TUBERCULOSIS(TB)

TB FACTS

- TB is curable
- Someone in the world is newly infected with TB every second
- Someone dies of TB every 15 seconds world wide
- Eight million people develop active TB every year
- Each one can infect between 10 and 15 people in one year just by breathing
- The best way to prevent TB is to treat and cure people who have it

TB AND HEALTH

TB is much more than a health concern. It is a complex of socio economic problem which impedes human development and traps world's poorest and most marginalized in a vicious circle of disease and poverty

Confronting TB requires inter-sectoral action combined with relevant health issues.

Impact of TB

- A person who have TB and is never diagnosed or treated loses an average full year of work
- Over 900 million women are infected with TB. This year 2 ½ million women will get sick from TB and one million will die.

TB AND HUMAN RIGHTS

- Discrimination against people with TB is a violation of their human right
- People with TB often do not know that TB is curable

- People with TB often lack information about available TB treatment
- People with TB often lack access to affordable TB treatment
- Access to information about prevention and treatment is a basic right.
- Promoting and respecting human rights including the rights of people with TB is the pre-requisite for health and development

Definition

Tuberculosis is a systemic mycobacteria disease generally caused by a bacillus mycobacterium tuberculosis but also occasionally caused by mycobacterium bovis. In most cases the lungs are involved causing pulmonary tuberculosis (PTB) however other organs such as the brain, intestines and bones may also be involved – extra pulmonary tuberculosis

Causative organism(s)

- Mycobacterium tuberculosis is quite demonstrated in laboratory by staining it with ziehl-neelsen (zn). It retains the red color even after decolourization with acid and alcohol – therefore being called AAFB /AFB – acid /alcohol fast bacillus. The organism is a slow growing acid hence responsible for it's long incubation period.
- Tuberculosis is characterized as chronic communicable disease. In order to kill all mycobacterium treatment must be continued for a long period of time so as to give time for all the dormant mycobacteria to become active again and to be affected by drugs. Stopping the drugs too soon will increase the dormant mycobacteria that become active again.

Infection and disease

- TB is a communicable disease – thus can spread from infectious patients to a susceptible contact. The risk of becoming infected with these organisms is determined by the number of organisms coughed (TB) or sneezed into the air as fine spray droplets.
- Not all persons who are infected this way will develop clinical disease. A great proportion of those infected will be able to overcome the infection and thus will develop a natural immunity. Those who do not will develop clinical disease, either as s direct progression from the initial infection or later in life.

Predisposing factors for infection

- Staying with infectious patients in poorly ventilated room
- Staying in contact with a patient who is not on treatment or has not completed his full course
- Overcrowding, since this increases the chances of getting the infection from many other people

Predisposing factors to disease

- Poor general nutritional and health status (poverty)
- Not having received a BCG vaccination
- Having HIV at the same time
- History of recent measles (for pediatric TB)
- Over age and tender age / diabetes

NB; with the current HIV epidemic many HIV positive patients who had successfully overcome mycobacterium infection in the past experience a fare-up of this infection leading to clinical disease. This is true for TB. HIV infections causes a breakdown in the immune defense mechanism

and thus the few mycobacteria that were kept under control by the immune system get a chance to become active again and cause disease.

The most frequent form of tuberculosis is pulmonary tuberculosis – the most form in relation to control of the disease because the infectious form are usually infections. But it can however attack any organ of the body except hair and nails.

When untreated tuberculosis is a disease with high mortality rate. However the current regime of treatment can achieve a very high cure rate (almost 100%) provided the patient is started on treatment at an early stage of the disease and takes drugs regularly.

Mode of Transmission

The disease is transmitted from an infected person through droplet from an acute person. The inhaled bacilli settle in the lungs, and cause infection (primary infection)

Incubation Period

Mycobacteria are relatively slow in growing organisms which divides at an average only once in every two weeks. This counts for long incubation period.

Incidence and Importance

TB is much more than a health concern, it is composed of social economic problem which impedes human development and traps the world poorest and most marginalized in a vicious circle of disease and poverty.

TB is endemic in most developing countries and most common mainly in African countries. Most adults will have been exposed to TB infection at some stage in their lives. The disease is on increase especially in those countries with a serious HIV problem. If left untreated the disease can

kill or at least disable the patient for the rest of their lives. Because of its frequency and severity TB is always an important disease – the most important public health problem.

TB is a very common opportunistic infection in persons with HIV positive or suffers from AIDS. On such a person TB is often the first disease that develops when body immunity starts to decrease. The impact of TB is – a person who have TB and is never diagnosed or treated loses an average full year of work**.**

Pathogenesis

The mycobacteriae are spread from infectious patient to healthy individuals (susceptible) by droplet infection. It happens when a patient with open pulmonary tuberculosis coughs out heavily infected sputum into the atmosphere. Many people are exposed to TB infection, but only a few will develop actual disease. The rest will overcome by acquiring active natural immunity to the disease.

