g/dl)
Children 6 to 59 months	Below 11.0
Children 5 to 11 years	Below 11.5
Children 12 to 14 years	Below 12.0
Adult men (15+ years or above)	Below 13.0

Adult women (15+ years or above, non-pregnant)	Below 12.0
Pregnant women (regardless of age)	Below 11.0

Severity of anaemia:

- Hb 11.0 – 10.0 g/dl to the cut-off point = mild anaemia
- Hb ≤ 10.0 – 7.0 g/dl = moderate anaemia
- Hb < 7.0 – 4.0 g/dl = severe anaemia
- Hb < 4.0 g/dl = very severe anaemia

Prevention of anaemia:

- Consumption of iron and vitamin rich foods. Iron in foods of animal origin (haem iron) is more easily absorbed compared with iron in foods of plant origin (which is mostly non-haem iron). Vitamin C enhances absorption of iron while tea and coffee inhibits iron absorption.
- Prevention and treatment of anaemia related diseases (malaria, worm infestation, other infections)
- Iron and folic acid supplementation to the most at risk groups – children, pregnant women, sickle cell patients (See National Guidelines for Micronutrient Supplementation)
- Use of micronutrients fortified foods (iron and folic acid included).

Iron Deficiency

The main function of iron is transport of oxygen at various sites in the body. Thus iron is a component of haemoglobin and myoglobin (protein molecule in the muscle which carries oxygen for muscle metabolism). Iron is a component of cytochromes (involved in cell respiration); component of xanthine oxidase (involved in catabolism of purines which make nucleic acids). Iron is a component of aconitase (involved in the Krebb's Cycle) and many other enzymes such as peroxidase and catalase.

While Hb concentration is used to define anaemia, it does not define the body's iron status. Three stages are distinguished in the reduction of the body's iron status:

1. Depletion of iron stores: the body's storage pool (deposits in the liver, spleen and bone marrow) diminishes due to insufficient dietary intake. This has no effect on the Hb yet.
2. Iron deficiency erythropoiesis: storage levels substantially reduced, inadequate iron is available in the bone marrow for the synthesis of Hb. Still, no overt effect on the Hb level.
3. Iron deficiency anaemia: last and most severe stage of iron deficiency – iron stores are insufficient to maintain Hb synthesis. Hb level decreases leading to anaemia.

Signs and symptoms of deficiency

- Pallor
- Glossitis
- Fatigue
- Dizziness
- Decreased mental alertness
- Anaemia (microcytic)

Dietary measures

- Rich sources of iron include meat (especially liver), poultry, fish, and seafood. These contain heme iron, which is easily absorbed in the gut.
- Others are fruits, vegetables, eggs, milk and dairy products, which contain non-heme iron. Absorption can be enhanced by vitamin C (taking meal with fruit).
- Use of foods fortified with iron

NB: Certain substances (phytates in cereals and vegetables, tannins in tea and coffee) decrease iron absorption. Also when food is boiled in water iron is leached and is lost if the water is discarded.

Treatment of anaemia

Refer to the management of anemia (Haematology) section

3.0 IODINE DEFICIENCY DISORDERS (IDD)

Iodine is an essential component of the thyroid hormones – Triiodothyronine (T₃) and Tetraiodothyronine (T₄ or Thyroxine). The hormones have profound influence on energy metabolism, protein synthesis, growth and development. They also play part in the conversion of carotene to Vitamin A and synthesis of cholesterol. Insufficient level of iodine leads to inadequate production of the hormones. This, in turn, affects brain development, physical growth and functioning of muscles, heart, liver and kidneys. Goitre is an enlarged thyroid gland – a result of thyroid over-activity as it strives to capture sufficient iodine from the blood. Deficiency of iodine results in pathological conditions referred collectively as iodine deficiency disorders (IDD).

Manifestation of iodine deficiency:

Iodine deficiency disorders (IDD) include the following:

- *Goitre*: Enlarged thyroid gland from over-activity
- *Hypothyroidism*: Dry skin, weight gain, puffy face, frequent constipation and lethargy – from under-active thyroid
- *Hyperthyroidism*: Exophthalmia, rapid pulse and weight loss – from over-active thyroid
- *Cretinism*: Child born to a mother who was iodine deficient during pregnancy. Has mental retardation, retarded growth and neurological problems (spasticity).

Dietary measures

The iodine content of the individual foods varies considerably according to the type of soil, fertilizer, animal feed and processing methods used.

- Natural food sources of iodine include:
 - Drinking water (reflecting amount of I_2 present in the soil)
 - Fish,
 - Sea weeds (Sea weeds are rich in iodine but are a rare component of the diet).
 - Iodized salt (table salt fortified with iodine compound) is the strategy for control of iodine deficiency worldwide. Potassium iodate (KIO_3) or potassium iodide (KI) is added to edible salt. The recommended iodization level in Tanzania is 40 to 70 parts per million (ppm).

Drug treatment

- Injectable iodized oil: given as intra-muscular injection. The iodine is retained in the body tissues for a long period of time (three to five years), maintaining the thyroid hormones at normal levels
- Iodinated oil capsules: 400 mg iodine administered orally, repeated after one to two years
- Lugol's solution: 3 drop (21 mg) once a month, up to one year

4.0 VITAMIN A DEFICIENCY (VAD)

Vitamin A is a fat-soluble vitamin. It plays important roles in the body, including role in vision, maintenance of epithelial tissue, synthesis of mucous secretion, growth, reproduction and immunity.

Causes of VAD include the following:

- Low consumption of vitamin A rich foods (yellow fruits, green and pigmented vegetables, red palm oil, foods of animal origin)
- Dietary deficiency due to poor food processing, preservation and preparation.
- Limited consumption of fats and oils (for example, non-use of cooking oil) leading to poor absorption of vitamin A from food.
- Poor breastfeeding (non-use of colostrum, insufficient breastfeeding).
- Diseases that deplete vitamin A from the body (measles, ARI).
- Diseases affecting food absorption (chronic diarrhea, intestinal parasites).

