## CHAPTER II. BACTERIAL DISEASES

## II.1 TUBERCULOSIS

### A.DEFINITION

**Tuberculosis** is an infectious disease and transmissible due to **Mycobacterium.**

Typical causative agents (i.e usual agents) of tuberculosis are;

1. **Mycobacterium tuberculosis or Koch bacillus**

- It causes most morbidity and mortality due to TB
- It is spread through droplets coughed up by sputum positive person i.e patient to patient transmission
- They are sensitive to Ultra-Violet Light, so daytime outdoor transmission is rare.

**2. Mycobacterium bovis**

- Spread by ingesting non-pasteurized milk
- Domesticated and wild animals are the usual host
- It causes intestinal tuberculosis in humans

### B.MYCOBACTERIUM TUBERCULOSIS OR KOCH BACILLI

1. **GENERAL CHARACTERISTICS**

-Slender (slim) curved rods

-Acid-fast, and Alcohol-fast bacilli in Zielhl-Nielsen stain (Acido-Alcohol Resistant Bacilli; AARB or BAAR in French).

-Slow multiplication ( 4-6 weeks are required for a culture).
- Very sensitive to heat, and Ultra-Violet Light
-Resisting to the cold and dryness
-Strict aerobic (require oxygen for living)
-In the host they cause very small (microscopic) swellings called **tubercles** (or granulomata) in any organ that is infected.
-Very virulent and multiply inside macrophages

**The Mycobacterium tuberculosis (Koch bacilli)** causes tuberculosis which can be;

**1. Chronic disease** that can affects mainly;
-The lungs to result in **Pulmonary Tuberculosis**
- Other organs such as the brain, intestines, kidneys, bones and spine where it is called **Extra Pulmonary Tuberculosis.**

2. **Acute disease** and is called **Miliary Tuberculosis**

Miliary tuberculosis occurs when Mycobactera enter the blood and are disseminated widely in the lungs and other organs of the body especially in infants and immunocompromized persons than in immunocompetent adults.

Miliary tuberculosis is so called because it causes the appearance of numerous small tubercles (which look like millet i.e Miliary) in organs, and it normally kills the patients if untreated. X-Rays show the numerous small tubercles.

2. **TRANSMISSION OF TUBERCULOSIS**

Primarily by air (others are uncommon like digestive,)

Direct transmission through Droplet spread. When the patient coughs, spits, sneezes, laughs, sings or speaks, he emits an aerosol of droplets charged with bacilli. Direct inhalation of those droplets cause an infection to a new host.

Indirect transmission through Droplet nuclei (residue of dried droplet spread, that passes a long time suspended in the air).

**3. RESERVOIR**

Human:

- Healthy infected
- Patient with pulmonary tuberculosis with positive microscopy

Animals:

- Bovines
- Monkeys, cats, dogs, etc.

**4. PATHOGENESIS**

After inhalation of bacilli, the development of tuberculosis in a previously uninfected child or adult person can be of three types;

1. PRIMARY TUBERCULOSIS
2. POST-PRIMARY( OR REACTIVATION) TUBERCULOSIS
3. MILIARY TUBERCULOSIS(or Disseminated tuberculosis)

**1. PRIMARY TUBERCULOSIS**

i.A susceptible person becomes infected by inhaling Mycobacterium through Droplets of sputum( liberated by coughing or speaking) from an infectious tuberculosis patient after repeated exposure . Risk factors include bad ventilation and overcrowding.

ii.The Mycobacteria breathed in inhabit in the distant(i.e peripheral) part of the lung tissue. There they multiply and cause some tissue destruction especially in the mid zone of the lung (cause the peripheral lesion). While this happens, the immune system sends many lymphocytes to the site in an attempt to conquer the invading organism.

iii.Then the cells of the immune system including Macrophages and Lymphocytes try to eliminate the mycobacteria by surrounding and killing them, whereby some mycobacteria are transported to regional lymph nodes e.g Hilar lymph nodes, which tend to enlarge due to some inflammation.

The isolated lesion (i.e. solitary peripheral lesion) in the lung together with enlarged regional lymph node is called **PRIMARY COMPLEX or GHON'S COMPLEX**.
iv.Most primary complex heal spontaneously by action of cell mediated immunity resulting in fibrosis and sometimes calcification to form tubercles (i.e. swelling) 2-6 weeks after infection.

v.The bacilli in the tubercles die slowly but some remain alive even for 20 years or more.

vi.The primary infection ends there in most people who have a well functioning cell mediated immunity.

vii.For people who have or later may have weak cellular immunity, the infection progresses into Post primary (i.e reactivation) tuberculosis.

**Clinical features of Primary Tuberculosis**

It is usually asymptomatic, but may be fever, malaise; cough with or without sputum, painful red rashes (erythema nodosum), sweating, and anorexia.

## 2. POST PRIMARY(OR REACTIVATION) TUBERCULOSIS

a.This occurs as a result of reactivation of formally dormant Mycobacteria in the turbecles formed during primary tuberculosis period. It can occur 20 years or more later after primary tuberculosis healed.

b.Risk factors for post primary tuberculosis include;

- Age( infants, small children and old people of 50 years and above due to weak cellular immunity)
- Malnutrition
- Concurrent infection ( i.e occurring at the same time; e.g malaria, worms, measles..)
- Toxic factors e.g alcohol and smoking
- Immunosuppression (e.g HIV, Steroids,..)
- Host genetic factors
- Poverty
- Stress

c.If the immune response is weak or immunity declines, the mycobacteria may gain the upper hand and spread to other party of the lungs. There they cause characteristic lesion. These solid lesions, containing mycobacteria and immune response cells, then gradually involve more and more lung tissue.

d.The apex of the lung is the common site of infection. Other parts such as kidneys, bone and bone marrow, lymph nodes, brain, meninges and intestines may be involved

e.Before long, because of continuing tissue destruction, the middle parts of the solid lesions begin to liquefy into cheese-like material which is released into the small airways.The patient starts to cough. The cough soon begins to produce purulent sputum.

d. As the mycobacteria destroy lung tissue, the destruction may cause disruption of some small (or sometimes large) blood vessels. This result in blood appearance in the sputum. This is known as **haemoptysis**. The disease can spread outside the lungs in several ways. The mycobacteria may migrate into the blood stream and thus be carried to other party of the body.

e.The patient loses weight, coughing blood, has night sweats, and chronic fever, malaise, anorexia (lack of appetite).

### 3. MILIARY OR DISSEMINATED TUBERCUKOSIS

i.It occurs when a large blood vessel is eroded by TB whereby the bacilli get disseminated in the blood to almost every organ system of the body resulting in fulminating (i.e of sudden onset, and rapid in progress and acute) infection.

ii.It occurs in very young children, very old people and those who are immunocompromised

iii.Symptoms resemble those of reactivation (post primary) tuberculosis, but meningitis and brain involvement are common.

iv.This form of TB is commonly fatal (i.e kills)/

### 4 .CLINICAL MANIFESTATIONS

-Productive Cough for more than 2 weeks
- Haemoptysis, dyspnoea, thoracic pain
- Fever,
- Night sweating
- Anorexia
- Asthenia
- Weight loss
- Other signs according to the affected organ (extrapulmory tuberculosis).

-Enlarged or tender lymph nodes in the neck or other areas

-Unusual breath sounds (crackles): by auscultation

In French; **3 T**;-Toux productive≥2semaines,-Temperature elevée,-Transpiration nocturne

**3 A**;-Asthenie,-Amaigrissement,-Anorexie
**2D**;-Douleur thoracique,-Dyspnee
**1H** : Hemoptysis

## 5. DIAGNOSIS

1. **Bacilloscopy** (sputum examination )

It is mainly based on finding acid –fast mycobacterium in the sputum smear with Fluorescence microscopy ( old method Ziehl Nielsen stain) At least 2 sputum specimens must be collected and examined in 2days.

1st day: Sputum N$^{O}$ 1: Immediately on the first day of consultation and under supervision
2nd day: Sputum N$^{O}$ 2: The following day early morning

2. **Tuberculin (Mantoux) skin test**; or Intra-Dermo-Reaction (IDR)

-10 Tuberculin unities of purified protein derivative antigen of Mycobacteria tuberculosis are injected on the front of the forearm.
-There appears a swelling (i.e weal) like a pimple ( Spot). This is monitored for hardening (i.e induration) 3 days later. The diameter of induration is recorded as follow;

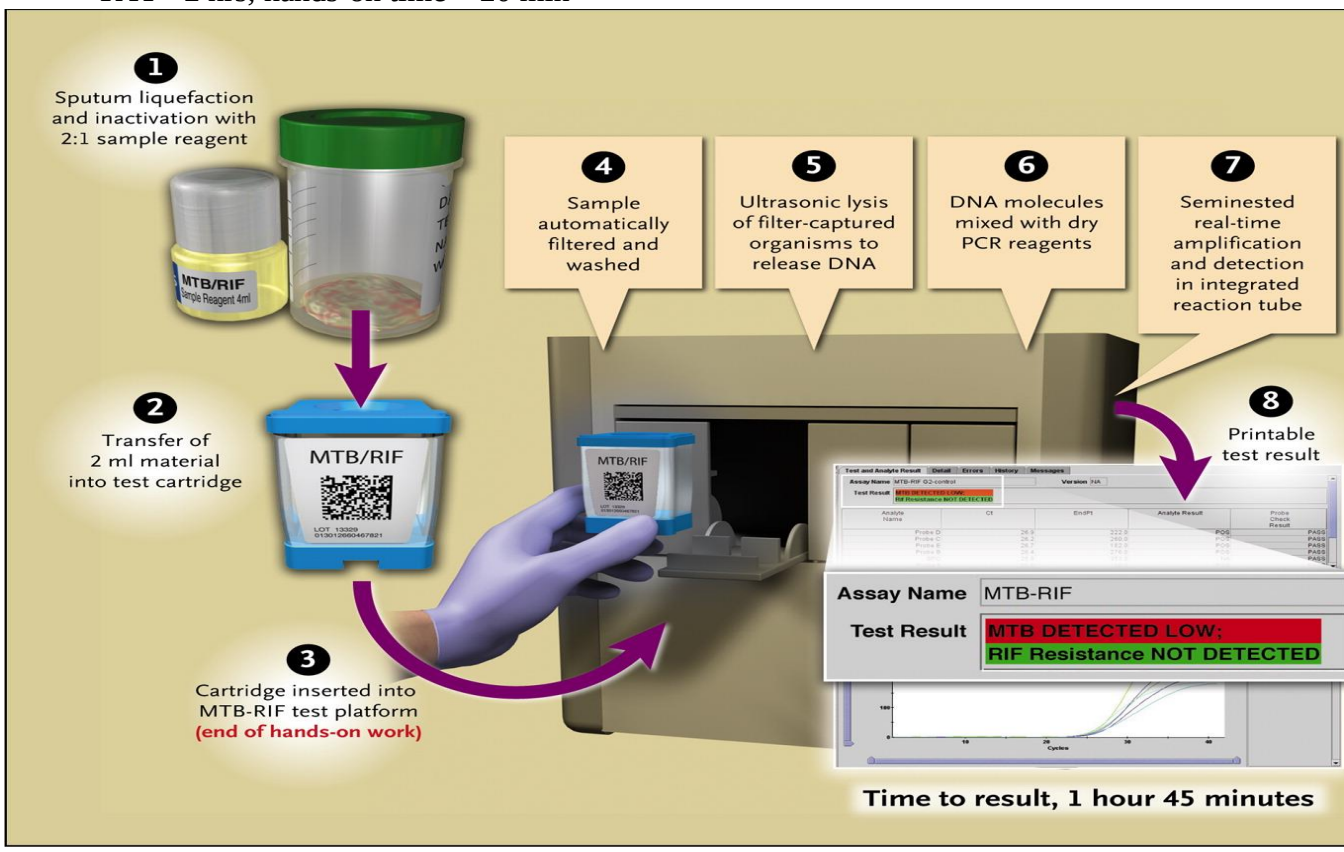
a.Diameter over 10 mm indicates positive reaction (i.e Previous exposure to tuberculosis in BCG or naturally, or active tuberculosis disease).

b.Diameter 5-10 mm is questionable i.e uncertain and so test must be repeated using 10 Tuberculin unities

c.Diameter of induration less than 5mm is a negative test.