Not all mycobacterium are eliminated from the body by acquiring natural active immunity, but their number is kept at control by the body immune system, thus they don't cause the disease. If the immunity is depressed by the predisposing factors TB may flare up and cause obvious clinical disease. The clinical presentation varies with the organ infected but initially all had been infected by positive – sputum patient. These patients represent the infectious reservoir for TB in the community. The spread and transmission of TB can only be significantly reduced if the reservoir is reduced. The organisms (infected) are breathed into lungs and settle in a distant part of the lungs tissues. There they multiply and cause some of these tissues destruction. While this happens the immune system sends many leucocytes to the site in the attempt to conquer the invading organisms. The place in the lungs where mycobacteria settle first is surrounded by the leucocytes and other

immune response cells. These cytotoxic cells try to eliminate the mycobacteria by surrounding and killing them. This fight is associated with enlargement of the regional lymph nodes at the bifurcation of the trachea.

The combination of a solitary lesion in the lung with enlargement of regional lymph nodes is known as the **primary complex**. Most primary complexes heal spontaneously but some mycobacteria may still remain alive in the lung tissue or in the lymph nodes in a more or less dormant state for a while afterwards. They cause no further problems because the immune system has had them under constant surveillance and control.

If the immune response is weak or immunity declines, the mycobacterium may gain an upper hand and spread to the other parts of the lungs. There they cause characteristic lesions. These solid lesions, contaminating the mycobacterium and the immune response cells, they gradually involve more and more lung tissue.

Before long, because of lung tissue destruction, the middle parts of the both lesions begin to liquefy into cheese like material which is released into the small airways. The patient starts to cough, then soon begins to produce purulent sputum. The sputum contains the material from inside the lesions and can be heavily laden with infected mycobacteria. This is the infectious stage of the disease. The patient has now become infectious to others. As the mycobacteria destroy lung tissue, the destruction may cause destruction of some small or sometimes larger blood vessels and bleeding starts. This is known as **Hemoptysis** and it is highly suggestive of tuberculosis. Occasionally a patient may die as a result of massive hemorrhage.

The disease can spread outside the lungs in several ways. The mycobacterium may migrate into the blood stream and this be carried to other parts of the body. They can also spread through the

lymph nodes especially those of the neck (cervical lymphadenopathy). The patient may swallow some of the sputum as coughs up and thus introduce germs into the intestines.

Whenever the mycobacteria spread and settle, they cause similar lesions, whether it is in the meninges (lining of the brain and spinal cord), kidneys, bones and joints, the pericardium or the peritoneum. The TB in these areas is referred to as extra pulmonary TB to distinguish it from PTB. When the mycobacteria enter the blood stream and are disseminated widely to both lungs, this is called **Milliary Tuberculosis** a serious form of the disease.

The infection, as it spreads causes an inflammatory reactions in the tissue and organs it has spread to. Involvement of the pleura, pericardium and peritoneum results in TB pleuritis, pericarditis and peritonitis respectively. Subsequent accumulation of inflammatory fluid at these sites causes pleural and pericardial effusion and ascites respectively. These complications and are dealt with in the hospital.

The course of TB Infection

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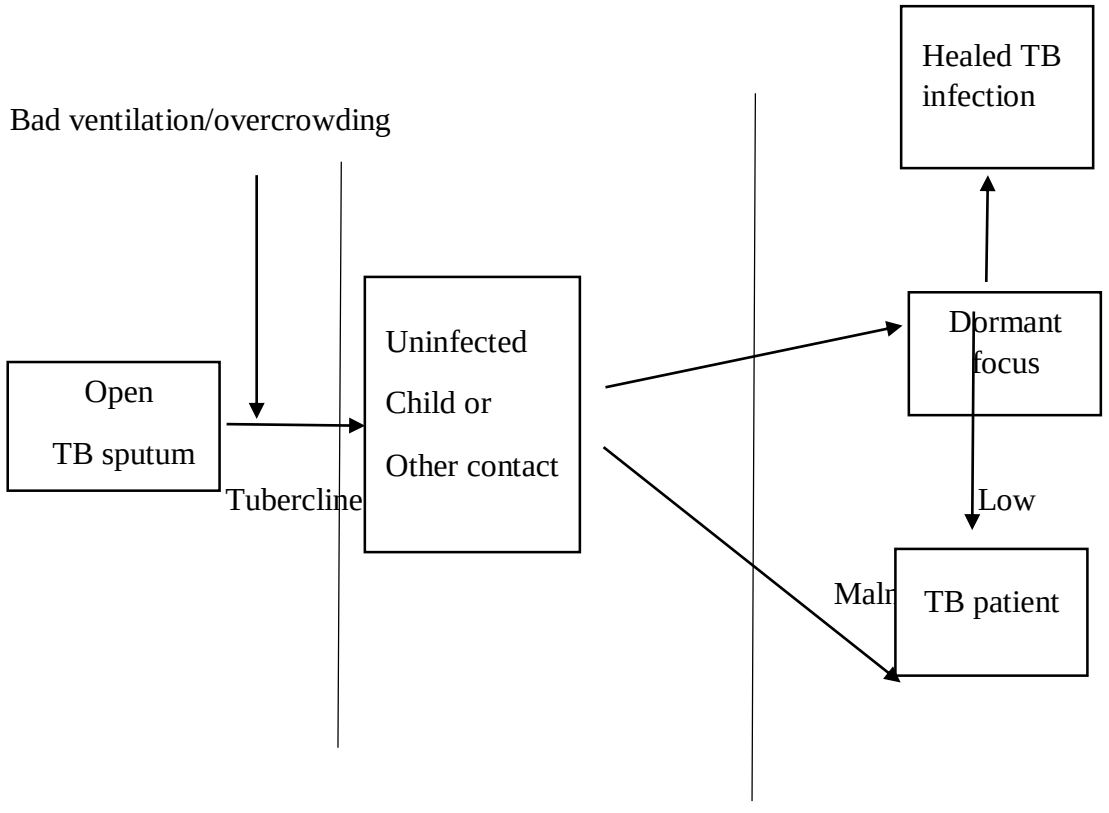


Pregnancy etc

Diagnosis

Adults:

1. **History taking:**

- signs of pulmonary tuberculosis

-Patient may present with vague complaints with or without close contact with family member or close associate with a patient of TB.