Manifestation of VAD

- Xerophthalmia (the eye manifestations of VAD)
 - Night blindness
 - Xerosis (corneal, conjunctival)

- Bitot's spots.
- Corneal ulcer
- Xerophthalmic fundus.
- Keratomalacia (often leading to blindness).
- Slowed growth and development
- Reduced reproductive health
- Increased risk of anaemia
- Follicular hyperkeratosis.

Prevention of VAD

- Increase consumption of horticultural foods (fruits, vegetables)
- Consumption of red palm oil
- Improve child feeding practices (breastfeeding, complementary feeding)
- Use of cooking oil
- Early and proper treatment of diseases (measles, ARI, diarrhea, worms).
- Vitamin A supplementation (see National Guidelines for Micronutrient Supplementation)
- Use of food fortified with vitamin A.

Treatment of VAD (disease targeted supplementation)

Different treatment regimens are prescribed for patients presenting with different conditions, as follows:

- Children presenting with xerophthalmia, measles or persistent diarrhea or severe acute malnutrition.
- Children presenting with moderate acute malnutrition, acute diarrhea or lower respiratory tract infections.
- Pregnant women presenting with xerophthalmia
(*See National Guidelines for Micronutrients Supplementation*)

5.0 DEFICIENCY OF VITAMIN B₁ (THIAMINE)

Vitamin B₁ is utilized in carbohydrate, fats and protein metabolism for production of energy. It, contributes to body's supply of niacin (another B vitamin) by facilitating in the conversion of tryptophan (an amino acid) to niacin. Promotes appetite and supports the functioning of the central nervous system. Thus deficiency leads to shortage of energy and lesions in nervous tissues. Deficiency is commonly caused by consumption of highly polished cereals or foods containing thiaminase (anti-thiamine factor). Alcoholics are also prone to deficiency of thiamine.

Signs and symptoms of deficiency

- Characterized by enlargement of nerves, weight loss (due to loss of appetite), oedema and disturbance in heart function
- Lack of energy
- Lesions in nervous tissues.

Dietary measures

- Whole grain cereals and pulses
- Green vegetables (such as green peas)
- Fruits
- Fish, meat, milk, oil seed, yeast

Drug treatment

- Mild chronic thiamine deficiency & for those with malabsorption:
C: Vitamin B₁ 5 – 25 mg i/m every 12 hours for 3 days then orally for 1 month
- For severe deficiency:
C: Vitamin B₁ 200 – 300 mg daily for 3 days

6.0 VITAMIN B₂ (RIBOFLAVIN) DEFICIENCY

Vitamin B₂ is utilized in the metabolism of carbohydrates, fats and proteins for production of energy. Also it plays part in synthesis of corticosteroids and production of red blood cells. Deficiency occurs in populations consuming highly polished cereals.

Signs and symptoms of deficiency

- It characterized by sore throat, pharyngeal and oral mucous membrane hyperaemia, angular stomatitis, cheilosis, glossitis and anemia
- Riboflavin deficiency almost invariably occurs in combination with other vitamin deficiencies.

Dietary measures

- Animal products (milk, meat liver, fish, eggs, cheese)
- Vegetable products (green leafy vegetables)
- Cereal grains and pulses

Drug treatment

C: Vitamin B-complex 1 tablet 8 hourly for 1 month.

7.0 VITAMIN B₃ (NIACIN) DEFICIENCY

Niacin is utilized in carbohydrate, fat and protein metabolism for production of energy. In Tanzania deficiency occurs in communities whose main staple food is maize or sorghum and particularly during rainy season when food diversification is at its lowest. Deficiency leads to Pellagra.

Signs and symptoms of deficiency

It is a disease characterized by a triad, referred to as three Ds:

- Dermatitis (darkened scaly skin on the parts exposed to the sun)
- Diarrhea
- Dementia (memory loss)
- Some patients may present also with glossitis

Dietary measures

- Animal products (especially liver), pork, poultry
- Groundnuts, beans, peas, other pulses, yeast
- Cereal grains (but not maize or sorghum)

Note

- Treatment of maize with alkalis such as limewater makes the niacin much more available
- Protein is good source as the amino acid tryptophan can be converted to niacin in the gut.

Drug treatment

C: Nicotinamide: Adult gives 100 mg every 6 hours for 7 days followed by multivitamin preparation containing 50 to 60 mg of nicotinamide daily for 1 month.

Children: 10 to 25 mg every 8 hours for 7 days, followed by multivitamin preparation as above.

8.0 VITAMIN B₆ (PYRIDOXINE) DEFICIENCY

Pyridoxine is involved in synthesis and breakdown of amino acids (hence important in protein metabolism), in the conversion of glycogen in the liver and muscle tissue to glucose (hence maintenance of blood glucose levels), and in reaction that produces a heme precursor, necessary for formation of haemoglobin. Pyridoxine also aids in the conversion of amino acid tryptophan to niacin. Disease or clinical features associated specifically with pyridoxine are rare. However, various medical conditions and drugs affect vitamin pyridoxine metabolism, for example, deficiency of the vitamin occurs in patients who are on chloramphenicol and TB patients who are on isoniazid

Signs and symptoms of deficiency

- Dermatitis, glossitis, cheilosis
- Macrocytic anaemia
- Convulsions

Dietary measures

- Animal sources – meat, liver, pork, fish, milk.
- Vegetables – spinach, turnips, broccoli
- Fruits – bananas, oranges, water melon
- Yeast

Drug treatment

C: Pyridoxine 50 mg every 8 hours until recovery

- In case deficiency is isoniazid induced, it should be replaced with ethambutol.

