DEDER GENERAL HOSPITAL

ADULT OUTPATIENT DEPARTMENT

STANDARD TREATMENT GUIDELINE PROTOCOL

"Adapted from National STG 2021 4th Edition"

October 2024

Deder, Eastern Ethiopia

SMT APPROVAL SHEET

TITLE	**Title:** ADULT OUTPATIENT DEPARTMENT STANDARD TREATMENT GUIDELINE PROTOCOL **Version:** 1.0			
	NAME	**POSITION**	**ROLE**	**SIGN**
AUTHORS	Abdi Tofik (BSc, MPH)	Quality Director	Team leader	
	Abdella Aliyi (BSc MW)	Quality Officer	Member	
	Mahammad Aliyi (BSc N)	Reform head	Member	
	Draft Date: July 15, 2016E.C	**Approved Date: July 20, 2016E.C**		
	Name	**Position**	**Role**	**Sign**
SMT APROVAL	Nuredin Yigezu (BSc, MPH)	CEO	Chair person	
	Dr. Derese Gosa (GP)	Medical director	Member	
	Dr Isak Abdi (G/Surgeon)	Staff Representative	Member	
	Dr. Dawit Seifu (GP)	IPD Director	Member	
	Abdi Tofik (BSc, MPH)	Quality Director	Member	
	Hamza Jamal (BSc N)	Metron	Member	
	Abrahim Tahir (BSc N)	HR Head	Secretary	
	Obsa Usma'il (BA)	Finance and procurement head	Member	
	Bellisa Usma'il (BSc Pharm)	Pharmacy head	Member	
	Alamudin Usma'il (BSc Lab)	Laboratory head	Member	
	Dine Bakar (BA)	Internal Auditor	Member	
	Redwan Sharafuddin (BSc Pharm)	Planning Head	Member	
	Nure Jamal (BA)	General service head	Member	
	Mahammad Shamshaddin (BSc)	Qondaala Naamusaa	Member	
REVIEW	**Reviewed and updated** **Review date: July 2018E.C**			

TABLE OF CONTENTS

SECTION 1:

INTRODUCTION

1.1 Background

Deder General Hospital is a secondary-level health facility located in East Hararghe Zone, Oromia Region, Ethiopia. The hospital serves a catchment population of over 1.5 Million people, including urban and rural communities. It operates with a capacity of approximately 120 inpatient beds and provides a full range of outpatient, inpatient, surgical, maternal, and diagnostic services.

The Adult Outpatient Department (OPD) is the hospital's largest service unit, functioning as the first point of contact for most adult patients. It handles a wide spectrum of medical conditions, ranging from minor acute illnesses to chronic diseases and complex cases requiring referral.

In line with the Ethiopian Ministry of Health's Health Sector Transformation Plan (HSTP II) and the Essential Health Service Package (EHSP), the OPD plays a critical role in:

- Providing first-line diagnosis and treatment for common conditions
- Identifying and referring complex cases to specialized services
- Delivering preventive and promotive health education
- Supporting continuity of care for patients with chronic illnesses

The importance of the OPD in the continuum of care means that consistency, quality, and efficiency of services have a direct impact on patient outcomes and hospital performance.

1.2 Burden of Disease in Adult OPD

1.2.1 Overview

An analysis of District Health Information System 2 (DHIS2) data for the Ethiopian Fiscal Year (EFY) 2016 identified the top ten leading causes of morbidity and mortality in the Adult OPD. These conditions account for the majority of patient visits and a significant proportion of serious outcomes.

Table 1: Top Ten Leading Causes of Morbidity and Mortality – Adult OPD (EFY 2016)

Rank	Disease	Percentage (%)
1	Dyspepsia	79.0
2	Pneumonia	64.0
3	Asthma	24.0
4	Gastroenteritis / Colitis	24.0
5	Tuberculosis (TB) Infection	12.0
6	Malignant Otitis Externa	11.0
7	Refractory Anaemia	9.0
8	Typhoid Fever	4.2
9	Streptococcal Pharyngitis	3.3
10	Urinary Tract Infection (UTI)	2.4

Source: DHIS2 – Deder General Hospital, EFY 2016

1.2.2 Epidemiological Significance

- The disease profile reflects both communicable and non-communicable conditions, indicating a double burden of disease in the catchment area.
- High prevalence of dyspepsia (79%) suggests possible links to dietary patterns, *Helicobacter pylori* infection, and overuse of non-steroidal anti-inflammatory drugs (NSAIDs).
- Pneumonia (64%) remains a leading cause of morbidity, driven by seasonal outbreaks, late presentation, and co-morbidities such as HIV.
- Asthma (24%) and gastroenteritis/colitis (24%) are common, reflecting environmental factors, sanitation challenges, and allergen exposure.
- Tuberculosis (12%) prevalence underscores ongoing transmission in the community despite national TB control programs.
- Malignant otitis externa (11%) may be associated with poor ear hygiene, uncontrolled diabetes, or resistant organisms.
- Refractory anaemia (9%) highlights the importance of nutritional deficiencies, chronic diseases, and late diagnosis.
- Typhoid fever (4.2%) reflects ongoing water and sanitation issues.
- Streptococcal pharyngitis (3.3%) and UTIs (2.4%) remain significant but preventable causes of outpatient visits.

1.2.3 Trends Over Time

Comparison with hospital records from previous years suggests:

- Gradual decline in typhoid fever prevalence due to improved water safety interventions.
- Stable but high rates of pneumonia and dyspepsia.
- Slight increase in non-communicable respiratory diseases (asthma, COPD) likely linked to environmental pollution and smoking.
- No significant decline in TB infection rates, indicating a need for sustained community-based TB detection and adherence programs.

1.2.4 Public Health Implications

The identified disease burden has direct implications for:

- **Service Planning:** Prioritizing resources for high-burden conditions.
- **Training**: Ensuring OPD staff are proficient in diagnosing and managing the top 10 diseases.
- **Medicines & Supplies:** Maintaining adequate stocks of first-line treatments for these conditions.
- **Community Interventions**: Strengthening health education, hygiene promotion, and preventive measures.
- **Policy & Strategy**: Aligning OPD activities with Ethiopia's Health Sector Transformation Plan II and AMR containment strategies.

1.3 Health System Context

1.3.1 Ethiopian Health System Overview

Ethiopia's healthcare delivery system is structured into a three-tier system:

1. **Primary Level**: Comprising health posts, health centers, and primary hospitals.
2. **Secondary Level:** General hospitals serving as referral centers for primary facilities.
3. **Tertiary Level:** Specialized and teaching hospitals providing advanced care.

Deder General Hospital functions at the secondary level, receiving referrals from nearby health centers and primary hospitals while also providing direct services to walk-in patients. As such, it bridges the gap between primary healthcare and specialized tertiary care.

1.3.2 Role of the Adult Outpatient Department in the Health System

The Adult OPD is pivotal in:

- Acting as a gatekeeper to the hospital's inpatient and specialty services.
- Managing common and moderately complex cases directly at the secondary level.
- Serving as a training site for health professionals in the region.
- Facilitating referrals to higher-level care when indicated.

1.3.3 Service Integration

The OPD works in close collaboration with:

- Laboratory & Imaging Departments for diagnostics
- Pharmacy Department for medicines dispensing and stock management
- Emergency Department for urgent cases
- Specialty Clinics (TB, ART, chronic care, surgical, ENT) for follow-up and long-term management
- This integration ensures continuity of care and efficient patient flow, but requires clear protocols to avoid duplication, delays, and inconsistent management.

1.4 Need for Hospital-Specific STG

While the National STG 2021 provides the foundation for evidence-based treatment across Ethiopia, it is designed to be broad and adaptable to different contexts. Local adaptation is necessary for Deder General Hospital due to:

1. **Epidemiological Profile** – The disease pattern in the hospital's catchment area differs from national averages, with certain conditions (e.g., dyspepsia, malignant otitis externa) ranking higher locally.
2. **Resource Availability** – Diagnostic tools, medicines, and specialist services are determined by hospital capacity, which may limit implementation of some national recommendations.
3. **Antimicrobial Resistance Patterns** – Local antibiogram trends influence antibiotic selection for common infections.
4. **Staff Training & Capacity** – The STG serves as a reference and training tool for new and existing staff, helping bridge knowledge gaps.
5. **Referral System Realities** – Distance, transport availability, and referral acceptance influence how long certain patients are managed at the general hospital level.

In short, this hospital-specific STG ensures that treatment guidelines are not only evidence-based but also feasible and practical within Deder General Hospital's operational environment.

1.5 Guiding Principles

The Deder General Hospital Adult OPD STG is built upon six guiding principles:

1. **Evidence-Based Practice** – Recommendations are drawn from the latest scientific evidence, national STG, and WHO guidelines, adapted to local realities.
2. **Equity and Access** – All patients should have access to standard care regardless of socioeconomic status, gender, or location.
3. **Quality and Safety** – Emphasis is placed on correct diagnosis, rational prescribing, infection prevention, and avoidance of medical errors.
4. **Patient-Centered Care** – Management considers patient preferences, cultural context, and social circumstances.
5. **Cost-Effectiveness** – Choices in diagnostics and treatments balance clinical benefit with efficient use of limited resources.
6. **Sustainability** – Protocols are designed to be maintained over time with existing hospital resources and capacity.

1.6 Scope of the Document

1.6.1 Target Users

This Standard Treatment Guideline is intended for use by:

- Medical Doctors working in the Adult OPD
- Integrated Emergency Surgeons providing outpatient services
- Nurses involved in triage, assessment, and follow-up
- Pharmacists responsible for medicine dispensing and counseling
- Laboratory Professionals assisting in diagnostic investigations
- Specialists (Internal Medicine, Surgery, ENT, etc.) providing consultations
- Interns and Students on clinical attachment

1.6.2 Patient Population

- **Age Group:** Adults aged 14 years and above (per national OPD definition)
- **Setting**: Ambulatory patients attending the OPD; referral and follow-up cases from other units
- **Clinical Scope:** Common medical, surgical, and ENT conditions seen in OPD, with emphasis on the top 10 high-burden diseases identified in EFY 2016
- **Exclusions:** Pediatric cases (managed under Pediatric OPD STG), obstetric/gynecologic conditions (managed under MCH protocols), and emergency cases requiring immediate resuscitation (managed in Emergency Department)

1.6.3 Conditions Covered

This document specifically includes detailed diagnostic and treatment protocols for the following conditions:

1. Dyspepsia & Peptic Ulcer Disease
2. Community-Acquired Pneumonia
3. Asthma
4. Gastroenteritis / Colitis
5. Tuberculosis
6. Malignant Otitis Externa
7. Refractory Anaemia
8. Typhoid Fever
9. Streptococcal Pharyngitis
10. Urinary Tract Infection

Other OPD conditions may be managed using the National STG 2021, which remains the overarching reference.

1.7 Expected Benefits

The adoption and consistent use of this STG is expected to result in:

1.7.1 For Patients

- More accurate diagnosis and timely treatment
- Reduced complications and avoidable hospital admissions
- Improved satisfaction due to clear communication and follow-up care
- Access to safe, effective, and affordable medicines

1.7.2 For Healthcare Providers

- A practical, locally relevant reference tool for daily practice
- Reduced clinical uncertainty in case management
- Improved skills in rational prescribing and disease management
- Better coordination between OPD and other hospital units

1.7.3 For Hospital Management

- Optimized resource utilization and cost savings
- Reduced wastage of medicines and laboratory supplies
- Improved quality indicators and patient outcomes
- Stronger alignment with Ministry of Health targets and standards

1.8 Development Process

1.8.1 Source of Recommendations

This document is **adapted** from the National Standard Treatment Guidelines, 4th Edition, 2021, and other evidence-based resources including:

- WHO guidelines for common OPD conditions
- Ethiopian Essential Medicines List (EML), 6th Edition
- National Antimicrobial Resistance Containment Strategy
- Local Deder General Hospital morbidity and mortality data (EFY 2016)

1.8.2 Stakeholder Involvement

The development of this STG involved multidisciplinary input from:

- OPD clinicians and nurses
- Pharmacists and pharmacy managers
- Laboratory heads
- Public health officers
- Hospital management and medical directorate
- Regional Health Bureau representatives

1.8.3 Methodology

1. **Needs Assessment**: Review of OPD case mix and leading causes of morbidity and mortality
2. **Document Review**: Analysis of National STG and other reference guidelines
3. Local Adaptation: Adjustment of recommendations to match available diagnostics, medicines, and referral pathways
4. **Stakeholder Validation:** Review workshops with clinical teams for feedback
5. **Approval:** Endorsement by hospital management and Regional Health Bureau
6. **Implementation Planning**: Training schedules, distribution of printed copies, and integration into daily OPD workflow

1.9 Relationship to National & Regional Policies

This STG is designed to align with Ethiopia's national health strategies and regional priorities. Its implementation supports:

- National Standard Treatment Guidelines (4th Edition, 2021): This hospital-specific STG is directly adapted from the national document, ensuring consistency with the Ministry of Health's evidence-based recommendations.
- Essential Medicines List (EML), 6th Edition: All recommended pharmacologic treatments are based on medicines included in the Ethiopian EML and available in Deder General Hospital's pharmacy formulary.
- Health Sector Transformation Plan II (HSTP-II, 2020/21–2024/25): This STG supports the HSTP-II goals of improving quality of care, reducing preventable mortality, and enhancing equity in access to services.
- National Antimicrobial Resistance Containment Strategy (2017–2027): The STG promotes rational antibiotic use and infection prevention practices, contributing to Ethiopia's AMR containment targets.
- Oromia Regional Health Bureau Guidelines: Adaptation considers the regional health priorities and resource availability, ensuring that the STG is feasible within local operational constraints.

1.10 Limitations

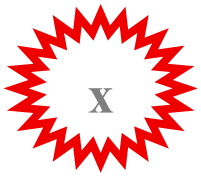
While comprehensive, this STG has certain limitations:

1. **Resource Constraints:** Availability of medicines, laboratory tests, and imaging may vary depending on stock levels, supply chain disruptions, and budget constraints.
2. **Infrastructure Limitations:** Inconsistent power supply, limited internet access, and occasional equipment downtime may impact timely diagnosis and treatment.
3. **Human Resource Capacity:** The hospital experiences periodic staff shortages, turnover, and the presence of newly graduated staff requiring ongoing mentorship.
4. **Scope Restriction:** This document focuses on the top 10 diseases in the Adult OPD; it does not replace the National STG for less common conditions.
5. **Evidence Evolution:** Medical evidence changes over time; recommendations may become outdated if not regularly reviewed and updated.

1.11 Sustainability & Updating Plan

To ensure this STG remains current and functional:

- **Review Frequency:** This STG will be reviewed every two years or earlier if there are significant changes in clinical evidence, national policy, or hospital epidemiology.
- **Responsibility:** A Hospital STG Review Committee—comprising representatives from OPD, Pharmacy, Laboratory, and Management—will oversee revisions.
- **Feedback Mechanism:** Healthcare workers are encouraged to provide feedback via an STG suggestion logbook maintained in the OPD.
- **Capacity Building:** Regular in-service training sessions will be conducted to keep staff updated on protocol changes.
- **Integration into Orientation:** New staff and interns will receive STG training as part of their orientation process.
- **Digital & Printed Copies:** The STG will be available in both printed manuals and digital format for ease of access.



1.12 Ethical Considerations

Ethical practice is central to the application of this STG. All healthcare providers must adhere to:

1. **Informed Consent:** Patients must be given adequate information about their diagnosis, treatment options, and associated risks to make informed decisions.
2. **Confidentiality:** Patient information must be protected and shared only with authorized personnel involved in their care.
3. **Non-Discrimination:** All patients must receive care regardless of age, gender, ethnicity, religion, socioeconomic status, or disability.
4. **Cultural Sensitivity:** Management plans should respect cultural beliefs and practices, provided they do not compromise patient safety.
5. **Professional Integrity:** Healthcare providers must avoid conflicts of interest and base treatment decisions solely on patient needs and evidence-based practice.
6. **End-of-Life Care:** For patients requiring palliative care, decisions should be guided by compassion, dignity, and respect for patient and family wishes.

1.13 How to Use This STG

The Deder General Hospital Adult OPD Standard Treatment Guideline is designed for practical, daily use by clinicians, nurses, pharmacists, and other healthcare staff. To ensure it is applied effectively:

1.13.1 General Use

- Use this document as the primary reference for diagnosis and treatment of the top 10 OPD conditions.
- For conditions not included here, refer to the National STG 2021 and relevant specialty protocols.
- Always adapt treatment to the individual patient's condition, co-morbidities, and preferences, within the limits of available resources.
- Keep this STG accessible in OPD consultation rooms, triage stations, pharmacy, and laboratory units.

1.13.2 Format & Navigation

Each disease chapter is organized as follows:

1. **Definition:** Clear and concise explanation of the condition.
2. **Causes & Risk Factors:** Common etiologies relevant to local context.
3. **Clinical Features:** Key symptoms and signs for diagnosis.
4. **Investigations:** Diagnostic tests prioritized by availability and necessity.
5. **Management:** Evidence-based pharmacologic and non-pharmacologic interventions.
6. **Follow-Up:** Criteria for reassessment and continuity of care.
7. **Referral Criteria:** Indications for referral to higher-level care or specialists.
8. **Prevention & Health Education:** Strategies for reducing recurrence and community spread.

1.13.3 Symbols & Notations

- [E] – Medicine available in the Ethiopian Essential Medicines List (EML)
- [PO] – Oral route of administration
- [IM] – Intramuscular route
- [IV] – Intravenous route
- [UR] – Use with caution in renal impairment
- [P] – Use with caution in pregnancy

1.13.4 Clinical Decision Support

- Use triage algorithms for rapid identification of severe illness.
- Apply dose calculation charts for weight-based dosing.
- Consult the hospital antibiogram before prescribing antibiotics when possible.

1.13.5 Documentation Requirements

For every patient encounter:

- Document diagnosis, investigations, treatment plan, and follow-up date clearly in the patient's record.
- Record any adverse drug reactions in the national pharmacovigilance reporting format.

1.14 Definitions & Acronyms

1.14.1 Definitions

- **Adult**: For the purpose of this STG, any patient aged 14 years or older.
- **Ambulatory Care**: Outpatient care that does not require overnight hospital admission.

- **Essential Medicines**: Medicines that satisfy the priority healthcare needs of the population, as defined in the Ethiopian EML.
- **First-Line Treatment**: The preferred initial treatment for a condition based on effectiveness, safety, and cost.
- **Referral**: The process of directing a patient to higher-level or specialized care for further management.
- **Standard Treatment Guideline (STG)**: A systematically developed protocol to assist healthcare providers in making appropriate clinical decisions.

1.14.2 Acronyms

Acronym	Meaning
ADR	Adverse Drug Reaction
AMR	Antimicrobial Resistance
ART	Antiretroviral Therapy
CAP	Community-Acquired Pneumonia
DHIS2	District Health Information System 2
EDL	Essential Drug List
EFY	Ethiopian Fiscal Year
EML	Essential Medicines List
ENT	Ear, Nose, and Throat
HSTP-II	Health Sector Transformation Plan II
IPD	Inpatient Department
IV	Intravenous
IM	Intramuscular
MOH	Ministry of Health
OPD	Outpatient Department
OTC	Over-The-Counter
PO	Oral
STG	Standard Treatment Guideline
TB	Tuberculosis
UTI	Urinary Tract Infection
WHO	World Health Organization

SECTION 2:

PURPOSE, RATIONALE, AND PRINCIPLES OF GOOD PRESCRIBING & DISPENSING PRACTICE

2.1 Purpose

The Deder General Hospital Adult OPD Standard Treatment Guideline (STG) serves as a practical clinical tool to:

1. **Standardize clinical practice** — ensuring that all healthcare providers follow consistent, evidence-based protocols for diagnosis, treatment, and follow-up of common adult OPD conditions.
2. **Improve quality of patient care** — by reducing unnecessary variation in clinical management, ensuring timely diagnosis, and optimizing patient outcomes.
3. **Promote rational medicine use** — ensuring that medicines are prescribed appropriately, safely, and cost-effectively in alignment with the Ethiopian Essential Medicines List (EML).
4. **Support antimicrobial stewardship** — to help prevent and control antimicrobial resistance (AMR).
5. **Optimize resource utilization** — prioritizing interventions that are effective, affordable, and feasible within the hospital's available infrastructure and budget.
6. **Serve as a training and reference tool** — for newly deployed clinicians, interns, and other health professionals, strengthening diagnostic and prescribing skills.
7. **Facilitate monitoring and evaluation** — by providing a framework against which the quality of clinical care can be measured.

2.2 Rationale

The rationale for a hospital-specific STG at Deder General Hospital includes:

- **High Burden of Disease:** Analysis of EFY 2016 OPD data shows a concentration of morbidity and mortality in a limited set of conditions, making focused, disease-specific protocols essential.
- **Variation in Clinical Practice**: Without standardized protocols, management of the same condition may vary widely between providers, leading to inconsistent patient outcomes.
- **Antimicrobial Resistance Threat:** Misuse and overuse of antibiotics in OPD settings is a key driver of AMR, which can render standard treatments ineffective.
- **Resource-Limited Context:** The hospital must prioritize treatments and investigations that are affordable, available, and sustainable.
- **Policy Alignment**: The local STG ensures that hospital practice aligns with the National STG 2021, the Essential Medicines List, and the National AMR Strategy, while adapting to local needs.
- **Staff Capacity Building**: A locally relevant STG helps ensure all clinicians, including those newly assigned, are trained in the same evidence-based protocols.

2.3 Principles of Good Prescribing

Good prescribing is central to delivering safe, effective, and cost-efficient care. It requires a structured approach that includes clinical judgment, evidence-based selection of medicines, and patient-centered decision-making.

2.3.1 General Principles

- Prescribe only when there is a clear clinical indication.
- Choose medicines from the Ethiopian Essential Medicines List (EML) and hospital formulary.
- Select the most appropriate medicine considering efficacy, safety, cost, and patient factors.
- Avoid unnecessary polypharmacy (use of multiple medicines without clinical justification).
- Adjust dosages for special populations such as the elderly, pregnant women, and patients with renal or hepatic impairment.
- Document all prescriptions clearly, including generic name, dose, route, frequency, and duration.

2.3.2 Antimicrobial Prescribing Principles

- Only prescribe antimicrobials for confirmed or strongly suspected bacterial infections.
- Base choice of antibiotic on likely pathogens and local resistance patterns.
- Prefer narrow-spectrum antibiotics when possible to minimize AMR development.
- Avoid prescribing antibiotics for viral illnesses such as common cold or influenza.
- Follow recommended duration of treatment; avoid unnecessarily prolonged courses.
- Review antibiotic prescriptions regularly and adjust based on patient response or laboratory results.
- Educate patients on the importance of completing the prescribed antibiotic course.

2.3.3 Steps in Rational Prescribing *(Adapted from WHO Good Prescribing Guide)*

1. Define the patient's problem through history, examination, and investigations.
2. Specify the therapeutic objective — what you aim to achieve with treatment.
3. Choose the standard treatment based on the STG.
4. Verify suitability for the patient, considering co-morbidities and contraindications.
5. Start treatment with clear instructions for use.
6. Give information about possible side effects, follow-up needs, and warning signs.
7. Monitor and review treatment response and make changes if necessary.

2.4 Principles of Good Dispensing Practice

Dispensing is the process of preparing and providing medicines to patients with appropriate counseling. Errors in dispensing can lead to treatment failure or harm.

Key principles include:

- **Prescription Verification:** Ensure prescriptions are legible, complete, and appropriate.
- **Accuracy:** Provide the correct medicine, dose, formulation, and quantity.
- **Labeling:** Clearly label with patient name, medicine name (generic), dosage, frequency, duration, and special instructions.
- **Patient Counseling:** Explain medicine purpose, how to take it, possible side effects, what to do if a dose is missed, and storage conditions.
- **Documentation:** Record dispensed medicines in pharmacy registers for accountability.
- **Pharmacovigilance:** Monitor and report any adverse drug reactions using the national ADR reporting form.

Section 3:

Antimicrobial Resistance, Patient Care, and Palliative Care

3.1 Antimicrobial Resistance (AMR)

3.1.1 Background

Antimicrobial Resistance (AMR) occurs when microorganisms such as bacteria, viruses, fungi, and parasites evolve to withstand the effects of antimicrobial medicines, rendering standard treatments ineffective. AMR is recognized by the World Health Organization (WHO) as one of the top ten global public health threats.

In Ethiopia, factors contributing to AMR include:

- **Overuse** and **misuse** of antibiotics in both human and animal health
- **Self-medication** and **over-the-counter sale of antibiotics** without prescription
- **Incomplete courses** of antibiotic treatment
- **Poor infection prevention** and control (IPC) practices in healthcare facilities
- **Inadequate surveillance** of resistance patterns

At Deder General Hospital, local antibiogram data shows emerging resistance to commonly prescribed antibiotics, making it essential to preserve the effectiveness of available antimicrobials through rational prescribing and proper use.

3.1.2 Principles for Combating AMR in OPD

To reduce the risk and spread of AMR:

- Prescribe antimicrobials only when indicated — avoid empirical antibiotics for clearly viral infections.
- Choose antibiotics based on likely pathogens and local resistance data.
- Use narrow-spectrum antibiotics whenever possible.
- Prescribe the right dose, route, and duration according to STG recommendations.
- Educate patients about adherence and the dangers of sharing or saving antibiotics.
- Report treatment failures and unusual resistance patterns to the hospital's AMR focal person.

3.1.3 The Role of the Hospital Antimicrobial Stewardship Program (ASP)

Deder General Hospital will integrate this STG with its Antimicrobial Stewardship Program to:

- Conduct regular prescription audits in OPD
- Provide feedback and mentoring to prescribers
- Monitor antimicrobial consumption patterns
- Disseminate updated local antibiograms annually
- Organize continuous professional development (CPD) sessions on AMR

3.2 Patient Care in Ambulatory and Hospitalized Settings

3.2.1 Ambulatory (Outpatient) Care

Ambulatory care in OPD focuses on timely diagnosis, treatment, and follow-up without hospital admission.