-Patient may present with D/M or other chronic diseases

- Cough persisting for 3 weeks or more
- Hemoptysis — specific
- Chest pain
- Shortness of breath
- Loss of body weight — non-specific
- Fever and night sweat

2. **Physical examination (symptoms)**
    - Crepitation
    - Bronchial breathing

- Diminished breath sounds or dullness

NB: the above not very diagnostic

3. **Sputum smear examination**
    - Very essential for every patient suspected of having pulmonary TB
    - Sputum exam by direct smear for AFB is the most important tool and very diagnostic of PTB is cheap and fast.

Mode of Sputum collection

Three specimens of sputum are collected

1st specimen- when patient comes to the hospital and given the sputum container

2nd specimen – collected in the morning of the second day and brought to the hospital

3rd specimen – when patient brings the 2nd specimen to the hospital should give the 3rd specimen

4. Radiology (chest X-ray other ray)
5. Sputum culture
6. Monteux testing
7. Biopsies
8. CT scans

Signs and symptoms

Early signs and symptoms

- Productive or non-productive prolonged cough – 3 or more weeks

- Unexplained loss of weights
- Night sweat
- Positive tuberculosis test

Late signs and symptoms

- Chest pains
- Hemoptysis – blood stained sputum
- Enlarged lymph nodes
- Severe loss of body weight
- Pleural pain
- Difficulty in breathing
- Symptoms of other organs involved
- General body weakness
- Fever

Complications of PTB

- Pleurisy,
- laryngitis,
- pleural effusion,
- bronchial and tracheal complications,
- pericardial effusion,
- pneumothorax,
- enteritis,
- milliary TB

- severe hemoptysis,
- respiratory failure,
- pleural/pericardial effusion,
- , kidney failure, vertebral collapse (potts disease)

Management of Pulmonary Tuberculosis

Purpose of tuberculosis treatment

- To cure the individual patient.
- To prevent spread of the disease to others.

The aim of the treatment is to kill the mycobacteria as efficiently as possible and within the shortest time.

- There are different treatment regimens, which are basically combinations of several anti-tuberculosis drugs.
- Combinations of two or more drugs is far superior to single drug treatment because of risk of the mycobacteria developing resistance to single drug is much higher than the the risk of their risk developing resistance to several drugs at the same time.
- The regimens depends on the country policy for the treatment of TB in each of them. Treatment of tuberculosis is always in two phases.
- Depending on the individual patient condition he/she may be treated as an inpatient or outpatient,
- Admit the patient into the ward and put in bed rest in well ventilated room to avoid droplets infections.

- Take vital observations of TPR or BP record, interpret and report. The frequently depends on patient's condition- 4 houly or bd .
- Assist and prepare in carrying out investigations, sputum, chest, x-ray sputum and mantoux test.
- Give high protein diet with extra milk.
- Weigh the patient weekly to note any increase or further decrease indicating deterioration of condition.
- Administer drugs according to the national regime policy and the phase.
- WHO-came up with a TB treatment strategy known as DOTS{Directly Observed Treatment short course
- When using DOTS strategy you must adhere to the following rules-

  -Follow the national treatment guidelines

  -Ensure that there is adequate supply of anti-TB drugs

  -Ensure each patient is on the correct treatment regime

  -Administer the initial (intensive) phase of treatment under supervision

  -Encourage all the patients to attend the TB clinic regularly during the continuation phase

  -Promptly trace the defaulters

  -Maintain accurate records on patient personal data and clinic attendance

  N/B For **the DOTS strategy to succeed the government must be committed to the programme.**

  Treatment Phases

  The intensive- therapy phase
  The continuation-therapy phase

- **Intensive therapy phase**- is composed of combination of most powerful drugs aimed at killing as many tubercle bacilli as quickly as possible.
- However these drugs are also the most expensive. During this phase the patient may be treated as an inpatient or be seen daily as an out-patient.
- At present, short-course therapy (SCC) is given to all TB registered by NLTP.
- The first two months (intensive phase) the treatment should be administered under direct observation.
- The continuation phase (4-6 months) the patient collect a supply of drugs four weekly for daily self-medication at home and under supervision of a family member or guardian.