9.0 VITAMIN B₁₂ (COBALAMIN) DEFICIENCY

Vitamin B₁₂ is involved in the synthesis of the thymine nucleotides of DNA (along with folic acid) and therefore in the synthesis of red blood cells. It plays part in the metabolism of fatty acids, hence in the formation of myelin (the sheathing around the axons of nerve cells). The vitamin is involved also in the carbohydrate metabolism (stabilizes glutathione – a component of enzymes needed in carbohydrate metabolism).

Signs and symptoms of deficiency

- Macrocytic megaloblastic anaemia
- Decreased white blood cells
- Angular stomatitis, glossitis
- Delusions, nerve problems, unsteady gait.

Dietary measures

Main source is animal foods – meat, liver, seafood, eggs, milk, and cheese.

Note

- Animals or plants do not synthesize the vitamin – it is synthesized by bacteria in animals.
- Humans can not obtain the vitamin by action of bacteria in the gut because it can not be absorbed very far down the intestine
- Some plants (legumes that contain nodule bacteria) can synthesize the vitamin

Drug treatment

Adult

C: Cyanocobalamin 50 to 150µg (O) daily, taken between meals.

Children give orally 5- to 105µg in 1-3 divided doses.

Intramuscular injection: Initially 1mg, repeated 10 times at intervals of 2 – 3 days. Maintenance dose: 1 mg every month.

In malabsorption patients use injectable Vitamin B complex 0.25ml– 2.0ml IM

10.0 FOLIC ACID DEFICIENCY

Folic acid is involved in the metabolism of amino acid (conversion of histidine to glutamic acid). It is also involved in the synthesis of thymine (a distinctive component of DNA) and therefore in the formation of red blood cells and maintenance of nervous system.

Signs and symptoms of deficiency

- Macrocytic megaloblastic anaemia
- Stomatitis, glossitis
- Diarrhea
- Neural tube defects (spina bifida, anencephaly, encephalocele)

Dietary measures

- Green leafy vegetables
- Legumes
- Liver, meat, fish, poultry

Drug treatment

Adults and children over one year

A: Folic acid 5 mg (O) daily for 4 months, then maintenance dose of 5 mg every 1-7 days depending on underlying disease.

Children up to one year: 0.5 mg/kg body weight daily

11.0 VITAMIN C (ASCORBIC ACID) DEFICIENCY

Vitamin C helps the body use calcium and other nutrients to build bones and the walls of blood vessels, helps form collagen which is important for connective tissues, increases absorption of iron from foods, increases resistance to infection, enhances protein metabolism, is an antioxidant.

Signs and symptoms of deficiency

- Scurvy (bleeding gums, dry skin, dry mouth, impaired wound healing).
- Gingivitis (bleeding sore and inflamed gums)
- Stomatitis (sores on corners of the mouth)
- Anaemia (of iron deficiency)

Prevention (dietary measures)

- Fruits: citrus fruits, berries, pawpaw, mangoes, melons, guavas, bananas.
- Vegetables: green vegetables, tomatoes, potatoes (with skin), sprouted cereals, pulses.

Note: Substantial vitamin C can be lost during food processing, preservation and preparation.

Drug treatment

- Therapeutic:
 - A:** Ascorbic acid tablets 250 mg daily, in divided dose, until recovery
- Prophylactic:
 - A:** Ascorbic acid tablets 25 – 75 mg daily
- In malabsorption patients injectables Ascorbic acid IV/IM 500mg

12.0 VITAMIN D DEFICIENCY

Vitamin D facilitates calcium and phosphorus absorption and utilization, hence formation of bones and teeth.

Signs and symptoms of deficiency

- Rickets – a disease of bones in infants and children
- Osteomalacia in adults

Prevention

- Exposure of the skin to sunshine (vitamin D is produced by the action of the sun on the skin)
- Vitamin D rich foods: wheat germ, fish, liver, egg yolk, organ meats, cheese, milk (breast milk other milks), butter, margarine, mayonnaise.

Drug treatment

C:Ergocalciferol 1000 – 5000 iu/daily (PO) for 2 weeks then 4000 iu/daily for 2 months

13.0 VITAMIN E (TOCOPHEROL) DEFICIENCY

Vitamin E is an antioxidant. It plays role in reproductive health (enhances fertility) and also in haemoglobin synthesis.

Signs and symptoms of deficiency

- Leg cramps,
- Muscle weakness,
- Nerve problems and
- Hearing problems.

Dietary measures

- Consumption of vegetable oils
- Whole grain cereals

Drug treatment

Adult

C: Alpha tocopherol acetate 50 - 100mg daily until recovery

Below 1 yr: 50mg until recovery

14.0 VITAMIN K DEFICIENCY

Vitamin K is essential for the synthesis of prothrombin in the liver, factor VII, IX and X. It also helps in the production of proteins necessary for bone calcification. Primary deficiency of vitamin K occurs only in neonates. Secondary deficiency may be associated with malabsorption syndrome, liver cirrhosis and the use of Coumarin derivatives such as dicumarol, warfarin and other analogues.

Signs and symptoms of deficiency

- Injuries/wounds taking long to stop bleeding.
- Infants are relatively deficient in vitamin K and therefore at risk of serious bleeds including intracranial bleeding.

Dietary measures

Vitamin K exists in two forms (K₁ and K₂) and is obtained in foods of plant and animal origins:

- Vitamin K₁ (phylloquinone), synthesised by plants
- Vitamin K₂ (menaquinone), synthesized by bacteria in animal intestine

Drug treatment

- Adults:
 - D:** Phytomenadione 10 mg i/v stat
 - To prevent vitamin K deficiency bleeding (haemorrhagic disease of the newborn):
 - D:** Phytomenadione 0.5-1 mg i/m once, at birth
- OR**
- D:** Phytomenadione 2 mg, two doses given in the first week. Third dose given at 1 month.