Core elements include:

- Rapid triage to identify critically ill patients needing emergency referral
- Thorough history-taking and focused examination
- Ordering only essential investigations guided by STG
- Initiating appropriate treatment promptly
- Providing clear discharge instructions including medicine use, warning signs, and follow-up plans
- Coordinating with specialty clinics for chronic care follow-up (e.g., TB, asthma, anemia)

3.2.2 Hospitalized Care (Inpatient Management)

Patients may require hospital admission from OPD if they meet criteria such as:

- Severe disease presentation
- Complicated or advanced stage of illness
- Need for continuous monitoring or parenteral therapy
- Poor response to initial outpatient treatment
- High risk of deterioration (e.g., elderly, comorbid conditions)

Principles for inpatient care:

- Immediate stabilization of airway, breathing, and circulation (ABC approach)
- Comprehensive diagnostic workup
- Daily patient review by the admitting clinician
- Multidisciplinary care involving nursing, pharmacy, nutrition, and laboratory teams
- Early initiation of discharge planning and post-discharge follow-up arrangements

3.3 Palliative Care

3.3.1 Definition

Palliative care is the active, holistic care of individuals with life-limiting illnesses, focusing on relief of pain and other distressing symptoms, while addressing psychological, social, and spiritual needs.

3.3.2 Goals of Palliative Care

- Alleviate pain and other physical symptoms
- Support emotional and psychological well-being
- Facilitate open and compassionate communication about prognosis and care options
- Enhance quality of life for patients and their families
- Provide bereavement support to families after the patient's death

3.3.3 Core Principles

- **Holistic Assessment**: Evaluate physical, psychological, and spiritual needs.
- **WHO Analgesic Ladder:**
  - ✓ Step 1 – Non-opioids (e.g., paracetamol, NSAIDs) for mild pain
  - ✓ Step 2 – Weak opioids (e.g., codeine, tramadol) ± non-opioids for moderate pain
  - ✓ Step 3 – Strong opioids (e.g., morphine) ± adjuvant medicines for severe pain
- **Symptom Management:** Address dyspnea, nausea, constipation, depression, and anxiety.
- **Family Involvement**: Engage families in care planning and decision-making.
- **Ethical Practice:** Respect patient autonomy and cultural beliefs.

3.3.4 Integration in Deder General Hospital OPD

Although palliative care is often associated with inpatient or hospice services, OPD clinicians play a crucial role in:

- Identifying patients with palliative needs early (e.g., advanced cancer, end-stage organ failure)
- Initiating symptom relief before referral
- Coordinating with community and home-based palliative care programs
- Providing follow-up consultations for stable patients

SECTION 4:

DISEASE SPEFIC TOPICS

CHAPTER1:

DYSPEPSIA AND PEPTIC ULCER DISEASE

BRIEF DESCRIPTION

- Dyspepsia describes a wide and common clinical entity which presents in one of the three ways:
  1. Epigastric pain/burning (epigastric pain syndrome)
  2. Postprandial fullness
  3. Early satiety
- Dyspepsia is caused by a number of disorders.
- The most common cause is functional (non-ulcer) dyspepsia followed by peptic ulcer disease.
- Gastro esophageal reflux disease (GERD), gastric cancer, medication induced dyspepsia, biliary pain, chronic abdominal wall pain and pancreatitis are other possible causes.

CLINICAL FEATURES

- Depending on the type of dyspeptic syndrome patients may present with predominant epigastric burning sensation/pain/discomfort, postprandial discomfort and fullness or be unable to finish a regular meal.

ALARM SIGNS (need to be further investigation for cancer)

- Advanced age (>55years)
- Previous gastric surgery
- Unintended weight loss
- Persistent vomiting
- Hematemesis
- Progressivedysphagia/Odynophagia
- Otherwise, unexplainedanemia
- Palpable abdominal mass
- Lymphadenopathy
- Jaundice

INVESTIGATIONS

- **H. Pylori test**: IgG serology or stool antigen or 13C-urea test
- Hemoglobin/hematocrit, stool for occult blood-when indicated
- Upper GI endoscopy

- H. Pylori test needs to be done for the following patients
  - Long standing dyspepsia
  - Younger than 55 years
  - No alarm symptoms
  - No use of non-steroidal anti-inflammatory drugs
  - No features of GERD (Gastro Esophageal Reflux Disease)
- "Test and treat" for H. Pylori can be practiced in these group of individuals

TREATMENT
OBJECTIVES OFTREATMENT

- Decrease symptoms/improve quality oflife
- Prevent development of complications

NON PHARMACOLOGIC

- Avoid offending foods/drinks

PHARMACOLOGIC

- **H. Pylorinegative**
  - First line:**Proton pump inhibitors**
    - Omeprazole, 20mg P.O., twice per day for 4-8 weeks
    - Esomeprazole, 40mg P.O., daily for 4-8 weeks
    - Pantoprazole, 40mg P.O., BID for 4-8 weeks
  - Alternatives:**H2 receptorblockers**
    - Cimetidine, 400mg P.O., BID for 4-8 weeks
    - Ranitidine, 150mg P.O. BID for 4-8 weeks
    - Famotidine, 20-40mg P.O. daily for 4-8 weeks
- **H. Pylori positive: H. pylori eradication therapy**
  - **First line therapy**:All drugs for 7-14 days
    - Amoxicillin, 1gm, P.O. BID

      PLUS
    - Clarithromycin, 500mg, P.O., BID

      PLUS
    - Proton pump inhibitors (see the daily doses above)
  - **Alternative** (for penicillin allergic patients):This regimen has a higher failure rate.All drugs should be given for 7-14 days

- Clarithromycin, 500mg P.O.BID

  PLUS

- Metronidazole, 500mg, P.O.BID

  PLUS

- Proton pump inhibitors (see the daily doses above)

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

BRIEF DESCRIPTION

- Gastroesophageal reflux refers to the return of stomach contents in to the esophagus.
- Some degree of brief reflux occurs physiologically; usually after a meal or during sleep.
- GERD refers to a pathologic reflux associated with symptoms and complications.
- GERD is a common in primary care practice. Due to its symptoms, it can also be misdiagnosed.
- Based on the endoscopic appearance GERD is classified in to two types: Erosive and non-erosive.
- Erosive GERD (Erosive esophagitis) is diagnosed when there are endoscopically visible breaks in the esophageal mucosa while non-erosive GERD shows no visible mucosal injury on endoscopy.
- GERD is associated with significant esophageal or extraesophageal complications.
  - **Esophageal complications**
    - Barrett's esophagus: a precancerous change in the esophageal mucosa (from squamous epithelium to columnar epithelium)
    - Esophageal stricture: which manifests with solid food dysphagia

and intermittent food impaction?

- **Extraesophageal complications**
  - Triggering Asthma
  - Laryngeal and pharyngeal reflux: which manifests with chronic cough, repetitive throat cleaning, hoarseness of voice

CLINICAL MANIFESTATIONS

- **Symptoms**
- The two major symptoms of GERD which are considered classic (typical) are heartburn and regurgitation.
  - Heartburn is a commonly described by patients as a burning sensation behind the sternum (retrosternal area).
  - Regurgitation is defined as back flow of gastric contests in to the mouth or pharynx. Patients feel an acidic (sour) content coming to the mouth mixed with small amounts of undigested food.
- **Other symptoms**
  - Chest pain: GERD associated chest pain can mimic angina (pain from ischemic heart disease)
  - Triggering asthma attacks (wheezing)
  - Hoarsens of voice
  - Persistent cough
  - Nausea
  - Sensation of a lump in the throat (Globus sensation)
  - Increased salivation (Water brash)

DIAGNOSIS

- In patients with typical symptoms i.e., heartburn or regurgitation, the diagnosis of GERD can be considered on clinical grounds without additional investigations, if there are no alarm signs. In such cases empiric

therapy should be started.

INVESTIGATIONS

- **Upper GI (gastrointestinal) endoscopy**
  - Endoscopy is not necessary to make a diagnosis of GERD but it is indicated in patients with alarm features to see evaluate for possible malignancy.
  - The alarm features are weight loss, age above 60 years, iron deficiency anemia, dysphagia, persistent vomiting or family history of cancer in parents or siblings.
  - If GERD symptoms have been there for more than 5-10 years,endoscopy can be considered to look for evidence of Barrett'sesophagus.

TREATMENT
OBJECTIVES OFTREATMENT

- Relive symptoms
- Decrease the risk of complications such as Barrett's esophagus, esophageal stricture

NON-PHARMACOLOGIC TREATMENT

- **Life style modifications**
  - Weight loss in overweight and obsess patients.
  - Avoiding meals 2 -3 hours before bed is alsoadvisable.
  - Head elevations to 15-20 cm duringsleep.
  - Dietary selection should not be forced or recommended universally unless patients identify the specific food item as triggering factor. e.g., caffeine, spicy foods, food with high fat content, carbonated beverages, and chocolate)
  - Other life style modifications are not supported by evidence.
- **Surgery**
  - Surgical intervention (usually fundoplication) in GERD patients is rarely

indicated. Surgery ma may be considered in the following circumstances:

- Large hiatal hernia causing the reflux symptoms
- Evidence of aspiration
- Esophagitis refractory to medical therapy
- Persistent symptoms documented as being caused by refractory GERD: after checking compliance to PPI and optimizing PPI use.

PHARMACOLOGIC TREATMENT

- **First line: Proton-pump inhibitors (PPIs)**
  - No major difference in between the available PPIs
    - Omeprazole 40mg PO daily for 8 -12 weeks

      OR
    - Esomeprazole 40mg PO daily for 8-12 weeks

      OR
    - Pantoprazole 40mg PO daily for 8-12 weeks
- Stop therapy on symptom resolution to assess response
- After the first 8 -12 weeks, resume therapy as needed
  - Intermittent

    OR
  - On demand
- **Alternatives:** If PPIs are not available and the symptoms are mild Histamine-2 receptor blockers (H2 blockers) can be considered as alternatives.
  - Cimetidine 400mg BID for 8 weeks

    OR

- Ranitidine 150mg BID for 8 weeks

  OR

- Famotidine 20mg BID for 8 weeks

REFERRAL

- Patients with alarm symptoms need to be referred without any delay after the initial evaluation.
- Patients with persistent symptoms after 8 weeks of therapy should be referred for specialist evaluation and follow up.

FURTHER READING

1. Philip O. Katz Lauren B. Gerson, and Marcelo F.Vela. Guidelines for the Diagnosis and Management of Gastroesophageal Reflux Disease. Am J Gastroenterol 2013;108:308 – 328
2. World Gastroenterology Organization Global Guidelines: GERD, Global Perspective on Gastroesophageal Reflux Disease. Update October 2015.

GASTROINTESTINAL BLEEDING

UPPER GASTROINTESTINAL (GI) BLEEDING

BRIEF DESCRIPTION

- Upper GI bleeding refers to gastrointestinal blood loss originating from the gastrointestinal tract is proximal to the ligament of Treitz at the duodenojejunal junction.
- It can be overt or occult bleeding.
- Overt upper GI bleeding can manifest in the following ways:
    - Hematemesis: vomiting of frank red blood or a "coffee-grounds" material.

- Melena: Passage of black, tarry stool
- Hematochezia: passage of bright red or maroon (dark red) blood from the rectum. Upper GI bleeding causes hematochezia rarely, when it is massive and very acute.

- Occult upper GI bleeding present with symptoms anemia such as lightheadedness, or a positive fecal occult blood test on routine testing.
- The causes of upper GI bleeding are summarized in the table below.
- The two major causes that should be considered in every patient with overt upper GI bleeding are peptic ulcer disease and esophageal varices.

Table 6.1: Causes of Upper GI bleeding

1. Peptic ulcer disease
2. Esophageal varices
3. Gastroduodenal erosions
4. Mallory-Weiss tears (esophageal mucosal tear due to vomiting or retching)
5. Esophagitis
6. Vascular malformations,
7. Neoplasm
8. Coagulopathy
9. Obscure upper GI bleeding: often from small intestinal lesions

CLINICAL FEATURES

- **Symptoms**
  - Nausea
  - Vomiting of bright red blood or coffee-ground matter
  - Melena

- **Hematochezia**: rare in upper GI bleeding but can occur in massive acute bleeding.
- Symptoms related to the underlying cause;
  - Medication history: antiplatelet (aspirin, clopidogrel), non-steroidal anti-inflammatory drugs or anticoagulants
  - Symptoms of portal hypertension or liver cirrhosis in patients with variceal bleeding e.g., ascites, fatigue.
  - The bleeding in varices is generally bright red, painless, brisk, and voluminous.
  - Long standing epigastric pain: Suggestive of peptic ulcer disease
  - Preceding forceful vomiting or retching suggests Mallory-Weiss tears
  - **Weight loss**: may indicate neoplasm

- **Signs**
- In general, the physical signs (physical examination focuses on the following two elements)

4. **The hemodynamic status of the patient and the degree of anemia**
   - Blood pressure: check for supine BP. If supine BP is normal check for postural hypotension (supine, followed by measurement after 3 minutes of standing
   - Pulse rate: assess for resting tachycardia
   - Degree of pallor
5. **Signs of the underlying cause of the upper GI bleeding**
   - Signs of chronic liver disease or portal hypertension indicating the possibility of bleeding varices: Ascites, splenomegaly, encephalopathy.
   - Other site bleeding: platelet related disorders or coagulopathies

INVESTIGATIONS

- CBC (complete blood count)
- Serial hemoglobin/hematocrit every 8 hour: the initial hemoglobin/ hematocrit may be normal as the loss is whole blood (both plasma and cells)
- Coagulation profile: PT (INR) and PPTT
- Urea and Creatinine
- Liver enzymes
- Upper GI endoscopy; see above on diagnosis

DIAGNOSIS

- The diagnosis of upper GI bleeding is made on clinical grounds mainly based on history: a history of hematemesis or melena establishes the diagnosis.
- The next step in the diagnosis is trying to establish the cause of the upper GI bleeding.
- In addition to history and physical examination, identifying the cause of the upper GI bleeding requires upper GI endoscopy.
- Upper GI endoscopy has both diagnostic and therapeutic value.

RISK STRATIFICATION

- There are a few risk stratifications tools which are useful to assess the likelihood of a person with upper GI bleeding to need further interventions like endoscopic treatment and transfusion.
- The Glasgow-Blatchford bleeding score (GBS) is one of the scores. It is simple risk stratification tool which does not require endoscopy. We recommend using the score.

Table. 6.2: Glasgow-Blatchford bleeding score (GBS)

Risk marker	Value	Score
• Urea (blood urea) (mg/dl)	38 - 46	2
	47 - 57	3
	58 - 146	4
	≥147	6
• Hemoglobin (g/dl) in males	12 - 13	1
	10 - 12	3
	< 10	6
• Hemoglobin (g/dl) in females	10 - 12	1
	< 10	6
• Systolic BP (mmHg)	100 - 109	1
	99 - 90	2
	< 90	3
• Pulse ≥100 (per min)		1
• Presentation with melena		1
• Presentation with syncope		2
• Hepatic disease		2
• Heart failure		2
Note: Score 0: low risk Score > 0: high risk, keep in hospital as the patient is likely to require transfusion or endoscopic intervention Score > or = 8:requires ICU admission		

TREATMENT
OBJECTIVES OFTREATMENT

- Hemodynamic restoration
- Arresting or decreasing bleeding
- Preventing recurrence ofbleeding

PHARMACOLOGIC AND NON-PHARMACOLOGIC TREATMENT

- **Hemodynamic stabilization**
  - Monitor airway, blood pressure and heartrate.
  - Do NOT give patient anything by mouth
  - Establish two large bore IV lines (16 gauge)
  - Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid
  - Provide transfusion if either of the following ispresent
    - Hemodynamic instability (hypotension) ispresent.
    - Hemoglobin <9 g/dL in high-risk patients (e.g., elderly, coronary artery disease)
    - Hemoglobin <7 g/dL (70 g/L) in low-risk patients
- **Pharmacotherapy for allpatients**
  - **Intravenous proton pumpinhibitor**
    - Omeprazole 80mg IV loading followed by 40mg IV BID

      OR
    - Esomeprazole 40 mg IV BID

      OR
    - Pantoprazole 40 mg IV BID

- **Arresting bleeding**
- **Endoscopic** therapy is the main stay of therapy to arrest bleeding.
  - After hemodynamic stabilization consult or refer to facility with endoscopic services.
  - Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage after tracheal intubation.
- **Open surgery**
- **Indica**tions for surgery;
  - Hemodynamic instability despite vigorous resuscitation (> 3 units three of transfusion)
  - Shock associated with recurrenthemorrhage
  - Perforated PUD along with bleeding
  - Failed endoscopic therapy for bleeding PUD
- No access to endoscopy therapy with ongoing bleeding
- Relative indications: difficult crossmatch, refusal of transfusion, shock on presentation,

- **Treating the underlying cause**
  - Patients with H. Pylori associated ulcer bleeding should receive eradication therapy.
  - In NSAID or Aspirin associated bleeding ulcers: stop the drug and re-evaluate the need.
  - Anticoagulants: stop and re-evaluate for continued need, dose adjustment if c
  - Variceal bleeding: band ligation and non-selective beta-blocker therapy (propranolol)

- Idiopathic (non-H. pylori, non-NSAID) ulcers: long-term PPI is recommended

LOWER GASTROINTESTINAL (GI) BLEEDING

BRIEF DESCRIPTION

- Gastrointestinal (GI) Bleeding refers to any bleeding that occurs from the mouth to the anus. Anatomically GI bleeding is divided in to upper and lower.
- The ligament of Treitz is used as the anatomic reference to differentiate lower and upper GI bleeding.

  The incidence of lower GI bleeding is higher in older age groups, particularly in those taking anti-platelet agents like aspirin, non-steroidal anti-inflammatory drugs or anticoagulants.
- Lower GI bleeding can be overt or occult.
  - Overt lower GI bleeding presents with either frank red bleeding (hematochezia) or dark, tarry stool (melena).
  - Occult GI bleeding presents with evidence of iron deficiency anemia but no hematochezia or melena.
- Over GI bleeding is labeled to be massive when it is associated with hemodynamic instability.
- The major causes of lower GI bleeding are categorized asfollows:
  - Vascular causes
  - Hemorrhoids
  - Ischemic bowel
  - Vascular dysplasia (angiodysplasia)
  - Post procedure (postpolypectomy)
    - **Neoplastic causes**
  - Colon cancer
  - Polyps
    - **Anatomic causes**

— Diverticulosis

- **Inflammatory causes**

— Inflammatory bowel disease

— Infectious colitis

'

CLINICAL MANIFESTATIONS

- **Symptoms**
    - **Hematochezia:** passage of bright red or dark red (maroon) blood or clots per rectum.
  - Bleeding from the left colon tends to be bright red in color while bleeding from the right colon appears to be dark or maroon colored and may be mixed with stool.
  - Bleeding from the right colon might rarely cause melena (the stool itself is dark)
    - **Symptoms of anemia or hemodynamic compromise**: fatigue, postural dizziness, light headedness
- **Signs**
    - **Signs of hemodynamic compromise**:
  - Hypotension (supine or postural)
  - Resting tachycardia.
    - **Signs of anemia**
  - Pallor
  - Tachycardia
  - Ejection systolic murmur

INVESTIGATIONS

- The following important investigation in patients with lower GI bleeding
  - CBC (complete blood count): in massive acute bleeding the hemoglobin may appear normal.
  - Serial hemoglobin; every 8 hours
  - Coagulation studies: INR (PT) and PTT
  - Liver enzymes
  - BUN and creatinine
  - **Colonoscopy:** when the clinical diagnosis is lower GI bleeding

- **Upper GI endoscopy**: when the clinical diagnosis is upper GI bleeding.

DIAGNOSIS

- The diagnosis of lower GI bleeding requires the following important steps:

1. **Identifying whether the bleeding is upper or lower GI in origin.**
    - Massive upper GI bleeding can cause hematochezia; hence, differentiating Upper from lower GI bleeding is necessary.
    - The presence of hemodynamic instability favors upper GI bleeding
    - The presence of clots suggests lower GI bleeding
    - When there is suspicion of upper GI source: insert NG tube and do gastric lavage with normal saline
        - Gastric lavage with coffee-ground material or bright red blood= upper GI bleeding
        - Gastric lavage is bilious = lower GI bleeding
        - If the gastric lavage is neither of the above = indeterminate (it can be either of the two)

Identifying possible causes or precipitants of the bleeding.

- The history should focus on the following
    - **Medicat**ions: Antiplatelets (e.g., Aspirin or clopidogrel), non-steroidal anti-inflammatory drugs (e.g., Diclofenac, indomethacin, ibuprofen), anticoagulants
    - Prior history of bleeding
    - Significant abdominal pain: suggests inflammatory or ischemic bowel disease or perforation
    - Significant **weight loss**: suggests malignancy
- Digital rectal examination

3. Localization of the bleeding and definitive diagnosis

    - All patients with a clinical diagnosis of lower GI bleeding require colonoscopic examination to identify the cause of bleeding, arrest the bleeding if identifiable.

TREATMENT

OBJECTIVES OF TREATMENT

- Restore hemodynamic status
- Correct precipitating factors

INITIAL TREATMENT AND REFERRAL

- The following are components of the initial treatment of patients suspected of acute lower GI bleeding;

Hemodynamic status evaluation and resuscitation

- In patients with hemodynamic compromise secure two wide bore IV cannuale and resuscitate with crystalloids.
- While crystalloids are being given, blood should be requested for transfusion.
- Do not depend on the initial hemoglobin or hematocrit to for transfusion, as it is apparently ("falsely") normal.

- **Discon**tinue antiplatelets, non-steroidal anti-inflammatory drugs or anticoagulants
- Correct **coagulopathies**
    - E.g., If INR is high or patients are on warfarin, give fresh frozen plasma and/or vitamin K

REFERRAL

Patients should be referred to a facility with gastroenterology specialty service for colonoscopy, after hemodynamic stabilization.

In patients who continue to bleed massively and who are unstable to be transferred to a center with colonoscopy facility, surgical consultation should be made.

CHAPTER 2:
PULMONARY INFECTIONS

1. COMMUNITYACQUIRED PNEUMONIA (CAP)

BRIEF DESCRIPTION

- Pneumonia refers to acute inflammation of the distal lung-terminal airways, alveolar spaces, and interstitium.
- Pneumonia is a common
- The clinical presentation and the etiology vary greatly depending on the age of the patient, the infecting organism, the site/s the infection has involved, immune status of the patient and the place of acquisition of infection.
- Etiology of CAP
  - *Streptococcus pneumonia* is the most common etiology.

    Others: *Mycoplasma,* Chlamydia, *H. influenzae*, *M. catarrhalis*, *Legionella* (especially in elderly, smokers), *viral* (especially in young) , *Klebsiella* & other gram-negative bacteria (mainly in alcoholics & during aspiration),

    S. aureus (especially post-viral infection)
  -

CLINICAL FEATURES

- Although signs, symptoms & imaging do *not* reliably distinguish between "typical" and "atypical" pneumonia, the following can give clues.
- **"Typical"** (*S. pneumonia*, *H. influenzae*)
  - Acute onset of fever, cough with purulent sputum, dyspnea
  - Consolidation on CXR.
- **"Atypical" (***Mycoplasma*, *Chlamydia, Legionella,* viral)
  - More insidious onset of dry cough
  - E xtrapulmonary symptoms may be present (nausea/vomiting, diarrhea, headache, myalgias, sore throat)
  - Patchy interstitial infiltrates on CXR
  - Elevated transaminases & low serum sodium with *Legionella.*

IDENTIFYING SITE OF CARE

- Although it can't completely replace clinical judgement, clinicians need to use prognostic criteria to decide at the setting of treatment pneumonia (inpatient versus outpatient).
- The CURB-65 is relatively easy to use criteria for this purpose.

Table 8.35: CURB-65 criteria for deciding the setting of treatment in CAP	
C	**C**onfusion* (1 point)
U	**U**rea >20 mg/dL (7 mmol/L)** (1 point)
R	**R**espiratory rate ≥30 breaths per minute (1 point)
B	Low systolic (<90 mmHg) or diastolic (≤60 mmHg) **B**lood pressure (1 point)
65	Age ≥**65** years (1 point)

[*] Defined as an Abbreviated Mental Test Score ≤8 or new disorientation to person, place, or time.

***Urea is blood urea nitrogen [BUN], expressed in mg/dL or serum urea concentration, expressed in mmol/L*

CURB-65 score	Site of management
0	Outpatient treatment, PO antibiotic
1-2 point	Inpatient management, with IV antibiotics, in a general ward. Those with a score of 1, due age ≥ 65 can be managed as an outpatient.
3 to 5 points	Inpatient management with IV antibiotics; evaluate for ICU admission.

- Indications for ICU admission: ICU admission is required if the patient has severe CAP only. See the table below for criteria defining severe CAP.

Table 8.36:Criteria for defining severe CAP

Major criteria: the presence of one of the following two major criteria 1. Septic shock requiring vasopressor 2. Respiratory failure requiring mechanical ventilation
Minor criteria: three or more of the following (nine) criteria found in a patient 1. Confusion/disorientation 2. Hypotension requiring aggressive fluid resuscitation 3. Respiratory rate ≥ 30 breaths/min 4. Multilobar infiltrates 5. Blood urea nitrogen level ≥20 mg/dl 6. White blood cell count <4,000 cells/ml 7. Platelet count <100,000/ml 8. Hypothermia (core temperature,<36^0 C) 9. PaO2/FI O2 ratio ≤250

INVESTIGATIONSAND DIAGNOSIS

- **Chest X-ray (CXR)**: it is the most important investigation in the diagnosis pneumonia and all patients considered to have pneumonia should have CXR.
- **Sputum gram stain and culture**:
  1. It is not routinely recommended for majority patients.
  2. In patients with severe pneumonia requiring hospitalization, pretreatment sputum or endotracheal aspirate (if intubated) along with blood culture need to be sent.
  3. Patients to be treated empirically for MRSA or P. aeruginosa need also pretreatment sputum gram stain and culture plus bloodculture.
- PCR for specific viruses (e.g., SARS COV-2): in the right epidemiologic setting.
- Thoracentesis and pleural fluid analysis
  1. Do thoracentesis and pleural fluid analysis, if the size of pleural effusion is >2.5 cm in lateral decubitus position or obvious large (more than ½ of hemithorax), loculated, or with presence of pleural thickening
  2. Do pleural fluid: cell count with differential, gram stain and culture, chemistries (glucose, LDH, protein), if there is blood gas analyzer do PH,
- **Other laboratories**
  1. CBC with diff
  2. In patients with severe pneumonia: BUN/Cr, LFT, serum electrolytes, and blood glucose.