3. **Radiography or CHEST X –RAY**; (especially for patient with negative microscopy and for children. Even if this test contributes to the diagnosis of tuberculosis, it is not enough to pose the diagnosis. Radiologic images suggestive of tuberculosis are not specific
4. **Biopsy** of the affected tissue
5. **Culture and sensibility test:** it is the most sensible and most specific which gives the certitude diagnosis of tuberculosis. But this technique is very difficult and requires 2 to 8 weeks for obtaining the results. The culture is actually indicated essentially for searching of multiresistence case (in case of suspection of multiresistence TB). This test is done by the Referral National Laboratory
6. **GeneXpert technology:**
   - Requires minimal training and lab infrastructures
   - Simple sputum processing steps

- Automated amplification and detection steps
- Includes internal amplification control
- Closed system
- Rapid
  TAT <2 hrs; hands-on time ~ 20 min




## 6. PREVENTION AND CONTROL OF TUBERCULOSIS

1. Treatment of the sick in order to cure illness and prevent spread of disease to others by killing Mycobacteria as efficiently as possible and within the shortest possible time.

a. **WHO** strategy for the treatment of TB is known as **DOTS,** i.e Directly Observed Treatment Short Course. Practically it means the following activities;

   i. Directly; identifying the sick, infections cases.
   ii. Observed; Health workers or trained volunteers observe the patients as they swallow the drugs
   iii. Treatment; The TB patient must be provided with complete treatment and monitored to assess progress until cured. There must be good reporting and record keeping
   iv. Short course; The correct combination and dosages or anti-TB drugs must be used for the correct length of time.

b. Combination treatment using two or more anti-TB drugs must be used at the same time to prevent resistance to one anti-TB drug.
c. Treatment of TB is done in two phases for a total of 6 months
    i.Intensive therapy phase, 2 months
    ii.Continuation therapy phase , 4 months

**INTENSIVE THERAPY PHASE;**

i.Combination of most powerful drugs is used to kill as many bacteria as possible as quickly as possible.

ii.A patient may be admitted or treated as out patient but seen daily and treatment supervised

iii.Intensive therapy phase lasts 2 months with daily intake of (RHZE)

iv.After intensive therapy phase patient is discharged from hospital to continue treatment at home supervised, but continuation phase ( 4 months). It is long and necessary to eliminate the persistant bacilli and to avoid the relapse or multiresistant TB.

v.Patient reports at health facility every month for being checked, and to get fresh supply of drugs

vi.Follow up period involves taking from smear positive patients sputum at month 2, and 5

**2. CHEMOPROPHYLAXIS**

- ***** Isoniazid (H) can be used for 6-9 months to prevent development of subclinical infection to active TB in exposed children and people who have recently become tuberculin positive , of the HIV positive. Isoniazid reduces the risk of active tuberculosis in infected people by up to 90%.

3. Immunoprophylaxis with BCG (Bacillus, Calmette-Guerin). It is a vaccine made of attenuated living Mycobacterium bovis. It is given to children during the first year of life, and adults at risk of infection
4. Case finding and treatment.
5. Health Education to enable people to avoid spitting anywhere carelessly, overcrowding and poor ventilation.
6. Isolation

**TREATMENT OF TUBERCULOSIS IN RWANDA**

## A. PRIMO-TREATMENT; Duration 6 months

**2 (RHZE)$_7$/ 4 (RH)$_7$** (Category 1)
Indicated for all new cases $TPM^+$, $TPM^-$, TPMO, TEP

**R**; Rifampicin **H**; Isoniazid **Z**; Pyrazinamide **E;** Ethambutol 275mg
The duration of treatment is 6 months divided in 2 phases.
**Intensive phase** lasts 2 months and must be supervised daily.
**Continuation phase** lasts 4 months with daily intake of drugs. At least 3 doses among 7 doses must be supervised

N.B. The treatment of extra pulmonary tuberculosis is the same as that of Pulmonary tuberculosis.

## B. RETREATMENT; Duration is 8 months

**2S$_7$ (RHZE)$_7$/ 1(RHZE)$_7$/ 5(RHE)$_7$** (Category 2)

**S**; Streptomycin (Injectable) . N.B. Streptomycin is contraindicated for pregnant women

The duration of the treatment is 8 months.
**Intensive phase** lasts 3 months and is daily supervised.
**The continuation phase** lasts 5 months and at least 3 doses among 7 must be supervised.

**INDICATIONS FOR RETREATMENT;**

-Relapse,-Failure,- Resumption of treatment (after abandonment):

**RELAPSE;**

Patient treated in the past and declared cured or having finished the treatment successfully, and whose examinations of expectoration are again positive.

**FAILURE:**

Patient whose sputum remain or become again positive in the 5$^{th}$ month or later during the treatment
(2 samples at least at 15 days of interval)

**Resumption of treatment (after abandonment**):

Patient who interrupted the treatment during 2 or more consective months and return again for treatment

### C. TREATMENT FOR CHILDREN

**2(RHZE)** $_7$**/ 4 (RH)** $_7$ **(**Category 3)

| Phase | months/dosage | drugs | Pediatric tablets | | | Adults tablets | |
|---|---|---|---|---|---|---|---|
| | | | 5-7 kg | 8-14 kg | 15-20kg | 21-30 kg | |
| Intensive | 2 months (56 dosages) | $(R_{60}H_{30}Z_{150})$ | 1 | 2 | 3 | $(R_{150}H_{75}Z_{400}E_{275})$ | 2 |
| | | $(R_{60}H_{60})$ | 1 | 1 | 2 | $(R_{60}H_{60})$ | 2 |
| | | $E_{100}$ | 1 | 2 | 3 | | |
| Continuation | 4 months (112 dosages) | $(R_{60}H_{30})$ | 1 | 2 | 3 | $(R_{150}H_{75})$ | 2 |
| | | $(R_{60}H_{60})$ | 1 | 1 | 2 | $(R_{60}H_{60})$ | 2 |

Ethambutol 100mg is given for children less than 5 years old according to indications: $TPM^+$, meningeal and bone tuberculosis.

- Meningeal tuberculosis and Bone tuberculosis: **2(RHZE)** $_7$**/ 10 (RH)**$_7$

### D. TREATMENT IN CASE OF MULTIRESISTANCE (MULTIRESISTANCE TB)

**6**$K_{m6}Pt_{o7}CS_7Of_{x7}Z_7$**/ 14**$Pt_{o7}C_{S7}Ofx_7$**. (**category 4)

**6** months taking Kanamycin injectable , Prothionamide, Cycloserine, Ofloxacin and Pyrazinamide followed by 14 months taking Prothionamide, Cycloserine and Ofloxacin.

N.B. This treatment is uniquely administered to a specialized unit with a specialized team (KABUTARE, KIBAGABAGA , KIBUNGO HOSPITAL, KINYINYA and BIRYOGO HEALTH CENTRES).

**N.B.** The quinolones (Ciprofloxacin, Ofloxacin) **must not be used** in the treatment of respiratory infections based on the possibility of creating resistance in Tuberculosis.

## 7. EXTRA PULMONARY TUBERCULOSIS

Definition and localizations

- Any case of TB apart from the pulmonary parenchyma.
- Preural, pericardium, peritoneum, ganglia, bone, articulations, meninges, internal organs, skin, miliary, etc.
- The most frequent forms: Ganglionic TB (neck and axillary), pleural TB, disseminated TB
- Less frequent: Pericardial and meningeal TB.

1. **GANGLIONIC TUBERCULOSIS.**

- Most frequent cause of adenopathies in the countries with high prevalence of TB
- Cervical ganglia are touched (65%)
- The adenopathies are unilateral or bilateral, asymmetrical, painless, adherent, initially elastic and then hard, cold, and chronic.
- Loss of weight, night sweats, fever

Essential examinations:

- Bacilloscopy if cough
- Bacteriological examination of the secretions or the liquid of aspiration of the ganglion (direct microscopic examination)
- Histological biopsy = > examination, culture

Treatment: Chemotherapy without waiting results of the culture.

2. **PLEURAL EFFUSION OR PLEURAL TUBERCULOSIS**

- By pleural attack with Mycobacterium tuberculosis via lymphatic way.
- Often unilateral and massive.
- Young adults more touched.
- Associated to PTB in 1/3 of the cases.

Diagnosis

Clinical signs: Dry cough, thoracic pain++ at often brutal beginning, reduction of vesicular murmurs, matity to the percussion.
Loss of weight, night sweats, fever

Radiologic signs and ultrasonography for confirmation.

Bacilloscopy
Pleural puncture = > clear liquid or citrine yellow which coagulates in the tube (exudate); rate of high Albumin (> 30gr/dl), low glucose , increased leucocytes with lymphocytes in abundance , the direct examination under the microscopy is not very sensitive 25%, culture 75%( late).
Pleural biopsy = > granulomata confirming the diagnosis

**Treatment:**
Chemotherapy
Corticotherapy at a rate of 40 mg/j by decreasing the doses gradually per a month.
Pleural drainage

## 3. THE MILIARY TUBECULOSIS

- Massive dissemination by circulatory way
- The lesions develop simultaneously in all organisms.
- Especially attacks the children and immunocompromized persons.
- Acute form of TB
- Non specific clinic: cough, dyspnea, slimming (weigth loss), sometimes adenopathies, gastro intestinal disturbance.
- Diagnosis is done on basis of Chest X-ray = > small shadowy nodules disseminated uniformly in the two pulmonary fields;
- Treatment: chemotherapy
- Bad prognosis without Treatment

## 4. **MENINGEAL TUBERCULOSIS**

Severe form of EPTB, more often reaches the young children not vaccinated and immuno-depressed

Clinical signs: general signs like the fever, lethargy, vomiting, photophobia, irritability, convulsions, neck stiffness, coma, fatal without treatment.

Diagnosis:
Lumber puncture = > liquid under pressure, clearly, increased proteins, decreased glucose, increased leucocytes (lymphocytes).
The direct examination for KB is not very sensitive, the culture is sensitive but late result (2-6 weeks).

**Essential examinations**:
HIV test
LP = > liquid under pressure, clearly, increased proteins, lowered glucose, increased leucocytes (lymphocytes).
Search KB in the CSF

Treatment:
As soon as possible without waiting the results of the culture.

## 5. **BONE TUBERCULOSIS**

- The attack of the bones and the articulations.
- Articular TB often touches the large articulations (hips and knees).
- The **Pott's vertebra**; theTB that touches usually the médio-thoracic vertebrae (by blood or lymphatic way)

- Clinic: Back pain, anterior erosion of the vertebral body = > compressing and kyphosis, possible paraplegia by medullar compression, para vertebral cold abscesses .

- Diagnosis: clinic, radiological image and tissue biopsy.

- Treatment: Chemotherapy and mobilization of the column, surgery if the rachis is unstable.

6. **UROGENITAL TB**

- Can touch any body organ of the urogenital system of the man and the woman.
- Renal TB is presented in the form of pyuria, microscopic hematuria
- The standard culture of purulent discharge is sterile ( sterile pyuria) , it is necessary to seek KB in the urines ( at least 3 early morning urine samples are taken to identify acid fast bacilli).
- Ultrasonography.
- The salpingite TB = > female sterility, ectopic pregnancy.
- In the man Tb touches the prostate, the blisters seminal vesicles, and the epididymis
- Diagnosis by culture of the urines.

7.**OTHER LOCALIZATIONS**

- O Pericardium:TB is responsible for 90% of the pericardial effusion among patients HIV positive and for 50 à.70% in the negative HIV.
- O Peritoneum and intestines
- O Larynx and bronchi
- O Eye
- O Skin

8. **MULTIRESISTANT TUBERCULOSIS**

**DEFINITIONS**

Resistant case: case of tuberculosis which is resistant to one or more antituberculosis drugs.
Multiresistant Case (Mr-TB):case resistant to Rifampicine and Isoniazid (the 2 most powerful antituberculosis drugs).

**CAUSES**

It is a problem created by the human.

**The treatment prescribed is inadequate:**

Insufficient dose, insufficient duration ,incorrect diagram according to the category of the case
**DOTS badly observed**; bad follow-up of the patients: consequently the patient does not regularly take his treatment for the necessary required time.
**Bad management of programme**: irregular supplying of drugs and out-of-stock condition
**The patient was infected starting from patient carrying resistant bacilli.**

**When suspect a case of multi resistance?**

- In the event of retreatment failure well taken (chronic case)
- When a patient does not improve clinically and remains positive in spite of a correct, regular and supervised treatment.
- When a case of tuberculosis is diagnosed among the contacts of a known multiresistant patient.