Not use in patients with suspected Warfarin overdose and neonates

15.0 ZINC DEFICIENCY

Zinc is known to be essential nutrient for the body. It is a component of insulin and many enzymes, including:

- Carbonic anhydrase (which transports CO₂ from RBCs to the lungs).
- Carboxypeptidase (necessary for peptide digestion)
- Alcohol dehydrogenase

It plays role in the synthesis of nucleic acids and protein, metabolism of vitamin A from the liver and wound healing (synthesis of collagen) and enhancement of absorption of folic acid

Zinc occurs in all tissues, higher concentrations being in:

- The choroid membrane of the eye.
- Male reproductive organs (especially the prostate gland).
- In the red blood cells.
- In the pancreas (as component of insulin).
- Relatively lower concentrations in the liver, skeletal muscle, bone, skin and hair.

Signs and symptoms of deficiency

- Slow growth
- Loss of smell and taste
- Loss of appetite
- Diarrhoea
- Poor wound healing
- Skin lesions

Dietary measures

Zinc is present in most foods of animal and plant origins.

- The richest sources tend to be protein rich foods e.g. meat, seafood, eggs yolk and oysters.

- Cereal grains and legumes also contain zinc (but milling reduces the zinc content. Also phytates found in whole grain products and vegetables reduces the bioavailability of zinc.
- Fruits, vegetables and egg white are poor sources of zinc.

Treatment

A: Zinc tablets 50mg 2 to 3 times daily until recovery

Zinc supplementation- Refer to National Guideline Micronutrient supplementation

16.0 SELENIUM DEFICIENCY

Selenium functions as a component of glutathione peroxidase – a powerful antioxidant. Kwashiorkor children have shown improved weight gain with selenium supplementation. In China selenium deficiency has led to “Kesharis disease” – a serious condition affecting heart muscle.

Signs and symptoms of deficiency

- Muscle weakness
- Pancreatitis (blockage of the pancreatic ducts)
- Impaired growth
- Impaired hearing
- Impaired immune system
- Faster HIV infection progression and reduced survival

Dietary sources

- Selenium is found in most body tissues, highest concentrations being in the kidney, liver, spleen, pancreas and testes
- Selenium content of food varies with their protein content. Meats, seafoods, egg yolk and milk are good sources of selenium
- In cereals, selenium content depends on the concentration of the mineral in the soil
- Mushrooms and asparagus are rich sources. But boiling these vegetables causes the mineral to be leached

Drug treatment

A: Selenium IM/IV / oral 100 -500 microgram daily until recovery

17.0 CALCIUM DEFICIENCY

Calcium strengthens bones and teeth, facilitates normal functioning of the heart and helps blood clotting. Calcium also helps in the maintenance of normal blood pressure.

Signs and symptoms of deficiency

- Delayed blood clotting
- Osteoporosis (weak breakable bones)
- Osteomalacia
- Teeth problems
- Low resistance to infection
- Stunting

Dietary measures

- Foods of animal origin: milk, yoghurt, cheese
- Fish with bones that are eaten (*dagaa*)
- Vegetables: green leafy vegetables such as broccoli
- Legumes, peas.

Drug treatment:

Adults

D: Calcium gluconate 10% I.V (94.7 mg elemental calcium) at a rate of not exceeding 5ml/minute. 10ml

Pediatric dose: Calcium gluconate 10% I.V (47.5 mg elemental calcium) at a rate of not exceeding 5ml/minute

OR

C: Calcium gluconate 500mg daily until recovery

18.0 COPPER DEFICIENCY

All body tissues contain some copper. But highest concentrations are in the liver, brain, heart, kidneys and in the blood. Copper in the form of ceruloplasmin (a copper-protein complex in the blood plasma) is involved in various stages of iron nutrition. Copper enhances iron absorption and stimulates mobilization of iron from stores (in the liver and other tissues). Plays part in the conversion of ferrous iron to ferric (important during various stages of iron metabolism). Copper-containing enzymes play part in carbohydrate and fatty acid metabolism. Copper deficiency has been linked to anaemia in premature infants and in people with severe protein-energy malnutrition. Menke's disease (a rare congenital condition) is caused by failure of copper absorption.

Signs and symptoms of deficiency

- Mental deterioration
- Hypothermia
- Hair depigmentation
- Microcytic anaemia (indistinguishable from iron deficiency anaemia) affecting infants and people with severe PEM.

Dietary measures

- Foods richest in copper are nuts, shellfish, liver, kidney, raisins and legumes. Milk is a poor source of copper.
- Milling, grinding and cooking in water tend to reduce copper content.

- Copper content of foods is also influenced by environmental factors such as:
 - Copper content in the soil
 - Geographical location, for example, close to a copper industry.
 - Kind of fertilizer used.
 - Water equipment made of copper.

19.0 MAGNESIUM DEFICIENCY

In the body magnesium is found in the bone, muscle, in the soft tissues and in blood. Many of the physiological functions of Mg are based on the mineral's ability to interact with calcium, phosphate and carbonate salts. Magnesium catalyses many essential enzymatic reactions (glucose, fatty acid, amino acid metabolism), takes part in bone metabolism and protein synthesis. Mg is important in nervous activity and muscle contraction. *NB: Under certain circumstances (e.g. diarrhea and severe PEM etc.) excessive body losses of Mg may occur. This leads to weakness and mental changes and, occasionally, to convulsions.*

Signs and symptoms of deficiency

- Muscle spasms, cramps
- Tremors, seizures, coma

Dietary measures

- Most foods contain adequate amounts of magnesium
- Animal foods: good source is dairy products, meats and poultry
- Vegetables: green vegetables (okra, broccoli), cucumber skin
- Fruits: especially avocado
- Cereals (whole grain)
- Legumes
- Seafood

Drug treatment

D: Magnesium sulphate 0.5 to 1 mmol/kg I.V/I.M up to 160 mmol per day for 5 days.