TREATMENT OBJECTIVES

- Achieve clinical cure
- Prevent complications and associated morbidity and mortality.

NON PHARMACOLOGIC

- Bed rest
- Frequent monitoring of all the vital signs in order to detect complications early and to monitor response to therapy, for all patients.
- Give attention to fluid and nutritional replacements as required.
- Administer Oxygen via nasal prongs or face mask (e.g., if saturation <94%)

EMPIRIC ANTIBIOTIC

- Evaluate the patient for the following two parameters before starting empiric antibiotic

  1. Look for risk factors include of MRSA (Methicillin Resistant *Staphylococcus aureus)* or P. *aeruginosa*
     - Recent hospitalization
     - Receipt of parenteral antibiotics in the last 90 days
     - Prior isolation of the organisms from the respiratory samples
  2. Comorbidities
     - Chronic heart, lung, liver, or renal diseases
     - Diabetes mellitus
     - Alcoholism
     - Malignancy
     - Asplenia

- The duration of antibiotic therapy is generally 5 to 7 days; however, it needs to be guided by clinical response as well. Improvement in vital sign abnormalities (HR, RR, BP, oxygen saturation, and temperature), ability to eat, and normal mentation are needed for hospitalized patients.
- The antibiotic should be continued until the patient achieves stability but for no less than a total of 5 days.

Table 8.37: Empiric antibiotic recommendations for outpatient management of CAP			
CAP categories	**Etiology**	**First line**	**Second line**
CAP :outpatient No-comorbidities AND No risk factors*	S.pneumoniae H.influenzae Atypicals	Amoxicillin X 5- 7 days	Doxycycline OR Clarithromycin/Azithromycin
CAP: out patient With comorbidities	Above + beta-lactamase-producing organisms catarrhalis	**Combination therapy** Amoxicillin-clavulanate AND Clarithromycin/ Azithromycin X 5-7 days	**Combination therapy** Cefuroxime or Cefpodoxime AND Clarithromycin/Azithromycin
CAP for hospitalized patients	Gram positive and negative and atypical microorganisms	Ceftriaxone OR cefotaxime IV + Clarithromycin or Azithromycin for 5 to 7 days	Amoxicillin-clavulanate + azithromycin or Clarithromycin
*Risk factors = risk factors for MRSA or P. *aeruginosa* infections (see the text above)			

Table 8.38. Empiric antibiotic recommendations for inpatient management of CAP

CAP category	First line	Second line	Recent Hospitalization and parenteral antibiotics use (in the last 90
CAP: requiring admission to wards Does not have severity signs requiring ICU admission (see table 2 above)	IV Third generation cephalosporin + Macrolide Ceftriaxone OR Cefotaxime PLUS Azithromycin/Clarithromycin	Levofloxacin or Moxifloxacin alone	Take culture and Add coverage for P. aeruginosa*
CAP requiring ICU admission (Severe CAP)	IV Third generation cephalosporin + Macrolide Ceftriaxone OR Cefotaxime PLUS Azithromycin/Clarithromycin	IV Third generation cephalosporin + Levofloxacin/ Moxifloxacin	Take culture and and Add coverage for MRSA and P. aeruginosa*
* Options for P.aeruginosa coverage: piperacillin-tazobactam , cefepime , ceftazidime , imipenem, Meropenem * MRSA coverage:Vancomycin			

Table 8.39: Adult dose recommendations for CAP		
Antimicrobial agent	**Adult dose**	**Comments**
Amoxicillin (A)	1000mg PO TID	First line for outpatient with no comorbidities
Amoxicillin-clavulanate (A)	625mg PO TID or 875 mg/125 mg BID, or 2,000 mg/125 mg BID	First line for outpatient with comorbidities
Benzyl penicillin (A)	2-3 million IU I.V. QID	Can be used as an alternative to amoxicillin
Ampicillin-sulbactam	1.5 to 3 g IV QID	Alternative to cephalosporin's in hospitalized patient, also consider for aspiration pneumonia if anaerobic coverage needed
Cefuroxime (Wa)	500mg PO BID	Alternative to Amoxicillin-clavulanate
Cefpodoxime (Wa)	200mg PO BID	Alternative to Amoxicillin-clavulanate
Ceftriaxone (Wa)	1-2 g IV 12 hourly	First line in severe cases with macrolides
Cefotaxime (Wa)	2 g IV 6 hourly	First line in severe cases with macrolides
Clarithromycin (Wa)	500mg PO BID	WHO recommend it over Azithromycin due to safety concerns
Azitromycin (Wa)	500mg PO, first day then 250mg PO, for 4 days	Associated with cardiovascular issues
Doxycycline (A)	100mg PO, BID	Second line for mild to moderate cases

Levofloxacin (Wa)	500-750 mg PO/IV BID	More side effect (including masking TB), thus only used when no alternative, e.g in sever penicillin allergy
Moxifloxacin (Wa)	400 mg PO BID	See above comment for levo-floxacin
Cloxacillin	500mg PO QID	Alternative add on for staph
Vancomycin (Wa)	1g IV BID	Reserved only for MRSA suspicion and used only after culture sample taken
Ceftazidime (Wa)	2g IV q6-8h	Reserved only for pseudomo-nas suspicion and used only after culture sample taken
Cefepime (Wa)	2 g IV 8-12 hourly	See above comments for ceftazidime
Piperacillin + tazobactam (Wa)	2g IV Q8hr	See above comments for ceftazidime
A = access group antibiotics *Wa = watch group antibiotics* *Re= reserve group antibiotics*		

PREVENTION

- Smoking cessation should be encouraged during the initial visit
- Other infection prevention measures

SPECIAL POPULATION CONSIDERATIONS

- Pregnant:Floroquinolones and tetracyclines (if used) are not recommended in pregnancy.
- Elderly: elderly patients are more likely to have an altered/unusual clinical presentation and increased risk of severe disease. Frequent monitoring schedules are recommended for this group ofpopulation.

2. HOSPITAL-ACQUIREDAND VENTILATOR-ASSOCIATED PNEUMONIA

DEFINITIONS

- Hospital-acquired (or healthcare associated) pneumonia (HAP) is pneumonia that occurs 48 hours or more after admission and did not appear to be incubating at the time of admission.
- Ventilator-associated pneumonia (VAP) is a type of HAP that develops more than 48 hours after endotracheal intubation.
- Healthcare associated pneumonia (HCAP):is no more used currently.
- HCAP is now classified under CAP, with risk factors MDR pathogens, particularly MRSA and *P aeruginosa*.

INVESTIGATION AND DIAGNOSIS

- Clinical diagnosis: Clinical diagnosis of HAP and VAP requires the following three parameters to be present.
  1. A new or progressive lung infiltrate on chest X-ray.
  2. Clinical evidence to indicate the lung infiltrate is of infectious origin: one or more of the followings
     - A new onset of fever
     - Purulent sputum
     - Leukocytosis/leukopenia
     - Decline in oxygenation.

- Time of presentation: after 48 hours of admission for HAP and after 48 hours of intubation forVAP.

- Identification of pathogen: Cultures from lower respiratory samples should be taken before initiation ofantibiotic.
- Risk factors for increased mortality in HAP
  1. Septic shock
  2. Requirement for mechanical ventilator support
- Risk factors for Multidrug resistant pathogens in HAP/VAP.
  1. Risk factors for MDRVAP:
     - Prior intravenous antibiotic use within 90 days
     - Septic shock at time ofVAP
     - ARDS precedingVAP
     - Five or more days of hospitalization prior to the occurrence of VAP
     - Acute dialysis needed prior toVAP onset
  2. Risk factors for MDR HAP
     - Prior intravenous antibiotic use within 90 days

EMPIRIC TREATMENT FOR COMMONLY SUSPECTED ETIOLOGIES OF HAP/VAP

- The empiric antibiotic choice for HAP and VAP: needs to consider *Pseudomonas aeruginosa*, other gram-negative bacilli, and *Staphylococcus aureus*.
- Since delayed and inappropriate therapy is associated with high mortality, early and aggressive treatment with early and aggressive de- escalation is an important composite for HAP/VAP (particularly for VAP) management
- If possible, hospitals need to regularly generate and disseminate a local antibiogram,tailored to their HAP population.
- Duration of therapy: a seven days course is generally adequate for both HAP and VAP. A shorter or longer duration can be considered based on clinical response.

Table 8.40: Empiric antibiotics for HAP (not VAP)

Population	Antibiotic choices	Remarks

HAP with one of the following
• Septic shock
• Requiring mechanical ventilation
• Received antibiotic in the last 90 days
	A combination of three antibiotics: one from of the following lists
	• One of the following
	  ○ Cefepime 2 g IV q8h
	  ○ Ceftazidime 2 g IV q8h
	  ○ Meropenem 1 g IV q8h
	  ○ Imipenem 500 mg IV q6hd
	  ○ Piperacillin-tazobactam 4.5 g IV q6h
	PLUS
	• One of the following
	  ○ Gentamicin 5–7 mg/kg IV q24h
	  ○ Ciprofloxacin 400 mg IV q8h
	PLUS
	• Vancomycin 15 mg/kg IV q8–12h (for severe cases, loading dose 25–30 mg/kg, maximum 3g)
		• Double antipseudomonal coverage + MRSA coverage
		• Antibiotics need to be deescalated based on culture and sensitivity results, if there is clinical improvement.
		• Doses need to be adjusted in patients with impaired renal function.

HAP with none of the following • Septic shock • Requiring mechanical ventilation • Received antibiotic in the last 90 day	A combination of two antibiotics • One of the following ○ Ceftazidime 2 g IV q8h ○ Meropenem 1 g IV q8h ○ Imipenem 500 mg IV q6hd ○ Piperacillin-tazobactam 4.5 g IV q6h **PLUS** ○ Vancomycin 15 mg/kg IV q8–12h (for severe cases, loading dose 25–30 mg/kg, maximum 3g)	If the proportion of the unit's *Staphylococcus aureus* isolates thus far has been known to be < 20%, the vancomycin can be omitted and one of the following can be used: ○ Piperacillin-tazobactam 4.5 g IV q6h ○ Cefepime 2 g IV q8h

Table 8.41: Empiric antibiotics for VAP

Population	Antibiotic choices	Remarks
VAP with one of the following risk factors • IV antibiotic use within the previous 90 days • Septic shock at the time ofVAP • ARDS preceding VAP • ≥5 days hospitalization prior to the occurrence of VAP • Dialysis prior to VAP onset	A combination of three antibiotics: one from of the following lists • One of the following ○ Cefepime 2 g IV q8h ○ Ceftazidime 2 g IV q8h ○ Meropenem 1 g IV q8h ○ Imipenem 500 mg IV q6hd ○ Piperacillin-tazobactam 4.5 g IV q6h **PLUS** • One of the following ○ Gentamicin 5–7 mg/kg IV q24h ○ Ciprofloxacin 400 mg IV q8h **PLUS** Vancomycin 15 mg/kg IV q8–12h (for severe cases, loading dose 25– 30 mg/kg, maximum 3g)	➢ Double antipseudomonal coverage + MRSA coverage • Antibiotics need to be deescalat- ed based on culture and sensitivity results, if there is clinical improvement. . • Doses need to be adjusted in patients with

VAP with none of the above risk factors	A combination of two antibiotics • One of the following ○ Ceftazidime 2 g IV q8h ○ Meropenem 1 g IV q8h ○ Imipenem 500 mg IV q6hd ○ Piperacillin-tazobactam 4.5 g IV q6h **PLUS** Vancomycin 15 mg/kg IV q8–12h (for severe cases, loading dose 25–30 mg/kg, maximum 3g)	If the proportion of the unit's *Staphylococcus aureus* isolates is< 10%, the vancomycin can be omitted and one of the following can be used: ○ Piperacillin tazobactam 4.5 g IV q6h ○ Cefepime 2 g IV q8h

CHAPTER 3: ASTHMA

1. BRONCHIAL ASTHMA

Definition

- Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation.
- It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable airflow limitation.

CLINICAL MANIFESTATION AND DIAGNOSIS

- The diagnosis of Asthma is made clinically using history and physical examination after excluding alternative diagnosis.
- Characteristic pattern of symptoms with wheezing, shortness of breath, cough, chest tightness varying over time and in intensity.
- Symptoms usually get worse during night or early morning and/or with known triggers (Eg, infection, allergy exposure, weather change, exercise, medications, occupational risk and emotional disturbances).
- Peak Expiratory Flow (PEF) can aid the diagnosis.
- Most asthmatics has family history of asthma and has history of wheezing starting from early child hood.

Table 14.1: Symptom based diagnosis of Asthma	
Increased probability that symptoms are due to asthma	*Decreased* probability that symptoms are due to asthma
• More than one type of symptom (wheeze, shortness of breath, cough, chest tightness) • Symptoms often worse at night or in the early morning • Symptoms vary over time and in intensity • Symptoms are triggered by viral infections, exercise, allergen exposure, changes in weather, laughter, irritants such as car exhaust fumes, smoke, or strong smells	• Isolated cough with no other respiratory symptoms • Chronic production of sputum • Shortness of breath associated with dizziness, light-headedness or peripheral tingling • Chest pain • Exercise-induced dyspnoea with noisy inspiration (stridor)

- Physical Examination helps in excluding alternative diagnosis. Normal finding doesn't exclude the possibility of asthma.
- Examine the nose, throat and upper airways for (nasal polyp, nasal congestion and/or blockage. Look also for features of Atopy or eczema on the skin.
- The most frequent finding is wheezing on auscultation, especially on forced expiration.
- Silent Chest on chest auscultation might signify severe asthma.
- Wheezing is also found in other conditions, for example: Respiratory infections (Viral), COPD, Upper airway dysfunction, Endobronchial obstruction
- The diagnosis of asthma in general hospital should be based on:

- A history of characteristic symptom patterns mentioned above with evidence of variable airflow limitation, from PEF, bronchodilator reversibility testing or response totreatment.
    - A 20% or more change in PEF values from morning to afternoon or from day to day or before and after bronchodilator therapy suggests a diagnosis of asthma or inadequately controlled asthma.
    - PEF values less than 200 L/min indicate severe airflow obstruction.
- A trial of glucocorticoids (e.g. 30 mg daily for 2 weeks) may be useful in establishing the diagnosis, by demonstrating an improvement in either PEF or symptoms.

TREATMENT

GOALS OFASTHMA MANAGEMENT

- The long-term goals of asthma management are:
    - Symptom control: to achieve good control of symptoms and maintain normal activity levels
    - Risk reduction: to minimize the risk of asthma-related death, exacerbations, persistent airflow limitation and medication side-effects
- Achieving these goals requires a partnership between patient and their health care providers
    - Ask the patient about their own goals regarding their asthma. Shared decision-making is associated with improvedoutcome.
    - Good communication strategies are essential.
    - Consider the health care system, medication availability, cultural and personal preferences and health literacy.

TREATMENT TO CONTROL SYMPTOMS AND MINIMIZE RISK

- Establish a patient-clinician partnership
- Train every patient in essential skills and guided asthma self-management including:

- Asthma information
- Inhaler skills
- Adherence
- Guided self-management education
- Written asthma action plan
- Self-monitoring
- Regular medical review.

- Follow the continuous control-based asthma management cycle:
  - Assess symptom control and risk factors
  - Adjust treatment (pharmacological and non-pharmacological)
  - Review the response: symptoms, exacerbations, side effects
- Approaches of Asthma Management
  - Medications: Every patient with asthma should have a reliever medication
  - Most adults and adolescents with asthma should have a controller medication to reduce the risk of serious exacerbations, even in patients with infrequent symptoms
  - Treating modifiable risk factors and comorbidities
  - Non-pharmacological therapies and strategies

INITIAL CONTROLLER TREATMENT

- Start controller treatment early;
  - For best outcomes, initiate controller treatment as early as possible after making the diagnosis of asthma because delayed initiation decreases lung function.
  - All adults and adolescents with asthma should receive ICS-containing controller treatment to reduce their risk of serious exacerbations and to

control symptoms.

- For safety, treatment of asthma in adults and adolescent with SABA alone is no longer recommended.

- For patients presenting with mild symptoms (asthma symptoms or need for reliever twice a month or more), treatment with regular low-dose ICS, with SABA is highly effective.
- Consider starting at a higher step (e.g., Medium dose ICS or Low dose ICS/LABA) if;
  - Patient has troublesome asthma symptoms on mostdays.
  - Waking from asthma once or more a week, especially if any risk factors for exacerbations.
- If initial asthma presentation is with Severely uncontrolled asthma or an exacerbation;
  - Give a short course of oral corticosteroids (OCS) and start regular controller treatment (e.g. high dose ICS or medium dose ICS/LABA, then step down after 3 months when well controlled).

INITIATION AND MONITORING OF CONTROLLER TREATMENT

- Before starting initial controllertreatment;
  - Record evidence for diagnosis of asthma, if possible.
  - Record symptom control and risk factors, including lung function.
  - Consider factors affecting choice of treatment for this patient.
  - Ensure that the patient can use the inhalercorrectly.
  - Schedule an appointment for a follow-upvisit.
- After starting initial controllertreatment;
  - Review response after 2-3 months, or according to clinical urgency.
  - Adjust treatment (including non-pharmacological treatments).
  - Consider stepping down when asthma has been well-controlled for 3 months.

Presenting symptoms	Asthma symptoms up to 2 times per month (Mild asthma), GINA step 1 & 2	Troublesome asthma symptoms most days (Moderate asthma) GINA step 3	Severe uncontrolled asthma GINA step 4 & 5
Treatment	Step GREEN(G) (Use 1 or more controller medication)	Step YELLOW (Y) (Use 2 or more controller medications)	Step RED (R) (Use 3 or more controller medications)
	Daily Low dose ICS with as-needed SABA	Medium dose ICS with as-needed SABA	High dose ICS with as-needed SABA (concomitantly use 1 or 2 of the add on therapy)
	Low dose ICS taken whenever SABA is taken	Low dose ICS-LABA as maintenance and reliever therapy with ICS-formoterol	Daily medium to high dose ICS-LABA, with as-needed SABA Add on tiotropium
	As-needed low dose ICS-formoterol (Budesonide-formoterol)	Low dose ICS-LABA, with as-needed SABA	Medium to high dose ICS-LABA with as-needed SABA Add on LTRA
	Daily LTRA, with as-needed SABA	Low dose ICS with daily LTRA, with as-needed SABA	Medium to high dose ICS-LABA with as-needed SABA Add on Theophylline sustained-release preparation
		Low dose ICS with daily Theophylline sustained-release preparation, with as-needed SABA	Medium to high dose ICS-LABA with as-needed SABA Any of the add on drugs Add on low dose OCS Add on azithromycin

Figure 14.1: Stepwise approach to asthma therapy according to the severity of asthma and ability to

control symptoms *(adopted from the Ethiopian Asthma and COPD management guideline, 2020)*

NOTES ON STEPWISE APPROACH TO ASTHMA THERAPY

- Provide guided self-management education
- Treat modifiable risk factors and comorbidities
- Advise about non-pharmacological therapies and strategies
- Consider stepping up if … uncontrolled symptoms, exacerbations or risks, but check diagnosis, inhaler technique and adherence first.
- Consider stepping down if … symptoms controlled for 3 months + low risk for exacerbations. Stopping ICS is not advised in adults with asthma because of risk of exacerbations

ASSESSINGASTHMA SEVERITY

- How do you assess Asthma severity?
  - Asthma severity is assessed retrospectively from the level of treatment required to control symptoms and exacerbations
- When do you assess Asthma Severity?
  - Assess asthma severity after patient has been on controller treatment for several months
  - Severity is not static it may change over months or years, or as different treatments become available
- Categories of asthma severity.
  - *Mild asthma:* well-controlled with GINA Steps 1 or 2 or step G (low dose ICS, with as-needed SABA)
  - *Moderate asthma:* well-controlled with GINA Step 3 or step Y (low- dose ICS/LABA)
  - *Severe asthma:* requires GINA Step 4/5 or step R (moderate or high dose ICS/LABA ± add-on), or remains uncontrolled despite this treatment. It may appear similar to asthma that is uncontrolled due to lack of treatment.

REVIEWING RESPONSE AND ADJUSTING TREATMENT

- How often should asthma be reviewed?
  - 1-3 months after treatment started,then every 3-12 months.
  - During pregnancy, every 4-6weeks.
  - After an exacerbation, within 1week.
- Stepping up asthma treatment;
  - Sustained step-up, for at least 2-3 months if asthma poorly controlled;
    - Important: first check for common causes (symptoms not due to asthma, incorrect inhaler technique, poor adherence, persistent environmental exposures and drugs, comorbidities that may contribute to respiratory symptoms).
  - Short-term step-up, for 1-2 weeks, e.g. with viral infection or allergen;
    - May be initiated by patient with written asthma action plan.
  - Day-to-day adjustment;
    - For patients prescribed low-dose ICS/formoterol maintenance and reliever regimen*
- Stepping down asthma treatment;
  - Consider step-down after good control maintained for 3months.
  - Find each patient's minimum effective dose that controls both symptoms and exacerbations.

TREATING MODIFIABLE RISK FACTORS

- Exacerbation risk can be minimized by optimizing asthma medications, and by identifying and treating modifiable risk factors.
- Provide skills and support for guided asthmaself-management
  - This comprises self-monitoring of symptoms and/or PEF, a written asthma action plan and regular medical review.

- Encourage adherence to medications and appointments
- Give asthma information

- Prescribe medications or regimen that minimizes exacerbations
  - ICS-containing controller medications reduce risk of exacerbations
  - For patients with ≥1 exacerbation in the last year, consider low-dose ICS/formoterol maintenance and reliever regimen*

- Encourage avoidance of tobacco smoke (active or environmental tobacco smoke (ETS))
  - Provide smoking cessation advice and resources at every visit

- For patients with severe asthma
  - Refer to a specialist center, if available, for consideration of add-on medications and/or sputum-guided treatment

- For patients with confirmed food allergy:
  - Appropriate food avoidance
  - Ensure availability of injectable epinephrine for anaphylaxis

NON-PHARMACOLOGICAL STRATEGIES AND INTERVENTIONS

- Reduce indoor air pollution by cooking outside or using smokeless cooking stoves

- Avoid allergens that the patient is sensitive to:
  - contact with furry animals (e.g. cats, dogs)
  - Reduce pollen exposure
  - Reduce exposure to house dust mite

- Avoidance of tobacco smoke exposure
  - Provide advice and resources at every visit; advise against exposure of children to ETS (house, car).

- Occupational asthma
    - Ask patients with adult-onset asthma about work history. Remove sensitizers and irritants like dust and fumes as soon as possible. Refer for expert advice, if available.
- Encourage Physical activity
    - Encouraged because of its general health benefits. Provide advice about managing exercise-induced bronchoconstriction.
- Avoid medications that may worsen asthma
    - Always ask about asthma before prescribing NSAIDs or beta-blockers.
- Remediation of dampness or mold in homes
    - Reduces asthma symptoms and medication use in adults.

PATIENTS WITH POOR ASTHMA CONTROL SHOULD BE ASSESSED FOR THE FOLLOWING:

- Reasons for poor adherence and misunderstanding the difference between relievers and controllers
- Poor inhaler technique
- Exposure to trigger factors at home and work
- Presence of gastro-esophageal acid reflux disease (GERD)
- Rhinitis and sinusitis
- Use of medications that may aggravate asthma such as aspirin, non-steroidal
- anti-inflammatory drugs and ß blockers
- Other medical conditions mimicking asthma symptoms (e.g. cardiac disease).

INDICATIONS FOR CONSIDERING REFERRAL

- Difficulty confirming the diagnosis of asthma
- Suspected occupational asthma

- Persistent uncontrolled asthma or frequent exacerbations
- Risk factors for asthma-related death
- Significant side-effects (or risk of side-effects)
- Symptoms suggesting complications or sub-types of asthma
- Asthma with confirmed food allergy.

ASTHMA EDUCATION

Goals of asthma education include:

- An explanation of the nature of asthma and its inflammatory basis
- A description of the different classes of drugs and their purpose in treatment (i.e. as-needed "relievers" and regular "controllers")
- Advice on prevention strategies (allergen, irritant, and tobacco smoke avoidance)
- The correct choice and use of inhalers and the opportunity to practice under supervision
- How to recognize worsening asthma and how and when to implement their action plan
- In some patients, particularly those requiring stabilization or patients who have had a recent exacerbation or deterioration, the use of a PEF meter and chart.

ASTHMA MEDICATIONS AND COMMON SIDE EFFECTS

- Asthma medications can be divided into two categories, quick-relief and long-term control medications
- Quick-relief medications that act principally by direct relaxation of bronchial smooth muscle, thereby promoting prompt reversal of acute airflow obstruction to relieve accompanying symptom.
    - Short acting beta agonists (SABA) are the main stay of treatment.
    - SABAs are the most effective bronchodilators during exacerbations and provide immediate relief of symptoms.