**8. COMPLICATIONS OF TUBERCULOSIS**

- Massive hemoptysia
- Pneumothorax; due to the rupture of cavern into pleural cavity
- Chronic respiratory failure

**The Difference between Latent TB Infection and TB Disease**

| A Person with Latent TB Infection | A Person with TB Disease |
|---|---|
| • Has no symptoms | • Has symptoms that may include:<br>- a bad cough that lasts 3 weeks or longer<br>- pain in the chest<br>- coughing up blood or sputum<br>- weakness or fatigue<br>- weight loss<br>- no appetite<br>- chills<br>- fever<br>- sweating at night |
| • Does not feel sick | • Usually feels sick |
| • Cannot spread TB bacteria to others | • May spread TB bacteria to others |
| • Usually has a skin test or blood test result indicating TB infection | • Usually has a skin test or blood test result indicating TB infection |
| • Has a normal chest x-ray and a negative sputum smear | • May have an abnormal chest x-ray, or positive sputum smear or culture |
| • Needs treatment for latent TB infection to prevent active TB disease | • Needs treatment to treat active TB disease |

**II.2. LEPROSY (HANSEN'S DISEASE):**

The ancients seats of leprosy in Rwanda are: Gisagara (Kirarambogo H.C), Rubavu (Nyundo H.C), Bugesera, Rusizi (Masheshe H.C), Ngoma (Jarama H.C), Karongi (Mugonero H.C, Kibuye H.C), Nyaruguru ( Nyamyumba, Kabirizi, Ruramba and Ruheru Health centers).

## A. DEFINITION AND ETIOLOGY

It is an infectious disease caused by **Mycobacterium Leprae (HANSEN BACILLI).** It attacks the skin, mucus and the nerves. Untreated, it causes the disabilities.

Mycobacterium Laprae are weakly acid-fast and alcohol-fast bacilli in Ziehl-Nielsen stain. When stained they appear as thin pink rods.

Have slow growth with generation time of 20 days in experimental animals.

M.Leprae has not been successfully cultivated (cultured) on any bacteriological media or tissue culture

In humans incubation period **is 5-8 years**, and the disease is chronic. It affects the skin and the peripheral nerves.

## B.RISK FACTORS FOR LEPROSY INFECTION

i. Staying with an infectious patient in a poorly ventilated room

ii. Staying in contact with a patient who is not being treated or has not completed treatment

iii.Overcrowding. This increase the chance of catching the infection from many other people.

## A. RISK FACTORS FOR DEVELOPING LEPROSY DISEASE AFTER BEING INFECTED.

- Poor general nutrition and health status (i.e influence of poverty)
- Not having been vaccinated with BCG
- HIV infection presence
- Recent measles
- Metabolic disorders e.g diabetes mellitus

## B. TRANSMISSION OF REPROSY

- The mode of transmission of leprosy is not clearly understood, but it is thought to be due to prolonged direct contact with infectious people through;

  **1.** Prolonged direct contact with infectious by
     a. Droplet infection by sneezing, coughing, spitting, unhygienic nose cleaning habits.
     b. Broken(i.e breached) skin

- Once in the body leprosy bacilli divide inside macrophages of the skin and in nerve fiber Schwann cells
- Leprosy is a disease that is not easily caught by other people, mainly because of natural resistance to Mycobacterium leprae (i.e cellular immunity)
- That is when people are exposed to leprosy bacteria,

  -Most (i.e 75%) of people do not develop leprosy

  -Only some develop leprosy, a few of whom develop non-infectious leprosy, and still fewer develop infections type of leprosy.

## E. CLINICAL FEATURES OF LEPROSY

Leprosy is categorized into 4 clinical forms determined by the level of cellular immunity of the individual as follow;

### 1. INDETERMINATE LEPROSY

Features are;

- Lesions(1-3) appear as small discolored(i.e hypopigmanted) areas (i.e macules)
- Lesions are called indeterminate because they do not indicate how they will continue to develop
- Most lesions heal spontaneously and many lesions are never noticed
- No loss of sensation or it is slight
- No bacilli are found in smears
- Organs and nerves are not involved

- Lesions which do not heal may develop into tuberculoid, borderline or lepromatous leprosy.

### 2. TUBERCULOID LEPROSY

Features are;

- Skin lesion patches are few, raised or flat large or small, with dry surface
- There is some loss of feeling due to damage of peripheral nerves
- Loss of hair and of sweating
- Patches show central healing
- Patches have marked hypopigmentation
- Cutaneous nerves may be enlarged under patches of lesions
- Paralysis is common
- Skin smear is negative
- It is seen in patients with high degree resistance(i.e cell mediated immunity)
- Most common in dark skinned people than white (light) skinned people.

### 3. LEPROMATOUS LEPROSY

Features are;

- Skin lesions are numerous , small, a slightly raised or flat patches, macules, or nodules
- Skin may appear thicker than normal especially on face and ears
- No loss of hair
- No loss of sweating
- No loss of sensation
- Nerves are affected later and get thickened
- There is positive skin smear
- Other organs are involved including eyes, hands, feet, testicles, nose, fingers, teeth loss
- Paralysis due to damage of motor neurons

### 4. BORDERLINE LEPROSY

- Has both features of tuberculoid and lepromatous leprosy
- Skin lesions are very many, raised or flat
- There is some loss of feeling
- Nerves are affected very early
- Smears range from negative to positive

## CLINICAL DIAGNOSIS

**A.** Demonstrate hypopigmented patches with loss of sensation;

i.1-5 patches means **paucibacillary leprosy** characterized by

-Having scanty (inadequate) bacilli in smears

-Being non-infectious e.g. Indeterminate and Tuberculoid forms

ii.6 or more patches means **multibacillary leprosy** characterized by

-Having many bacilli in smears

-Being infectious e.g Borderline and Lepromatous leprosy

**B.**Abnormally large peripheral nerves at points of predilection(i.e points mostly favoured by the infection) e.g facial nerves, trigeminal nerves, greater auricular nerves, ulnar nerve, median nerve, radial nerve ,peroneal nerve and posterior tibial nerve

## F. LABORATORY DIAGNOSIS

It is based on finding acid-fast bacilli in the skin smears.

a.All positive smear cases, and Borderline leprosy cases that have negative smears are considered multibacillary e.g Lepromatous and Borderline leprosy

b.All other negative smears cases are considered paucibacillary. E.g Indeterminate and Tuberculoid leprosy

## G. PREVENTION AND CONTROL OF LEPROSY

A. **Treatment and management**

i.Early diagnosis and treatment as well as management of reactions prevent all disabilities.

Treatment recommended by WHO combine several drugs which are taken at the same time. This form of treatment is called Multidrug therapy (MDT).

**Table to show MDT for pauci-bacillary Leprosy according to WHO; Duration 6 months**

| MDT | 0-5 YEARS | 6-14 YEARS | 15YEARS AND OVER |
|---|---|---|---|
| 1.**DAPSONE** daily<br><br>-Self administered (or by mother) at home (and) | 25mg | 50mg | 100mg |
| 2.**RIFAMPICIN** | 300 mg | 300mg | 100mg |

| | | | |
|---|---|---|---|
| once in 4 weeks, supervised (i.e. Once monthly) | | | |

Treatment should be done as close as possible to where patients live

**Treatment schedule for Multi-bacillary Leprosy; Duration 12 months**

| **MDT** | **0-5 YEARS** | **6-14 YEARS** | **15 YEARS AND OVER** |
|---|---|---|---|
| **1.DAPSONE** daily<br><br>-Self administered<br><br>(and) | 25 mg | 50 mg | 100 mg |
| **2.RIFAMPICIN**<br><br>-Once in 4 weeks, supervised (and) | 300 mg | 300 mg | 600mg |
| **3.CLOFAZIMINE**<br><br>-Once in 4 weeks,<br><br>Supervised (and) | 100 mg | 200 mg | 300 mg |
| **4.CLOFAZIMINE**<br><br>-Self administered | 50 mg<br><br>Alternating days | 50 mg<br><br>Daily | 50 mg<br><br>Daily |

i. Prevention of injury, infection and deformity by

   c. Proper care of the skin of patient
   d. Proper care minor wounds and skin infections
   e. Provision of shoes
   f. Physiotherapy
   g. Health education

ii. Case finding among the contacts of Leprosy patients through school and community surveys
iii. Improvement in housing, social economic status
iv. BCG immunization is slightly protective but no effective vaccine as yet

**H.COMPLICATIONS**

- In severe cases**, disfigurement and deformities** occur in the hands, feet and face.
- Blindness.

- Testicular atrophy and sterility
- Liver involvement may manifest as gynaecomastia
- Paralysis
- Injuries: due to loss of sensibility

What can you do when you suspect a case of leprosy:

1. Interrogate the person
2. Examine the skin
3. Test the sensibility of the spots
4. Inform the supervisor of PNILT for confirming the diagnostic.

   Attention: PB leprosy with positive test: is classified in MB leprosy

## II.3 TYPHOID FEVER (SALMONELLOSIS}

a. **DEFINITION:** Typhoid fever or Salmonelosis is a contagious disease caused by Salmonella typhi or Eberth bacilli and Salmonella paratyphi A, B or C. It is a sepsis originated in intestines.

   The Salmonella are Gram negative bacilli and allocated in Enterobactor family.

   They produce three kinds of antigens.

- Antigen O; Flagellar
- Antigen H; Somatic
- Antigen Vi; Capsular

Both antigen O and antigen H serve in Serological Diagnosis called Serodiagnosis of **Widal and Felix.**

The serodiagnosis of Widal and Felix is positive since $8^{th}$ day for antigen O and $12^{th}$ day for antigen H. The body produces **anti O antibodies and anti H antibodies** as a body defense mechanism. These antibodies are detected during Widal and Felix serodiagnostic test.

Anti O antibodies disappear in blood after 2 to 3 months, while anti H antibodies persist within years.

Antigen Vi, virulent, is used in vaccination (immunization]

## b. EPIDEMIOLOGY

The reservoir of bacteria is the sick or convalescent human and the carrier. The last can harbor germs for months or years in his bile ducts. The individual eliminates the germs through feces and urine and the contamination occurs via the gastrointestinal tract by ingesting food and water contaminated by infected feces or urine from cases and carriers. The role of dirty hands and flies is very important

## c. PATHOGENESIS

In brief: The germs multiply in the lymphoid tissue of the small intestine and in mesenteric lymph nodes which lead to mesenteric adenolymphititis .By there they pass the lymphatic system into the blood stream where bacteremia, septicemia occur. Then they move into kidney and the liver from where they pass out in urine and faeces respectively.

In detail: the bacteria in contaminated food or water are swallowed and if they resist to the action of hydrochloric acid of gastric secretion, they arrive in the small intestine where they multiply and invade the intestinal mucosal barrier. If the typhoid bacilli can not resist to the bacterial competition, typhoid infection can not occur

If the multiplication of bacilli is sufficient to overcome the activity of phagocytic cells of the intestinal mucosa, pathogens can penetrate the mucosa, During this period, the patient is asymptomatic but the typhoid bacilli are excreted in feces.

- After germs penetration of the intestinal mucosa, these germs that have passed the intestinal barrier stop and multiply in the mesenteric lymph nodes, which currently increase in volume and present signs of acute inflammation ( redness, swelling, pain and heat].
- The most important lesions are found at the level of intestinal lymphoids formations (in PEYER patches i.e Lymph nodules in the lower part of small intestines where the typhoid bacilli multiply) which become necrotic. These lesions may be responsible for severe life-threatening complications (intestinal perforation, gastrointestinal hemorrhage).
- From these nodes, the infection spreads through the lymphatic route and then through the blood circulation, where septicemia occurs. Then, the blood culture becomes positive.

## d. PATHOPHYSIOLOGY

- Most of the pathological phenomena of typhoid fever are caused by the action of **endotoxin** released from killed bacteria (lysis in lymph nodes) where the signs of the disease occur.
- The most important physiological problem and most serious is the **endotoxinic septic shock** which can appear during the disease. Sometimes, the signs of endotoxinic shock occur soon after starting treatment; in this case, it is precipitated by the use of high doses

of chloramphenicol at the beginning of treatment, these doses determinate the rapid death of large numbers of germs with the release of significant amounts of endotoxin.