Abbreviation of the regime	2ERH4/6EH	
Phase	**Intensive phase**	**Continuation phase**
Duration	Daily supervised for 2 months	Daily self-adding extraction for 6 months
Drugs used	Ethambutol	

NB: children below age 10 years should not receive ethambutol but should instead receive streptomycin sulphate

DOSAGES OF ANTI-TUBERCULOSIS DRUGS ACCORDING TO BODY WEIGHT

DRUGS		**PRE-TREATMENT WEIGHT**		
DOSAGE	**FORMULATION**	**OVER 49KG**	**49-33 KGS**	**LESS THAN 33**
streptomycin	in injection	1gm	0.75mg	0.5g

Rifampicin isoniazid 50mgs pyrazinamide 300mg	Combination tablet	5	4-3	2-1
Rifampicin 150mg isoniazid 100mg	Combination tablet	4	3	2
Ethambutol 400mg intensive phase	Tablet	2-3	1-2	1
Ethambutal 400mg continuation phase	tablet	2 ½ -3	1 ½ - 2	1 ½
Ethambutal 400mg isoniazid 150mg	Combination tablet	2 ½ -3	1 ½ -2	1 ½

CAUTION;

- Pregnant and patients older than 40years should be given more than 0.5gms of streptomycin daily injection
- Ethambutal should not be given to children

RHZ – combination of **Rifampicin, isoniazid, pyrazinamide, - RIFATER**

RH – RIFINAH – **rifampicin and Isoniazid**

DRUG DOSAGES ACCORDING TO BODY WEIGHT

DRUG	**FORMULATION**	**OVER 50KGS**	**40-49KGS**	**30-39KGS**

RHZ (RIFATER) Rifampicin, Isoniazid pyrazinamide	Combined tablet	5	4	3
RH –RIFINAH Rifampicin, isoniazid	Combined tablet	4	3	2
Ethambutal	Tablet	2 ½	2	1 ½
Streptomycin	Injection	1gm	0.75gm	0.5gm
	CHILDREN	**20-30KG**	**10-20KG**	**5-10KG**
RHZ (RIFATER) Rifampicin, isoniazid Pyrazinamide	Combination tablet	2	1	1 ½
RH (RIFINAH) Rifampicin, isoniazid	Combination tablet	2	1	1 ½
Streptomycin				

Thiazina – cheap and effective drug and combination of thiacerazone and isoniazid

Treatment regimens and drug dosages

Treatment regimen for new smear the patients of other seriously all cases of

TB , milliary TB, TB vital organs

Abbreviation of the regimen	**2ERHZ/6EH**	
Phase	Intensive phase	Continuation phase
observation	Daily supervised for 2 months	Daily self –administered for 6 months
Drugs used	Ethambutol(E) rifampicin(R) isoniazid(H) pyrazinamide(Z)	Ethambutol(E) isoniazid(H)

Retreatment regimen for relapse (R), treatment failure (F) or treatment resumed (TR) with active tuberculosis disease and who have a positive sputum smear or culture results

2SRHZE/IRHZE/5RHE

Abbreviation of the regimen	**2SRHZE/IRHZE/5RHE**		
phase	**Intensive phase**		**Continuation phase**
Duration	Daily supervised for 2 months	Daily supervised for one month	Daily self-administered for 5 months
Drugs used	Streptomycin (S) Ethambutal (E) Rifampicin (R) Isoniazid (H) Pyrazinamide (Z)	Ethambutal (E) Rifampicin (R) Isoniazid (H) Pyrazinamide (Z)	Ethambutal (E) Rifampicin (R) Isoniazid (H)

Tuberculosis treatment- 1st line

6 months treatment composed of (over15years old)

Intensive phase 2 months –

- Rifampicin -150mgs
- Isoniazid -75mgs
- Pyrazinamide -400mgs
- Ethambutol – 275mgs

Continuation phase 4 months

- Rifampicin – 150 mgs
- Isoniazid -75mgs
- Pyrazinamide -400mgs

Re-treatment regimen

Adult over 50kgs

Intensive phase 2 months – 2SRHZE

- 1RHZE

<u>Children</u>

Intensive phase 2 months -– 2SRHZE

Intensive phase 1 month - 1RHZE

Continuation phase – 5 months – adult

5RHE

- Rifampicin
- Isoniazid
- Ethambutol

Children - 5RH

Sputum collection

– start sputum collection
– early morning before breakfast

NB : D/S over 40 years ⎫
Xxxx pregnant ⎭ do not exceed ¾gms of streptomycin

Reassure the patient and the relatives. After the patient has finished the course of drugs during the intensive phase is discharged and followed up in chest clinic as an outpatient.

Provide health education to the patient's family and the community on cause, transmission, prevention and control of tuberculosis. Follow up contacts and defaulters and non-complainants at home.

Prognosis: good when early diagnosis and treatment are made. However TB has case fatality rate of 12-15% because of complications – these are managed in the hospital.

Prevention and Control of Tuberculosis case finding and treatment

- **Case Finding and Treatment**

-Control of TB depends on the success of treatment of registered TB patients and contact tracing.

- Emphasis is placed on those who are sputum smear positive to make them negative. -Put all patients with open tuberculosis on treatment as early as possible.

-Early case finding – health workers should recognize early symptoms of TB and diagnosing - wherever patients reports in health facility with suggestive signs/symptoms (passive case finding)

- **Home visiting**

-In addition ,health workers must visit homes of all new TB cases to look for any other cases this is called **contact tracing or the case finding**.

-A patient is rendered non-infectious after completing the treatment without defaulting – one who defaults increase risk of relapse and may once again become infectious to others. This is prevented by case holding.

- **Selecting a particular day in a health facility**

During this selected day all the TB cases are seen- this prevents long queuing for the patients.