- Salbutamol (Albuterol) Inhaler is available in Ethiopia.
- Regularly scheduled use is not generally recommended.
- SABA alone therapy is no longer recommended. Common but benign side effects include tremor and tachycardia

- Long-term control (controller) medications that act primarily to attenuate airway inflammation and that are taken daily independent of symptoms to achieve and maintain control of persistent asthma.
- Steroids (ICS or OCS), long-acting beta agonists (LABA), and leukotriene modifiers comprise the important long-term control medications.
- LABAs provide bronchodilation for up to 12 hours after a single dose.
  - Salmeterol and formoterol are the LABAs available for asthma.
  - LABAs should not be used as monotherapy since they have no anti-inflammatory effect, use with inhaled corticosteroids.
  - They should not be used for symptom relief or exacerbations.
- Corticosteroids are the most potent and consistently effective anti-inflammatory agents currently available.
  - They decrease both acute and chronic inflammation, resulting in reduced symptoms and improved lung function.
  - These agents may also potentiate the action of beta-adrenergic agonists.
  - ICS (See tables 14.2 below) and OCS (prednisolone 5mg tabs) are used for asthma treatment.
  - Inhaled corticosteroids are preferred, first-line agents for all patients with persistent asthma.
  - Patients with persistent symptoms or asthma exacerbations who are not taking an inhaled corticosteroid should be started on one.
  - Inhaled corticosteroids have few side effects at standard treatment doses.
  - Some of the local side effects include oral candidiasis, dysphonia, reflex

cough and bronchospasm.

- High dose ICS and long-term use of oral steroids predisposes to systemic side effects which includes adrenal suppression, osteoporosis, skin thinning, easy bruising, diabetes, hypertension, infections, glaucoma and cataracts.

- Theophylline provides mild bronchodilation in asthmatic patients.
  - Theophylline also has anti-inflammatory and immunomodulatory properties, enhances mucociliary clearance, and strengthens diaphragmatic contractility.
  - Sustained-release theophylline preparations (e.g., theophedrine 120/11mg tablets 1-4 times per day) are effective in controlling nocturnal symptoms
  - As an added therapy in patients with moderate or severe persistent asthma whose symptoms are inadequately controlled by inhaled corticosteroids.
  - When added to an inhaled corticosteroid, theophylline may allow equivalent control at lower corticosteroid doses.
  - Theophylline use needs to be monitored closely owing to the medication's narrow therapeutic-toxic range, individual differences in metabolism, and the effects of many factors on drug absorption and metabolism.
  - At therapeutic doses, potential adverse effects include insomnia, aggravation of dyspepsia and gastroesophageal reflux, and urination difficulties in men with prostatic hyperplasia.
  - Dose-related toxicities include nausea, vomiting, tachyarrhythmias, headache, seizures, hyperglycemia, and hypokalemia.

- Leukotriene receptor antagonists (LTRA) are less effective than ICS particularly for exacerbations.
  - They may be appropriate for initial controller treatment for some patients who are unable or unwilling to use ICS; for patients who

experience intolerable side-effects from ICS; or for patients with concomitant allergic rhinitis.

- Before prescribing montelukast (adult dose 10 mg once daily), health professionals should counsel patients about the risk of neuropsychiatric events.
- Add-on tiotropium (long-acting muscarinic antagonist) in patients whose asthma is not well controlled with ICS-LABA.
- It (mostly 5 µg once daily by mist inhaler) modestly improves lung function and modestly increases the time to severe exacerbation requiring oral corticosteroids.
- Add-on azithromycin (three times a week) for adult patients with persistent symptomatic asthma despite moderate-high dose ICS and LABA reduced asthma exacerbations.
- Common side effects include diarrhea, ototoxicity and cardiac arrhythmia.
- Before considering add-on therapy with azithromycin, ECG should be checked for long QTc.

Table 14.2: Inhaled corticosteroids (ICS) and Combinations for Adults and adolescents (≥12 years) (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

Inhaled corti-costeroid	Total daily dose (mcg)		
	Low	Medium	High
Beclomethasone dipropi-onate (CFC)	200–500	500–1000	>1000
Beclomethasone dipropi-onate (HFA)	100–200	200–400	>400
Budesonide (DPI)	200–400	400–800	>800
Fluticasone propionate (DPI or HFA)	100–250	250–500	>500
Fluticasone/salmeterol (DPI)	100/50	250/50	500/50
Budesonide/formoter-ol*(HFApMDI)	80/4.5	160/4.5	320/9
Mometasone furoate (HFA-pMDI)	200-400		>400

NB: DPI-Dry Powder inhaler, MDI -Metered dose inhaler CFC-Chloroflourocarbon HFA-Hydroflouroalkane

*When Budesonide/formoterol is prescribed as maintenance and reliever therapy, the maximum recommended dose of formoterol in a single day is 72 mcg.

HOW TO USE INHALERS FOR ASTHMA MANAGEMENT

- An inhaler is a medical device used for delivering medication into the body via the lungs.
- It is mainly used in the treatment of asthma and chronic obstructive pulmonary disease.
- The two most common forms are: metered-dose inhaler; dry powder inhalers
- Some of the types of inhalers include: Auto halers (Breath Activated aerosol devices), Nebulizers mists and nasal inhalers
- Most patients (up to 80%) cannot use their inhaler correctly. This contributes to poor symptom control and exacerbations.
- To ensure effective inhaler use:
    - Choose the most appropriate device for the patient before prescribing
    - Check in haler technique at every opportunity
    - Correct using physical demonstrations
    - Paying attention to incorrect steps and
    - Confirm that you have checklists.

METERED-DOSE INHALER (MDIS)

- The medicine is in a small canister, inside a plastic case.When the inhaler is pressed, a measured dose of medicine comes through the mouthpiece.
- MDIs require good technique and coordination by pressing down on the inhaler and breathing in at the same time.

- Because using the inhaler correctly can be difficult, spacer devices are recommended for use with MDIs.
- The spacer is attached to the MDI to make it easier to use the inhaler and get more medicine into the lungs.

HOW TO USE METERED DOSE INHALER

- Remove the cap and check the mouthpiece is clean and free of objects.
- Shake the inhaler four or five times.
- Holding the inhaler upright with your thumb on the base, breathe out as far as comfortable
- Place the mouthpiece in your mouth closing your lips around it to form a good seal, do not bite.
- Start to breathe in slowly; press down firmly on the top of the canister to release a dose; while continuing to breathe in slowly and deeply.
- Removing the inhaler from your mouth; hold your breath for about 10 seconds, or as long as is comfortable.
- Breathe out gently away from your inhalermouthpiece
- For a second dose, wait approximately 30 seconds before repeating steps
- Replace the cap

Figure 14.2: Metered Dose Inhaler *(Adopted from the Ethiopian Bronchial asthma and COPD management guideline)*

DRY POWDER INHALER (DPI)

- Dry powder inhalers are handheld devices that deliver medication to the lungs and airways as you inhale through it.
- Examples of dry powder inhalers include:Turbuhaler;Accuhaler;Handihaler; Ellipta inhaler and Breezhaler.
- The common forms available in Ethiopia are Turbuhaler (eg.Symbicort) and Accuhaler (eg. Seritide)

Figure 14.3: Different forms of devices delivering DPIs (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

HOW TO USE ACCUHALER® (DRY POWDER INHALER-DPI)

- Check dose counter.
- Open cover (Use thumb grip)
- Hold the casing of the Accuhaler® in one hand while sliding the thumb grip away until a click is heard
- Holding your Accuhaler® with the mouthpiece towards you slide the lever away from you until a click is heard.This makes the dose available for inhalation and moves the dose counter on.
- Holding the inhaler horizontally, breathe out as far as comfortable
- Place the mouthpiece in your mouth; closing your lips around it to form a good seal - do not bite

- Breathe in as strongly and deeply as possible
- Removing the inhaler from your mouth; hold your breath for about 10 seconds, or as long as is comfortable
- Breathe out gently away from your inhalermouthpiece
- Toclose the Accuhaler®, slide the thumb grip back towards you as far as it will go until it clicks.

Figure 14.4: Dry Powder Inhaler (Accuhaler) (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

TURBUHALER (DPI)

- Since the turbuhaler is a breath-activated device, to use the turbuhaler properly, you must be able to breathe indeeply.
- Adults and children 7 years of age and older should be able to use the turbuhaler.

Figure 14.5: Dry Powder Inhaler (Turbuhaler) (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

HOW TO USE TURBUHALER (DPIS)

- Open by unscrew and remove the cap.
- Hold the turbuhaler upright
- Load the dose: twist the base anticlockwise and then back in the other direction until you hear a click. Your turbuhaler is now loaded with one dose of medicine
- Breathe out, away from the turbuhaler, do not blow directly into the turbuhaler
- Place the mouth piece in your mouth and form a seal with your lips and breathe in deeply.
- Remove the turbuhaler and hold your breath for up to 10 seconds.
- Toclose, replace the cap and twist until it is on properly.

- Cleaning and storing your turbuhaler: wipe the mouthpiece with a clean dry tissue.
- Do not wash or allow the mouthpiece it to get wet when cleaning.
- Keep the cap on when not in use.
- The device may clog if exhaled or dribbled into or if stored in an area of high humidity with the cap off or unsealed.

COMMON PROBLEMS WHEN USING A TURBUHALER

- To get the most benefit, it is important to use the correct technique.
- Here are a few commonproblems:
    - Not holding your turbuhaler upright (vertical) while loading the dose.
    - Covering the air inlets with your lips.
    - Breathing in through your nose instead of your mouth.
    - Shaking the inhaler to see how much is left.
    - Storing your turbuhaler in a damp place with the cap off.

HOW TO USE SPACERS

- If patient unable to use an inhaler correctly, add a spacer to increase drug delivery to the lungs, especially if using inhaled corticosteroids. This may also reduce the risk of oral candida.
- Clean the spacer before first use and every second week: remove the canister and wash spacer with soapy water. Allow it to drip dry. Avoid rinsing with water after each use.
- Spacers are not commonly available in Ethiopia so a plastic water bottle. See figure 14.6 below to modify a 500ml plastic bottle for use as an effective spacer

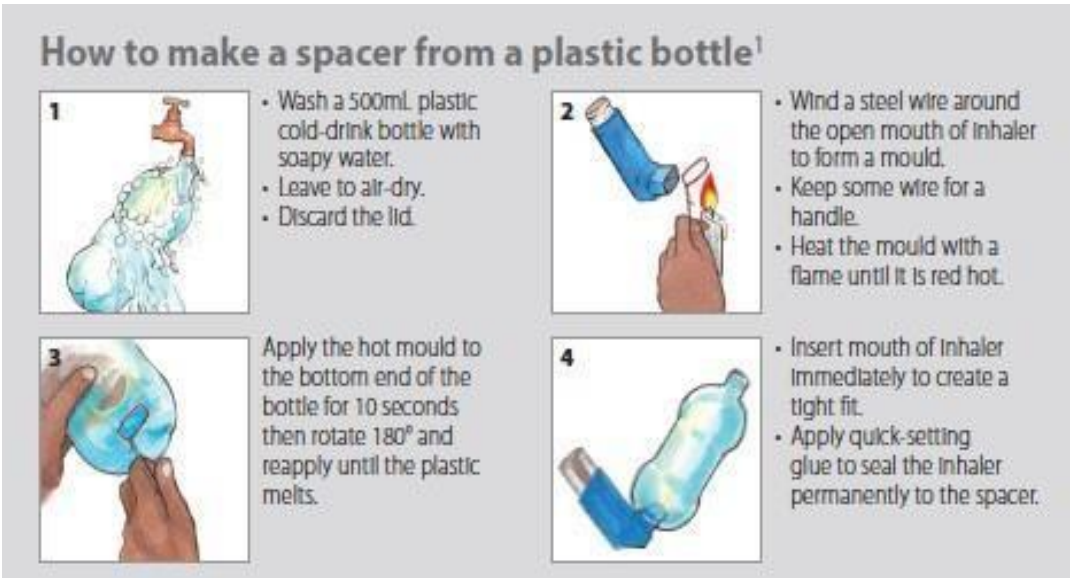


Figure 14.6: How to make a spacer from a plastic bottle (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

How to use a bottle spacer

- Use a modified 500ml plastic bottle in a similar way to a conventional spacer

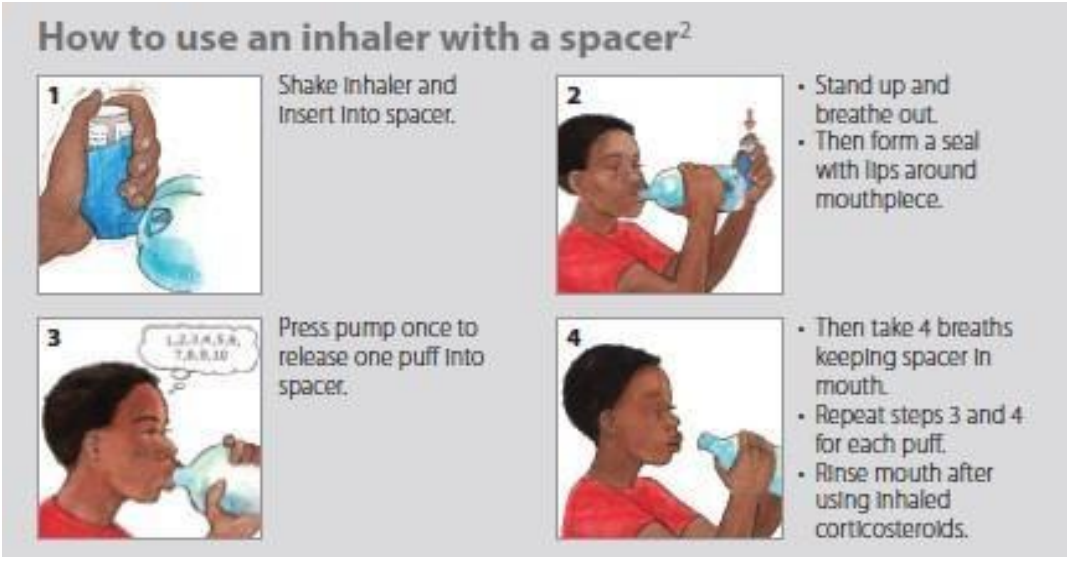


Figure 14.7: How to use an inhaler with a spacer (Adopted from the Ethiopian Bronchial asthma and COPD management guideline, 2020)

CHRONIC OBSTRUCTIVE PULMONARY DISEASES (COPD)

BRIEF DESCRIPTION

- Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease
- It is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

CLINICAL FEATURES

- Chronic and progressive dyspnea
- Cough
- Sputum production
- Wheezing and chest tightness
- Others – including fatigue, weight loss, anorexia, syncope, rib fractures, ankle swelling, depression, anxiety.
- Chest auscultation may demonstrate bilateral wheeze or crackles.

DIAGNOSIS

- The diagnosis of COPD is based on signs and symptoms and is confirmed by spirometry.
- Spirometry is required to make the diagnosis of COPD
- The presence of a post-bronchodilator FEV1/FVC < 0.70 confirms the presence of persistent airflow limitation.

- However, spirometry is often not available hence clinical criteria can be used to determine probability of COPD.
- COPD should be considered in any patient over the age of 40 years who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease.
- Other diagnostic tests can be employed to rule out concomitant disease or tailor additional treatment
    - CXR, CBC to exclude anemia or polycythemia
    - ECG and echocardiography in patients with signs of cor pulmonale
- Pulse oximetry at rest, with exertion, and during sleep should be performed to evaluate for hypoxemia and the need for supplemental oxygen.

MANAGEMENT OF COPD

An effective COPD management plan includes four components:

- Assess and monitor disease
- Reduce risk factors
- Manage stable COPD
- Manage exacerbations

The goals of effective COPD management are to:

- Prevent disease progression
- Relieve symptoms
- Improve exercisetolerance
- Improve health status
- Prevent and treat complications
- Prevent and treat exacerbations
- Reduce mortality

MANAGEMENT OF STABLE COPD

- The management of stable COPD focuses on improving breathlessness, reducing the frequency and the severity of exacerbations, and improving health status and prognosis.
- It includes avoidance of modifiable risk factors, vaccinations, pharmacologic therapies, oxygen therapy and pulmonary rehabilitation.
- Avoidance of modifiable risk factors: smoking cessation and reduction of indoor air pollution.
    - Smoking cessation: This is a known to affect the natural course of COPD and should be advised to all patients irrespective of the level of symptom control or severity of disease. For details on smoking cessation, see the session under "COPD Prevention" below.
    - Reduction of indoor air pollution through introduction of non-smoking cooking devices or alternative fuels should be encouraged.
- Vaccinations:
    - Influenza vaccination reduces serious illness and death.
    - It is recommended for all patients with COPD.
    - Pneumococcal vaccination (PCV13 and PPSV23) is recommended for all patients ≥65.
    - The PPSV23 is also recommended in younger patients with significant comorbidity including chronic lung or heart diseases.
- Pharmacological therapies: These are used to reduced symptoms, reduce the frequency and severity of exacerbations, and improve exercise tolerance and health status.
    - Beta-2 agonists:
        - Short-acting bronchodilators (SABA) like Salbutamol may be used for patients with mild disease
        - Longer-acting bronchodilators (LABA) like Salmeterol and

Formoterol inhalers are usually more appropriate for those with moderate to severe disease.

- Regular and as needed SABAs improves symptoms and FEV1.
- The effect of SABAs usually wears off in 4 – 6 hours whereas those LABAs have duration of action up to 12 hours.
- SABAs can also be used as needed (in between doses of LABAs). Important side effects include tachycardia and somatic tremor.

- Antimuscarinic (Anticholinergic) drugs:
  - These include the Short Acting Muscarinic Antagonist (SAMA) like Ipratropium and the Long-Acting Muscarinic Antagonist (LAMA) like Titropium.
  - Ipratropium has a duration of action that is between 6 – 8 hours whereas Tiotropium lasts for up to 24 hours.
  - LAMAs generally improve symptoms and health status, and reduce exacerbations and related hospitalizations.
  - Dryness of the mouth is the most important side effect of these drugs.
- Oral bronchodilator therapy:
  - Methylxanthines (theophedrine) may be contemplated in patients who cannot use inhaled devices efficiently.
  - Side effects include palpitations caused by atrial and ventricular arrhythmias, grand mal convulsions, headaches, insomnia, nausea, and heartburn.
- Combined inhaled glucocorticoids and bronchodilators:
  - The fixed combination of an inhaled glucocorticoid and a LABA (e.g. fluticasone with salmeterol, budesonide with formoterol)
  - It improves lung function, reduces the frequency and severity of exacerbations and improves quality of life especially in patients with moderate to very severe COPD and exacerbations.

- These advantages may be accompanied by an increased risk of pneumonia, particularly in the elderly.
- Use this combination especially when there is history of hospitalization for exacerbation, two or more exacerbations per year, blood eosinophils >300/micL or history of (concomitant) asthma.
- Because there is no loose preparation of LABAs in the market, we generally tend to use combination of LABA/ICS.

- Oral glucocorticoids:
  - Oral glucocorticoids are useful during exacerbations but maintenance therapy contributes to osteoporosis and impaired skeletal muscle function, and should be avoided.
  - Oral glucocorticoid trials assist in the diagnosis of asthma but do not predict response to inhaled glucocorticoids in COPD.

- Pulmonary rehabilitation:
  - Exercise should be encouraged at all stages and patients reassured that breathlessness, while distressing, is not dangerous.
  - Physical training, disease education and nutritional counseling reduce symptoms, improve health status and enhance confidence.
- Oxygen:
  - The long-term administration of oxygen (>15hrs/day) to patients with severe resting hypoxemia increases survival.
  - It is indicated for patients with a PaO2 <55mmHg or SaO2 <88% confirmed twice over three-weeks period
  - It is also indicated for patients with PaO2 55 – 60mmHg or SaO2 <88% if there is evidence of pulmonary hypertension, peripheral edema suggesting congestive heart failure or polycythemia (Hct >55%).

MANAGEMENT OF EXACERBATION OF COPD

Most COPD patients in our country are undiagnosed, a few of them are told they have "bronchitis" without confirmatory spirometry and still fewer have confirmed COPD.

- Therefore, COPD with acute exacerbation should be considered in every patient presenting with a recent worsening of his/her long standing cough or dyspnea or sputum color change (purulence).
- COPD exacerbations can be classified into 4 categories based on severity
    - Mild: can be managed at home with SABAs only.
    - Moderate: can be managed as outpatient with SABAs + antibiotics+ steroids.
    - Severe without respiratory failure: can be treated in the wards with SABAs + antibiotics+ steroids.
    - Severe with respiratory failure: Needs ICU admission for respiratory support (Noninvasive or invasive ventilation).

PHARMACOLOGIC MANAGEMENT INCLUDES:

- Bronchodilators
    - Short acting bronchodilators:
        - Metered dose inhaler (MDI)= Salbutamol inhaler: 2-3 puffs every hour and then tapered to 2 puffs every 4hrs.
        - Nebulization of salbutamol or combined salbutamol/ipratropium bromide solution.
    - Long-acting bronchodilators + ICS: should be continued if patient was using them and should be started at discharge if they were not being used.
- Corticosteroids
    - Only used when having a significant exacerbation (moderate or severe disease), as they may lead to development of pneumonia and sepsis
    - Can be given orally (prednisolone 40mg) or IV (hydrocortisone or methylprednisolone)

ORAL AND IV ROUTES ARE EQUALLY EFFECTIVE

- Recommended for 5-7days only

- Antibiotics
  - Recommended for moderate to severe illness or when the sputum is purulent.
  - Antibiotics are usually given for 5-7days
  - The specific antibiotic given should depend on the sensitivity pattern of the hospital.
  - Commonly Amoxiciline/clavulanic acid, cefalosporins, quinolones or macrolids can be used.
  - Sputum culture is generally not helpful except in few conditions which may be associated with Gram negative infections like pseudomonas earoginosa.
    - Patient has recurrent exacerbation.
    - Patient is on invasive mechanical ventilation.

- Oxygentherapy
  - Delivers a flow rate of <15L/min.
  - Source of oxygen can be a cylinder (>99% pure oxygen) or oxygen concentrator (90% oxygen,10% nitrogen).
  - Titrated to achieve a saturation of oxygen of 88-90% to avoid oxygen induced hypercapnia.

REFERRAL

- Patients who need further therapy and optimization
- Those requiring
  - High flow oxygen
  - Non-invasive ventilation: BIPAP devices
  - Invasive Mechanical ventilation

Pregnant women who cannot avoid travel to a malarious area during pregnancy

- Mefloquine is relatively safe and can be used for prophylaxis
- Avoid doxycycline.
- Safety data on atovaquone-proguanil is limited.

A. Pediatrics:

- For infants < 5 kg of body weight and with uncomplicated *P. falciparum* malaria: Give Arthemete Lumfantrene (AL) as for children weighing 5 kg.
- Chloroquine is a safe drug that can be used in all children with only P. vivax infection.

CHAPTER 4

INTESTINAL HELMINTHIC INFESTATIONS

BRIEF DESCRIPTION

- These are infestation caused by intestinal worms (nematodes and cestodes), which are commonly associated with poor personal and environmental hygiene.
- Although they may not be fatal, they contribute to malnutrition and diminished work capacity.

CLINICAL FEATURES

- Include abdominal cramps, nausea, bloating, anorexia
- Anemia
- Perianal itching
- Passage of adult worms

INVESTIGATIONS

- Mainly by direct stool microscopy:A single stool microscopy may not be diagnostic.A repeated stool microscopy may be needed.

TREATMENT

OBJECTIVES

- Reduce symptoms
- Break the cycle of transmission

Pharmacologic (See table below)

Table 8.55: Treatment of common intestinal helminthic parasitic infestations

Name of infestation Etiology Mode of transmission	Treatment	Remark
Ascariasis ***Ascaris lambricoids*** Ingestion of the larvae of the parasite together with food	**First line-options** **Albendazole,** 400mg P.O. as a single dose, for children: 1 – 2 years, 200mg as a single dose. **Mebendazole,** 100mg P.O.BID for 3 days or 500mg, once **Alternative (pregnant women)** **Pyrantel pamoate**, 700mg P.O. as a single dose	Presence of migrating larvae in the lungs can provoke pneumonia

Enterobiasis ***Enterobius Vermicularis*** Ingestion of the eggs of the parasite together with food	**First line-options** **Mebendazole,** 100mg P.O. BID for 3 days, repeat in two weeks OR **Albendazole,** 400mg P.O. as a single dose, repeat in two weeks, Alternative **Piperazine,** 4g in a single dose. • Simultaneous treatment of the entire household is warranted due to high transmission possibilities	Common in children and auto infection may occur
Hookworm infestation *Necator americanus or Ancylostoma duodenale* Penetration of the larvae of the parasite through skin	**First line-options** **Albendazole,** 400mg P.O. as a single dose (**preferred**) OR **Mebendazole,** 100mg P.O. BID for 3 days or 500mg stat **Alternatives:** **Pyrantel pamoate**, 700mg P.O. as a single dose	

Strongyloidiasis ***Strongloidexs stercolaries*** Penetration of the larvae of the parasite through skin	**First line** **Ivermectin,** 200mcg/kg daily for 2 days. For disseminated strongyloidiasis, treatment with ivermectin should be extended for at least 5–7 days or until the parasites are eradicated. **Alternatives-**options **Albendazol**e 400mg P.O.BID for three consecutive days (less effective than ivermctin). OR **Thiabendazole,** 1500mg, P.O. BID, for children: 25mg/kg p.o. for three consecutive days (comparable efficacy to ivermectin).	Larvae migrate to the lungs where they cause tissue destruction and bleeding. Treat concomitant anemia if any
Trichuriasis ***T.tricura***	**First line-options** **Mebendazole**, 500mg P.O., single dose **(preferred over Albendazole)** OR **Albendazole,** 400mg, P.O. for three days oxantel pamoate 15 to 30 mg/kg (if available) plus albendazole 400 mg on consecutive days is superior than other therapies	Heavy infestation leads to bloody diar- rhea, bleeding & weakness

Taeniasis ***T.saginata*** ***T.solium***	**First line-Intestinal infestation** **Praziquantel** P.O. 600mg or 10mg/Kg, single dose **Alternative** **Niclosamide,** 2g in a single dose P.O. **Treatment of neurocysticercosis** **Albendazole** P.O. 15mg/kg per day for 8- 28 days **or** **Praziquantel** 50–100mg/kg daily in three divided doses for 15–30 days**.** Longer courses are often needed in patients with multiple subarachnoid cysticerci **PLUS** -High-dose glucocorticoids -Anti epileptics (if there is seizure)	*T. solium* (pork tapeworm) may cause fatal cysticercosis
	First line	
Hymenolepis nana	**Praziquantel**, 25mg/kg or 1800mg P.O. single dose, followed by repeat dose 10 days later	
	Alternatives	
	Niclosamide, 2g P.O. on the first	

	day followed by 1g QD for 6 days	

CHAPTER 5

TUBERCULOSIS

DRUG SUSCEPTIBLE TUBERCULOSIS BRIEF DESCRIPTION

- Tuberculosisis is a chronic bacterial infection caused by a group of bacteria, *Mycobacterium tuberculosis complex,* the most common of which is Mycobacterium tuberculosis. Less frequently, it can be caused by *Mycobacterium bovis* and *Mycobacterium africanum.*
- Tuberculosis usually affects the lungs in which case it is called pulmonary TB.
- In addition to the lungs, any part of the body can be affected with this bacterium and in this case, it is called extra-pulmonary TB.
- Common extra-pulmonary sites affected include the lymph nodes, pleura, spine, urinary tract, the brain, joints, bone and abdomen.
- HIV infection is one of the most important risk factors for the development of active tuberculosis.