## e. CLINICAL SIGNS

**Incubation**: 10 to 15 days, biologically detectable, contagious since 2nd day.

**1st week; Invasion period,** there is

- Severe Headache
- Asthenia
- Vertigo
- Insomnia
- Constipation
- Epistaxis
- The fever increases progressively up to 40 $^{o}$ c at the end of the week while the pulse accelerates slowly(dissociation pulse-T$^{o}$)

**2nd week, State period**

- The temperature remains on top with pulse-temperature dissociation.
- **Tuphos state**: the patient is completely adynamic, uninterested to everything that surrounds him, prostration, delirium, sometimes the patient can be agitated and aggressive.
- Abnormal movement e.g. shaking of extremities
- Diarrhea ; color jus de melon
- Mild abdominal swelling (meteorisme abdominal]
- Rose spots occur on the trunk of 40%
- Splenomegally occur in 30 to 50% of the cases
- The right iliac fossa is gurgling
- Abdominal pain

**3rd week**: **Defervescence phase or resolution**

- The fever decreases progressively and other signs disappear progressively , general state of the patient is improving
- However ,it is during this phase that the complications may occur

**4th week**: **convalescence phase**

-It is often long and marked by asthenia

## g. COMPLICATIONS

There are many and related to 2 mechanisms;

1. **Septic complications;** due to bacteria dissemination

- Hepatic abscess
- Hepatitis
- Cholecystitis
- Purulent meningitis
- Osteomylelitis and arthritis

2. **Complications due to endotoxine;** they ere more frequent

- **Intestinal hemorrhage;** 2 to 6% of cases, due to ulcerations of Peyer patches. It is marked by a drop in temperature, a rapid pulse and a drop in blood pressure. The patient may fall in **cardiovascular collapses(** the temperature fall brutally from 40 to 35 degree celecius, pulse very high increased, BP decreased with undetectable minimum, cold extremities}
- **Intestinal perforation**: 2 to 3% of the cases. It is often accompanied by abdominal pain, vomiting and in the long time signs of **peritonitis**. This perforation occurs often silently and its prognosis is very serious
- **Myocarditis**, which may complicates into cardiac failure. It is characterized by dyspnea, palpitations, precordial pain, and rapid heart rate.

- **Neurologic complications;** encephalopathy with coma

## g. CLINICAL FORMS OF TYPHOID FEVER

- ✓ **Mild or attenuated form**: the signs are restricted, there is no tuphos.
- ✓ **Severe forms**: there is a choleralike diarrhea, nervous disorders (coma,). Tuphos state is present, hemorrhage. It is often fatal
- ✓ **Relapsing forms**; it is manifested as attenuated form of the disease. It is like as there are forms with incomplete treatment.

## h. DIAGNOSIS

- Clinical diagnosis; see above
- Biologic diagnosis;
  -Full Blood Count (FBC]; Leucopenia with neutropnea
- Certitude diagnosis; for isolating the germ
  **-Hemoculture**; (blood culture] positive during the $1^{st}$ and $2^{nd}$ week, before antibiotherapy
  **-Coproculture**; stool culture
  **-Serodiagnosis of Widal and Felix**; it detects the anti O and anti H antibodies. Problem of interpretation because it is positive for those who were vaccinated (only for anti H antibodies].

**i. TREATMENT**

Etiologic treatment; Frequently used ANTIBIOTICS;

1. CHLORAMPHENICAL IV 3x1gr/ day for adults
2. CIPROFLOXACIN Per os, 20 to 30 mg/ kg/ day two times a day during 10-15 days
3. CEFOTAXIME in IV 100mg/ kg/ day and 3x 1gr/ day for adult
4. CEFTRIAXONE in IV 100mg/ kg/ day and 2x2gr/ day for adult

Other treatment;

1. Corticotherapy
2. Infusions ( rehydration}in case of shock or in case of fever persistence above 5 days
3. Sedation ( PHENOBARBITAL in case of prolonged insomnia]
4. Symptomatic treatment; analgesic, antipyretic,..
5. Surgical treatment in case of intestinal perforation

Hygieno-dietetic treatment;

-

- ✓ Bedrest
- ✓ Rigorous oral and body hygiene measures for any contagious patient.
- ✓ Regimen: it must include easily digestible food, more or less liquid or semi-liquid.

Surveillance of;

- BP, pulse, temperature
- Abdominal exam
- The state of the stool
- Anemia due to CHLORAMPHENICAL treatment

**j. PREVENTION**

i. Carriers must not be allowed to work as food handlers
ii. Provision of good water supply
iii. Improvement in hygiene
iv. Vaccination
v. Patient isolation and disinfection

N.B. -Avoid the laxative drugs and enema because they may precipitate intestinal perforation

-Avoid oral drugs in case of intestinal hemorrhage and give only liquid food in small quantities

II.4. **THE TETANUS**

## a. DEFINITION AND ETIOLOGY

**Tetanus** is a severe toxi-infection due to **Clostridium tetani or Nicolaier bacilli (anaerobic bacilli and Gram positive** characterized by muscle spasms and rigidity (Cardinal features of tetanus]. This anaerobic germ lives in the soil as resistance form (spore) for many years. It is found in the intestines of animals where lives as a saprophyte and is eliminated with the faeces of these animals.

## c. PATHOGENESIS;

Spores of Clostridium tetani live in faeces, soil, dust and on instruments. A tiny break in skin or mucosa, e.g cuts, burns, ear piercing may admit the spores. Spores may then germinate and only germinate in relatively hypoxic tissue such as necrotic or ischaemic tissue, or tissue surrounding a foreign body. As clostridium tetani grows, it produces at least two exotoxins (**Tetanospasmin and Tetanolysin**). The role of tetanolysin in human is unclear but it may promote growth of clostridium tetani or contribute to the autonomic dysfunction. Tetanospasmin causes the clinical manifestation of tetanus by travelling up peripheral nerves and by inhibiting neurotransmitter release from a presynaptic terminal of nerves and interferes with inhibitory synapses.

## EPIDEMIOLOGY AND PATHOLOGY

The transmission is direct and requires portal of entry:

- Skin wounds, even very small bites such as needles, thorns, which even have already healed. Also mentioning injections, surgical interventions, trauma, etc.
- The umbilical wound in the newborn
- The uterine cavity (post-partum, post-abortion)
- Accidental injuries even small neglected wounds ; 40 to 60% of the cases
- Chronic wounds; 10% of the cases

The germs once entered the body, they remain localized in the wound and secrete **neurotropic toxins** wich spread throughout all the body and cause the disease manifestations.

## d. CLINIC

- **The incubation period**; it is very variable; 4 to 30 days. As the incubation is short more the disease is severe.

- **The invasion period**

-**Trismus**; intense, painful permanent contracture of the masseters joining two jaws. This opposes to any opening of the mouth, feeding and speech are difficult because of the contractions of muscles responsible for mastication (masseters].

The presence of this sign must always make you to think about tetanus even in the absence of any local lesion and try to find the portal of entry.

**-Dysphagia;** difficult swallowing

- **At the state phase :**

  - Generalized contractures. They are intense, permanent and painful and interest:

  The face: where the risus sardonicus occurs (rire sardonique]

  The neck; stiff neck occurs

  The abdomen; abdominal muscles contractions (stick abdomen]

  The limbs; remain in extension

  The back muscles: opisthotonus attitude (arched body with hyper extended neck]

  These spasms are exaggerated by paroxysm induced by movement, injections, noise, light,...and they are very painful while the client is lucid (coherent].

These spasms may interest the viscera and can cause constipation, apnea, dysphagia, dysuria and even spasm of the larynx which can asphyxiate the patient and can be fatal if they don't proceed immediately to the tracheotomy.

**e) EVOLUTION:**

It is often fatal, especially if the treatment was delayed and in severe cases.
Favorable evolution is rare
**Untreated;** deaths in 80-90% of the cases
**Treated;** mortality is 50% in average

**f.International Classification of Tetanus (according to gravity]**

| **CRITERIA** | **SCORE 0** | **SCORE 1** |
|---|---|---|
| **1.Incubation** | ≥ 7 days | **< 7** days |
| **2.Invasion** | ≥ 2 days | **< 2** days |
| **3.Portal of entry** | others | Umbilical, IM,open fracture, surgery, uterus, wide burn |
| **4.Paroxysm** | 0 | Present |
| **5.Pulse** | ≤ 120 for adult | >150 for child |
| **6.Fever** | Normal | >38.4 degree celecius |
| **TOTAL SCORE; 0 to 6** | | |

| | | |
|---|---|---|
| **Tetanus Stage I** | **Score 0,1** | **Mild Tetanus** |
| **Tetanus Stage II** | **Score 2,3** | **Moderate Tetanus** |
| **Tetanus Stage III** | **Score 4,5,6** | **Severe Tetanus** |

### h. COMPLICATIONS

- **Respiratory complications**
  - Laryngeal spasm (apnea or respiratory distress)
  - Bronchial congestion
  - Aspiration bronchopneumonia
  - Bone fracture, especially vertebral fractures, muscle rupture.

- Other complications
  - Convulsions
  - Urinary Tract Infections by stasis or urinary catheterization
  - Dehydration
  - Renal failure
  - Poor nutrition
  - Cardio-vascular accident;
    -Cardiac arrest
    -Emboli or cardio- vascular collapses

### h. PARA CLINICAL EXAMINATION

There is no required examination for diagnosis

### i.NEONATAL TETANUS

**Portal of entry;** Umbilical (especially for home delivery]
Clinical signs occur from the $6^{th}$ to $7^{th}$ day with;
-Impossibility to breastfeed due to the contraction of the masseters
-Characteristic Risus sardonicus
-Opisthotonus attitude
These 3 signs are enough for Tetanus diagnosis
**Evolution;**
*** Untreated;** 100% of mortality
***Treated;** 60% of mortality

### g) TREATMENT

✓ **Preventive Treatment**

* First: vaccination
* The serum therapy: the antitetanic serum is required before any suspected wound. It is effective if done early, it must be associated with vaccination or boosters shots if the subject has already been vaccinated.

✓

This serotherapy may be accompanied by serious accidents and even fatal; **Anaphylactic shock**. To avoid this accident, they use BESREDKA method: it involves injecting gradually small doses in subcutaneous injection: First they give a quarter of the dose, 15 minutes after a half of the dose, another 15 minutes after 1 cc and finally 15 minutes after the rest of the dose. During this period they must monitor the alarm signs.

*Emergency treatment for any suspected traumatic wound: surgical cleaning of the wound (debride the wound) .Oxygenated water is better to kill anaerobic germs.

N.B: what to do before an injury:
* If the client has been properly vaccinated and that the booster shots is less than one year, nothing to do.
*If the booster shots is more than 1 year and less than 5 years, the booster shots is given again

*If the client has not been vaccinated or if the vaccination is longer than 5 years, **serovaccination** ( combination of serum therapy and vaccination} is practiced.

**Curative treatment**

- Obligatory **hospitalization** in the **dark room** without any **light or noise**.
- General antibiotherapy; **IV PENICILLIN G or METRONIDAZOLE IV** and apply also local care of the wound
- Give **human tetanus immune globulin** to neutralize free toxin or give **horse antitetanus serum** (ATS} if available
- Stop the contractures by giving the **myorelaxants** ; the more used are **DIAZEPAM** and the **PHENOBARBITAL**
- **Respiratory reanimation ;** intubation and/ or tracheotomy

**N.B. *** Don't give food orally in case of dysphagia

* Insert a nasogastric tube (NGT] (obligation for a new born] for liquid or semi-liquid feeding

*Infusions

*Nursing; hygiene,…

**For Neonatal Tetanus;**

- Calm the baby with a half ampoule of Diazepam( 5mg) in IM
- Insert a NGT for feeding and administration of sedative drugs( Diazepam and Phenobarbital).
- Care of the umbilical cord
- Artificial milk feeding; 60 to 100 cc , 6 times per day, and increase the quantity as far as the baby grows

**II.5 MENINGITIS**

**a. DEFINITION**

**Meningitis** is the inflammation of the arachnoid and pia mater (meninges) of the brain and spinal cord and the cerebrospinal fluid (CSF).

- Meningitis is an infection which causes inflammation of the membranes(meninges) covering the brain and spinal cord.
- Meningitis is an in infection and inflammation of the meninges and cerebrospinal fluid surrounding the brain and spinal cord.

MENINGES

- Connective tissue membranes that separate the skull from the brain.
- This three-layered
- Dura mater ("hard mother"),

- Arachnoid mater

**subarachnoid space**

- Pia mater ("soft mother").