Maintenance: oral dose 24 mmol per day in divided doses

21.0 FLUORINE DEFICIENCY

Fluorine is a mineral that plays a protective role to bone and dental tissues: It protects against dental caries (makes them resistant to weak organic acids formed from foods that get stuck between teeth). It prevents bones from developing osteoporosis. Fluorine enhances iron absorption (protects against anaemia) and enhances wound healing.

NB: High concentration of fluorides in water (above 6 ppm) causes mottling of teeth (dark brown stain). Chronic ingestion of high concentrations (from natural high content in the area or environmental pollution) can lead to bone and tooth malformations.

Signs and symptoms of deficiency

- Dental caries.
- (Mottling of teeth and skeletal malformations are a result of excessive fluoride).

Dietary measures

Most animal and plant foods contain amounts that reflect the content in the soil.

- Fish and seaweed are rich sources.
- Other rich sources include bone meal, meats and dairy products
- Grains, vegetables and nuts.

Drug treatment:

In areas where drinking water is fluoridated and the fluoride content is above 0.7 parts per million. Supplementation is not recommended.

S: Fluorine tabs: Under 6 yrs 250 micrograms daily
Over 6 years : 500 micrograms to 1mg daily

22.0 MICRONUTRIENT DEFICIENCIES

Micronutrient deficiencies are a major health problem in Tanzania. Deficiencies occur across all population groups but women and children are highly vulnerable because of rapid growth and inadequate dietary practices. These include deficiencies of vitamin A, iron, iodine and zinc.

Interventions to address micronutrient deficiencies include food based approaches whereby production and consumption of micronutrients rich foods are promoted. Micronutrient supplementation programs target most vulnerable groups such as pregnant and lactating women, and children aged below 5 years.

Food fortification with micronutrients is another approach aimed to deliver micronutrients to the general population, most vulnerable groups included. Food fortification includes iodization of edible salt and fortification of staple foods such as cereal flours and cooking oil. Other interventions target children aged 6 to 23 months with a single dose of packets containing multiple vitamins and minerals in powder form that can be sprinkled onto any semi solid complementary food at the point of use.

Dietary measures

Promote production and consumption of fortified foods

Intervention:

Food fortification has been defined as the addition of one or more essential nutrients to a food, whether or not it is normally contained in the food, for the purpose of preventing or correcting a demonstrated deficiency of one or more nutrients in the population or specific population groups (FAO/WHO 1994). Below are requirement for food fortification in Tanzania.

MINIMUM REQUIREMENT FOR FORTIFIED FOOD

Food vehicle	Nutrient	Fortificant compound	Specifications	
			Minimum	Maximum
Wheat flour	Iron	Sodium iron	30	50
		EDTA	mg/kg	mg/kg
	Zinc	Zinc oxide	30	50
			mg/kg	mg/kg
	Vitamin B ₁₂	Vitamin B ₁₂	0.0005	0.025
			mg/kg	mg/kg
	Folate	Folic acid	1	5
			mg/kg	mg/kg
Maize flour	Iron	Sodium iron	5	25
		EDTA	mg/kg	mg/kg
	Zinc	Zinc oxide	20	25
			mg/kg	mg/kg
	Vitamin B ₁₂	Vitamin B ₁₂	0.0002	0.01
			mg/kg	mg/kg
	Folate	Folic acid	0.5	2.5
			mg/kg	mg/kg
Edible fats and oils	Vitamin A	Retinyl	6	28
		Palmitate	mg/L	mg/L
	Vitamin E	Alpha	65	190
		Tocopherol	mg/L	mg/L
Edible salt	Iodine	Potassium iodide		
Complementary food	Micronutrients powder		Powder containing (Vitamin A (Dry Vitamin A Palmitate) microencapsulated 400µg of retinol (1000 IU); Thiamin (Thiamine Mononitrate) 0.5mg; Riboflavin (Fine powder) 0.5mg; Niacin (Niacinamide) 6.0mg; Folate (Folic Acid Food Grade) 0.15mg; Vitamin B6 (Pyridoxine Hydrochloride) 0.5mg Vitamin B12 (Vitamin B12 0.1% WS) 0.9µg; Vitamin C (Ascorbic Acid) 30 mg; Vitamin D (Dry Vitamin D3) Microencapsulated 50µg (200 IU) Vitamin E (Dry Vitamin E) 5.0mg; Zinc (zinc gluconate) 4.1mg; Iron (Encapsulated Ferrous Fumarate) 10mg of elemental iron (equivalent to 30mg	

			of ferrous fumarate); Copper (Copper (II) Gluconate) 0.56mg; Selenium 17.0µg; Iodine (potassium iodide) 90µg; Filler: Maltodextrin as needed.
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CHAPTER TWENTY FIVE

POISONING

1.0 Common poisonings

These can be intentional or accidental. Suspect poisoning in any unexplained illness in a previously healthy child/adult. Traditional medicines can also be a source of poisoning.

Diagnosis

This is made from relevant history elicited from patient, relatives or friends, from clinical examination, and the results of investigations, where appropriate.

- **Find out full details of the poisoning agent**, the amount ingested and the time of ingestion. Attempt to identify the exact agent involved requesting to see the container, where relevant.
- **Check for signs of burns** in or around the mouth or of stridor (laryngeal damage) suggesting ingestion of corrosives:
 - Admit all patients who have ingested iron, pesticides, paracetamol or aspirin, narcotics, antidepressant medicines;
 - Patients who have ingested corrosives or petroleum products should not be sent home without observation for 6 hours. Corrosives can cause oesophageal burns which may not be immediately apparent and petroleum products, if aspirated, can cause pulmonary oedema which may take some hours to develop.