CLINICAL FEATURES

- The clinical features of tuberculosis depend on the specific organ affected.
- The clinical features can be grouped: general (non-specific) and organ specific

- The general symptoms of TB (pulmonary or extra-pulmonary):
  - Weight loss, fever, night sweats, loss of appetite, fatigue, malaise
  - Malnourished and chronically sick appearance
- Organ specific
  - Pulmonary tuberculosis
    - Cough that lasts for more than 2 weeks with or without sputum production
    - Chest pain
    - Hemoptysis
    - Shortness of breath
  - Tuberculous lymphadenitis
    - Slowly growing painless lymph nodeenlargement
    - Initially firm and discrete, later become matted andfluctuant.
    - Formation of abscesses and discharging sinuses, which heal with scarring.
  - Tuberculous pleurisy
    - Pleuritic chest pain (pain whilebreathing/coughing/sneezing)
    - Intermittent cough
    - Shortness of breath
    - Signs of pleural effusion (dullness, decreased/absent air entry and decreased tactile fremitus)
  - TB of bones and/or joints
    - Localized pain and/or swelling +/- discharge,stiffness of joints
    - Spine (TB spondylitis): localized swelling over the back(gibbus), back pain paralysis (weakness of the lower extremities)

- Abdominal TB
  - Chronic non-specific abdominal pain with diarrhea or constipation,
  - Fluid in the abdominal cavity (ascites).
  - Mass (inflammatory mass) in the abdomen
- Tuberculous meningitis
  - Headache, fever, vomiting: insidious onset
  - Neck stiffness, impaired level of consciousness.
- Tuberculous pericarditis
  - Chest pain (pleuritic)
  - Shortness of breath
  - Pericardial friction rub or distant heart sounds

INVESTIGATIONS AND DIAGNOSIS

- The diagnosis of tuberculosis requires clinical suspicion and microbiologic identification of the bacilli.
- Microbiologic tests to identify the bacilli
  - **Sputum direct m icroscopy**: Ziehl Nielsen (acid fast bacilli) (**AFB**) staining
    - Three sputum specimens (SPOT-EARLY MORNING-SPOT) need to be collected and examined in two consecutive days
    - Result must be available on the second day.
  - **Gene Xpert MTB/RIF**
    - A fully automated DNA/molecular diagnostic test to detect TB and Rifampicin resistance simultaneously.
    - It is recommended as the initial diagnostic test for all persons being evaluated forTB.

Sputum culture and drug susceptibility

- Culture is the gold standard
- It takes weeks to get the results.
- If sputum AFB and/or Gene Xpert are negative and there is a strong suspicion, sputum culture can be sent to a referral laboratory. However, treatment for an alternative diagnosis or "clinical TB" should not be delayed.

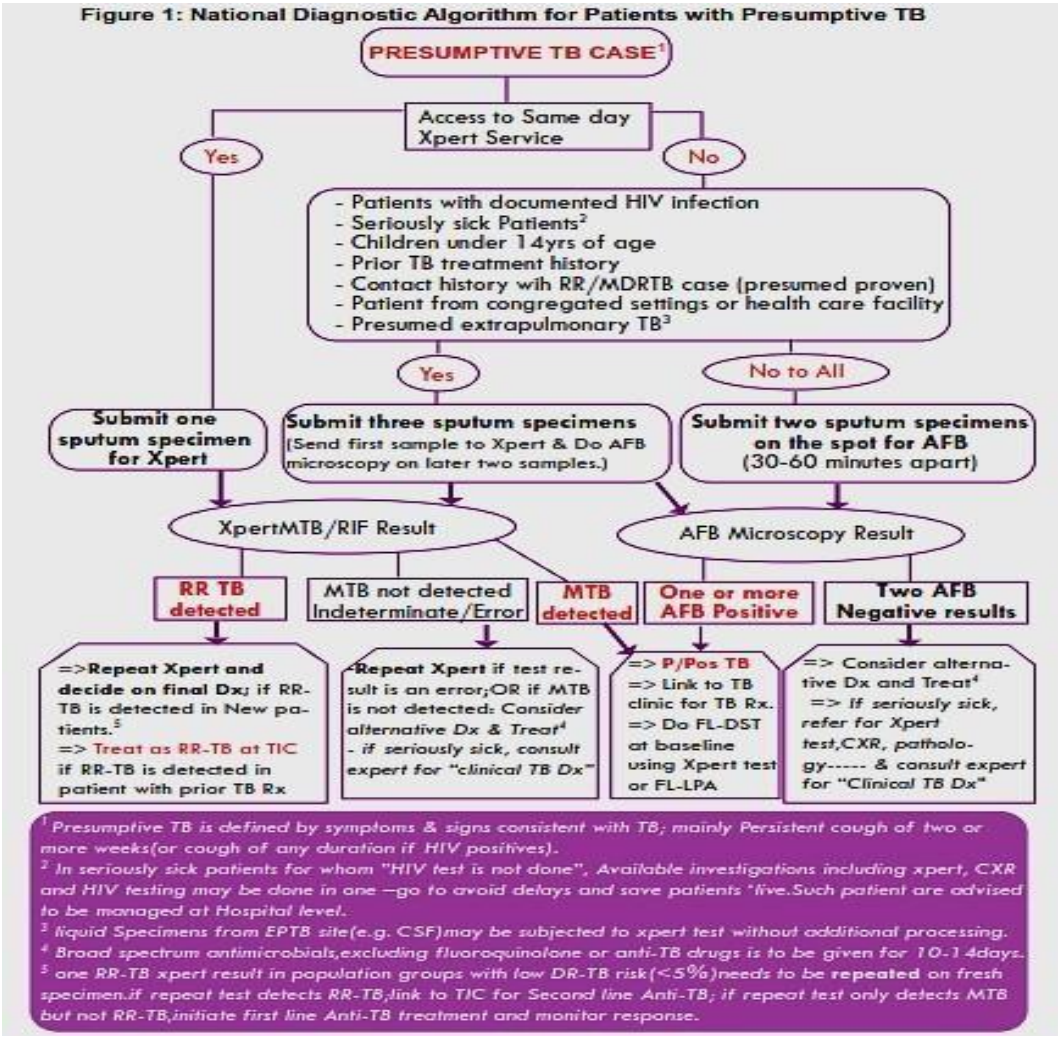
- **Line Probe Assay(LPA)**
  - A test to identify the presence of specific mutations on the genes of TB bacilli which are responsible for Isoniazid and Rifampicin resistance.
  - It is a rapid and accurate test to identify cases with MDR-TB.
  - The test may be available in referral laboratory for patients suspected of MDR-TB.

- Body fluid analysis and identification of pathogen
  - Pleural fluid, ascitic fluid, CSF
    - Cell count with differential
    - Protein, glucose
    - LDH
    - Gene Xpert
    - Cytology (cytopathology)
- Fine needle aspiration and histopathologic examination: enlarged lymph nodes
- Tissue biopsy and histopathology: pleural, skin, endometrium, bronchial, colon/cecum, liver
- Imaging:

- Chest X-ray.
- Depending on the suspected extra pulmonary sites other imaging modalities may be needed: abdominal ultrasound, brain/spinal MRI, Joint/bone X-ray, image guided FNA orbiopsy

- Other investigation: HIV test, CBC, ESR, CSF analysis

Figure 1: National Diagnostic Algorithm for Patients with Presumptive TB



Case definitions

- **A case of tuberculosis**: is a definite case of TB or a patient whom an experienced health worker has diagnosed TB and has decided to treat the patient with a full course of TB treatment.
- **A definite/proven case of tuberculosis** is a patient with either of the following
  - Two sputum smears (one sputum positive is enough for HIV pos- itive patients)

    OR
  - Sputum culture positive for Mycobacterium tuberculosis.

    OR
  - Mycobacterium tuberculosis is identified from a clinical specimen, either by culture or by a newer molecular technique.

TREATMENT OF DRUG SUSCEPTIBLE TB

Objectives

- Cure
- Prevent death from activeTB or its late complications
- Restore quality of life and productivity
- Decrease transmission
- Prevent relapse
- Prevent the development and transmission of medicine resistance

Non pharmacologic

- Counseling: adherence, the nature of treatment, contact screening
- Good nutrition
- Adequate rest

- Admission for severely ill patients e.g., Tb meningitis, pericarditis

Pharmacologic

- Standardized combination treatment: all patients in a defined group receive the same treatment regimen.
- A combination of 4 or more anti-TB medicines.
- Directly observed treatment (DOT)

First line anti-TB Medicines

- The first line anti-TB medicines available for TB treatment in Ethiopia are:
  - **Rifampicin (R):** the most bactericidal and potent sterilizing agent
  - **Isoniazid (H):** highly bactericidal especially in the first few days
  - **Pyrazinamide (Z):** only active in acidic environment and bacilli inside macrophages
  - **Ethambutol (E):** bacteriostatic and effective to prevent drug resistance when administered with other potent drugs

Table 8.64: The essential anti-TB drugs and their dose recommendations

Recommended adult dose and children ≥ 25 kg body weight		First line TB drugs	Recommended pediatric dose	
Daily dose (mg/kg weight)	Maximum (mg)		Daily dose (mg/kg weight)	Maximum (mg)
5 (4-6)	300	**Isoniazid (H)**	10 (7–15)	300
10 (8-12)	600	**Rifampicin (R)**	15 (10–20)	600
25 (20-30)	2,000	**Pyrazinamide (Z)**	35 (30–40)	-
15 (15-20)	1,600	**Ethambutol (E)**	20 (15–25)	-

- **Standardized first line treatment regimen for new drug susceptible (presumed to be drug susceptible) tuberculosis**
  - New pulmonary TB (PTB) patients presumed or known to have drug-susceptible TB
  - New extrapulmonary TB (EPTB) patients
    - Standardized regimen: 6 months total (2 months intensive and 4 months continuation phase) 2RHZE/4RH
    - Intensive phase: 2 months Rifampicin, Isoniazid, Pyrazinamide & Ethambutol (2RHZE)
    - Continuation:4 months Rifampicin and Isoniazid (4RH)

Table 8.65: First line TB treatment adult and pediatric dosing chart using body weight bands

Adult and pediatric ≥25 kg weight			**Pediatrics**			
Patient weight band (kg)	**Regimen and dose in two phases**		**Daily dose (mg/kg weight)**	**Regimen and dose in two phases**		
	Intensive: 2(RHZE)	Contin-uation: 4(RH)		Intensive:2(RHZE)		Contin-uation: 4(RH)
				RHZ75/ 50/150	**E 100**	**RH75/ 50**
20-29	1 ½	1 ½	4-7kg	1	1	1
30-39	2	2	8-11kg	2	2	2
40-54	3	3	12-15kg	3	3	3
≥55	4	4	16-24kg	4	4	4

- **Previously treated TB patients presumed or known to have drug-susceptible TB**
  - In all previously treated TB patients who require re-treatment, specimen for rapid molecular-based drug susceptibility testing for first line TB

drugs should be obtained at or before the start of treatment.

- While awaiting the result, the standard first line treatment regimen is recommended: 2(RHZE)/4(RH)
- A "retreatment regimen" with addition of streptomycin **is not recommended**.

- **Patients who presented with is for patients activeTB after known contact with a patient documented to have drug-resistant TB**
  - Sample should be sent for rapid Drug Susceptibility Test (DST)
  - Treatment should be decided based on rapid DST result.
  - While awaiting DST result, the patient may be initiated treatment with the regimen based on the DST of the presumed source case.
- **Extended continuation phase**
  - The following extrapulmonary forms of Tb require prolonged continuation phase
    - CNS (TB meningitis or Tuberculoma)
    - Bone or joint TB (Vertebral (TB spondylitis), joint and osteomyelitis), which require prolongation of the continuation phase for
  - Regimen (a total of 12 months: 2months intensive phase and 10 months continuation phase);2RHZE/10RH
- **Adjuvant corticosteroid therapy**
  - Adjuvant corticosteroid therapy, dexamethasone or prednisolone tapered over 6-8weeks should be used for patients with the following two extrapulmonary forms
    - TB meningitis
    - TB pericarditis

Table 8.66: Prednisone dose for adult TB patients with TB pericarditis

Weeks of treatment	Prednisolone dosage
1-4	60mg/day
5-8	30mg/day
9-10	15mg/day
11 week	5mg/day (then discontinue at end of 11th week)

- **Corticosteroid for TB meningitis**
  - Dexamethasone 12 mg/day OR Prednisolone 60mg/day for 3 weeks and then decreased gradually during the subsequent 3 weeks.

MONITORING OF PATIENTS ON TREATMENT

- **Clinical monitoring**: during scheduled visit, a patient receiving anti-TB treatment should be checked for the following
  - Persistence or reappearance of clinical feature of TB
  - Weight monitoring: weight is a useful indicator of clinical improvement especially in children and should be monitored monthly.
  - Occurrence of Adverse drug reaction
  - Development of TB complications.
  - Adherence: by reviewing the "treatment supporter card" or UNIT TB register
  - Risk for drug resistance, and need for drug susceptibility testing screening
    - Unsatisfactory response to treatment beyond two months of treatment should alarm the possibility of drug resistance or alternative diagnoses.

- **Bacteriologic monitoring for initially bacteriologically confirmed pulmonary TB**
  - Sputum AFB (direct microscopy) should be done at end of 2nd, 5th and 6th month of therapy.
  - Molecular technique like Gene Xpert MTB/RIF cannot be used to monitor treatment as the technique may give false positive result by identifying dead bacilli.
  - If AFB positive at the end of second month: send sputum sample for Xpert and line probe assay (LPA) for DST.
    - If at least Rifampicin sensitive:continue to the continuation phase.
    - If Rifampicin resistance: Mark as Rifampicin-resistant Tb (RR- TB) and the outcome is labelled as "MDR TB". Treatment will be started as MDR-TB.
  - If AFB is positive at the end of 5th or 6^{th} month: the outcome is treatment failure. DST testing and treatment will proceed as MDR-TB suspect.
- **Management of adverse reaction to First line Anti-TB drugs**
  - Health workers should regularly monitor for occurrence of side effects to theAnti-TB drugs administered to the patient.

Table 8.67: Adverse reactions and drug interactions of first line TB medicines

Medicine	Adverse reaction	Drug interactions
Isoniazid (H)	Skin rash, Sleepiness and lethargy, Peripheral neuropathy (paresthesia, numbness and limb pain), Hepatitis. Rare: Convulsions, pellagra, arthralgia, anemia, lupoid reactions	Al(OH)3 decreases its absorption INH inhibits metabolism of phenytoin, diazepam carbamazepine and warfarin hence increases the serum concentrations
Rifampicin (R)	Gastrointestinal reactions (abdominal pain, nausea, vomiting), Hepatitis, Generalized cutaneous reactions,Thrombocytopenic purpura. Rare**:** Osteomalacia, pseudomembranous colitis, acute renal failure, shock, hemolytic anemia	Increase metabolism of warfarin, corticosteroids, antifungal agents, protease inhibitors, non- nucleoside reverse transcriptase inhibitors, oral hypoglycemic agents, oral contraceptives hence reduces serum levels of these medicines
Ethambotol (E)	Retrobulbar/Optic neuritis, (impairment of vision, red–green blindness, blurring) Rare: Generalized cutaneous reactions, arthralgia, peripheral neuropathy	
Pyrazinamide (Z)	Arthralgia, Hepatitis; Rare: Gastrointestinal reactions, cutaneous reactions, sideroblastic anaemia	

Table 8.68: Symptom based approach to the management of An- ti-TB medicines induced adverse effects

	Adverse-effects	**Responsible Medicines**	**Management**
Minor (Continue Anti-TB medi-cine/s)	Anorexia, nausea, abdominal pain	Rifampicin; Pyrazin-amide	Give tablets with small meals or before bed-time
	Joint pains	Pyrazinamide	NSAIDs
	Burning sensation in feet	Isoniazid	Pyridoxine 100mg daily
	Orange/red urine	Rifampicin	Reassurance
Major (Stop the respon-sible medi-cine/s)	Itching, skin reac-tion	Rifampicin or Isoniazid	Stop and replace with ethambutol; Stop, then reintroduce with de-sensitization
	Jaundice; hepatitis	Most anti-TB medicines (R, H,Z)	Stop all anti-TB medi-cines and refer
	Vomiting and con-fusion	Most anti-TB medicines	Stop all anti-TB medi-cines and refer
	Visual impairment	Ethambutol	Stop Ethambutol and refer
	Shock, purpura and acute renal failure	Rifampicin	Stop Rifampicin and refer

- **Treatment of patients with renal failure:**
  - In patients with estimated GFR <30ml/min or those on dialysis the dose of Ethambutol and Pyrazinamide need to be reduced from daily to three times per week.
  - Rifampicin and Isoniazid do not need adjustment in patients with renal failure.
  - To reduce Ethambutol and Pyrazinamide without reducing Rifampicin and Isoniazide make using the available fixed drug combinations, use the following regimen.
    - Intensive phase: RHZE three days per week and RH the remaining four days(for 2 months)
    - Continuation phase: RH daily
- **Treatment of patients with (previously known) liver disorder (e.g. hepatitis, cirrhosis):**
  - Most anti-TB medicines can cause liver damage.
  - Do not give Pyrazinamide.

- Isoniazid & Rifampicin plus one or two non- hepatotoxic medicines (Ethambutol and/or Streptomycin) can be used, for a total duration of eight months: 2SERH/6RH
- If the patient has severe liver damage or jaundiced: Streptomycin plus Isoniazid plus Ethambutol in the initial phase followed by Isoniazid & Ethambutol a total duration of 12 months: **2 SHE /10 HE**.

- **Treatment during pregnancy and breast-feeding**
  - Avoid Streptomycin because of the risk of toxic effects on the fetus.
  - Other medicines should not be discontinued during pregnancy or breast-feeding.
  - When a breast-feeding mother has PTB, the infant should, regardless of prior vaccination with BCG, be given chemo-prophylaxis and get BCG vaccination, if not previously vaccinated.
- **Treatment of patients also infected with HIV**
  - All patients with HIV and active TB who are not on ART should be started on ART as described below:
    - CD4 <50 cells/mm3: Initiate ART within 2 weeks of starting TB treatment
    - CD4 counts ≥50 cells/mm: Initiate ART within 8 weeks of starting TB treatment.
    - During pregnancy, regardless of CD4 count: Initiate ART as early as feasible, for to prevent HIV transmission to the infant (AIII).
    - With **tuber**culous meningitis: Initiate ART after 8 weeks of TB treatment.
  - Rifampicin can interact certain ARVS, particularly, some protease inhibitors; hence, drug interaction should be checked.

DRUG RESISTANTTB

BRIEF DESCRIPTION

- TB is considered drug-resistant (DR) when the causative agent (mycobacterium tuberculosis) is not killed by one or more of the available anti-TB medicines.
- Medicine- resistant TB can be primary or secondary (acquired).
- Primary resistance is medicine resistance among new cases i.e. persons who have never been previously treated for TB.
- Secondary resistance is medicine resistance among previously treated individuals.
- There are five different types of medicine resistance:
    - Mono-resistance: Resistance to one anti-tuberculosis medicine.
    - Poly-resistance: Resistance to more than one anti-tuberculosis medicine, other than Isoniazid andRifampicin.
    - Multidrug-resistance (MDR)-TB: Resistance to at least isoniazid and rifampicin, two most important first-linedrugs.
    - Extensive drug-resistance (XDR-TB): Resistance to any of the fluoroquinolones, and at least one of the injectable Second Line Medicines (capreomycin, kanamycin and Amikacin), in addition to resistance to INH and rifampicin.
        - Since XDR-TB progresses from MDR-TB in two steps, the term "pre–XDR-TB" was introduced to recognize MDR-TB with additional resistance to either one but not both of these classes of medicines.

- Total drug-resistance (TDR-TB): resistance to all anti TB medicines. The clinical features of medicine susceptible and medicine resistant TB are the same.

INVESTIGATION AND DIAGNOSIS

- Direct smear microscopy
- Gene Xpert MTB/RIF test
- Line Probe Assay (LPA) directly from the sputum specimen or cultured sample
- Culture and Drug Susceptibility Test (DST)
- The definitive diagnosis of medicine-resistant TB depends on laboratory diagnosis through Medicine Susceptibility testing (DST); it requires that M.tuberculosis is isolated and medicine susceptibility test is completed.
- Other investigations: Chest X-ray, HIV test, CBC, urinalysis, FBS, LFT, RFT, Serum electrolyte,TSH, HCG, Audiometric test.

TREATMENT OF MDR-TB

Objectives

- Cure the TB patient and restore quality of life and productivity
- Prevent death from active TB or its late effects
- Prevent TB relapse
- Prevent the development and transmission of extensive medicine resistance
- Decrease transmission

Non pharmacologic

- Surgery (see the adjunct therapies section below)
- Adherence counseling
- Psychosocial and emotional support

Nutritional support

Pharmacologic

- Treatment **of isoniazid monoresistant TB**
  - Rifampicin, Ethambutol, Pyrazinamide and Levofloxacin for duration of 6 months.
- **Treatment of Rifampicin resistant or Multi drug resistant TB (RR/MDR-TB)**
  - Treatment with second-line TB regimens.
- **Recommended MDR-TB treatment approach**
  - **Standardized treatmentapproach:**a pre-definedshortertreatment regimen (STR) once the diagnosis of RR-/MDRTB is confirmed using rapid first line DST techniques (i.e., Xpert MTB/RIF or LPA).
  - **Individualized treatment approach:** a regimen tailored to the individual patient when they do not meet the preset criteria to receive the standardized shorter treatment regimen.
  - **Patients who are not eligible for standardized shorter treatment regimen**
    - Confirmed resistance to a medicine in the shorter MDR-TB regimen
    - Exposure to one or more second-line medicines in the shorter MDR-TB regimen for >1 month (unless susceptibility is confirmed)
    - History of Pre-XDR-TB or XDR-TB
    - Intolerance to medicines in the shorter MDR-TB regimen or risk of toxicity
    - Fluoroquinolone resistance
    - Pregnancy

- Extensive pulmonary TB or severe extrapulmonary TB like CNS TB.
- Any extrapulmonary disease in people living with HIV

- One or more medicines in the shorter MDR-TB regimen not available**General principles for the use the standardized shorter treatment regimen**
  - Intensive phase: Seven agents administered together for up to 4-6 months
  - Continuation phase (CP): four agents for a fixed duration of 5 months.
  - The"core drugs": Bdq, Mfx, Cfz, and Pto, while Z, E and H are considered as add-on components of the STR.
  - Intensive phase (IP) consists of Bdq, Mfx, Cfz, Z, E, HH, and Pto administered for 4 to 6 months (Bdq better given for 6 months).
  - The continuation phase (CP) consists of Mfx, Cfz, E, and-Z for the fixed duration of 5 months.
  - The regimen: 4 Bdq -Mfx-Pto-Cfz-Z-HH-E / 5 Lfx-Cfz-Z-E
  - Intensive phase may be prolonged up to six months, if the patient remains smear positive after month four oftreatment.
- **Individualized DR-TB regimens**
  - For patients for whom the standardized regimens cannot be initiated or cannot be continued with it for clinical reasons or as a result of DST results.
  - It is often needed to be adjusted based on patient clinical history, once additional history or when DST results becomes available.
  - Indications for use of individualized treatment regimens
    - Presumed or confirmed PreXDR-/XDR-TB
    - Known contact with patient failing second line treatment
    - Pregnancy

- Disseminated, meningeal or central nervous system TB
- Any extrapulmonary TB in people living with HIV
- Laboratory evidence of resistance to quinolone or injectable and/or other agents,
- Occurrence of severe drug toxicities,
- Failure of standardized DR-TB treatment
- Re-treatment after treatment interruption beyond eight consecutive weeks o
- Risk of intolerance because of possible serious drug-drug interactions
- Severe adverse drug reactions to core drugs used in regimen

Table 8.69: Constructing individualized treatment regimen (see the step wise approach below)

Steps to Design individualized MDR Treatment Regimen	
Step 1	**Choose one later generation fluoroquinolone (Levofloxacin or Moxifloxacin)** • Oflaxacin susceptibility is routinely done. In addition to ofloxacin, every attempt should be made to determine susceptibility to moxi-floxacin and Levofloxacin • If only ofloxacin DST is known (and resistant) use Levofloxacin unless thought to be compromised (e.g., previous fluoroquinolone use); Moxifloxacin should be a last resort.