- Between the arachnoid and the pia mater is the **subarachnoid space,** which contains cerebrospinal fluid (CSF), the tissue fluid of the central nervous system.
- Recall the ventricles (cavities) of the brain: two lateral ventricles, the third ventricle, and the fourth ventricle.
- Each contains a choroid plexus, a capillary

network that forms cerebrospinal fluid from blood plasma

The organisms responsible for meningitis enter the central nervous system (CNS) via the bloodstream at the blood brain barrier (BBB).

BBB is the barrier that selects which substances reach the brain. The BBB is both a physical barrier and a system of cellular transport mechanisms. It helps to maintain the delicate HOMEOSTASIS of neurons in the brain by restricting the entrance of harmful substances from the blood, and by allowing the entrance of essentials nutrients.

The invading organisms migrate throughout the CNS via the subarachnoid space. The presence of organisms in the subarachnoid space produces an inflammatory response in the pia mater, the arachnoid, the CSF, and the ventricles. The exudates formed may spread to both cranial and spinal nerves, causing further neurologic deterioration.

**b. TRANSMISSION OF MENINGITIS**

The organisms are commensals in the nose and pharynx of people. So, they are spread and caught by droplet method (i.e Airborne). In a susceptible host they become invasive i.e they enter the blood stream and reach the meninges of the brain, they infect, and cause pus that forms on surface of brain. The route of transmission is represented by the **Upper Respiratory Tract.**

**c. Predisposing factors**

- Head trauma
- Immunosuppression
- CSF fistula/leak
- Neurosurgical patients
- Alcoholism
- Congenital defects
- Local infections:
  **a. Sinusitis**
  **b. Otitis media**
  **c. Pharyngitis**
  **d. Bacterial pneumonia**
- Splenectomized patients

* Classically, there are **two major types** of meningitis:

**1. Septic meningitis or Bacteria meningitis or Purulent Meningitis (purulent CSF)**
**2**. **Aseptic meningitis or clear liquid Meningitis (clear CSF)**

**1. SEPTIC OR PURULENT MENINGITIS**

This is the bacterial infection of the meninges (pia mater and arachnoid) and the CSF in which the Lumbar Puncture brings the purulent liquid.
Bacterial meningitis is an acute purulent infection within the subarachnoid space.
It is associated with a CNS inflammatory reaction that may result in decreased consciousness, seizures, raised intracranial pressure (ICP), and stroke.
It is considered **as serious.**

**a.Route for entry**

- Haematogenous
- Anatomic defect

*Congenital
*Traumatic
*Surgical

- Intraneural patways

**b. Pathogenesis**

- Colonization of the oropharynx → bacteremia → CNS invasion
- Local infection (pneumonia, endocarditis, UTI) → bacteremia → CNS invasion
- Direct CNS invasion from trauma, sinusitis, mastoiditis, or surgery

- The lysis of bacteria with the subsequent release of cell-wall components into the subarachnoid space is the initial step in the induction of the inflammatory response and the formation of a purulent exudate in the subarachnoid Space====purulent CSF

**c. Responsible germs;**

The most frequently organisms involved in bacterial meningitis are;
* MENINGOCOCCI (**Neisseria Meningitidis**); Gram negative Diplococci
* PNEUMOCOCCI (Streptococcus **Pneumonia**); Gram positive Diplococci
*HAEMOPHILUS INFLUENZA **(Haemophilus Influenza**); Gram negative bacilli
These 3 germs represent 80% of purulent meningitis.
The Meningococci (Neisseria Meningitidis) is less virulent but an epidemic type. Its contagiosity is very high.

**d. Principal germs in function of age**;

**1. Neonates (0-2 weeks);**
* Escherichia coli
* Streptococcus, GroupnB
*Listeria Monocytogenes
*Staphylococcus aureus
*Streptococcus, Group A

**2. Infants (½ - 3 months)**
*****Streptococcus, Group B
*Listeria monocytogenes
*Escherichia coli

**3. Children of 3 months to 6 years;**
* Often Haemophilus Influenza
*Staphylococcus aureus
*Neisseria meningitidis
*Streptococcus pneumoniae
*Mycobacterium tuberculosis

**4. Adults and Children > 6 years;**
*****Meningococci (**Neisseria Maningitidis**)
*Pneumococci (**Streptococcus Pneumonia**)

**5. Old persons;**
*****Enterobactors and Pneumococci

**-S. pneumoniae** ==pneumococcal meningitis
Severe meningitis------coma but vaccine exist

**-N. meningitidis**: meningococcal Meningitis: an epidemic meningitis (petechial or purpuric skin lesions)

**-L. monocytogenes** is acquired by ingesting foods contaminated by Listeria.

**-Staphylococci are** important causes of meningitis that follows invasive neurosurgical procedures

**e .CLINICAL SIGNS OF MENINGITIS**

Four syndromes define meningitis

- **Infectious syndrome**
- **Meningitidic syndrome**
- **Encephalitic syndrome**
- **HTIC syndrome (Hypertension Intracranial Syndrome)**

*** INFECTIOUS SYNDROME;**

-Hyperthermia of 39 -40 $^{o}$C
-Chills (due to venular constriction)
-Increased pulse
- Increased heart rate
-Labial Herpes
- Body malaise
-Myalgia or polyarthralgia
-Asthenia

***THE MENINGEAL (MENINGITIDIC) SYNDROME**

**.SIGNS/MENINGEAL IRRITATION**

-Stiff neck or Nuchal rigidity

-**Positive Kernig's sign;** the patient cannot fully extend his knee while the hip is flexed at 90 degrees. Inability to stretch leg at knee joint when thigh is at right angle(90 degree). When the knee is flexed at 90 degree, the client feels pain when you attempt to extend the knee.

-**Positive Brudzinski's sign;**

**1.** Passive flexion of one thigh causes spontaneous flexion of the opposite thigh

**2**. Passive flexion of the neck causes bilateral flexion of hips and knees

- Opisthotonos

**.SYMPTOMS**

-Severe headache

-Nausea and Vomiting

-Photophobia

**For infants;**

-Absence of stiff neck, but generalized hypotonia

-Digestive signs at the first plan: eg.vomiting

- Convulsion

-Refusal of breastfeeding

-The anterior fontanelle (i.e membranous space between parietal and frontal bones) may bulge (swell)

* **ENCEPHALITIC SYNDROME**

- Alteration in mental status
- Seizure/Drowsiness (sleepness)
- Delirium
- Stupor
- Coma

* **HTIC SYNDROME**

- Headache
- Projective Vomiting
- Diplopia(nerve VI)
- Mydriasis(nerve III)

**CUSHING'S TRIAD**

A response involving three classic signs:

- Widening pulse pressure: increased systolic BP with diastolic remaining the same or slightly elevated.
- Bradycardia
- Slowing respirations

Cushing's triad indicates increased severe ICP!

- Decorticate posturing

**POSITIVE dx**

The classic triad of acute bacterial meningitis consists of

- Fever,
- Nuchal rigidity, and
- A change in mental status
- Seizures

**N.B. 1. Signs of meningeal irritation**

**Nuchal rigidity “stiff neck”)**

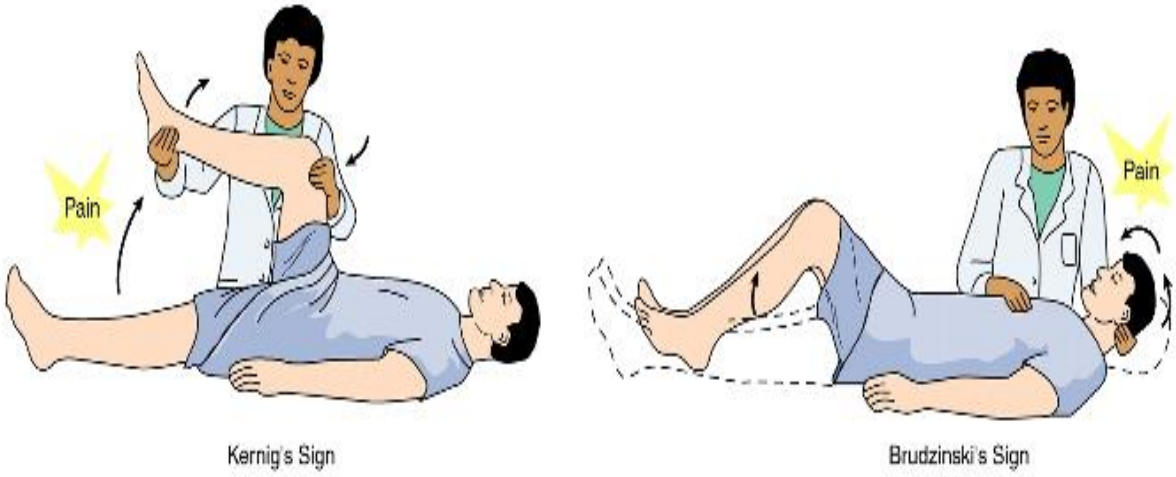
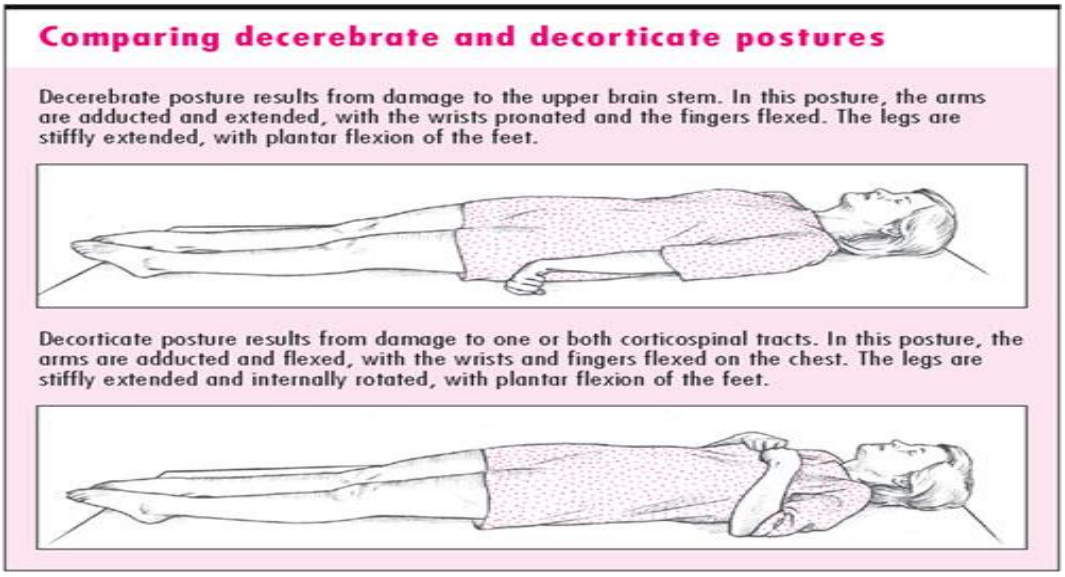



**Figure 64-1** Testing for meningeal irritation. (A) Kernig's sign. (B) Brudzinski's sign.



- **Brudzinski's sign:** spontaneous hip flexion during passive neck flexion
- **Kernig's sign:** the patient cannot fully extend his knee while the hip is flexed at 90 degrees.

2. **Decorticate posturing:** Abnormal flexion to pain



## f. DIAGNOSIS

The diagnosis of bacterial meningitis is made by

- **History and physical examination**
- **Lumbar puncture +CSF analysis**
- **Laboratory findings**

***CSF analysis (microscopic)**

**-Cytology (**blood cells: WBC**)**
**-Biochemistry (**glucose, protein, lactic acid**)**
**-Bacteriology (**CSF stains, culture**)**
**-Ph**

- **Lumbar Puncture** (LP) is done to obtain Cerebrospinal fluid (CSF) to be analyzed. This should be done before any antibiotherapy. The Lumbar Puncture is done with the patient in a flexed position to maximize the space between vertebrae. The lumbar puncture needle isusually inserted between **L3-L4** or **L4 and L5** to gain entry to the subarachnoid space where the CSF is located.

- N.B. Lumbar puncture must be done when no sign of increased intracranial pressure is present.