- **Intensive health education**

-Health education is given to the patients during the course of treatment to minimize defaulting and to ensure case holding. H/educate the community about TB so that control measures can be instituted and cases can be identified at an early stage.

- **Immunizing**

–Immunizing with BCG vaccine is effective before any natural infection.

-Newborn babies are given BCG at birth or as soon as possible after birth(within two weeks).BCG Should not be given to the babies who have AIDS –It has been reported it develops into serious systemic disease that look very much like TB

- **Avoidance of careless spiting**

-TB patients should be advised not to spit everywhere carelessly

- Should have sputum mug to spit in and disposed of carefully – burning or putting in pit latrine.

- **Overcrowding and poor ventilation**

Overcrowding in the houses/community should be avoided whenever possible and community educated on the dangers involved

TUBERCULOSIS

Tuberculosis is a systemic mycobacterial disease generally caused by mycobacterium Tuberculosis. In most cases, lungs are involved but virtually any can be affected. Referred to as Pulmonary Tuberculosis (PTB) when the lungs are affected. Occasionally m. bovis can also cause

TB in humans in developing countries. It is found in unpasteurized cow's milk causing GI TB in humans.

EPIDEMIOLOGY

Worldwide m. tuberculosis causes more death than any other single microbial agent. Approximately 30% of world population is infected with this organism.

Africa bears the highest burden of the disease at an incidence rate estimated 260-100,000 (1997, WHO). Most adults will have been exposed to the TB organism at some stage in their lives. TB is on the increase especially in countries with serious HIV problem. If left untreated or treated inadequately, TB can kill or render the patient disabled for life. Due to its frequency and severity, TB remains an important disease of public health significance.

SPREAD (MODE OF TRANSMISSION)

- TB is transmitted from one person to person by droplet infection. This occurs when an infectious person patient coughs out heavily infected sputum into the atmosphere.
- Most people becomes exposed to the TB infection yet few will progress to develop actual disease. The rest will overcome the infection by acquiring active natural immunity over the disease.

Immunity and Hypersensitivity

- After recovery from the primary disease many infection resistance to organism is meditated by cellular immune .

  NB; infection is different from disease.

- Circulating antibodies also form but play no role in resistance. During acquisition of active natural immunity, not all mycobacteria are eliminated from the body. Some may live on but can't cause the disease since the immune system restrict their activities. Therefore the infected person is asymptomatic.
- However when the body immunity is reduced by old age malnutrition, HIV infection, or use of immune suppressing drugs such as the corticosteroids, the m. tuberculosis become active again and causes a clinical disease. Therefore asymptomatic patient is a reservoir of TB in the community.

Development of Tuberculosis in a Patient

- A healthy person is infected with M tuberculosis when the organism are inhaled into lung and settle in a distinguished part of the lung tissue. There, they multiply and cause tissue destruction forming lesions. Macrophages and leucocytes attempt to conquer the organisms. In most individuals the mycobacteria is killed by the macrophages and other immune response cells. This process is associated with enlargement or regional lymph nodes at the bifurcation of the trachea. The combination of solitary lesion with enlargement of regional lymph nodes is known as the primary complex which heals spontaneously.
- Some mycobacteria may still remain alive in the lung tissue, but dormant state under the control of the immune system. If the immune system weakens, the mycobacteria may gain upper hand and spread to other parts of the lungs forming solid lesions also referred as tubercles/grann.
- After sometime, the solid lesions begin to liquefy into clike cheasy-material (caseation) which is then released to small airways. Xxxx develops a dry cough which soon becomes

productive of purulent sputum which is heavily laden with mycobacteria. At this stage the patient can be infectious to others. Lung tissue destruction by mycobacteria affect blood vessels resulting to hemoptysis (presence of blood in sputum). Mycobacteria may migrate into the blood stream and carried to the other parts of the body. They can also spread through lymph vessels causing swelling of lymph nodes especially those of neck (cervical lymphadenopathy). The patient may swallow some of the sputum coughed up and thus

- introduce bacilli into the intestines or have direct spread of the pleura. They cause similar lesions in meninges, kidney, bones, joints, pleura, pericardium, and peritoneum. TB in this area is referred as Extra-pulmonary Tuberculosis. When mycobacteria is disseminated widely to other organs of the body through the blood stream, a more serious form of TB develops known as miliary TB.
- Since mycobacteria causes inflammatory reactions, it results in accumulation of inflammatory fluid in pleura, pericardium and peritoneum leading to ascites in those areas. These are more serious complications that must be managed in hospital.

CLINICAL PICTURE

Early signs

- productive cough for more than 2-3weeks
- positive TB test
- unexplained weight loss
- night sweats

Late signs

- Blood stained sputum (hemoptysis). It is highly suggestive and patient may die due to massive hemorrhage
- Enlargement of lymph nodes
- Difficulty in breathing
- Severe weight loss
- Symptoms of other organs involved

DIAGNOSIS

1. Clinical features: cough more than 3 weeks, sputum production (blood stained) or purulent. No response to antibiotics. Weight loss
2. Sputum smear examination using Ziehl-Nielsen staining technique for acid-fast bacilli to diagnose PTB. Patient sputum is collected during the visit (spot specimen) then the patient is given a container to collect sputum the following morning. After delivery of the 2^{nd} specimen a third specimen is collected on the spot (2^{nd} spot specimen)
3. Staining of the contents from affected tissue using Ziehl-Nielsen staining material to diagnose Extra-Pulmonary TB e.g fine needle aspirate (in TB adenitis) CSF (in TB meningitis) skin biospis (in skin TB) lupus vulgaris.
4. Cultures when AFB staining is negative or when resistance is suspected.
5. Chest radiography: disadvantages- difficult to interpret and higher cost compared to microscopy
6. Tuberculin skin testing. To test whether the individual has been previously exposed to the M. Tuberculosis e.g Mantoux test. The resulting skin reaction does not necessarily imply current disease.