Step 2	**Choose both of these prioritized drugs: Bedaquiline (Bdq) and Linezolid (Lzd)** • If a drug is considered to have induced severe toxicity, do not include it in the regimen • Bdq is strongly recommended for adults >18 years, also can be used for 6–17 years.
Step 3	**Choose both of these prioritized drugs: Clofazimine, Cycloserine/terizidon**e • If a drug is considered not to be effective or it has induced severe toxicity, do not include it in the regimen • If effectiveness is difficult to judge, the drug can be added to the regimen, but should not be counted as one of the effective agents.
Step 4	**Choose one of the injectables (Amikacin (Amk) or streptomycin (S)** • If a regimen cannot be assembled with five effective oral drugs outlined in step 1 to 3, and if the isolate is susceptible to the injectable, use Amikacin for adults aged >17 years. • Streptomycin is an alternative under similar conditions. Ototoxicity should be closely monitored. • If resistant to all injectable drugs, do not include injectable.
Step 5	**If needed or if oral agents preferred over injectables in Step 4,** use the following drugs: **Delamanid, Pyrazinamide, Ethambutol** • Use pyrazinamide and ethambutol only when the isolate is documented as susceptible

Step 6	**Other SL agents** • **Ethionamide (Eto) or prothionamide (Pto)** • **Imipenem–cilastatin or meropenem with clavulanate (IV route),** • **p-Aminosalicylic acid** • **High-dose isoniazid** o Add drugs thought to meet the criteria of an effective drug and do not induce severe toxicity o If effectiveness is difficult to judge, the drug can be added to the regimen, but should not be counted as one of the effective drugs. o Ethionamide or prothionamide and/or p-aminosalicylic acid may be included in longer regimens only if bedaquiline, linezol- id, clofazimine or delamanid are not used or if better options are not possible. o Mutations in the inhA region of the M.tuberculosis genome can confer resistance to Eto/Pto and INH. In this situation, ethionamide/prothionamide may not be a good choice.
None	The following drugs are no longer recommended for inclusion in individualized MDR-TB regimens: Capreomycin and kanamycin; Amoxicillin/clavulanate (when used without a carbapenem); Azithromycin and clarithromycin; thioacetazone; gatifloxacin; perchlozone, interferon gamma or sutezolid

- **Extra pulmonary and central nervous system drug resistant TB**
  - The same strategy and duration of treatment as pulmonary drug-resistant TB with the only exception of CNS involvement.

- The treatment of drug resistant tuberculous meningitis should be guided by drug susceptibility results and the drugs to penetrate the central nervous system.
- The treatment duration in meningitis: a minimum of 20 months,

Table 8.70: Penetration of Anti-TB Drugs in Cerebrospinal Fluid

CNS Penetration level	Anti-TB drugs
Good penetration	Isoniazid, rifampicin, pyrazinamide, ethionamide, prothionamide, cycloserine, linezolid, imipenem, meropenem.
Penetration only through inflamed meninges	Aminoglycosides (streptomycin, kanamycin, amikacin), Fluoroquinolones (moxifloxacin, or levofloxacin,)
Poor or no penetration	Ethambutol, PAS
No or little data	Capreomycin, clofazimine, bedaquiline, Delamanid.

- **Treatment of MDR-TB in Special populations**
  - **HIV Infection**
    - Higher pill burden, drug–drug interactions, immune reconstitution inflammatory syndrome pose unique challenges Drug interactions between antiretroviral and anti-TBagents:
    - Efavirenz can produce a decrease in serum bedaquiline concentrations, avoid thiscombination.
    - Protease inhibitors can result in increased serum bedaquiline levels.
    - Check for overlapping toxicities and drug-druginteraction.
  - **Pregnant women**
    - Treating MDR-TB during pregnancy outweighs the harm of not treating to mother, child, and the community.

- Most of the drugs are pregnancy category C, Bedaquiline and meropenem, are category B, and aminoglycosides are category D
- Aminoglycosides and ethionamide can be avoided

- **Pyridoxine supplementation**
  - Pyridoxine (Vitamin B6) supplementation for all patients and for the period of the whole treatment duration.
  - For patient receiving the shorter standardized regimen: pyridoxine 25 to 50mg tablet.
  - Ptients receiving cycloserine containing regimens receive 50mg of pyridoxine for every
- **Adjunct therapy**
  - Corticosteroids
  - Beneficial as an adjunctive therapy in DR-TB patients with severe respiratory insufficiency, central nervous system or pericardial involvement.
  - Prednisone is commonly used, started at approximately 1 mg/kg of body weight with gradual tapering dosage after one to two weeks.
- **Surgery in treatment of drug-resistant TB**
  - Emergency indication: Sever hemoptysis and tension spontaneous pneumothorax.
  - Urgent indication: recurrent hemoptysis that cannot be stopped by other treatment methods.
  - Elective indications: Localized cavitaryTB with positive sputum after 4-6 months, M/XDR-TB characterized by failure of anti-TB chemotherapy, local complications and sequelae
- **Monitoring patient response to MDR-TB treatment using culture**
  - Treatment response should be monitored clinically, radiographically, and bacteriologically.

- Sputum smear microscopy and culture monthly.
- When cultures remain positive after 3 months of treatment, do drug susceptibility tests

- **Care and support for patients with MDR/RR-TB**
  - Health education and counselling on the disease and treatment adherence.
  - One or more of the following treatment adherence interventions (complementary and not mutually exclusive) may be offered to patients
    - Tracers and/or digital medication monitor;
    - Material support to the patient
    - Psychological support to the patient
    - Staff education.

- **Treatment of Contacts Exposed to MDR-TB**
  - For close contacts with presumed MDRTB latent infection, give 6 to 12 months treatment with a fluoroquinolone alone or with a second drug, on the basis of source-case isolate DST.
  - Pyrazinamide should not be used as the second drug.

Table 8 . 7 1 : Symptom based approach to the management of 2nd line Anti-TB medicines induced adverse-effects

ADR	Suspected agent	Management	Remarks
Nausea (N), vomiting (V)	Eto/Pto, PAS, H, E, Z, Cfz	1. Assess for dehydration; and rehydrate if indicated. 2. If mild symptoms and no signs of dehydration, O Advise patient to take medicines with porridge. O Initiate antiemetic therapy if needed (Metoclopromide) O Encourage patient to continue treatment O Encourage patients to increase fluid intake(water, juice, tea) 3. If there is dehydration or persistence of symptoms, · Initiate rehydration accordingly · Refer patient to treatment initiating center	1. N and V are very common in early weeks of therapy and usually abate with time and adjunctive therapy. 2. Electrolytes should be monitored and replaced if vomiting is severe. 3. Reversible upon discontinuation of suspected agent. 4. Clofazimine can cause severe abdominal pain and acute abdomen.This is rare, but if occurs, clofazimine should be suspended.

Gastritis	PAS, Eto/ Pto	1. Give antiTb medicines with small food, avoid caffeine, cigarettes and assess for signs of severity 2. If mild symptoms give H2-blockers, proton-pump inhibitors, or antacids. 3. If severe (severe persistent dyspepsia, hematemesis/coffee ground vomitus, black tarry stool, initiate rehydrationand refer.	1. Severe gastritis, as manifested by haematemesis, melaena or haematechezia, is rare. 2. Dosing of antacids should be carefully timed so as to not interfere with the absorption of antituberculosis medicines (take 2 hours before or 3 hours after antituberculosis medications). 3. Reversible upon discontinuation of suspected agent(s).
Hearing loss	Km,Am, Cm	1. Confirm that this is not due to ear wax or other conductive problems. 2.Check whether patient has history of hearing loss previously 3. Document hearing loss and compare with baseline audiometry if available. 4. Refer if it is new event or worsening of complaint.	1.Patients with previous exposure to aminoglycosides may have baseline hearing loss. In such patients, audiometry may be helpful at the start of MDR-TB therapy. 2. Hearing loss is generally not reversible. 3.The risk of further hearing loss must be weighed against the risks of stopping the injectable in the treatment regimen. 4.While the benefit of hearing aids is minimal to moderate in auditory toxicity, consider a trial use to determine if a patient with hearing loss can benefit from their use

Electrolyte disturbance (Low K&Mg) Manifesting as fatigue, muscle cramp, muscle spasm	Cm, Km, Am,	1. Check potassium (if available). 2. If potassium is low also check magnesium (& calcium if hypocalcemia is suspected). 3. Initiate potassium supplement if K+> 3.0meq/L) and monitor Potassium weekly 4. Correct if there are contributing causes of hypokalemia (Vomiting, diarrhea) 5. Refer if K+<3.0meq/L	1. If severe hypokalemia is present, consider hospitalization. 2. Amiloride 5–10 mg QD or Spironolactone 25 mg QD may decrease potassium and magnesium wasting and is useful in refractory cases. 3. Oral potassium replacements can cause significant nausea and vomiting. Oral magnesium may cause diarrhea.
Peripheral neuropathy	Cs, H, Km, Am, Cm, Eto/ Pto	1. Increase pyridoxine to maximum daily dose (200 mg per day). 2. Initiate therapy with tricyclic antidepressants such as amitriptyline. Non-steroidal anti-inflammatory medicines or acetaminophen may help alleviate symptoms. 3. If no improvement, refer.	1. Patients with co-morbid disease (e.g., diabetes, HIV, alcohol dependence) may be more likely to develop peripheral neuropathy, but these conditions are not contraindications to the use of the agents listed here. 2. Neuropathy may be irreversible; however, some patients may experience improvement when offending agents are suspended

Seizure	Cs, H, FQs	1. Suspend suspected agent pending resolution of seizures. 2. Initiate anticonvulsant therapy (e.g., Phenytoin,Valproic Acid). 3. Increase pyridoxine to maximum daily dose (200 mg per day). 4. Refer after controlling seizure	1.Anticonvulsant is g until MDR-TB treatm suspected agent disc 2. History of previou is not a contraindicat agents listed here if well controlled and/c ing anticonvulsant th 3. Patients with histo may be at increased of seizures during MI traindication to the u but may increase the sion developing durir
Hypothy-roidism (swelling, slowing, fatigue, day time sleepi-ness)	PAS, Eto/ Pto	Check TFT if available to confirm, Refer to TIC	1.Completely reversi ation of the medicine combination medicin
Blurring of vision	E, Eto	Refer	
Arthralgia	Z, FQ	1. Initiate therapy with NSAIDs (e.g Ibuprofen) 2. Refer if severe or no improvement.	

CHAPTER 6: EAR CONDITIONS

ACUTE OTITIS MEDIA

BRIEF DESCRIPTION

- Acute otitis media is the rapid onset of signs and symptoms of inflammation of the middle ear cleft mostly following URTIs.
- The most common causative bacterial organism are Streptococcus Pneumonia, Haemophilus Influenza A and Moraxella catarrhalis. Viral infection, commonest etiologies, may commonly prepare the way for secondary bacterial infection.
- The younger the child, the more severe the generalized symptoms are and the more discrete the local signs are. On occasions, the gastrointestinal symptoms are the most pressing.
- **Risk factors:** crowed conditions, day care, passive smoking, bottle feeding, low socioeconomic status,

CLINICAL FEATURES

Symptom

- Symptoms vary according to patients' age
- Neonates only present with irritability and/ or feeding difficulty
- Infants and older children can present with fever (with or without prior history of URTI) and otalgia, or tugging on the ear.
- In severe cases, rigors and occasionally meningismus can occur in children.
- Adults and older children can present with otalgia (mostly worse by night),

sometimes fever and impaired hearing.

- Both adults and children can present with ear discharge lasting <2 weeks Pain often improves after the onset of discharge.

Signs

- Otoscopy shows hyperemia, bulging and opacity of the surface of tympanic membrane,
- Perforation of the tympanic membrane can be seen in advanced case mostly in the posterior quadrant of the tympanic membrane.
- Pneumatic Otoscopy can be performed to asses for tympanic membrane mobility.

Investigation and Diagnosis

- The Diagnosis of acute Otitis media is mostly clinical.

Additional tests

- Ear swab for culture and sensitivity

TREATMENT

Goal

- Relieve symptoms or pain
- Return hearing to normal
- Prevent chronicity and complications (like perforation, meningitis, brain abscess, etc).

Non pharmacologic

- Advise on keeping the ear dry. I.e. apply Vaseline soaked cotton during bathing.

Pharmacologic treatment First line

- Pediatrics:Amoxicillin high dose 80-90 mg/kg/day po divided every 12 hour or every 8 hours for 10 days. (Adult: 1000mg BID or TID for 7 to 10 days)

Alternative, for non-responders to Amoxacillin

- Amoxicillin-clavulanate extra strength oral suspension 90/6.4 mg/kg/day po divided bid (preferred)

Second line drug

- **Ceftriaxone 50 mg/kg** IV or IM once daily x 3 days (is preferred in pediatrics), others agents in adults include: **cefuroxime axetil, cefpodoxime proxetil, cefixime**.
- If beta-lactamase allergy:**Azithromycin** 10 mg/kg per day orally (maximum 500 mg/day) as a single dose on day 1 and 5 mg/kg per day (maximum 250 mg/day) for days 2 through 5.

PAIN MANAGEMENT

- Paracetamol, 30-40mg/kg/24hrs in four divided doses to relieve pain. Alternative Ibuprofen

PREVENTION

- Parent education about risk factors
- Antibiotic prophylaxis – amoxicillin or macrolide shown effective at half therapeutic dose
- Pneumococcal and influenza vaccine
- Surgery: choice of surgical therapy for recurrent AOM depends on whether local factors (Eustachian tube dysfunction) are responsible (use ventilation tubes), or regional disease factors (tonsillitis, adenoid hypertrophy, sinusitis) are responsible

MASTOIDITES

BRIEF DESCRIPTION

- Poorly treated or untreated OM that last more than two weeks can lead to extension of the infection form mid era cavity to the mastoid air space leading to mastoiditis.

CLINICAL FEATURES

- Fever
- Profuse ear discharge
- Retro auricular swelling with tenderness

Investigations and diagnosis

- Culture and sensitivity tests

TREATMENT

Objective

- Eliminate the foci of infection in the temporal bone and the middle ear cavity.

Pharmacologic

- Patients with acute mastoidites should be admitted to the hospital and administered IV antibiotics active against resistant microorganisms, with bed rest and IV fluid. If not Acute mastoidites, it should be referred within 48 hours. Antibiotics should be considered with non-pharmacologic managements like drainage and surgery.
- If it is sub-per-ostial abscess, abscess drainage should be considered and referred for definitive management (cortical mastoidectomy)

ANTIBIOTICS FOR ACUTE MASTOIDITES

- If no recurrent AOM or no recent antibiotic use (3 to 6 months),
- **Vancomycin (15 mg/kg** intravenously [IV] every 6 hours; maximum 1 g per dose)
- If recurrent AOM (most recent episode within six months) or recent antibiotic administration,
- **Vancomycin PLUS Ceftazidime/cefepime 50 mg/kg** per dose IV every 8 hours (maximum 2 g per dose)

DURATION OF ANTIMICROBIAL THERAPY:

- Totally four weeks IV treatment may be continued for 7 to 1o days or IV to PO conversion should be done approximately after a clinical improvement.

Non pharmacologic

- Surgery if the inflammation is no longer confined to the mucosa but has extended to the bone

Prevention

- Early, adequate treatment of acute otitis media (AOM)
- Preventing recurrent AOM
- Immunization with the pneumococcal conjugate vaccine

Chronic Suppurative Otitis Media

- Chronic otitis media is defined as long-standing inflammation of the middle ear cleft in which characterized by chronically discharging ears for > 12 weeks (3 months).

CLINICAL FEATURES

Symptoms

- Constant or intermittent discharge (usually odorless) from the ear mostly not accompanied by otalgia.
- Hearing impairment in the affected ear

Signs

- Otoscopy: Tympanic membrane Perforation
- Tuning fork test (Rhinee and Webers Test): Conductive Hearing Loss
- Symptoms of impending complications like fever, lethargy, headache, vomiting, neck pain, changing mentation, dizziness, vertigo).

Investigations

- Culture of the ear discharge.

Treatment

Goals

- Keep the ear dry
- Eliminate the foci of infection in the temporal bone and the middle ear
- Construct the sound–conducting apparatus.

Non pharmacologic

- Instruct patients to keep the ear dry (Vaseline gauze, dry it after showering)
- Aural toilet (recommended together with topical antibiotics)

Pharmacologic

N.B. In acute exacerbations only Antibiotic treatment, whenever possible, must be directed by the results of culture and sensitivity of the ear discharge.

First line Topical:

- **Ciprofloxacin ear drop**, 0.3%, 5ml. 2 – 3 drops twice daily for 02 weeks.
- N.B; Ciprofloxacin and other quinolone like Norfloxacin or Ofloxacin with or without steroid combination can be used,
- If initial topical antibiotic therapy failed: culture directed systemic therapy should be tried (refer if no microbiology laboratory).
- **Refer** patients to ENT specialists for

Prevention

- Cornerstone of therapy
- Promptly and appropriately treating AOM
- Strict water precautions for prevention and management of recurrence
- Education on the risk factors like passive smoke exposure, contaminated water, and malnutrition might help.

OTITIS EXTERNA

- Otitis External diffuse inflammation of the external ear canal which may involve the pinna or the tympanic membrane.
- The most common causative agents being Pseudomonas A., Staphylococcus aureus and other gram-negative microbes occurring as a polymicrobial infection.
- Fungal otitis external can occur in the setting of repeated antibiotic use.
- Frequent Swimming, Rigorous ear cleaning, excessive use of air phone and underlying dermatological conditions can be risk factors for otitis externa
-

ACUTE OTITIS EXTERNA CLINICAL FEATURES

Symptoms

- Itching and Pain aggravated by movement and pressure on the auricle
- Rarely mucoid ear discharge
- Hearing impairment and aural fullness
- Posterior auricular lymphadenopathy

Signs

- Tragal tenderness,
- Otoscopic tenderness
- Erythematous and inflamed external ear canal,
- Posterior auricular Lymphadenopathy
- Diffuse edema of the External ear canal, with apparent granulation tissue and trisumus should alert the health care worker to consider malignant Otitis externa (Skull Base Osteomellitus) and seek immediate referral.

INVESTIGATIONSAND DIAGNOSIS

- Diagnosis of Otis external is clinical

- Culture and sensitivity studies can be mandated in recurrent cases and those unresponsive to antibiotics.

TREATMENT

Goal

To relive pain and other symptoms

To treat the infection

To prevent complications

Non-pharmacologic treatment

Keep the ear dry

Clean the ear until dry with ear wicks or suction if available cotton wicks by the physician

PHARMACOLOGIC TREATMENT

First line

Ciprofloxacin 0.2% and dexamethasone 0.1% otic suspension 2 – 3 drops twice daily for a total of 02 weeks (better tolerability).

OR

Neomycin 0.35%, Polymyxin B 10,000 units/mL, and hydrocortisone 0.5% otic solution 2 – 3 drops twice daily for a total of 02 weeks.

NB; Apply ear wicks to keep the external ear canal open for the first 3 days in diffusely edematous and narrow canal. Mild cases with only minor discomfort and pruritus, non-antibiotic topical preparation containing an acidifying agent and a glucocorticoid (eg, Acetic acid 2% and hydrocortisone 1% otic solution) can be used. Avoid use of acidifying antiseptic agents if tympanic membrane is perforated.

Aminoglycoside topical solutions can be used when ciprofloxacin and other quinolone are not available however avoid usage in perforated tympanic membranes due to ototoxicity.

Systemic antibiotic (in addition to topical) can be considered in severe cases and when Malignant OE is suspected (IV antibiotics recommended), immunosuppression, regional lymphadenopathy, systemic symptoms like fever until patient can be referred.

CHRONIC OTITIS EXTERNA

Usually caused by vegivores ear cleaning or total absences of ear wax.The cause can be infectious (bacterial or fungal) or non-infectious.

CLINICAL FEATURES

Symptom

Long standing ear itching

Sign

Dry, wide external oditerium mities with complete absence of wax (non-infectious)

Hypertrophic external olitory canal or Fungal Hyphae can be seen in the canal in cases of otomycosis.

TREATMENT

Non-infectious

Non-pharmacologic:

Acidifying agents like acetic acid

Treat the underling conditions like

applying cerumenolytic agent's alcohol, glycerin

dermatologic conditions

For infectious

Topical antifungals with cleaning or debridement

CERUMEN (WAX) IMPACTION

BRIEF DESCRIPTION

Ear wax is a mixture of secretions from ceruminous and sebaceous glands, epithelial debris and dust.

Ear wax is part of the body physiological defense mechanisms and needs removal only when it is symptomatic.

CLINICAL FEATURES

Aural fullness, Itching and decreased hearing.

Occasionally tints, vertigo

Otoscopy can reveal wax obliterating the ear canal.

TREATMENT

Cerumenolytic drops like Hydrogen per oxide, Olive oil

Syringing or manual irrigation (contraindications like previous history of ear discharge/tympanic membrane perforation, previous history of ear surgery, the only hearing ear). The only and rare complication of syringing is tympanic membrane perforation. It can be managed in a conservative way by advising the patient to keep the ear dry and distance themselves from other individuals with upper respiratory infections. NB traumatic tympanic membrane perforation usually heals by itself with conservative management.

Manual removal by an expert

N.B., Syringing and manual irrigation should be done by a Luke warm water (body temperature, 37 degree Celsius) and whenever possible after the usage of ceruminlytics for 2-3 days.

FOREIGN BODIES IN THE EAR

BRIEF DESCRIPTION

The majority of patients with foreign bodies in the ear are children.

The organic or inorganic objects may give rise to otitis externa (especially

organic) by local irritation of the epithelium of the mental walls.
CLINICAL FEATURES
Any suspicion for foreign body
Foreign body detected on Otoscopic examination.
INVESTIGATIONS
Diagnosis is clinical TREATMENT Goals
Open the ear canal which is completely or partially closed by removing the foreign body.
Eliminate secondary infections
Non pharmacologic
Irrigation of the suspected ear with water by ENT specialist if there is no perforation of the tympanic membrane.
Cerails can be irrigated if fride, unfride Cerails should not be irrigated because it get swollen
N.B. Foreign bodies that cannot be removed by irrigation should be removed manually, using general anesthesia in small children.
Ears with vegetable foreign bodies should not be irrigated, since this may cause the matter to swell.
Live insects can be killed rapidly by instilling alcohol, 2% lidocaine (Xylocaine), and Olive oil. Before removal is attempted.
Inorganic foreign body especially lithium button battery should be removed urgently within 2 hours
Manual removal is another approach.
If the foreign body is in the middle ear early referral is advisable

TINNITUS

BRIEF DESCRIPTION

An auditory meaningless perception in the absence of external source of sound, likely related to loss of stimuli to the central auditory pathways.

(Meaning less perception in the absence of external auditory sound is tinnitus, If meaning full perception in the absence of external auditory sound is auditory hallucination)

It can occur on 1 or both sides of the head.

Mostly happens in the setting of Sensory Neural Hearing Loss (SNHL).

Could be intermittent or persistent (> 6 month)

Could be Primary or idiopathic and Secondary i.e with identifiable underlying cause)

Could be subjective or objective

Could be pulsatile or non-pulsatile

Could be caused by local or systemic disease

Could affect the patient's quality of life and lead to depression, anxiety and other mental health issues.

THE SOUNDS CAN BE EXPRESSED AS THE FOLLOWING MEANINGLESS SOUNDS

Hissing, roaring Buzzing, tingling sounds in one or both ears

Local causes

It can have associated hearing Impairment, vertigo, aural fullness

Any history of ototoxic drug intake

SYSTEMIC CAUSES

Psychogenic: Associated mental health disturbances i.e sleep disturbance , emotional disturbances , anxiety , anger , frustration

Organic: History of Diabetic, Hypertension and Dyslipidemia, neurologic disorders

Signs

Examine for signs of inner, middle ear and external disease on Otoscopy i.e Tympanic membrane Perforation, Otorrhea, cerumen impaction, objective peripheral vertigo.

Neurological examination

Hear murmur, head and neck masses (carotid bruits), and Vascular sounds

Investigation and diagnosis For local causes-

Hearing assessment is part of tinnitus evaluation

Tunning Fork test

Audiometric evaluation (to assess for the type and degree of hearing loss)

Imaging Studies only when underlying organic lesion is suspected or pulsatile tinnitus.

Workup for systemic causes (CBC, VDRL, thyroid function test, Doppler ultrasound, etc)

TREATMENT

Non-pharmacologic

Avoid ototoxic medications

Treat underlying cause if identified

Provide counseling for patients with bothersome symptoms and if needed psychiatric evaluation and treatment.