General Principles of Management

- Observe person and patient safety
- Remove patient from source of poison
- Support vital function
 - Establish and maintain a clear airway
 - Ensure adequate ventilation and oxygenation
 - Monitor blood pressure, heart rate, temperature, respiratory rate, pupil size and responsiveness

2. 0 Principles for management of ingested poisons

- Gastric decontamination is most effective within one hour of ingestion.
- Gastric decontamination will not guarantee that all of the substance has been removed.

Contraindications to gastric lavage are:

- An unprotected airway in an unconscious patient
- Ingestion of corrosives or petroleum products e.g. kerosene

- Check the patient for emergency signs: coma, convulsions, acute confusion, hepatic and/or renal failure, skin eruption, psychiatric or neurologic disturbance of acute onset and check for hypoglycaemia.
- Identify the specific agent and remove or adsorb it as soon as possible.

Note: Treatment is most effective if given as quickly as possible after the poisoning event, ideally within 1 hour.

- If the the patient has swallowed kerosene, petrol or petrol-based products (note that most pesticides are in petrol-based solvents) or if the patient's mouth and throat have been burned (for example with bleach, toilet cleaner or battery acid) **do not vomit the patient** but give water orally.

Treatment

- **Never** use salt as an emetic as this can be fatal.
- Give activated charcoal, if available, and do not induce vomiting; give by mouth or NG tube according to table below.

Amount of activated charcoal per dose

- Children up to one year of age: 1 g/kg
- Children 1 to 12 years of age: 25 to 50 g

Adolescents and adults: 25 to 100 g

- Mix the charcoal in 8–10 times the amount of water, e.g. 5 g in 40 ml of water.
- If possible, give the whole amount at once; if the child has difficulty in tolerating it, the charcoal dose can be divided.
- If charcoal is not available, then induce vomiting but only if the patient is conscious by rubbing the back of the patient throat with a spatula or spoon handle; if this does not work, give an emetic such as ipecacuanha (10 ml for 6 months to 2 year-olds or 15 ml for over 2 years); if this does not work, then try rubbing the back of the patient's throat again.

Note: Ipecacuanha can cause repeated vomiting, drowsiness and lethargy which can confuse the diagnosis of poisoning.

Gastric lavage

- Only do it in health care facilities if staff has experience in the procedure, and if the ingestion was only a few hours ago and is life threatening, and there has been no ingestion of corrosives or petroleum derivatives
- Make sure a suction apparatus is available in case the patient vomits
- Place the patient in the left lateral/ head down position
- Insert a large NGT. Ensure the tube is in the stomach
- Perform lavage with 10 ml/kg body weight of warm normal saline (0.9%). The volume of lavage fluid returned should approximate to the amount of fluid given.

- Lavage should be continued until the recovered lavage solution is clear of particulate matter. Note that tracheal intubation may be required to reduce risk of aspiration.
- Give specific antidote if this is indicated
- Give general care.
- Keep the patient under observation for 4–24 hours depending on the poison swallowed

Referral

Consider transferring patient to next level referral hospital, where appropriate and where this can be done safely, if the patient is unconscious or has deteriorating conscious level, has burns to mouth and throat, is in severe respiratory distress, is cyanosed or is in heart failure.

3.0 PRINCIPLES FOR MANAGEMENT OF POISONS IN CONTACT WITH SKIN OR EYES

Skin contamination

- Remove all clothing and personal effects and thoroughly flush all exposed areas with copious amounts of tepid water.
- Use soap and water for oily substances.
- Attending staff should take care to protect themselves from secondary contamination by wearing gloves and apron.
- Removed clothing and personal effects should be stored safely in a see-through plastic bag that can be sealed, for later cleansing or disposal.

Eye contamination

- Rinse the eye for 10–15 minutes with clean running water or saline, taking care that the run-off does not enter the other eye.
- The use of anaesthetic eye drops will assist irrigation.
- Evert the eyelids and ensure that all surfaces are rinsed.
- In the case of an acid or alkali irrigate for 15–20 minutes
- Where possible, the eye should be thoroughly examined under fluorescein staining for signs of corneal damage. If there is significant conjunctival or corneal damage, the patient should be seen urgently by an ophthalmologist.

4.0 PRINCIPLES FOR MANAGEMENT OF INHALED POISONS

- Remove from the source of exposure.
- Administer supplemental oxygen if required.

Inhalation of irritant gases may cause swelling and upper airway obstruction, bronchospasm and delayed pneumonitis. Intubation, bronchodilators and ventilatory support may be required.

5.0 SPECIFIC POISONS

5.1 Management for corrosive compounds poisoning

Examples—sodium hydroxide, potassium hydroxide, acids, bleaches or disinfectants

- **Do not** induce vomiting or use activated charcoal
- Give milk or water as soon as possible.
- Then give the patient nothing by mouth and arrange for surgical review to check for oesophageal damage/rupture, if severe.

5.2 Management for petroleum compounds poisoning

Examples—kerosene, turpentine substitutes and petrol

- Do not induce vomiting as inhalation can cause respiratory distress with hypoxaemia due to pulmonary oedema and lipoid pneumonia. Ingestion can cause encephalopathy. Supportive treatment includes oxygen therapy if respiratory distress present

5.3 Management for Organo-phosphorus and carbamate compounds poisoning

Examples: organophosphorus – Malathion, Parathion, TEPP, mevinphos and carbamates – methiocarb and carbaryl.

These can be absorbed through the skin, ingested or inhaled. The patient may complain of vomiting, diarrhoea, blurred vision or weakness. Signs are those of excess parasympathetic activation: salivation, sweating, lacrimation, slow pulse, small pupils, convulsions, muscle weakness/twitching, then paralysis and loss of bladder control, pulmonary oedema, and respiratory depression.