A small amount of tuberculin (10 tuberculin units is 0.1ml of saline) is injected intradermally in front of the forem. A small swelling appears and the site is examined for individuals. 3 days later, the diameter of induration is recorded in mm. More than 10mm (suggests BCG vaccination, natural infection) less than 10mm (absence of active TB) or weakened immune system since it is so weak it cannot respond

MANAGEMENT

Treatment of TB serves 3 main purpose:

- To cure the individual patient
- To render the patient rapidly non-infectious
- To prevent emergence of drug resistance

WHO recommends use of DOTS strategy in TB control (directly observed treatment short course)

- Directly observed – observed patients as they swallow drugs
- Treatment- provided with complete treatment, monitored and progress assessed until the disease is cured (good reporting).
- Short course – correct combinations and dosages used in correct length of time

A combination treatment of 2 or more drugs is used since a single drug use will most likely lead to development of resistance by mycobacteria. Treated in 2 phases: intensive therapy and continuation therapy phase.

Intensive therapy; patient is treated as an inpatient and seen daily as an outpatient (takes 2 months). A combination of the most powerful drugs used to kill as many tubercle bacilli as quickly as possible.

Continuation therapy: patient continues treatment with supervision at home. Patient reports to the health facility every month for collection of fresh supply of continuation phase drugs, and review of general health and response to treatment. Follow up sputum taken every 2 months in 6 months short-course and in months 2^{nd}, 5^{th}, and 8^{th} month in 8 months

Drugs used are;

- Isoniazid (INH, H)
- Rifampicin (R)
- Pyrazinamide (PZA, Z)
- Ethambutol (E)

Combination drug regimen used to reduce number of tablets to swallow to

- Improve compliance
- Reduce chances of resistance 110

To be discharged patient need to be fully cured. However if relapses occurs patients should be alerted to come back to clinic as soon as possible. Therefore adequate health education is important throughout the treatment to ensure patient's co-operation.

Drug reaction

Most common is itching skin rash but alleviated by antihistamine drugs. Others include;

drug	**Side effect**
Streptomycin	Allergic reaction (rash fever) numbness, dizziness, hearing strange noises, ataxic gait, deafness

Rifampicin	Fln syndrome, jaundice, skin rash
Isoniazid	Nausea, diarrhoea and vomiting, peripheral neuritis, jaundice, pellagra –like dermatitis
Pyrazinamide	Anorexia, nausea, flushing , anemia (rare), gout. Gouty arthritis (quite frequent)
Ethambutol	Ocular toxicity presenting as blurred vision or red-green color blindness

Streptomycin avoided in pregnancy due to risk of congenital deafness. Patients with severe reactions need to referred to hospital for admission and proper management.

COMPLICATIONS

Since it has a high fatality rate of 12-15 %, it is important to detect the complication early and manage well for proper prognosis. Most common complications include;

- Severe hemoptysis
- Respiratory failure
- Pleural pericardial effusion
- Meningitis
- Kidney failure
- Vertebral collapse (Pott's disease)

HIV AND TUBERCULOSIS

Patients with HIV infection are very prone to TB because of the breakdown of their immune system. The disease may be due reactivation of primary infection, often long suffer the first infection took place or it may be due to a fresh infection easily acquired from an infectious contact since the patient has lost immunity to the disease.

HIV positive patients develop many kinds of opportunistic infections hence difficult to distinguish from TB. Symptoms vague and may be overshadowed by the pathology of AIDS rate disorders. A thorough diagnostic work-up is required in these patients.

It is also important when examining the patient to be on the look for other disease conditions which may be present in the same patient since a diagnosis of TB can be made without anyone being alerted that they have an underlying HIV infection.

One must make correct diagnosis before starting anti-treatment since HIV patients are prone to severe drug.

The actual treatment of TB does not differ from that of HIV negative patients

HIV and TB co-infection present challenges to successful treatment because of:

- Increased mortality in such patients
- Increased co-morbidity
- Increased number of drug side effects
- Serious interactions between ARU and Anti-TB drugs
- Immune reconstitution syndrome
- Increased recurrence rates after completion of treatment

NB TB treatment must be commenced as a matter of priority. The ARV therapy will be started depending on the patient's clinical and immune status.

TB in Children

Difficult to diagnose because of difficulty in obtaining specimen. Gastric lavage can be used to obtain specimen for ZN staining in order to demonstrate swallowed mycobacteria but the results are unreliable. Physical and x ray exams cannot reveal much. Therefore diagnosis is made primarily on history, clinical signs and symptoms and the result of tuberculin test. A score chart may help in making the diagnosis

ANTI –TB dosages for Children

drug	Max	kg	Points
Isoniazid	300mg	5-10	0-2
rifampicin	600mg	10-20	3-4
Pyrazinamide	2000mg	25-40	5-6
ethambutol	1200mg	15-25	
Streptomycin	1000mg	15-20	>7

DRUG RESISTANCE

TB bacillus can become resistant to therapy with one or more drugs. Almost all cases of drug resistance is due to failure to adhere to full course of anti-TB therapy.