Recommend Hearing Aids for individuals with hearing impairment (N.B; Hearing aids should be encouraged even for elderly patients as its improves their quality of life greatly)

CHAPTER 7: ANEMIA

APPROACHTOADULTSWITH ANEMIA

BRIEF DESCRIPTION

- Anemia is functionally defined as reduction in red blood cell (RBC) mass, accompanied by a decrease in oxygen carrying capacity.
- Laboratory wise anemia is defined as a reduction in one or more of the three RBC measurements in the CBC: hemoglobin (Hg), hematocrit (HCT), or RBC count. For practical purposes, hemoglobin or hematocrit are commonly used.
- WHO criteria for diagnosing anemia in men and women are hemoglobin values <13 and <12 g/dl, respectively.
- Anemia is not a single disease entity; it is rather a manifestation of several pathologies.
- Anemia can be classified based on RBC morphology (size), as measured by mean corpuscular volume (MCV).
- Classification based on of RBC size is useful for considering possible causes; however, it should never be taken as diagnostic.
    - Microcytic: MCV<80 fl
        - Common causes: iron deficiency anemia and anemia of chronic disease
    - Normocytic: MCV 80-100 fl
        - Common causes: anemia of chronic disease, CKD
    - Macrocytic: MCV> 100 fl
        - Common cause: vitamin B12 deficiency, alcohol abuse, chronic liver disease
- Base on the cause, anemia can be divided in to two broad categories:
    - Anemia due to increased RBC loss or destruction
        - Hemorrhage

- Hemolysis

- Anemia due to defective or decreased RBCproduction
  - Iron deficiency anemia
  - Vitamin B12 or folate deficiency
  - Anemia of chronic disease
  - Chronic kidney disease
  - Hypothyroidism,
  - Aplastic anemia
  - Bone marrow infiltration: Leukemia, lymphomas, other cancers, granulomatous diseases
  - Chemotherapy induced anemia

CLINICAL FEATURES

Symptoms

- Fatigue, dyspnea, palpitation, syncope
- Headache, lightheadedness, tinnitus, vertigo, difficulty of concentration
- Anorexia, nausea, indigestion
- Symptoms s of the underlying disease e.g., melena in GI bleeding, heavy menstrual bleeding, generalized body swelling in CKD signs

Signs

- Pallor, tachycardia, wide pulse pressure /ejection systolic murmur.
- Signs of Heart Failure (raised JVP, S3, hepatomegaly, edema)
- Signs of the underlying disease-causing anemia: lymphadenopathy, splenomegaly, angular chelitis, tumors (abdominal/ pelvic mass) etc.
- Investigation and diagnosis
- CBC with RBC indices
- Peripheral blood smear
- Reticulocyte count and index
- Further investigations: depends on the suspected cause/s of anemia based on the above tests,the history and physical examination findings.
    - **Suspected iron deficiency anemia**: serum ferritin, total iron binding capacity (TIBC), transferrin saturation ([serum iron ÷ TIBC] X 100%)
    - **Once iron deficiency is diagnosed**: stool for occult blood, stool microscopy for hookworm infestation, upper GI endoscopy or colonoscopy may be needed based on the clinical suspicion.
    - **Suspected megaloblastic anemia**: serum vitamin B12 level, serum folate and if serum folate level is normal.
    - **Suspected hemolytic anemia**: Reticolcultye count or percenetage bilirubin (indirect hyperabuleminmia), LDH, Coomb's test.

TREATMENT
OBJECTIVES OFTREATMENT

- Improve the functional status of the patient by correcting the hemoglobin.
- Treatment of the underlying cause

NON PHARMACOLOGIC

- Transfusion of packed RBC: Indications for transfusion
    - Hemoglobin $\leq$ 7g/dl: for most hospitalized medical or surgical patients

- For ambulatory patients with chronic anemia transfusion may not be needed even at hemoglobin is <7g/dl, unless the patients have severe symptoms e.g., heart failure
- Hemoglobin < 8g/dl: for those with pre-existing chronic cardiac disease, undergoing orthopedic or cardiac surgery
- In trauma or acutely bleeding patients
  - Do not use hemoglobin or hematocrit for transfusion decision as they are falsely elevated
  - Hemodynamic status and ongoing nature of bleeding should be used
  - Whole blood is preferable if there is acute or ongoing bleeding
- For the following patients higher hemoglobin target might be aimed
  - Acute coronary syndrome
  - Severe thrombocytopenia in hematology/hematology patients

- Nutritional support
- Non pharmacologic treatment pertinent to the underlying cause

PHARMACOLOGIC

- Pharmacologic treatment depends on the underlying cause of anemia.

REFERRAL

- Patients with anemia suspected due to primary bone marrow disease, malignancy, autoimmune disease, GI bleeding, and unknown/unclear cause should be referred to a referral hospital.

1. IRON DEFICIENCYANEMIA

BRIEF DESCRIPTION

- Iron deficiency anemia is a common cause of anemia worldwide.
- The major causes of iron deficiency anemia are nutritional deficiency, impaired absorption form the GI tract, and chronic blood loss from the GI or genitourinary tract Examples: hook worm infestation, colonic cancer, bleeding peptic ulcer or gastric cancer, prolonged or excessive menstrual bleeding, gynecologic malignancies.

CLINICAL FEATURES

- In addition to the general clinical features anemia (mentioned above), chronic iron deficiency anemia might show unique clinical features.
  - Pica: desire (craving) to eat unusual substances like soil, ice.
  - Koilonychia: Thin, brittle nail with depressed (concave or spoon) distal half.
  - Glossitis or angular stomatitis: the tongue and angel the mouth inflamed and sore.
  - Plumer-vinson syndrome: Difficulty of swallowing due to esophageal webs.
- **Investigations specific for iron deficiency anemia**
  - CBC: low Hg and hematocrit, low MCV, low MCH, and increased RDW.
  - Iron studies
    - Serum Ferritin: usually ow (it could be high in patients with vhronic inflammation or CKD in spite of iron deficiency)
    - Serum iron: may be low ornormal
    - Total iron binding capacity (TIBC): usually high
    - Transferrin saturation (TSAT) = serum iron/ TIBC X 100%: low (<20%)

- Clinical evaluation and investigation to identify the possible cause of bleeding
  - Stool for ova of parasites
  - Digital rectal examination
  - Gynecologic examination
  - Upper GI endoscopy and/or colonoscopy.

- **Pharmacologic treatment of iron deficiencyanemia**
  - **Treatment of the underlying cause**
    - The cause of the iron deficiency state should be identified and treated.
    - Oral iron (tablet): For at least three months following correction of the anemia
    - Ferrous sulfate, 325mg (has 65mg elemental iron), PO,TID.

      OR
    - Ferrous fumarate, 324mg, (has 106 elemental iron), PO, BID.

      OR
    - Ferrous gluconate, 325mg P.O. (39mg elemental iron), 1-2tabs, TID
    - Oral iron (solutions/syrup): if the tablets are not tolerated or patient preference
    - Iron hydroxide polymaltose syrup (Each 5ml contains 50mg elemental iron), 10ml PO, BID to TID.

      OR
    - Ferrous gluconate syrup (Each 5ml contains 24mg elemental iron), 15ml, PO TID.

      OR

- Ferric ammonium citrate syrup (Each 15ml contains 32.8mg elemental iron), give 30ml,TID.

- **How to instruct oral iron intake?**
  - Preferably to be taken 2 hours before or 4 hours after meal.
  - If separating from food is difficult due to gastrointestinal side effects, foods which significantly interfere with iron absorption should be avoided when the iron is given e.g. milk, eggs, tea, and coffee.
  - GI side effects are very common with oral iron administration. These include epigastric pain, nausea or vomiting, constipation or diarrhea, metallic taste.
  - For patients who do not tolerate, they may be advised to take it with meals, or to take a smaller dose, solutions or elixir forms.

- **Intravenous (IV) iron**

Indications for IV iron therapy

  - Intolerance to oral iron therapy.
  - Anemia secondary to chronic kidney disease with a requirement for erythropoietin.
  - No improvement in hemoglobin after 4 weeks of oral iron.
  - Existence of conditions that interfere with absorption of iron from the GI tract e.g., atrophic gastritis, gastrectomy, inflammatory bowel disease
  - Blood loss difficult to cope with oral iron therapy e.g., heavy menstrual bleeding, bleeding telangiectasia
  - Severe anemia during late second or third trimester of pregnancy

- **IV iron administration**
  - For **patients not on hemodialysis:**
    - **Iron sucrose** 200mg, IV, administer over 5 minutes, every 3 days for a total of 5 doses (a total of 1000mg). This dose is usually

sufficient but if hemoglobin is not corrected, additional doses can be given.

OR

- **Iron sucrose** 200mg diluted in 100ml NS; administer over 30 minutes.

- **For patients on hemodialysis**
  - **Iron sucrose** 100mg, IV, over 2-5 minutes, given early during dialysis sessions (within the first hour) until iron deficiency is corrected. It needs to be given again, if iron deficiency persists or recurs.

2. MEGALOBLASTICANEMIA

BRIEF DESCRIPTION

- Megaloblastic anemia is a morphologic term that describes abnormal red blood cells with maturation defects;the red blood cells tend to be large.
- The major causes of megaloblastic anemia are vitamin B12 (Cobalamin) and folate deficiency or a combination of both,
- The pathologic process which results in megaloblastic anemia can also result in leukopenia and thrombocytopenia. In case of Vitamin B12 deficiency the nervous system can be affected.
- Major causes of vitamin B12 deficiency
  - Gastric o**rigin (pernicious anemia, chronic atrophic gastritis)**
  - **Small intestine malabsorption (chronic diarrhea from small bowel/ileal pathologies)**
  - Strict vegetarians.
- Major causes of folate deficiency

- Poor nutritional status
- Increased demand during pregnancy and lactation

- Alcoholism
- Drugs: anti-epileptic drugs (phenytoin, phenobarbitone) or drugs which affect Folate metabolism (Methotrexate, cotrimoxazole)
- Malabsorption
- Critical illness

CLINICAL FEATURES

- In addition to the clinical features of anemia due to any other cause, some clinical manifestations may suggest megaloblastic anemia.
  - Gossitis (pain over the tongue with smooth, beefy red tongue)
  - Angular
  - Jaundice
  - Neurologic or neuropsychiatric manifestation: specific to vitamin B12 deficiency, but not folate deficiency
  - Neuropathic pain in the lower limbs
  - Decreased position sensation and gait disturbance
  - Weakness of the lower extremities
  - Irritability, depression, disorientation, dementia, frank psychosis

DIAGNOSIS AND INVESTIGATIONS

- Investigations for megaloblastic anemia
  - CBC
  - Anemia with high MCV (>100fl). When there is concomitant iron deficiency,the MCV can be normal or low.
  - Leukopenia and thrombocytopenia may also befound.
  - Peripheral morphology: hypersegmentation of neutrophils, large RBCs (macro-ovalocytes).
  - Hypersegmentation of neutrophils is defined as >5% of neutrophils with 5 or more lobes.
  - Bilirubin: Indirect hyperbilirubinemia
  - Determination of the levels of vitamin B12 andFolate
  - Serum vitamin B12 level
  - Serum folate level
  - RBC folate level: to be requested if serum folate level is normal
  - Bone marrow aspiration: indications
  - If the serum vitamin B12 and folate levels are normal but there is strong clinical suspicion.
  - When there is need to exclude other causes.
  - Determining the cause of the deficiency: if the cause is not clinically obvious, further wok up will be needed to identify the underlying cause.

TREATMENT

- **Objectives oftreatment**
  - Improve functional status of the patient by correcting theanemia
  - Correct existing and prevent further neuropsychiatric manifestations

- Identify and treat the underlying cause

- **Pharmacologic treatment of vitamin B12 (Cobalamin) deficiency**
  - **Cyanocobalamin (Vitamin B12)** 1000micrograms (1mg), IM, to be given according to the following schedule
    - Every day for one week
    - Every week for four weeks. If hemoglobin has not normalized, continue weekly until it gets normal.
    - If the underlying disorder persists, 1mg every month for the rest of the patient's life.

- **Pharmacologic treatment of folate deficiency**
  - **Folic acid**, 1 to 5mg P.O., daily for 1-4 months, or until complete hematologic recovery.
  - **Vitamin** B12 level should be checked before giving folic acid alone; as treatment with folic acid alone might worsen neurologic manifestation of vitamin B12 deficiency.
  - If vitamin B12 can't be checked, both Folic acid and vitamin B12 should be started at the same time.

- **Follow up of treatment response**
  - Symptomatic improvement
  - Hemoglobin level

ERYTHROCYTOSIS (POLYCYTHEMIA)

BRIEF DESCRIPTION

- Erythrocytosis, also called polycythemia, refers to abnormally increased red blood cell count as measured by hematocrit, hemoglobin or RBC count above the sex-specific normal range.
- The following terminologies are important
  - **Relative versus absoluteerythrocytosis**
    - **Relative erythrocytosis** is a clinical situation in which there is a decrease in plasma volume resulting in apparent erythrocytosis. It is the result volume contraction, not an increase in RBC count.
    - **Absolute erythrocytosis** indicates a true increase in the RBC mass.
  - **Primary versus secondary erythrocytosis:** Absolute erythrocytosis can be primary or secondary.
    - **Prim**ary erythrocytosis is an autonomous production of RBCs by the bone marrow without any physiologic stimuli. The most important cause of primary erythrocytosis is polycythemia vera (PV).
    - Secondary erythrocytosis results pathologies which increase serum erythropoietin level and stimulate RBC production (see the table below).

- Polycythemia Vera (PV) is a myeloproliferative neoplasm characterized by high RBC count, increased risk of thrombosis and vasomotor symptoms. A gain-of-function mutation in Janus kinase 2 (JAK2) is found in about 98% of patients with PV
- Distinguishing Polycythemia Vera (PV) from secondary erythrocytosis is very crucial.

Table 7.1: Major causes of secondary erythrocytosis	
Mechanism	**Major causes**
Hypoxia driven	Chronic lung diseases e.g., COPD, fibrotic lung diseases
	Obstructive sleep apnea
	Smoking
	Long term carbon monoxide exposure
	Cyanotic congenital heart diseases
Renal causes (renal hypoxia driven)	Renal cysts
	Hydronephrosis
	Renal artery stenosis
Paraneoplastic erythropoietin secretion	Renal cell carcinoma
	Hepatocellular carcinoma
	Uterine myoma
Miscellaneous	Post kidney transplant erythrocytosis
	Drug induced erythrocytosis: Erythropoietin, testosterone

CLINICAL FEATURES

- **Asymptomatic:** erythrocytosis is mainly a laboratory finding, the patient could be asymptomatic.
- **Symptomatic**: the symptoms and signs in patients with erythrocytosis can originate from the following reasons.
    - Due to increased blood viscosity (high RBC mass)

- Due to the underlying cause in secondary erythrocytosis
- Symptoms and signs specifically related to polycythemia vera

- Clinical features due to the increased viscosity of blood
  - Myalgia, fatigue, headache
  - Blurred vision or transient loss of vision, decreased cognition.
  - Paresthesia
  - Chest discomfort, abdominal pain
- Symptoms and signs suggestive polycythemia vera(PV)
  - Pruritus after bathing
  - History of arterial or venous thrombosis (e.g., ischemic stroke) or venous thrombosis (e.g., DVT, PE, hepatic vein thrombosis)
  - Erythromelalgia (episodic or persistent, intense pain over the toes or fingers with erythema or hotness)
  - Splenomegaly.
- Clinical features that suggest specific secondary causes
  - Symptoms of chronic lung diseases: cough chest tightness, wheezing, and shortness of breath.
  - Symptoms suggestive of obstructive sleep apnea: day time sleepiness, fatigue, apneic (breathless) spells at night, and snoring.
  - History of smoking or long-term exposure to carbon monoxide (indoor smoke).
  - Clinical evidence of neoplasm for paraneoplastic causes: e.g., hepatocellular carcinoma, renal cell carcinoma

DIAGNOSIS AND INVESTIGATION

Investigations

Investigations useful to identify possible causes

- CBC
  - Thrombocytosis and/or leukocytosis: suggestive of PV
  - Erythrocytosis with low MCV: suggestive of PV
- Chest X-ray: if the history and physical examination suggest underlying lung disease.
- Abdominal ultrasound: to look for liver mass, renal cysts or solid mass.
- **Serum erythropoietin (EPO) level**
  - When there are no obvious secondary causes, determination of the serum EPO level helps to differentiate primary from secondary causes.
    - Elevated EPO = secondary causes
    - Low or normal EPO = primary cause, mainly PV.
- **Testing for the JAK2 (Janus kinase 2) mutation**
  - It is essential to test for JAK2 mutation when PV is considered.
  - This test should only be ordered by a specialist who would treat and follow these patients i.e., a hematologist or an internist who follows these patients.
  - 95 – 100% of patients with PV have a JAK2 mutation involving either exon 14 or 12.
- **Bone marrow biopsy**
  - May be needed if secondary cause is not apparent and JAK2 cannot be done.

DIAGNOSIS

- Erythrocytosis is said to be present in adults when one or more of the followings is present:
  - Hematocrit >48% in women or >49 %in men.
  - Hemoglobin: >16.0 g/dL in women or >16.5 g/dL in men.

TREATMENT

OBJECTIVES OFTREATMENT

- Treating the underlying cause
- Decrease symptoms related to hyperviscosity
- Decrease the risk ofthrombosis

TREATMENT OF SECONDARY ERYTHROCYTOSIS

- **Treat the underlying cause:Examples**
  - Stop smoking,
  - Decrease/avoid indoor carbon monoxide exposure
  - Treatment of the underlying lung disease e
  - Surgery for renal cell carcinoma.
- **Limited phlebotomy for secondary erythrocytosis**
  - Limited phlebotomy is appropriate in secondary erythrocytosis, if there are symptoms of hyper viscosity (headache, slow mental function, transient loss of vision, paresthesias) and hematocrit is usually > 65%.

TREATMENT OF POLYCYTHEMIAVERA

- **Riskstratification**:patients with either of the following two characteristics are considered high risk
  - History of arterial or venous thrombosis, irrespective of age.
    - Age >60 years are considered high risk.The rest areconsidered low risk.
- **Phlebotomy (Therapeuticphlebotomy)**
  - For all patients with PV (both low and high risk)
  - Target hematocrit is < 45%
  - One to two units per week until target is achieved. For those who do not tolerate (elderly, women, cardiac or pulmonary disease) reduce to half to one unit per week.

- **Aspirin**
  - **For all patients with PV**
    - **Aspirin** 75-100mg, PO, day.
- **Cytoreductive therapy**
  - To be decided by a specialist.

3. THROMBOCYTOPENIA

THROMBOCYTOPENIA IN HOSPITALIZED PATIENTS

Brief description

- Thrombocytopenia is defined as a platelet count less than 150×10^3 per µl.
- The degree of thrombocytopenia can be divided from mild to severe. However, these numbers should be interpreted cautiously as severity definitions may vary.
  - Mild = 100,000 to 150,000/ µl
  - Moderate = 50,000 to 99,000/ µl
  - Severe <50,000/ µl
- The safest platelet count at which bleeding is unlikely to occur is not precisely known. It also varies significantly with the underlying cause.
  - Surgical bleeding risk is high when platelet counts <50,000/ µl.(<100,000/ µl for neurosurgery or major cardiac or orthopedic surgery).
  - Severe spontaneous bleeding: the risk is high when platelet counts <20,000 - 30,000/ µl
- Thrombocytopenia is not only a risk for bleeding but it can also be a manifestation of life-threatening thrombotic disorders.
  - Common clinical disorders which can cause thrombosis and thrombocytopenia at same time
    - DIC (disseminated intravascular coagulation)
    - HIT (Heparin induced thrombocytopenia)

- TTP/HUS (thrombotic thrombocytopenic purpura/hemolytic uremic syndrome
- APS (antiphospholipid antibody syndrome)

- Thrombocytopenia is a common in hospitalized patients. The risk is even much higher in critically ill patients.
- The causes of thrombocytopenia in hospitalized patients are numerous causing diagnostic challenges.Some of them are listed in the table below.

Table 7.2: The major causes thrombocytopenia in hospitalized pa*tients*

1. Spurious thrombocytopenia (Pseudothrombocytopenia)
2. Hemodilution: massive transfusion or crystalloid resuscitation
3. Sepsis
4. Malaria
5. Disseminated intravascular coagulation (DIC)
6. Heparin induced thrombocytopenia (HIT)
7. Drug induced thrombocytopenia
8. Pregnancy complications: gestational thrombocytopenia, preeclampsia, HELLP syndrome, acute fatty liver of pregnancy, DIC
9. Chronic liver disease and hypersplenism.
10. Alcohol or nutritional deficiencies (Vitamin B12 and/or folate deficiency)
11. Thrombotic microangiopathies (TMA): TTP/HUS (Thrombotic thrombocytopenic purpura/Hemolytic uremic syndrome), catastrophic APS
12. Autoimmune (Rheumatologic) diseases:SLE
13. Post transfusion purpura
14. Viral infections: HIV, Hepatitis C

CLINICAL EVALUATION OF THROMBOCYTOPENIA IN ACUTELY ILL HOSPITALIZED PATIENTS

- Confirmation of the thrombocytopenia:
  - Platelet clumping is a laboratory artifact than cause spurious thrombocytopenia.
  - Repeat CBC

- Using EDTA-free tubes (e.g. heparin or citrate tubes)
- Do peripheral morphology to see if the thrombocytopenia is genuine or not.

- Detailed clinical history and physical examination: Evaluate if the underlying disease (e.g., sepsis) is a possible cause
- Evaluating for life threatening causes
  - Sever malarias peripheral blood smear (thin and thick)
  - HIT: History of heparin administration, date of administration, the presence of thrombosis, the degree of decrease in platelet count from baseline in percentage.
  - TTP/HUS: Peripheral morphology for fragmented RBCS, serum LDH, BUN and serum creatinine, urinalysis
  - Acute leukemia: other cell line, peripheral smear and bone marrow aspiration
  - DIC: determine PT(INR) and PTT, peripheral smear
  - Transfusion history: transfusion associated pupura
- Detailed drug history
- Evaluate for other common causes
  - Liver disease: clinical evaluation and liver function tests
  - Viral causes: HIV and HCVscreening
  - Alcohol intake
  - Nutritional status: Peripheral blood smear to see evidence of megaloblastic anemia.

TREATMENT

- Treatment of the underlying cause: is the main stay of management in acutely ill hospitalized patients with thrombocytopenia.
- Platelet transfusion: Indications

- **Active bleeding:** If there is active bleeding and the platelet count <50,000cells/µl.
- **Prophylactic platelet transfusion**: In patients without active bleeding prophylactic platelet transfusion should be avoided unless the platelets count < 10,000 cells/µl.

- Hold antiplatelet agents and anticoagulants, if platelets count < 30,000cells/ µl.

REFERRAL

- Patients in whom the cause of thrombocytopenia is not identified.
- Patients in whom the cause of thrombocytopenia is identified but treatment cannot be provided in that setting

5. IMMUNETHROMBOCYTOPENIA (ITP)

BRIEF DESCRIPTION

- Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterized by a low platelet count resulting from platelet destruction
- Other conditions which can cause immune related thrombocytopenia should be excluded before the diagnosis ITP. E.g., HIV, HCV infection,

  H. Pylori infection, systemic lupus erythematosus, chronic lymphocytic leukemia should be excluded before the diagnosis of ITP. Hence, ITP is a diagnosis of exclusion.
- Patients with other associated conditions (e.g., other autoimmune diseases) are described as having secondary immune thrombocytopenia.
- The incidence of ITP is higher in children than adults. Preceding viral infections are common precipitants of ITP in children.
- Classification of ITP based on the duration of the disease
  - Newly diagnosed ITP:ITP duration of less than 3 months
  - Persistent ITP:ITP duration of 3-12 months
  - Chronic ITP: ITP duration of more than 12 months

CLINICAL FEATURES

- Asymptomatic: The vast majority of patients with ITP are not symptomatic, unless the platelet is very low.
- Bleeding mucocutaneous bleeding also called "Platelet-type" bleeding)
  - Petechiae, purpura, and easy brusing.
  - Epistaxis, gingival bleeding, menorrhagia, gross hematuria
  - Gastrointestinal bleeding: bloody vomitus, bleeding peer rectum
  - Intracranial bleeding: headache, change in mental status or focal neurologic deficit
  - Signs of anemia: Pallor, tachycardia, low blood pressure or postural drop in blood pressure (if massive bleeding)

INVESTIGATIONSAND DIAGNOSIS

- The diagnosis of ITP is made based on clinical grounds after exclusion of other causes of thrombocytopenia.
- A platelet count of < 100,000/µl is needed for consideration of the diagnosis.

  Peripheral blood smear: is required to exclude other causes of thrombocytopenia and to confirm the presence of true thrombocytopenia.
- Serologies: HIV and HCV (hepatitis C Virus) serology tests are needed in all patients
- H. Pyolri test: is indicated in all patients.
- ANA might be needed is there is a clinical evidence of SLE.
- TSH: autoimmune thyroid diseases are common in patients with ITP.
- Bone marrow aspiration/biopsy:
  - It is not generally indicated for the diagnosis of ITP
  - It is indicated in individuals with atypical features such as B-symptoms, lymphadenopathy, splenomegaly, unexplained leukocyte abnormalities or unexplained anemia, and age > 60 years.
  - It is also indicated before splenectomy.

TREATMENT
OBJECTIVES OFTREATMENT

- Increase the platelet count to a safe level to prevent major bleeding. Safe level of platelet is >30,000/µl.

N.B.:The aim of ITP treatment is not to bring the platelet to normal levels

-

NON PHARMACOLOGIC

- **Emergency platelet transfusion**
  - Generally, platelet transfusion should be avoided.
  - Indication for platelet transfusion: life-threatening bleeding only.
  - If platelet transfusion is indicated, intravenous steroids should be started immediately.
- **Splenectomy:** is an option of treatment for patients who have corticosteroid refractory or dependent disease.

PHARMACOLOGIC

Not all patients with ITP need treatment. Those with no indications to treatment should be followed with CBC and clinical assessment of bleeding.The patients should be given enough information about bleeding.

- Indications for tr**eatment**
  - Platelet count < 30,000/µl, irrespective of bleeding status.
  - Platelet count > 30,000/µl and significant bleeding (other than minor mucocutaneous bleeding)
- **First line:Corticosteroids**
  - **Dexamethasone,** 40mg, oral or IV, daily for 04 consecutive days with no tapering. Repeat this 4 day cycles every 2-4 weeks for 4-6cycles.

    OR
  - **Prednisolone,** 1mg/kg for 1-2 weeks, if there is response taper over a period of six weeks or less.
    - Typical tapering regimen: After response, reduce by 10 mg/week until 0.5 mg/kg is reached; then taper by 5mg/week.
- **Treatment response**
  - Response: is defined if there is a platelet count >30,000/µl and at least doubling from the baseline both must be fulfilled.
  - Durable response: if there is response persisting up to 6 months.
  - Remissio**n:** is defined if platelet count is >100,000//µl for >12 months

- **Steroid dependent:** Ongoing need for continuous prednisolone > 5 mg/d (or equivalent) or frequent courses of corticosteroids needed to keep a platelet count >30,000/µl.