Treatment

- Remove poison by irrigating eye or washing skin (if in eye or on skin).
- Give activated charcoal if ingested and within 1 hour of the ingestion.
- Do not induce vomiting because most pesticides are in petrol-based solvents.
- In a serious ingestion where activated charcoal cannot be given, consider careful aspiration of stomach contents by NG tube (the airway should be protected).
- If the has signs of excess parasympathetic activation (see above) give

B: Atropine 15–50 micrograms/kg IM or IV over 15 minutes. Repeat every 10- 15 minutes until no chest signs of secretions, and pulse and respiratory rate returns to normal.

Auscultate the chest for signs of respiratory secretions and monitor respiratory rate, heart rate and coma score (if appropriate).

- Consider obidoxime (a cholinesterase activator) 3-5mg/kg IV if <24 hours. It may be given 5minutes after the first dose of atropine.
- Check for hypoxaemia with pulse oximetry. Give oxygen if oxygen saturation is less than 90%.
- If muscle weakness give

S: Pralidoxime (cholinesterase reactivator) 25–50mg/kg diluted with 15 ml water for injection by IV infusion over 30 minutes repeated once to twice, followed by 10 to 20 mg/kg/hour, as necessary.

5.4 Management of Paracetamol poisoning

- If within 1 hour of ingestion of 150mg/kg or more paracetamol give activated charcoal, if available, or induce vomiting.

For conscious and no vomiting give

C: Methionine (<6 years: 1 gram every 4 hours - 4 doses; 6 years and above: 2.5 grams every 4 hours for 4 doses).

- If more than 8 hours after ingestion, or the patient cannot take oral treatment, give

C: Acetylcysteine 150mg/kg IV in 200mls 5% dextrose over 20 minutes, then 50mg/kg in 500mls 5% dextrose over 4 hours, then 100mg/kg in 1 liter of 5% dextrose over 16 hours.

In severe poisoning a further 100mg/kg may be given over the next 24 hours

For children <20 kg give the loading dose of 150 mg/kg in 3 ml/kg of 5% glucose over 15 minutes, followed by 50 mg/kg in 7 ml/kg of 5% glucose over 4 hours, then 100 mg/kg IV in 14 ml/kg of 5% glucose over 16 hours.

Monitor electrolyte especially potassium.

5.5 Management of Aspirin and other salicylates poisoning

The patient can rapidly become acidotic and are consequently more likely to suffer the severe CNS effects of toxicity.

Salicylate overdose can be complex to manage. These cause acidotic-like breathing, vomiting and tinnitus.

- Give activated charcoal within one hour of ingestion if available. If charcoal is not available and a severely toxic dose has been given, then perform gastric lavage or induce vomiting as above
- If available check the blood gases, pH, bicarbonates and serum electrolyte.

- Replace fluid losses (Plasma potassium concentration should be corrected before giving sodium bicarbonate as hypokalaemia may complicate alkalinisation of urine)
- Give IV sodium bicarbonate 1 mmol/kg over 4 hours to correct acidosis and to raise the pH of the urine to above 7.5 so that salicylate excretion is increased. Monitor urine pH hourly.
- Give IV fluids at maintenance requirements
- Haemodialysis is required if the concentration exceeds 700mg/litre or in presence of severe metabolic acidosis
- Monitor blood glucose every 6 hours and correct as necessary

5.6 Management of Iron poisoning

- Check for clinical features of iron poisoning: nausea, vomiting, abdominal pain and diarrhoea. The vomitus and stools are often grey or black. In severe poisoning there may be gastrointestinal haemorrhage, hypotension, drowsiness, convulsions and metabolic acidosis.
- Gastrointestinal features usually appear in the first 6 hours and a patient who has remained asymptomatic for this time probably does not require antidote treatment.
- Activated charcoal does not bind to iron salts; therefore consider giving a gastric lavage if potentially toxic amounts of iron were taken.
- Give antidote treatment

D: Deferoxamine 50 mg/kg IM up to a maximum of 1 g by deep IM injection repeated every 12 hours; if very ill, give IV infusion 15 mg/kg/hour to a maximum of 80 mg/kg in 24 hours.

5.7 Management of Carbon monoxide poisoning

- Give 100% oxygen to accelerate removal of carbon monoxide (note patient can look pink but still be hypoxaemic) until signs of hypoxia disappear.
- Check blood gases and serum electrolyte

Prevention of Poisoning

- Keep medicines and poisons in proper containers and out of reach of children
- Advise patients/care takers on first aid if this happens again in the future
- Do not make the patient vomit if they have swallowed kerosene, petrol or petrol based products or if patient's mouth and throat have been burned, nor if the patient is drowsy.
- Try to make the patient vomit if other medicines or poisons have been taken by stimulating the back of the throat.
- Take the patient to a health facility as soon as possible, together with information about the substance concerned such as container, label, sample of tablets, berries etc.

6.0 BITES

6.1 Management of Insects Bites

Important insect bites are those from scorpions.

Symptoms: Most bites and stings result in pain, swelling, redness, and itching to the affected area.

Treatment and Management

Treatment depends on the type of reaction

- Clean the area with soap and water to remove contaminated particles left behind by some insects
- Refrain from scratching because this may cause the skin to break down and results to an infection
- Treat itching at the site of the bite with antihistamine
- Give appropriate analgesics
- Where there is an anaphylactic reaction treat according to guideline.

6.2 Management of Scorpion sting

Scorpion stings can be very painful for days. Systemic effects of venom are much more common in children than adults.

Diagnosis of Scorpion poisoning (envenoming)

Signs of envenoming can develop within minutes and are due to autonomic nervous system activation. They include:

- Shock
- High or low BP
- Fast and/or irregular pulse
- Nausea, vomiting, abdominal pain
- Breathing difficulty (due to heart failure) or respiratory failure
- Muscle twitches and spasms.
- Check for low BP or raised BP and treat if signs of heart failure

Treatment

First aid

- Transport to hospital as soon as possible.