Secondary resistance can be caused by poor absorption of drugs due to diarrhoea, inadequate dosing, poor quality drugs, drug-drug interactions with other drugs that result in low levels of anti-TB drugs.

Primary resistance can result when patient develops resistance before exposure to treatment. Resistance to both Rifampicin and Isoniazid is referred to as (MOR-TB) multi-drug resistance TB. Resistance to one or two drugs but excluding to both Rifampicin and isoniazid together with 2 line agents is called (XDR-TB) Extensively drug resistant TB. Management of (MDR-TB) is a complex and most patients usually succumb to the disease. There is no current therapy for XDR-TB. Therefore ensuring and encouraging full compliance to therapy in all patients

PREVENTION AND CONTROL

Case finding and treatment

- Put all patients on treatment as early as possible to prevent
- Early case finding. Community health worker should visit all homes of all new TB cases to look for other cases

  Contact tracing and active case finding
- Advertise routine methods of TB treatment in the health center.
- Proper health education to patient and relatives and public

BCG provide active immunity against TB. Loses potency in sunlight, ulcer take 6 weeks to xxx xxxxx is a proof of BCG vaccination. (on the left forearm or light shoulder)

Not to spit anywhere carelessly. Sputum mug provided in which sputum is disposed carefully in fire or pit latrine. Soiled mug should be dried in bright sunlight after disposal of the contents. Avoid overcrowding and poor ventilation.

LEPROSY (HANSEN'S DISEASE)

Leprosy is one of the oldest diseases of human beings. It is caused by a bacteria belonging to the same family as the mycobacterium that causes TB, known as mycobacterium leprae. Leprosy is a major public health and socio economic problem because it is a disabling and deforming disease. Leprosy is not a killer disease in that it runs a chronic course and does not significantly reduce the life expectancy of infected individual.

In some communities patients suffering from leprosy are discriminated against or stigmatized due to ignorance and unfounded traditional beliefs. This causes a lot of distress and misery to those infected and their families. In Kenya, leprosy is almost been eradicated except for a few endemic areas in coastal, eastern, nyanza and western province.

Mode of transmission

Leprosy has a long incubation period and runs a chronic course if it is not adequately treated at an early stage. The mycobacterium leprae bacillus multiplies very slowly (dividing only once every 14-30 days). That is why the incubation period is long, about 5-8 years. Just like TB, the leprosy bacillus is transmitted by droplets, by sneezing, coughing, spitting and unhygienic nose cleaning habits. The organism is also suspected to enter the body through broken skin such as small wounds. Leprosy is a common among family members of the infected.

There are certain factors that increase the incidence of leprosy in the community. These are:

- Presence of many untreated cases
- Overcrowding in living houses
- Presence of susceptible new comers in leprosy endemic areas
- Hiding patient with leprosy and starting treatment late

Classification (Type) of Leprosy

Broadly speaking, there are two forms of leprosy: the tuberculoid form and the lepromatous form. Let's briefly discuss the two;

1. Pauci- bacillary leprosy(PBL) also called tuberculoid leprosy is characterized by;
    - Absence or presence of very few bacilli in the skin smears or skin biopsy (skin smear is negative)
    - Skin patches 1-5 cm
    - Reaction type I
    - Nerve involvement/damage effects one or more peripheral nerves
    - Disability and deformities affects one or more peripheral nerves
    - Deformities and disability are common as a result of irreversible nerve damage and most are disfiguring
2. Multibacillary leprosy (MBL) also called lepromatous leprosy, is characterized by
    - Presence of numerous bacilli in most tissues of the body, except brain and spinal cord
    - Skin patches six or more
    - Skin smear positive(numerous bacilli present)
    - Reaction both type I and type II
    - Nerve damage comes late
    - Disability and deformities usually develop at a later stage of the disease

Nerve involvement in Leprosy

The main cause of disability in leprosy is the destruction of the nerves. Damage to the sensory nerve fibres causes anaesthesia, while damage to the motor nerve fibres, causes paralysis. Impaired circulation, loss of sweating and skin atrophy is caused by damage to autonomic nerve fibres.

Leprosy patients may get burned or injured on their limbs and fail to notice because of anesthesia. The patient may walk on an injured foot without realizing it.

In the eye, the cornea may become anaesthetic so that foreign bodies may enter unnoticed leading to corneal damage. Anaesthetic eyelids may lose the blinking reflex or fail to close the eye (lagopthalmos) leading to dryness, iritis, adhesions, glaucoma and blindness.