ALTERNATIVE TREATMENTS:

- Intravenous immunoglobulin (IVIg)
- Anti-D
- Rituximab
- Splenectomy

CHAPTER 8
TYPHOID FEVER (ENTERIC FEVER)

BRIEF DESCRIPTION

- Typhoid fever (E nteric fever) is an acute febrile illness characterized by fever and severe systemic illness.
- The causative organism is *Salmonella enterica* serotype Typhi (formerly *S. typhi*).
- Other *Salmonella* serotypes like *S. enterica* serotypes Paratyphi A, B, or C, can cause a similar illness; however, differentiation is not easy but it is not clinically useful.
- Enteric fever is a collective term which refers to both typhoid and parathyphoid fever; however, the term enteric fever and typhoid
- Humans are the only reservoir for *S.* Typhi, the mode of transmission is via contaminated food or water.

Clinical features

- The clinical presentation of typhoid fever can vary from a mild to life threatening in severity.
- Fever is the most important clinical manifestations.
    - For consideration of typhoid fever there must be fever that lasted at least three days
    - The fever characteristically increases daily ("stepwise") reaching as high as in the forties ^{in} degree celsius.
- Relative bradycardia (pulse-temperature): normal pulse rate in spite of high-grade fever may be observed.
- Other manifestations: headache, fatigue, malaise, loss of appetite, cough, constipation or mild diarrhea and skin rash or rose spots, hepatosplenomegaly.
- In the late courses of the disease the patient may develop neurologic manifestations

- "Typhoid encephalopathy": altered consciousness, delirium, and confusion.
- Acute psychosis
- Meningeal signs

- Life threatening complications: intestinal perforations, gastrointestinal hemorrhages, andencephalitis.

Table 8.33: Case definitions of typhoid fever

Probable or Suspected case of typhoid fever	A patient with documented fever (38°C and above) for at least 5 days prior to presentation, with rising trend AND Having no other focus to explain the cause of the fever (e.g., malaria, meningitis, pneumonia, abscess, pyelonephritis etc.)
Confirmed case of typhoid fever	A patient with persistent fever (38 °C or above) lasting 3 or more days and *S. Typhi* isolated on culture (blood, bone marrow culture, stool or urine)
Chronic carrier	An individual excreting *S. Typhi* in the stool or urine for longer than one year after a blood culture confirmed episode of typhoid fever.

INVESTIGATIONSAND DIAGNOSIS

- The criterion standard for diagnosis of typhoid fever has long been culture isolation of the organism. Cultures are widely considered 100% specific.
- CBC: Usually normal, mild anemia relative leucopenia and occasional thrombocytopenia may be observed
- Liver transaminase and serum bilirubin: usually rise mildly.

DIAGNOSIS

- Microbiologic diagnosis
- The definitive diagnosis of typhoid fever based on culture:
  - Blood culture: the mainstay for the diagnosis
  - Bone marrow culture: has the highestyield
  - Stool or urine cultures
  - Serological tests (Widal test)

- It is not recommended for diagnosis of typhoid fever.
- Based on studies done in different parts of Ethiopia the positive predictive value of theWidal test,among febrile patients with suspected typhoid fever is <6%. Hence, a positive or high titer Widal test is by in large a false positive.

- Clinical diagnosis: a patient with documented high-grade fever which is persistent, after exclusion malaria and other causes of fever should be suspected of fever.
- Wrong diagnosis (malpractice)
  - Testing for typhoid fever in patients with non-specific complaints such as headache, malaise, and arthralgia but without high grade fever is malpractice.
  - Diagnosis of typhoid fever based on a positive or high titer Widal test alone in patients without high grade fever is a malpractice.
  - Diagnosis of typhoid fever in patients with high grade fever with Widal test alone without excluding malaria, clinical evaluation and investigation for other cause of fever is malpractice.

TREATMENT OBJECTIVES

- Treat acute infection: decrease morbidty and mortality
- Prevent chronic carriage

Non pharmacologic

Symptomatic treatment:

- Use of antipyretics e.g., paracetamol to control fever, cooling

Pharmacologic treatment

First line

Table 8.34: Drug of choice for complicated and uncomplicated typhoid fever

Drug	First line	Alternative
Uncomplicated Typhoid fever	Ciprofloxacin 500mg P.O., BID for 7 - 10 days	Azithromycin 1 g orally once then 500 mg orally daily OR 1 g orally once daily for 5 to 7 das
Complicated/sever Typhoid fever	Ceftriaxone 2gm IV daily or 1 gm Iv BID OR in 2 divided doses I.M. OR I.V. for 10 to 14 days	Only if no alternative: IV Ciprofloxacin: 20 mg/kg per day in two divided doses (maximum 800 mg per day) then, Oral: 30 mg/kg per day in two divided doses (maximum 1000 mg per day)

ADJUNCT CORTICOSTEROIDTREATMENT:

- Indications: Severe systemic illness (delirium, obtundation, stupor, coma, or shock)
- Dexamethasone (3 mg/kg sat, followed by 1 mg/kg every 6 hours for a total of 48 hours)

PREVENTION

- As enteric fever infects from the ingestion of contaminated food or water, sanitation and hygiene, access to clean water and carful consumption of non-cooked/raw foods is critical.
- Typhoid conjugate vaccine for infants and children six months or older is recommended by WHO in endemic areas like Ethiopia.

Special population considerations

- **Pregnant women:** Fluoroquinolone are contraindicated in pregnant mothers. Third generation cephalosporins should be used in pregnant mothers in place of fluoroquinolone or azithromycin.

CHAPTER 9
URINARY TRACT INFECTION

BRIEF DESCRIPTION

- Urinary Tract Infection (UTI) refers to the presence of microorganisms in higher numbers to cause invasion of the urinary tract (UT) epithelium and inflammation that cannot be accounted for contamination.
- UTI is classified in different ways that have implication to treatment and outcome

ACCORDING TO ANATOMIC SITE OF INVOLVEMENT:

- Lower UTI: cystitis, urethritis
- Upper UTI: pyelonephritis

ACCORDING TO THE PRESENCE OF STRUCTURAL URINARY TRACT PROBLEMS

- **Uncomplicated UTI**: UTI that occurs in individuals with no structural or functional abnormalities in the urinary tract that interfere with the normal flow of urine.
    - Typically, in women
- **Complicated UTI**: UTI that occurs in individuals with structural or functional abnormalities in the urinary tract.
    - E.g., congenital distortion, obstructive stone, indwelling catheter, vesicoureteral reflux, prostatic hypertrophy, or neurologic bladder.
    - UTI in men should be considered complicated unless proven otherwise.
    - UTI in patients with recent urinary tract instrumentation, postoperative UTI, in patients with kidney transplantation should be considered complicated.
- **Catheter associated UTI:** UTI occurring in a person whose urinary tract is currently catheterized or has been catheterized within the past 48 hours.
    - **Urosepsis** is defined as life threatening organ dysfunction caused by a

body's response to infection originating from the urinary tract or male genital organs.

- **Recurrent UTI-** symptomatic and culture-proven UTI occurring two or more times in six months or three or more times in a year. Recurrent UTI is not necessarily complicated.
- **Asymptomatic bacteriuria:** Bacteiruria > 105 bacteria/ml of urine without symptoms.
- **Symptomatic abacteriuria:** Symptoms of urinary frequency and dysuria in the absence of significant bacteriuria in urine culture.

The presence of pyuria (≥10 WBC/mm^3 of uncentrifuged urine) alone is not sufficient for diagnosis of UTI or bacteriuria. More than 60% of samples in women with asymptomatic pyuria have no bacteriuria.

ETIOLOGICAGENTS

- Uncomplicated UTI: *Escherichia coli* is the commonest pathogen.
- Complicated or hospital acquired UTI: In addition to *E. coli*, *pseudomonas, klebsiella*, *enterobacteria*, *proteus*, *serratia* and assume greater importance.

CLINICAL FEATURES

- **Lower UTI (Cystitis)**
  - Dysuria,frequent urination and urgency
  - Occasionally, gross hematuria
  - Suprapubic pain
  - Elderly people may have nonspecific symptoms: urinary incontinence chronic, malaise, abdominal pain and decreased eating or drinking)
  - N.B: cloudy or malodorous urine are nonspecific features that should not routinely swift for cystitis evaluation OR treatment.
- **Upper UTI(pyelonephritis)**
  - Flank pain

- Fever, chills and rigors
- Vomiting
- Significant costovertebral angle tenderness
- If associated cystitis (lower UTI), not always present: dysuria, urgency, frequency
- In elderly: fever or poor feeding or disorientation without another sign

- **Acute prostatitis**: acutely ill, fever, chills, malaise, myalgia, dysuria, frequency, urgency, urge incontinence), pelvic or perineal pain, and cloudy urine.
- **Chronic prostatitis:** usually, symptoms of recurrent UTI (frequency, dysuria, urgency0 perineal discomfort, a low-grade fever, with repeated isolation of the same organism from the urine

INVESTIGATIONS AND DIAGNOSIS

Investigation

- Urinalysis
  - Microscopy for WBC, RBC
  - Dipstick: leukocyte, nitrite
- Urine culture: clean catch mid-stream urine specimen
- CBC
- RFT
- Ultrasound kidneys and prostate (in men 40 years): If complicated only

DIAGNOSIS

- The diagnosis is based on clinical findings plus urinalysis
- Urine analysis
  - Pyuria (WBC > 10 cells/mm3) is present in almost all patients with UTI
- Urine culture:

- Significant bacteriuria = presence of 105 bacteria (CFU) /ml of urine
- In symptomatic UTI a lower bacterial count should still be considered positive
- The diagnosis of recurrent UTI requires culture confirmation

- Digital rectal examination (DRE): For men suspected of having prostatitis; tender or swollen/edematous prostate.
- Ultrasound: when complicated UTI is suspected.

TREATMENT OBJECTIVES

- Eradicate infection and improve quality of life
- Prevent recurrence

Non pharmacologic

- Recurrent UTI: Postcoital voiding, avoiding spermicide use and liberal fluid intake
- Complicated UTI: correction of the underling anatomical or functional abnormality. This requires urologic/gynecologic consultation.

Pharmacologic

A. Acute, Uncomplicated UTI in women

Table 8.54:Antimicrobial regimen selection for urinary tract infec- tions for adults

Scenario	First line options	Alternatives
Acute uncomplicated UTI (cystitis) in women	Ciprofloxacin 250- 500mg PO, BID or Norfloxacin, 400mg PO., BID, for 3 days. OR Nitrofurantoin 50mg P.O., QID for 5 days (effective if available) OR Cotrimoxazole (TMP-SMO) 160/800mg P.O, BID for 3 days (Only if the local resistance is known to be <20%)	Fosfomycin 3g single dose OR Cefpodoxime100mg PO, BID for 5 days *OR* Amoxicillin-clavulanate 500/125mg TID for 5 days
Uncomplicated mild to moderate pyelonephritis (Mild to moderate): able to take orally, no vomiting, no dehydration, no sepsis)	Ciprofloxacin, 500mg P.O., BID, oral for 7-10 days.	Cefpodoxime, 200mg P.O., BID for 10 days preceded by a single dose of ceftriaxone 1gm IV/IM.
Severe pyelonephritis *vomiting, dehydration, or evidence of sepsis)* without risk of resistant infections– *start IV*	Ciprofloxacin, 400mg, IV, BID. Complete 10-14 days with oral Ciprofloxacin 500mg BID at discharge OR Ceftriaxone, 1- 2gm, IV, daily, followed by oral ciprofloxacin. OR Cefotaxime 1gm IV TID, followed by oral ciprofloxacin	If no response to these options within 48- 72 hours, use options listed in the category (sever pyelonephritis with risk of resistant pathogens)

Severe pyelonephritis with the either of following three risk factors for of resistant pathogens in the last 3 months 1. Multidrug-resistant urinary isolate 2. Inpatient stay at a health care facility 3. Use of a broad-spectrum antibiotic (fluoroquinolone, third or later generation cephalosporin trimethoprim-sulfamethoxazole)	Meropenem 1gm IV TID OR Imipenem 500mg IV QID	OR Cefepime 2 gm IV TID OR Ceftazidime 2 gm IV TID OR Piperacillin-tazobactam 3.375 gm IV QID Addition of aminoglycoside: Gentamycin 3-5mg/Kg, IV, once daily can be sonsidered.

RECURRENT UTI

- Antibiotic prophylaxis is recommended for women who experience two or more symptomatic UTIs within six months or three or more over 12 months.
- The degree of discomfort experienced by the woman needs to be considered in the decision.
- Recurrent pyelonephritis deserves prophylaxis.
- Any prophylaxis should be given after current active infection is treated.
- Prophylaxis regimen options
  - Continuous: daily (sometimes three times per week)
  - Postcoital: a single dose of antibiotic after every sexual activity; if there is clear temporal relationship between episodes of the recurrent UTI and sexual intercourse.

- Self-treatment (Not actual prophylaxis): A 3 days short course treatment in a patient who clearly understand the symptoms. To be started at the first onset of symptoms. Risk of recurrence remains high.

- **The antibiotic prophylaxisoptions**
  - **Trimethoprim-sulfamethoxazole** 40 mg/200 mg once daily or 3 times per week
  - **Nitrofurantoin** 50 mg or 100mg once daily
  - **Cefaclor** 250 mg once daily
  - **Cephalexin** 125 mg -250mg once daily
  - **Norfloxacin** 200 mg once daily
  - **Ciprofloxacin** 125 mg once daily

PROSTATITIS

- **Acute bacterial Prostatitis**: Floroquinolones or Trimethoprim/ sulfamethoxazole is first line agent. Duration of treatment is 4 weeks (28 days).
- **Chronic bacterial prostatitis:** Difficult to treat. Similar antibiotics to acute bacterial are use but for a duration of 1–4 months.

ASYMPTOMATIC BACTERIURIA

- It should not be treated with antibiotics.

- The only two indications to treat with antibiotics: pregnancy and prior to invasion urinary tract instrumentation.

PREGNANCY AND UTI

- Antibiotics to be avoided for pregnant women: fluoroquinolones, aminoglycosides and trimethoprim/sulfamethoxazole
- Antibiotics recommended for uncomplicated UTI in pregnant women: amoxicillin/clavulanate, cephalexin or cefpodoxime for 3-5 days
- Antibiotics recommended for severe or complicated scenarios:

CHAPTER 10

THROAT CONDITIONS

ACUTE TONSILLITIS

BRIEF DESCRIPTION

Acute infection of the lymphoepithelial tissue of the faucial isthmus, the palatine tonsil, and pharyngeal (adenoid) tonsil, lingual and tubal tonsil is known as tonsillitis.

The commonest causes are viral, followed by less likely beta-Streptococci, Staphylococci, Streptococcus Pneumoniae (Diplococcus pneumoniae) and Haemophilus.

CLINICAL FEATURES

Symptoms

- Low/High Fever and possibly chills, especially in children.
- Persistent pain in the oropharynx or Naso pharynx
- Pain on swallowing that radiates to the ear.
- Opening the mouth is often difficult and painful if it is complicated.
- Usually associated with systemic symptoms like Headache and marked feeling of malaise, chills, and rigor.

Signs

- Inflamed and reddened enlarged tonsils
- Exudates are apparent in bacterial tonsillitis
- Tender cervical lymphadenopathy

INVESTIGATIONS

- CBC and ESR
- Culture from throat swab.

TREATMENT

Objective

- Treat infection
- Shorten the duration of the disease
- Prevent complication
- Relieve pain

Non pharmacologic

Gargling with warm Normal saline solution

Pharmacologic

The management is conservative

The indication for bacterial tonsillitis, high grade fever, exudative tonsillitis, and tender cervical lymphadenopathy. Several symptoms that are not suggestive of group A Streptococcus are cough, conjunctivitis, and coryza.

First line

Amoxicillin 250-500mg P.O.,TID for 7 – 10 days. 125mg/5ml, 250mg/5ml P.O., TID for 7 –10 days. (pedi; 50 mg/kg PO once daily to a maximum of 1000-1200 mg x 10 days)

Alternatives

Consider cephalosporin (e.g. **Cephalexin** 500 mg po bid x 10 days)

OR

If there is poor response go for **Amxoicillin – Clavulanate:** pediatric dose: 45 mg/kg/day in two divided doses for ten days (Adult dose: 875/125 mg PO bid x 10 days)

FEVER MANAGEMENT

Paracetamoll, 500mg P.O, PRN.

Local complications

peritonsillar abscess (the complications may also include, paraphayrengial abscess and retropharyngeal abscess, not addressed here)

In adiaguatly treated acute or chronic tonsillitis can spreed to the surrounding tissue and form abscess called peritonsillar abscess

CLINICAL FEATURES OF PERITONSILLAR ABSCESS

Severe pain such that the patient often refuses to eat

The head is held over to the diseased side, and rapid head movements are avoided.

The patient has sialorrhea and oral fetor

Swelling of the regional lymph nodes,

Fever with high temperatures of 39°C to 40°C and the general condition deteriorates rapidly.

Redness, and protrusion of the tonsil, the faucial arch, the palate and the uvula

Marked tenderness of the tonsillar area

TREATMENT OF PERITONSILLAR ABSCESS

Early referral for drainage of the absccess and tosnillectomy

Systemic complication

Septicemia

Rheumatic heart disease

Post streptococcal plomerulonephritis

ADENOID TONSILLAR HYPERTROPHY

BRIEF DESCRIPTION

Enlargement of the adenoids with or without concomitant palatine tonsillar enlargement.

Leads to obstructive symptoms in children peaking at ages 4-5, and is rare after the age of 12.

CLINICAL FEATURES

Symptoms

Nasal obstruction, snoring

Recurrent rhino sinusitis or otitis media

Nasal discharge, post nasal drip and cough

Obstructive sleep apnea characterized but loud snoring at night, recurrent apneic episodes enuresis, and day time solomonence, poor school performance, cognitive impairment

Signs

Adenoid Facies (Open mouth, high arching palate, narrow mid face, malocclusion)

Hypo nasal voice

Bilateral palatial tonsillar enlargement

INVESTIGATION AND DIAGNOSIS

Diagnosis can be made through direct visualization of the adenoids via flexible nasopharyngoscope or mirrors (Posterior Rhinoscopy)

Lateral Neck X-RAY can show the adenoid shadows

TREATMENT

Referral of children to ENT care centers is mandated, if the child has symptoms of obstructive sleep apnea or recurrent tonsillitis. Recurrent tonsillitis is defined as an acute attack of tonsillitis 7 times in a year or five times a year for two consecutive years or three times per year for their consecutive years.

Indication for referral of adeno tonsillar hypertrophy for surgical management

obstructive sleep apnea

Recurrent tonsillitis

Failure to thrive

Recurrent peritonsillar absence

Malignancy suspicion

If the patient has severe dysphagia

ACUTE LARYNGITIS

BRIEF DESCRIPTION

This is an inflammation strictly localized to the vocal cords, usually of viral origin. Acute laryngitis is usually due to ascending or descending infection from the other parts of the airway. In children there is a danger of airway obstruction.

CLINICAL FEATURES

Hoarseness of the voice

Aphonia

Pain in the larynx and coughing attacks

In children there is a danger of airway obstruction

Investigations

Laryngoscopy shows red and swollen vocal cords lose their normal color

Treatment Objective

Relieve airway obstruction

Non pharmacologic:

Voice hygiene (voice rest, rehydration)

Mist therapy (application of steam or inhalant)

PHARMACOLOGIC

First line

Antibiotics are not usually recommended due to the viral onset of the disease If there is an upper airway obstruction

Prednisolone, 40-60mg P.O., daily based on severity, the dose is reduced by 5mg every 5 days. NB: Glucocorticoids should only be given if there is concern for airway compromise.

FOREIGN BODY IN THE THROAT

All pharyngeal foreign bodies are medical emergencies that require airway protection.

CLINICAL FEATURES

Symptoms

History of chocking,

Dysphagia and odynophagia

Dysphonia and hoarseness

Coughing and stridor

INVESTIGATION AND DIAGNOSIS

High degree of suspicion is required especially in children with partial obstruction

Neck and Chest X-RAY for radiopaque objects.

GOAL OF TREATMENT

Secure airway

N.B. Refer patients for immediate endoscopic removal

SALIVARY GLAND CONDITIONS

MUMPS (EPIDEMIC PAROTITIS)

Mumps is a disease characterized by swelling of one or more salivary glands.

The parotid glands are the salivary glands most commonly involved with mumps, In 75-80% of cases both glands are involved.

CLINICAL FEATURES

Symptoms

Ear pain localized to the ear lobe aggravated by chewing

Swelling at the angle of the jaw

Sour taste in the mouth

Fever (usual subsides within 7 days)

Rarely sudden hearing loss can occur

In adult males orchitis (testicular swellings can occur occasionally)

Signs

Tender swelling of the parotids or other salivary glands

Erythematous and edematous submandibular duct or stepsons (parotid duct)

INVESTIGATIONS

Diagnosis is clinical **TREATMENT Objective**

Relieve symptoms

Non pharmacologic:

Massage the gland **Pharmacologic First line**

Paracetamol, children; 30-40mg/kg/24 hr. divided into 4 – 6 doses adults;

1000mg P.O, every 6 hrs PRN

Alternatives

Tramadol, 100 mg P.O., every 6 hrsPRN for adults.

N.B. Not recommended for chidren below 12 years of age

Acute and non-chronic non-obstructive suppurative Sialadenitis

Acute bacterial infection of the salivary glands usually involves the paratoid glands. This condition is usually seen in debilitated patients. The usual causative organism is Staphylcoccus aureus.

CLINICAL FEATURES

Pain and swelling of the involved gland

Purulent secretions can be expressed from the orifice of the duct

Fever

INVESTIGATIONS

Sologra phy shows a tree in leaf appearance

Tissue should be taken for histology in doubtful cases

TREATMENT

Objective

Control pain

Prevent recurring episodes

Non pharmacologic

Bed rest

Restricted jaw movement

Pharmacologic First line

Cloxacillin, 500mg P.O., QID for 7 – 10 days. 50 – 100mg /kg/24hrs. P.O., divided into 4 doses for 7 - 10 days.

Alternatives

Cephalexin, children; 6 to 12 mg/kg PO Q 6 hours. Maximum 25mg/kg Q

6 hours adults; 50mg to 1gm QID PO Q6 hours

If penicillin allergy, Clindamycin, 150 to 450mg P.O., QID or 300mg IM or IV QID or TID. In severe infections 20mg/kg/24hr. IM or IV into 4 doses.

UPPER AIRWAY OBSTRUCTION

It is difficulty of breathing caused by obstruction occurred above the thoracic airway (which could be nasopharynx, oropharynx, laryngeal or extra-thoracic tracheal airway)

Caused by obstructive mass or post inflammatory

CLINICAL FEATURES

Hoarseness of voice, hypo nasal or pharyngeal (hot potato speech) based on location of obstruction.

stridor (inspiratory, expiratory or biphasic) based on site obstruction.

tachypnea, tachycardia.

Cyanosis & loss of consciousness are late signs.

MANAGEMENT

Try to calm the patient & keep breathing (avoid crying in pediatric age & talk in adults.

Administration of intranasal or face mask oxygen.

Administration of adrenaline inhalation (nebulization) & steroid if we suspect post-inflammatory obstruction.

Secure the airway: non-surgical (intubation) or surgical (cricothyrotomy and tracheotomy).

Monitoring Aspect	Yes	No	Comments
PROTOCOL UTILIZATION MONITORING TOOL **Objective**: To monitor adherence to the Standard Treatment Guidelines (STG) within the Adult OPD, ensuring appropriate use and timely review of clinical protocols.			
1. Protocol Awareness			
Are healthcare providers familiar with the protocol?			
Was a briefing on the top 10 diseases conducted?			
2. Protocol Utilization			
Are diagnoses in alignment with protocol guidelines?			
Are treatments initiated based on protocol recommendations?			
3. Documentation			
Are treatment plans documented as per protocol?			
Are follow-up plans in line with protocol timelines?			
4. Outcomes and Feedback			
Has there been an improvement in patient outcomes?			
Are there any challenges in implementing the protocol?			
5. Protocol Compliance			
Are deviations from the protocol documented with justification?			
Are treatment adjustments aligned with updates in patient condition?			
6. Education and Training			

Are new staff members oriented to the protocol?			
Are continuous training sessions provided to reinforce protocol application?			
7. Resource Availability			
Are all necessary medications available as per the protocol?			
Are equipment and diagnostic tools required by the protocol readily accessible?			
8. Quality Improvement Measures			
Are challenges in protocol implementation reviewed regularly?			
Is there a system in place to update the protocol based on hospital or national guideline changes?			
9. Patient Education and Adherence			
Are patients informed about their condition and treatment according to the protocol?			
Is patient adherence to treatment and follow-up monitored and supported?			
10. Review and Reporting			
Are monitoring reports submitted to department heads or quality committees?			
Are audit results and findings discussed in department meetings for continuous improvement?			

Implementation and Review Process

To ensure the effective application of this protocol, a structured implementation plan will be followed:

1. Training and Orientation: All clinical staff within the Emergency Department and Adult OPD will be oriented on the new protocol, with regular refresher training provided to keep them updated on any changes or enhancements.

2. Resource Allocation: Regular assessments of resource availability (e.g., medications, diagnostic tools) will be conducted to ensure that all items required by the protocol are accessible. Any gaps identified will be reported to the procurement department to facilitate timely acquisition.

3. Quarterly Monitoring and Feedback Sessions: Utilization of the monitoring tool will be reviewed quarterly. Findings will be shared with relevant teams, and challenges or deviations will be addressed in feedback sessions. This continuous loop will support a culture of accountability and improvement.

4. Quality Improvement Reporting: Results from the monitoring tool will be documented and submitted to the hospital's quality committee. Trends in compliance, identified issues, and success stories will be highlighted in these reports to guide decision-making at the hospital level.

This systematic approach will ensure adherence to the STG protocols and will enable Deder General Hospital to maintain high standards in patient care, fostering both consistency and quality in the management of the most prevalent health conditions in the Adult OPD Department.

DEDER GENERAL HOSPITAL

ADULT OUTPATIENT DEPARTMENT

STANDARD TREATMENT GUIDELINE PROTOCOL

"Adapted from National STG 2021 4th Edition"