**ANALYSIS OF CSF;**

- **Aspect of the liquid;**

  - It flows under pressure,-Purulent, -Trouble, -Sometimes yellow

- **Cytology of CSF:**

  -Increased WBC: Neutrophils in abundance

- **Biochemistry of CSF**:

  -Increased protein in CSF

  - Decreased glucose in CSF

  -Increased Lactic acid in CSF

- **Culture**:

  - Meningococci

  -Pneumococci…

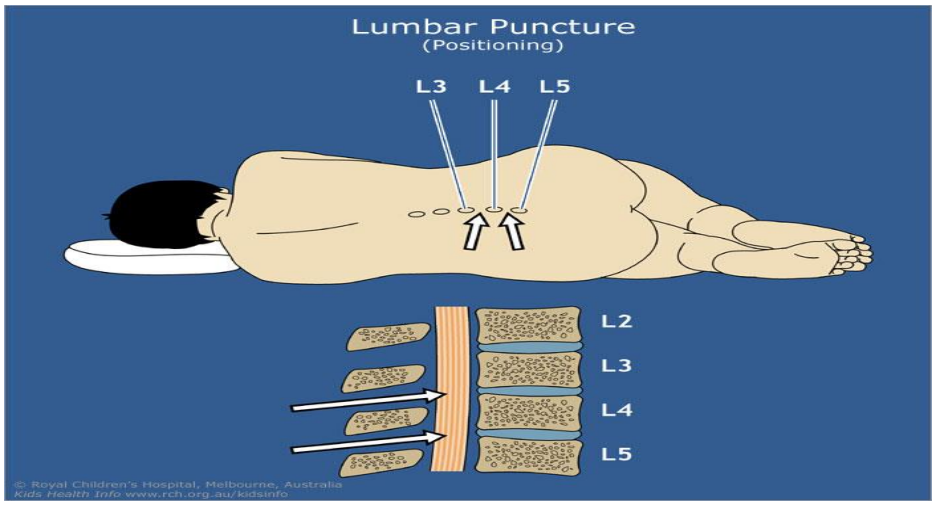
. **Ph**: decreased

**CSF changes in bacterial meningitis**

| COMPONENT | NORMAL CSF | BACTERIAL MENINGITIS |
|---|---|---|
| Glucose | 30-70mg/ dl | <50mg/dl |
| Protein | <50mg/dl | >150mg/dl |
| WBC | <5/$mm^3$ | >1200/$mm^3$ |
| $p^H$ | 7.3 | 7.1 |
| Lactic acid | <14mg/dl | >35mg/dl |

**Diagnostic test for meningitis: Lumbar puncture**

**Note:** Needle is usually inserted between **L3-4, or L4-5**



- A Lumbar Puncture is an invasive diagnostic test, in which CSF is extracted for examination, and the pressure of the spinal column fluid is measured.

**.** Permits the urgent distinction of bacterial meningitis from viral meningitis and examination of the CSF allows precise diagnosis.

**CSF CULTURE**

- CSF culture is much less sensitive after antibiotics are given.
- Neisseria meningitidis will disappear from CSF within one hour of appropriate antibiotic therapy.
- Strep pneumoniae will disappear in 4-10 hours after appropriate antibiotics.

**g. TREATMENT:**

**Therapeutic guideline**

| Age | **Probable etiology** | **First line antibiotics** |
|---|---|---|
| **Adults and children > 6 years** | **Meningococci**<br><br>**Pneumococci** | **Penicillin** 100.000IU/kg or<br><br>**Chloremphenical** ;1g×3/day for adults or 100mg/kg/ day in infant or<br><br>**Cefotaxime** 200-300mg/kg/24hours in 4 times<br><br>Association or not with **gentamycin** to fight associated sepsis |
| **Infants and children of 3 months to5 years** | **H. influenzae** | **Chloremphenical –** children ;100mg/kg day or **Ampicillin** 200mg/kg /day or **amoxicillin** 200 mg/kg/ day or **Cefotaxime** 200mg/kg /dayor **ceftriaxone** 100mg/kg/day |
| **New born** | **E.Coli, Streptococcus , GroupB; Listeria Monocytogene** | **Cefotaxime** 200mg/kg/day<br><br>**Ceftriaxone** 100mg/kg/day |

*__Procedures commonly employed include__

1. Correction of fluid and electrolyte deficits (rehydration)

2. Provision for adequate oxygenation.

3. Monitoring of cardiovascular function

   a. Pulse

   b. Arterial blood pressure

   c. Central venous pressure

4. Monitoring intracranial pressure - administer **mannitol** to reduce cerebral edema.

5. **Diazepam;** in case of convulsion

6. **Antipyretic; e.g.** aspegic

*__Adjunctive Therapy__

- Corticotherapy e.g **DEXAMETHASONE 0.6mg/ kg** 20 min before antibiotic therapy for to reduce meningeal irritation and cerebral edema
- Increased Intracranial Pressure
- Includes elevation of the patient's head to $30^{O}$ to $45^{O}$, intubation and hyperventilation and mannitol.

**Duration of the treatment;**

2-3 weeks for Meningococci

3-6 weeks for Pneumococci and H.influenzae

## 2. ASEPTIC OR CLEAR LIQUID MENINGITIS

**a.Definition:**

A syndrome characterized by acute onset of meningeal symptoms, fever, and cerebrospinal fluid with bacteriologic ally sterile cultures**.**

**Comment**

Aseptic meningitis is a syndrome of multiple etiologies, but most cases are caused by a viral agent

## b. CLINICAL MANIFESTATIONS

The symptoms of clear liquid meningitis are similar to those of purulent meningitis. The diagnosis is obtained from the result of Lumbar puncture by analyzing the CSF.

*Viral meningitis presents as fever, headache usually frontal or retroorbital and often associated with photophobia and pain on moving the eyes, and

*malaise, myalgia, anorexia, nausea and vomiting, abdominal pain, and/or diarrhea.

*****Nuchal rigidity

*****Evidence of severe meningeal irritation, such as

**Kernig's and Brudzinski's signs,** is generally absent

## c. PRINCIPAL ETIOLOGIES OF CLEAR LIQUID MENINGITIS

**a. Viral**

**b. Fungal(cryptococcus,histoplasma,candidiasis,….)**

**c. Parasitic**

**d. Tubercular**

**e. Syphilis, Listeria, brucella, leptospire**

*** Bacterial etiology;**

**-Meningitis due to Listeria monocytogenes;** Symptoms similar to those of tubercular meningitis

**-Tubercular meningitis; Diagnosis;**

**.** Clear liquid meningitis with abundant lymphocytes in the CSF, decreased glucose and increased protein in the CSF.

**-Other agents;** Brucellosis, syphilis, Leptospirosis, Rickettsiosis,…

*** Viral Meningitis;**

It is not considered as **serious** or potentially life threatening**.** It often occurs as a sequella to a variety of viral illnesses, including measles, mumps, herpes simplex and herpes zoster.

The formation of exudate that is common in bacterial meningitis doesn't occur, and no organisms are obtained for culture from the CSF. Inflammation occurs over the cerebral cortex and the meninges.

***Fungal meningitis**

It is meningitis due to **CRYPTOCOCCUS NEOFORMAS.** The disease is called **Cryptococcus meningitis**. It is the most commonly seen fungal infection that affects the CNS of clients with acquired immunodeficiency syndrome (AIDS). Other possible fungi are histoplasma, candida albicans…

CLINIC; headache, nausea, vomiting and show a decline in mental status, fever or not

***Protozoal Meningitis**

Meningitis following parasitic affection; Amoeba, trypanosoniasis, toxoplasmosis…

**d.TREATMENT OF CLEAR LIQUID MENINGITIS**

It depends on the etiology;

e.g. In case of herpetic meningoencephalitis ; give **Acyclovir** in 10 to 15 days.

In case of Cryptococcus meningitis; give **B AMPHOTERICINE.** Alternative is the **FLUCONAZOLE**

**N.B. EMPHOTERICINE** is given in Infusion and it must be protected against the light. Give **a premedication (Antihistamine and corticoid)** to the patient before giving emphotericine for preventing patient sensibility to the drug.

**Typical CSF findings in Meningitis**

| **BACTERIAL** | **VIRAL** |
|---|---|
| 1. Presence **of neutrophils** in the CSF is associated with infection by N. meningitidis, S. pneumoniae etc.<br>2. CSF protein level reflects the degree of meningeal inflammation:-10 X in bacterial infections | 1. Presence **of lymphocytes** is associated with infection by viruses or mycobacteria.<br>2. CSF protein level reflects the degree of meningeal inflammation:- |

| | |
|---|---|
| 3. CSF **glucose levels** :-<br>very low in bacterial infections | 2-3 X in viral CNS infection<br>3. CSF **glucose levels** :-<br>normal with viral infections |

**COMPARISON OF CSF FOR PURULENT AND CLEAR LIQUID MENINGITIS**

| **CHARACTERISTIC OF CSF** | **PURULENT MENINGITIS** | **CLEAR LIQUID MENINGITIS** |
|---|---|---|
| **Aspect** | Purulent CSF. Xanthochromic( yellow) | Clear CSF |
| **Flow pressure** | Elevated | Normal or less elevated |
| **Leucocytes** | Increased >1000 WBC/mm3 , **Neutrophils** in abundance | Increased (10-2000 WBC/mm3, **Lymphocytes** in abundance |
| **Glucose** | Decreased <40mg/ dl | Normal >45 mg/ dl |
| **Protein** | Increased > 100mg/dl | Normal or slightly increased |
| **Lactic acid** | Increased | Normal |
| **Gram Stain** | Positive or negative | - |
| **Culture** | Often positive | Negative |

**e.COMPLICATIONS OF MENINGITIS;**

Increased ICP resulting from exudation can lead to hydrocephalus and cerebral edema.

-Hydrocephalus

-Cerebral edema

-Herniation of the brain; due to increased ICP

-Hemiplegia

-Deafness

-Death

**f.PREVENTION OF MENINGITIS**

*****Vaccination

*Health education on avoiding crowded places and improving ventilation of houses

## II.6 BACILLARY DYSENTERY (SHIGELLOSIS)

### a. DEFINITION AND ETIOLOGY:

It is acute human infection limited to the digestive tract, caused by the germs of the genus **Shigella.** This is the bacteria, that is non motile bacilli (because of the lack of cilia), Gram-negative, of the genus Shigella including the following species;

- Shigella dysenteriae ( the more common and severe one)
- Shigella flexneri
- Shigella boydii
- Shigella sonneii

They produce **endotoxin** and **exotoxin,** but Shigella dysenteria is the only one that secretes the **exotoxin**.

- Humans are the only reservoir of shigella bacilli.
- The bacilli cause **acute diarrhea with blood** (i.e dysenteria) also called shigellosis.

### b. TRANSMISSION OF BACILLARY DYSENTERY

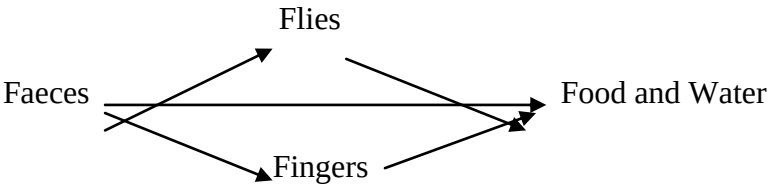
**a. Sources of infection** include;

i. Sick persons of bacillary dysentery
ii. Convalescent carriers of bacillary dysentery-they can spread the bacilli for up to 3 months
iii. Other carriers

**b.Transmission route**

***** Transmission is by; **Faeco-oral route**

e.g; **The rule of F for faeco-oral route**




- Faeces transmitted to food by flies or fingers result into infection when the food is ingested.
- Faeces can also be contracted from
    i. Visiting the toilet during anal cleaning
    ii. Lavatory seats
    iii. Door handless
    iv. Crockery( plates, cups,saucers,bowls)
    v. Cutlery( knives, forks, spoons)
    vi. Bedding (e.g bed sheets, blankets)

**c. RISK FACTORS FOR BACILLARY DYSENTERY**

1. Unsanitary disposal of human faeces
2. Shortage or lack of water which is safe
3. Presence of house flies
4. Seasonal changes
   -In dry season;there is shortage of water
   -In the wet (rainy) season; there is faecal contamination of water sources from run off water.

5. Malnutrition-reduces resistance

6. Old age or early age

7. Overcrowding e.g in prisons, refugee camps

**d. PATHOGENESIS**

Shigella are the pathogens specific to the digestive tract. They are eliminated through the faeces and can contaminate water and food. The human is contaminated by ingesting food or drinking water contaminated by the faeces. The incubation period is short (1-4 days).After ingesting the germs, the bacilli multiply in the large intestine, invade the intestinal mucosa, penetrate in the enterocytes and destroy them using their toxin. The endotoxin of the bacilli causes the irritation of the intestinal wall where the inflammatory reaction of the intestinal mucosa occurs. This inflammation of the mucosa explain the symptomatology of the disease.