Hospital care

Antivenom

- If signs of severe envenoming give scorpion antivenom, if available (as above for snake antivenom infusion).

Other treatment

- Treat heart failure, if present

Supportive care

- Give oral paracetamol or oral or IM Morphine according to severity. If very severe, infiltrate site with 1% lignocaine, without epinephrine.

6.3 Management of Snake bite/Poisoning

Less than 10% of 3500 snake species are poisonous and they include cobras and mambas (Elapidac), see snakes (hydrophidac) and the boomslang and vine snakes (columbidac). It is a common problem in Tanzania. Clinical condition depends on the type of snake bite and amount of poison (venom) injected. Hence envenomation (poisoning) will be neurotoxic in cobra and mambas and sea snakes and haemotoxic in vipers and boomslang.

Snake bite should be considered in any severe pain or swelling of a limb or in any unexplained illness presenting with bleeding or abnormal neurological signs. Some cobras spit venom into the eyes of victims causing pain and inflammation. Contact with snakes, scorpions and other insects result in two types of injuries: those due to direct effect of venom on victim and those due to indirect effect of poison e.g. hypersensitivity reaction to bee sting.

Diagnosis of snake poisoning (envenoming)

- General signs include shock, vomiting and headache. Examine bite for local necrosis, bleeding or tender local lymph node enlargement.
- Specific signs depend on the venom and its effects. These include:
 - Shock
 - Local swelling that may gradually extend up the bitten limb
 - Bleeding: external from gums, wounds or sores; internal especially intracranial
 - Signs of neurotoxicity: respiratory arrest or paralysis, ptosis, bulbar palsy (difficulty swallowing and talking), limb weakness
 - Signs of muscle breakdown: muscle pains and black urine
- Check haemoglobin (where possible, blood clotting should be assessed).

Treatment

First aid

- Reassure the patient;
- Splint the limb to reduce movement and absorption of venom. If the bite was likely to have come from a snake with neurotoxic venom, apply a firm bandage to the affected limb from fingers or toes to proximal of site of bite;
- Clean the site with clean water to remove any poison and remove any fangs;
- If any of the above signs, transport to hospital which has antivenom as soon as possible. If snake has already been killed, take this with patient to hospital to hospital.

Treatment

Hospital care

Treatment of shock/respiratory arrest

- Treat shock, if present.
- Paralysis of respiratory muscles can last for days and requires intubation and mechanical ventilation or manual ventilation (with a mask or endotracheal tube and bag) by relays of staff and/or relatives until respiratory function returns. Attention to careful securing of endotracheal tube is important. An alternative is to perform an elective tracheostomy.

Antivenom

- If there are systemic signs or severe local signs (swelling of more than half of the limb or severe necrosis), give antivenom, if available
- Prepare IM Epinephrine and IV Chlorpheniramine and be ready if allergic reaction occurs.
- Give monovalent antivenom if the species of snake is known.
- Give polyvalent antivenom if the species is not known. Follow the directions given on the antivenom preparation.
- Dilute the antivenom in 2–3 volumes of 0.9% saline and give intravenously over 1 hour.
- Give more slowly initially and monitor closely for anaphylaxis or other serious adverse reactions.
- If itching/urticarial rash, restlessness, fever, cough or difficult breathing develop, then stop antivenom and give Epinephrine 0.01 ml/kg of 1/1000 or 0.1 ml/kg of 1/10,000 solution subcutaneously and IM or IV/SC Chlorpheniramine 250 micrograms/kg. When the patient is stable, re-start antivenom infusion slowly.
- More antivenom should be given after 6 hours if there is recurrence of blood incoagulability or after 1–2 hr if the patient is continuing to bleed briskly or has deteriorating neurotoxic or cardiovascular signs.
- Blood transfusion should not be required if antivenom is given.
- Response of abnormal neurological signs to antivenom is more variable and depends on type of venom.
- If there is no response to antivenom infusion this should be repeated.
- Anticholinesterases can reverse neurological signs in some species of snake (see standard textbooks of medicine for further details).

Other treatment

- **Surgical opinion**

Seek surgical opinion if there is severe swelling in a limb, it is pulseless or painful or there is local necrosis. Surgical care will include:

- Excision of dead tissue from wound
- Incision of fascial membranes to relieve pressure in limb compartments, if necessary
- Skin grafting, if extensive necrosis
- Tracheostomy (or endotracheal intubation) if paralysis of muscles involved in swallowing occurs

Supportive care

- Give fluids orally or by NG tube according to daily requirements. Keep a close record of fluid intake and output.
 - Provide adequate pain relief
 - Elevate limb if swollen
 - Give antitetanus prophylaxis
 - Antibiotic treatment is not required unless there is tissue necrosis at wound site
- Monitor very closely immediately after admission, then hourly for at least 24 hours as envenoming can develop rapidly.

8.0 MANAGEMENT OF OTHER SOURCES OF POISONING (ENVENOMING)

- The same principles of treatment, as above. Give antivenom, where available, if severe local or any systemic effects.
 - In general, venomous spider bites can be painful but rarely result in systemic envenoming.
 - Antivenom is available for some species such as widow and banana spiders. Venomous fish can give very severe local pain but systemic envenoming is rare.
 - Box jellyfish stings are occasionally rapidly lifethreatening. Apply vinegar on cotton wool to denature the protein in the skin.
 - Adherent tentacles should be carefully removed. Rubbing the sting may cause further discharge of venom.
 - The dose of antivenom to jellyfish and spiders should be determined by the amount of the venom injected.
 - Higher doses are required for multiple bites, severe symptoms or delayed presentation.
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