The mucous membrane can develop necrotic patches (areas) which turn into ulcers and intestinal perforation

Exotoxin when absorbed results in toxemia (toxic material in blood) leading to high fever and rapid pulse.

**e.CLINICAL MANIFESTATIONS**

After ingestion of many germs, they multiply in the large intestine where **colitis** with **bloody diarrhea** containing **the mucus**, abdominal pain, frequent diarrhea that can exceed 20 stools/ day, followed by **tenesmus** (i.e painful contraction of anal sphincter) without production of faecal matter except for blood and mucus.

Frequent diarrhea can cause **dehydration**. This disease is often accompanied by **fever**.

**f.DIFFERENTIAL DIAGNOSIS**

We must differentiate this disease from other diseases which are accompanied by dysentery such as;

- The acute intestinal amoebiasis (amoebic dysentery),
- Salmonellosis (acute gastroenteritis)
- Hemorrhagic rectocolitis ulcer
- Schistosomiasis…

e.g.

| DISEASE | FEVER | N$^{o}$ OF STOOLS/ DAY | PARACLINIC EXAM | DEHYDRATION |
|---|---|---|---|---|
| **AMOEBIC DYSENTERY** | - | < 20 | Fresh stool<br><br>(direct stool) | - |
| **BACILLARY DYSENTERY** | + (positive) | >20 | Stool culture | + (positive) |

| **BACILLARY DYSENTERY** | **AMOEBIC DYSENTERY** |
|---|---|
| Incubation period short less than a week | Long incubation period more than three weeks |
| Occurrence is epidemic | Occurrence is endemic |
| Tenesmus very severe | Tenesmus not usual |
| Tenderness of the whole abdomen | Tenderness localized in sigmoid colon |

| | |
|---|---|
| Onset acute | Onset is insidious |

N.B. The death is due to **severe hydro-electrolytic imbalance**

**g.DIAGNOSIS**

- Stool examination by microscopy may show macrophages (leucocytes) containing erythrocytes, but these can be confused with Entamoeba histolytica

Thus, the **Diagnosis of bacillary dysentery** is aimed to isolate the bacteria by **STOOL CULTURE**

**h.TREATMENT**

*****Rehydration therapy

*Ciprofloxacin tablet500mg twice daily for 3-5 days.

*N.B. Avoid anti-diarrheal drugs such as Loperamid (Imodium)…

**i.PREVENTION**

i.Observing strict personal hygiene among family contacts and the nursing staff

ii.Community preventive measures:

1. Health education on hygiene

    a. Use of latrines
    b. Use of safe(boiled/ filtered) water, safe food
    c. Washing of hands with soap
    d. Proper waste disposal
    e. Proper excreta disposal

2. Examine if water supply is contaminated, and treat it accordingly

3. Early testing and treatment of carriers

iii.The carriers who work in alimentary sectors should be stopped to work until they are no longer carriers.

N.B. There is no vaccine to shigellosis

**g.COMPLICATIONS;** are uncommon

* Perforation of the colon

*Digestive hemorrhage

## II.7 CHOLERA

### a. DEFINITION AND ETIOLOGY

It is a human toxi-infection caused by the presence in the intenstine **of vibrio cholerae**. Vibrio Cholera is a Gram negative, comma shaped rods, which is motile with a single terminal flagellum.

### d. TRANSMISSION AND PATHOGENESIS

***** It is an acute infectious disease
***** It is transmitted through water or food contaminated with infected faeces from a case or a carrier
*****Incubation period is 1-4 days
*****Infection remains in the intestine and doesn't involve the blood
*****The germs multiply in the small intestine where they release enterotoxin which acts on the intestinal mucosa and disrupts the electrolyte balance (elimination of chloride and potassium ions and inhibition of Sodium absorption)

### c. CLINICAL MANIFESTATIONS

The illness begins rapidly and is characterized by **watery diarrhea** without abdominal pain, **stool is very liquid, looking like "rice water**" very frequent (more than 20-50 / day), **abundant vomiting without effort** and **nausea**, vomit of "**rice water"** appearance. Diarrhea and vomiting lead to significant **dehydration** with **painful muscle cramps**, the hydro -electrolytic disorders are severe and the patient is often in acidosis and hypopotassemia. There is also **oliguria** or **anuria** (**hypovolemic shock** + **acute renal failure**). Ultimately the patient falls into a state of algidity**: Cold extremities, severe hypotension, hypothermia, rapid pulse, persistent skin fold.**

### d.DIAGNOSIS

It is essentially clinical and is supported by **stool culture**.

### e. CURATIVE TREATMENT

i. The most important part of treatment is to correct the fluid electrolyte balance by giving intravenous fluids (replacing fluid and salts losses). IV fluids containing 5g of NaCl, 4g of $NaHCO_3$ and 1 g of KCl per liter are enough to reduce diarrhea. OR oral rehydration solution (ORS) for patients who are still having strength.

N.B. Avoid plain Ringer's lactate; it may cause fatal $K^+$ ↓
Oral rehydration with WHO formula (20g glucose/ L) is not so effective as cooked rice powder solution (50-80g/ L) in reducing stool volume. Its high osmolarity (310 mmol/L $_{VS}$ 200mmol/L) is also unfavourable to water absorption.
ii.Antibiotics should be given to only the most severe cases
-Tetracycline 10mg/ kg/ 6h PO for 48h reduces fluids loss.
-Other drugs are indicated by antibiogram (Furadantine, Negram..)

**f. PREVENTION AND CONTROL**

1. Measures to improve food and personal hygiene and sanitation are the most important factors
2. Killed whole cell vaccine give only 50-60% protection
3. Health education about;

a. Food and water hygiene and sanitation
b. Sanitary disposal of human excreta by burying or mixing with disinfectant, incineration of solid waste.
c. Disinfecting patients clothing by boiling for 5 minutes
d. Drying bedding in the sun
e. Burial (Funeral) should be done quickly and near to the place of death
f. Discourage ritual washing of the dead
g. Discourage funeral feasts ( it is culturally possible)

Cholera is a notifiable disease. Therefore

a. Patients should be treated without being referred
b. Notify District med. Officer
c. Take stool specimens for culture

**g.COMPLICATIONS**

-Renal failure

-Death

**h.DEHYDRATION**

**1. DEFINITION;** Dehydration is defined as hydro- electrolytic loss by the organism

**2. CAUSES;** *Watery diarrhea

*Dysentery

*Fever

*Vomiting

*Abundant sweating

**3. DEGREE OF DEHYDRATION**

A. MILD DEHYDRATION

B. MODERATE DEHYDRATION

C. SEVERE DEHYDRATION

**4. DIFFERENCE BETWEEN DIFFERENT DEGREES OF DEHYDRATION**

| CHARACTERISTICS | **MILD DEHYDRATION**<br><br>**(PLAN A)** | **MODERATE DEHYDRATION**<br><br>**(PLAN B)** | **SEVERE DEHYDRATION**<br><br>**( PLAN C)** |
|---|---|---|---|
| *Diarrhea | - 4liquid stools/ day | - 4-10 liquid stools/day | - >10 liquid stools/day |
| *Vomiting | -Not or in few quantity | -Few | -More frequent |
| *Thirst | -Normal | -Increased than normal | -Unable to drink |
| *Urine | -Normal | -Few, concentrated | -No urine |
| *General aspect | -Alert | -Irritable, somnolent | -Unconcious |
| *Tears | -Present | -Absent | -Absent |
| *Eyes | -Normal | -Penetrated inside | -Dry and penetrated inside |
| *Mouth | -Humid(wet) | -Dry | -Very dry |
| *Respiration | -Normal | -Rapid than normal | -Very rapid and deep |
| *Skin fold | -Elastic | -Skin fold disappear slowly | -Persistent of skin fold |
| *Pulse | -Normal | -Rapid than normal | -Very rapid and weak |
| *Fontanel( infants) | -Normal | -Depressed | -Very depressed |
| *Temperature | - | - | -Fever $+38.5^0$C |
| *Weight | -No weight loss | -Loss of 25-100g/ kg of body weight | -Weight loss >100g/kg |
| *Decision | -The patient doesn't manifest the signs of dehydration | -At least 2 of the above signs, the patient has moderate dehydration | -2 or more of the above signs, the patient has severe dehydration |
| *Interventions | -ORS 50ml/ kg at home | -ORS 1 packet in 1L of water, 100ml/kg in 4-6h<br><br>-If vomiting or unable | -Infusion ; RL 100ml/ kg (if not available ; Normal saline) |

| | | | |
|---|---|---|---|
| | | to drink; infusion | |

## II.8. THE SYPHILIS

### a.Definition:

**Syphilis is** a sexually transmitted disease caused by bacteria. The highly infectious disease may also be passed, but much less often, through blood transfusions or from mother to fetus in the womb.

Without treatment, syphilis can cause irreversible damage to the brain, nerves, and body tissues

### b.Etiology

Syphilis is an infectious; often sexually transmitted disease caused by the bacteria ***Treponema pallidum*.** The bacteria penetrate chafed skin or the mucous membranes

### c.CLINIC

Syphilis is an infection that can stay in the body for years if not recognized and treated quickly. There are two forms: **Acquired** and **congenital syphilis**

**1. ACQUIRED SYPHILIS**: observed in adults, it evolves in three stages of syphilis, and each stage has distinct signs and symptoms

- ✓ **Primary stage**: it usually occurs 3 weeks after the infectious contact. It is characterized by **a chancre**, indurated and painless ulceration, single, localized at the point of inoculation of the external genitalia (penis, vulva).
  The chancre generally appears on the genital area but can also form on the lips, tongue or rectum if these areas have been exposed to a syphilis chancre on another person during oral or anal sexual contact. A chancre in the vagina, mouth or rectum is generally not easily seen.
  The chancre is accompanied by a regional satellite lymphadenopathy, inguinal and often painless. Untreated, the chancre heals spontaneously in 4 to 6 weeks.

- ✓ **Secondary stage**: corresponds to bacterial dissemination phase. If left untreated, primary syphilis progresses to a second stage called secondary syphilis. In this stage, the bacteria that cause syphilis spread throughout the body and cause additional symptoms. It usually occurs around 2 months after the contamination. Symptoms begin to appear after the chancre has resolved and include:
  - Skin lesions that are very contagious:
    + Syphilitic roseola
    + Papules = syphlides
  - Mucosal lesions that are very contagious:
    + Painless erosions especially at the oral mucous and genital mucous (mucous patches, condylomata...). These signs disappear spontaneously in 1 to 2years.
  - General symptoms: fever, general malaise, headache, Flu-like symptoms, Headache, Weight loss, fatigue...

**Tertiary stage**: The third and final stage of syphilis is called tertiary or late-stage syphilis, which develops from untreated secondary syphilis. When untreated secondary syphilis symptoms disappear, the infection still continues in the body. Symptoms of tertiary syphilis may appear for 10 to 20 years after initial infection with syphilis. During this time, syphilis can damage organs, such as the brain (general paralysis), cardiovascular (aortitis, aortic aneurysm), liver (the disease resembles to cirrhosis), bones, and nerves, leading to serious complications.

- The gums can be located anywhere (in the liver, kidneys, bones, stomach)
  Note: In the stomach there is often ulceration

### 2. CONGENITAL SYPHILIS

Transmission of *T. pallidum* from a syphilitic woman to her fetus across the placenta may occur at any stage of pregnancy, but fetal damage generally does not occur until **after the fourth month of gestation**, when fetal immunologic competence begins to develop. There are two types:

1. **Early congenital sphylis**: that of the New born.

The earliest signs occur between 2-10 weeks after birth and are infectious.

The earliest signs of congenital syphilis include **rhinitis** with purulent or bloody secretions which is soon followed by other mucocutaneous lesions: **bullae** (syphilitic pemphigus) in the palm of the hands and soles of the foot, vesicles, superficial desquamation, petechiae, and (later) papulosquamous lesions, mucous patches, and condylomata. The most common early manifestations are bone changes (61%), including osteochondritis, osteitis, and periostitis detectable by x-ray examination of long bones; hepatosplenomegaly (50%); lymphadenopathy.

2. **Late congenital syphilis**: it develops for grand children. Signs and symptoms appear after 2 years and are non infectious. It is characterized by:

***** Teeth deformation

*Osteo-arthritis

*Eye affection (keratitis): more common and occurs between ages 5-25.

*Perforation of ovula
*Etc…

N.B. **SPECIAL CLINIC AL FORM**; is syphilis in **pregnant women**. It may cause:
-Spontaneous abortion,
- Still birth
- Premature birth or at term but presenting signs of congenital syphilis.

### d. PARACLINICAL DIAGNOSIS

-**Primary stage**: collect the fluid at the chance and the direct examination by microscopy to search for spirochetes (Treponema pallidum)

- **Secondary and tertiary stage**: serological tests are made. Serological tests are the most effective methods.They are aimed to search for **antisyphilitic antibodies.** There are two types of serological tests for syphilis:
A.Non-treponemal antigen (cardiolipia): Non specific
B.Treponemal antigen: Specific

**1. Non-treponemal antigen tests**:
-To detect **anticardiolipide antibodies.**
**-Cardiolipide** is a constituent of the cytoplasmic membrane of treponema pallidum.
Non treponemal antigen tests include:

**a.VDRL** (Venerial Disease Research Laboratory)
-Cardiolipin antigen plus serum produces clump (agglutination) in positive result

**b.RPR Test** (Rapid Plasma Reagin): already eradicated

**2. Treponemal antigen tests**
**-**Use antigens of treponema pallidum and they are more specific.
They include:

**a.T.P.H.A** (Treponema Pallidum Hemagglutination Assay)
**b.F.T.A-Abs test (**Fluorescent Treponema Antibody Absorbed): permits putting in evidence of IgM antibodies. Highly specific and sensitive, and so widely used.
**c.ELISA** (Enzyme Linked Immunosorbent Assay)
-It is rarely used because it is very expensive.
N.B. In case of neurosyphilis, the CSF (cerebral spinal fluid) is used for examination.

**e. TREATMENT**

**1. Primary and Secondary stage of Syphilis:**
-Benzathine Penicilline 2.4 Mega Units (MU) in IM as a single dose
**2. Tertiary stage of Syphilis (i.e cardiovascular and neurosyphilis)**
**-**Benzathine Penicilline 2.4 MU in IM weekly for 3 weeks

**f.PREVENTION AND CONTROL**

i. Condoms use

ii.Avoid having sex with multiple partners

iii.Screen pregnant women for syphilis infection where possible

**g.COMPLICATIONS**

Complications of syphilis include:

- An increased risk for contracting HIV, which causes AIDS
- Aortic aneurysm: Dilation of aorta
- Birth defects
- Blindness
- Dementia
- Paralysis
- Stillbirth

## II.9. CHANCROID (OR SOFT SORE)

### 1. GENERALITIES

-It is caused by **Haemophilis ducreyi** bacteria which are Gram negative coccobacilli

-It causes a soft sore (chancroid) on sex organs

-It is sexually transmitted

-Women do not show symptoms in vagina when infected. So infected males cannot have sexual intercourse because of pain, and therefore they do not transmit infection. Buboes (lymphnodes with pus) can also develop as one of the symptoms.

### 2. CLINICAL MANIFESTATION

Infection is acquired as the result of a break in the epithelium during sexual contact with an infected individual. After an incubation period of 4-7 days, the initial lesion (a papule with surrounding erythema) appears. In 2 to 3 days, the papule evolves into a pustule, which spontaneously ruptures and forms a sharply circumscribed ulcer that is generally not indurated. The ulcers are painful and bleed easily, little or no inflammation of the surrounding skin is evident.Approximately half of patients develop enlarged, tender inguinal lymph nodes, which frequently become fluctuant and spontaneously rupture. Patients usually seek medical care after 1-3 weeks of painful symptoms.

### 3. DIAGNOSIS

-Gram negative coccobacilli is identified in fluid from chanchroid

### 4. TREATMENT OF CHANCROID

-Norfloxacin 800 mg orally in a single dose

-or Ciprofloxacin

-or Erytromycin

**5. PREVENTION**

i.Condoms use

ii.Not have sex at all until after treatment

iii.Avoid having sex with many partners

**II.10.GONORRHEA**

**a.DEFINITION:**

Gonorrhea is an acute or chronic purulent infection of urogenital tract .

**b.ETIOLOGY:**

- Etiologic agent: ***Neisseria gonorrhoeae***
- Gram-negative intracellular diplococcus
- Found only in humans with different epidemiological presentations for females and males

**c.TRANSMISSION**

It is transmitted through sexual intercourse, but also perinatal transmission (mother to child) is possible.

**d.RISK FACTORS**

- Multiple sex partners or inconsistent condom use
- Urban residence in areas with disease prevalence
- Lower socio-economic status
- Use of drugs

**e.CLINICAL MANIFESTATION**

*** IN MEN: (**90% of infected men develop symptoms)

- Typically purulent or mucopurulent urethral discharge. Often accompanied by dysuria ( Inflammation of the urethra)
- Untreated , can complicate in:

- **Epididymitis** – Inflammation of the epididymis :Symptoms: unilateral testicular pain and swelling
- **Prostatitis:** Inflammation of prostate gland

- **Male sterility:** Due to inflammation of vas deferens
- **Vesicle seminal inflammation**
- **Urinary retention:** due to narrowing of urethra
- **Hydronephrosis:** associated with urinary retention and increased pressure in kidney

***GonococcalUrethritis: Purulent Discharge**

***IN WOMEN(** 40% of infected women develop symptoms)

- Most infections are asymptomatic
- The vagina is normaly not infected because of reproductive age. However in prepuberty girls and women after menopause the acid production in vagina is very limited, and therefore the vagina and vulva inflammation ( vulvo-vaginitis) can occur in epidemic proportions of these age groups. The vagina of an adult woman of reproductive age is lined with squamous cells which conain glycogen. Through bacterial action , this glycogen is metabolized into lactic acid . Thus producing a low $P^H$ . This low $P^H$ protect the vagina wall from invading gonococci. The glycogen content of the cells is determined by oestrogen levels and is therefore low before puberity and after menopause. Then the

vagina is less resistant to gonococcal infections in prepuberital girls and postmenopausal women.

Then, the symptoms in women may include:

- Cervicitis – inflammation of the cervix
- Urethritis with mictalgia and dysuria – inflammation of the urethra
- Vulvo-vaginitis: muco-purulent vaginal secretions and vaginal itching
- Proctitis: inflammation of the anus and rectum

Untreated , can complicate in:

*Accessory gland infection

- Bartholin's glands-Bartholin's abscess
- Skene's glands

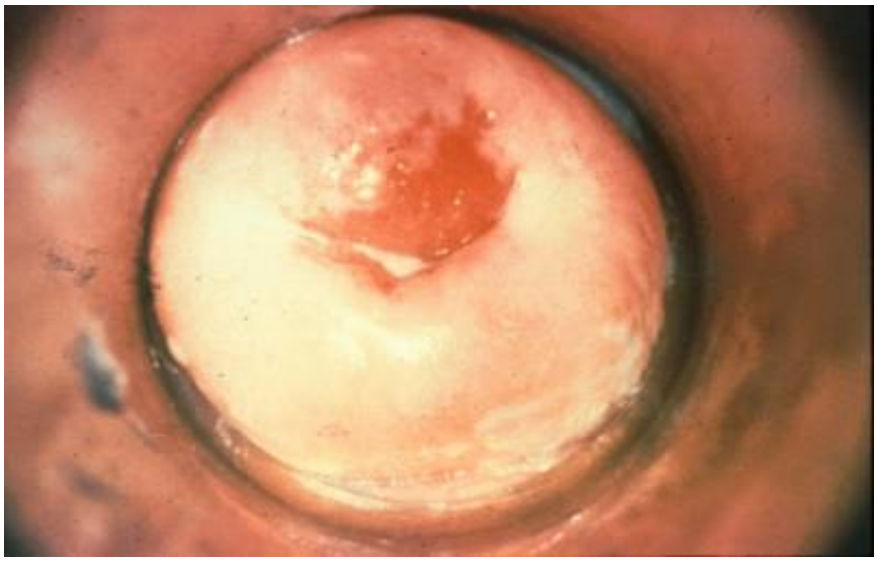
*Pelvic Inflammatory Disease (PID

*Peritonitis

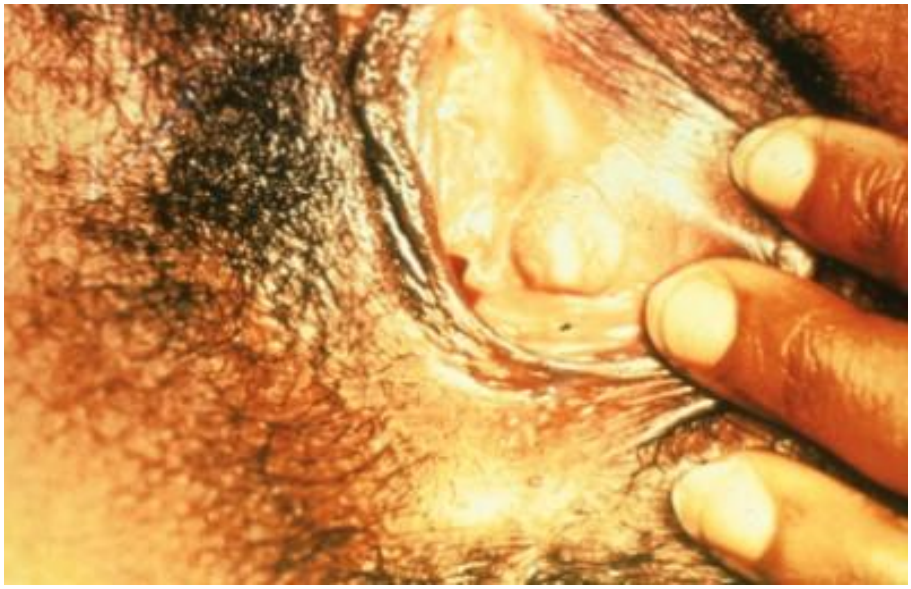
*Endometritis: inflammation of the endometrium

*Sterility: due to narrowing of fallopian tubes in case of chronic salpingitis

***Gonococcal Cervicitis**



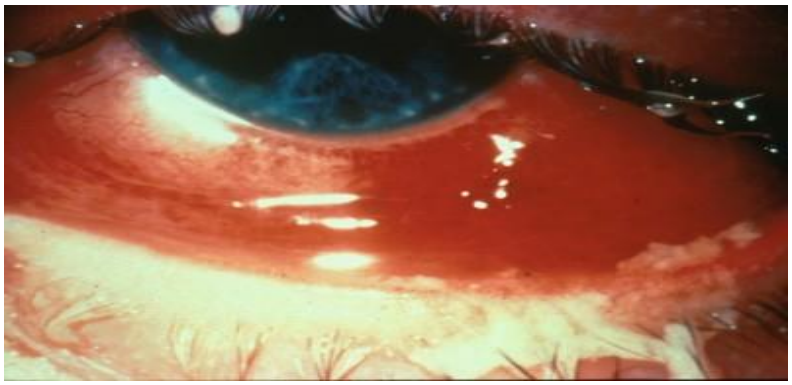
***Bartholin's Abscess**



**N.B. Gonorrhea Infection in Children**

- **Perinatal:** The child is infected during delivery when he is passing through infected maternal genital tract. It can cause infections of the conjunctiva **(Gonococcal Ophthalmia) , pharynx, respiratory tract**
- Untreated, **blindness** can occur

***Gonococcal ophtalmia: purulent eye discharge**



**f.DIAGNOSIS**

**-Bacteriological examination:** To find Gram negative diplococcic inside neutrophils (phagocytes) in smear of discharge from urethra (**urethra**l **swab**) or vaginal (**vaginal swab**).

- Legal standard is **culture of discharge** to confirm the identity of *Neisseria gonorrhoeae*

**g.TREATMENT**

**-First line treatment ( not in pregnant woman)**

*****Norfloxacin 800 mg orally in a single dose

Or Ciprofloxacin 500 mg orally in a single dose

Or Ofloxacin 400 mg orally in a single dose

**-Second line treatment**

- Spectinomycin 2 g in a single IM dose

  OR Cefotaxime 2 gr IM in a single dose

  Or Azithromycin 1 gr orally in a single dose

- Single-dose cephalosporin regimens
  - Ceftizoxime 500 mg IM
  - Cefoxitin 2 g IM with Probenecid 1 g orally

**h.PREVENTION**

*****Health education: use of condoms, sexual abstinence …

*Early diagnosis and treatment

*Regular eye washing with **1% Tetracycline eye ointment** in the newly born to prevent infection

**Partner management**

- Evaluate and treat all sex partners for *N.G*
- Avoid sexual intercourse until therapy is completed and both partners no longer have symptoms.