Clinical features

After infection, the mycobacterium leprae bacilli multiply in macrophages of the skin and the Schwann cells of the peripheral nerve fibres. The bacillus has a preference for the relatively cool places in the body such as the face and the limbs. The early signs of leprosy are as follows:

Hypopigmented patches on the skin with loss of sensation to pain, touch and temperature;

- Loss of sweating or loss of hair over affected part
- Burning sensations in the skin
- Weakness of eyelids, hands or feet
- Thickening of cutaneous nerves especially the ulnar, median and lateral popliteal nerves
- Nodules in the skin especially of the nose, face and ears
- Painless wounds(ulcers) and burns on the hand and feet

Reaction types

Reactions are sudden unexpected changes which occur in all types of patients with leprosy. These reactions are caused by a change in the balance between the immunity of a patient and the bacilli. There are two main types of reactions, type 1 or reversal reaction and type II or erythema Nodusum Leprosum. Let us briefly consider each type:

Type I Reaction (reversal reaction) - is common in Pauci-Bacillary leprosy (PBL). It occurs after a sudden increase in immunity results in a rapidly increased response of the body to the leprosy bacilli. This reaction cause sudden inflammation in places where the leprosy bacilli are present. It causes nerve damage, inflamed and raised red skin lesions and oedema of hands, face or feet.

Type II Reaction (Erythema Nodusum Leprosum) this appears 6 months or more after treatment and is caused by a reaction between dead leprosy bacilli and circulating antibodies. Nerve damage is not common in this reaction. Eyes, joints and testes become inflamed, nerve become tender and ulcerating tender nodules appear on the skin. Thus reaction is usually of sudden onset and tends to recur.

Generally, reactions in leprosy are provoked by a number of factors. These include;

- Malaria, malnutrition, anaemia
- Severe emotional of physical stress
- Menstruation, pregnancy, abortion, puberty and childbirth.
- Using drugs containing iodine
- BCG vaccination
- Osteomyelitis
- Septic wounds

NB Drugs do not cause reactions and therefore should not be stopped

Late deformities of leprosy

The following are late deformities of leprosy;

- Paralytic deformities including claw hand, claw fingers, wrist drop, claw toes, lagophthalmia, corneal, ulcers and facial paralysis
- Depression of the nasal bridge
- Wrinkling of facial skin
- Disfigured ears
- Stiffness of finger joints
- Shortening and loss of fingers and toes

Diagnosis

The diagnosis of leprosy can be made using the following;

- Clinical signs: presence of pigmented anaesthetic patches on skin and thickened nerves
- Bacteriological examination: skin slit crap, nasal smears for leprosy bacilli
- Chemical test: histamine test, Lepromin test

Management

The aim of leprosy treatment is to prevent nerve damage, deformity, blindness and defaulting. The national leprosy and TB programme (NLTP) in Kenya uses the WHO recommended multiple drug therapy for the treatment of the two classes of leprosy. Let's start by looking at the treatment for tuberculoid leprosy

Pauci-bacillary (tuberculoid) leprosy (PBL)

This type of leprosy is treated for 6 months as shown in table 10 below;

Table 10: six months treatment for pauci-bacillary leprosy for all ages

	0-5years	6-14 years	0ver 14 years
Rifampicin every 4 weeks supervised	150mg	300mg	600mg
Dapsone daily	25mg	50mg	100mg

Adapted from the Kenya National Leprosy and Tuberculosis Programme (NLTP)

Multi-bacillary leprosy

Multi-bacillary or lepromatous leprosy is also treated for months as shown in the following table.

Table 11: six months treatment for multi-bacillary leprosy for all ages*

	0-5 years	6-14 years	Over 14 years
Dapsone daily	25mg	50mg	100mg
Clofazimine (lamprene) four weekly supervised	100mg	200mg	300mg
Clofazimine (lamprene) Unsupervised	50mg on alternate days	50mg daily	50mg daily
Rifampicin every 4 weeks, supervised	150mg	300mg	600mg

Adapted from the Kenya National Leprosy and Tuberculosis programme (NLTP)

NB: The treatment of tuberculosis keeps changing depending on current research findings. Please check on the current treatment and adjust your notes accordingly

Having looked at drug therapy let us now find out what else can be done to prevent blindness and deformity.

Wound Prevention in Leprosy

Wounds are caused and made worse by the loss of sensation to pain, pressure or burning. Therefore to prevent further damage you should advice the patient to do the following;

- Wear protective footwear
- Wear heatproof gloves when working and handling hot objects
- To inspect the feet and legs regularly for swelling, cracks, bruises, injuries, dryness – a small mirror can be used to inspect the soles of feet.
- To soak feet for minutes twice daily in salty water, then rub oil on the skin to keep moist and prevents cracks
- To remove grit from inside the shoes

Eye care

For patients who are suffering from lagophthalmos, you should advise them as follows;

- To wear sun glasses
- To check the eye daily in front of a mirror for inflammation and foreign bodies
- To cover eyes with pads at night
- To avoid rubbing the insensitive eyes

Exercises

It is a common knowledge that joints which are not used become stiff, while muscles atrophy and become weak. Also scar tissue tends to retract resulting in contractures. That is why all patients with weak or damaged hands should do suitable exercises. For paralyzed muscles, passive exercises help to loosen the stiff joints and lengthen the skin. The exercise should be done for 5-10 minutes daily on regular basis.

Prevention and control

The cornerstone of leprosy control is to reduce the number of effective cases and interrupt transmission. These can be achieved through the following preventive measures

- Treatment of effective cases until cured
- Searching for unknown cases, registering and treating them
- Administration of BCG vaccine which gives some immunity against leprosy

HELMINTHIC DISEASE (HELMINTHIASIS WORM INFESTATION)

The worms of medical importance are divided into three main group according to their form.

Group 1

1. Nematodes (roundworms)

They are cylindrical and xxxxx includes

- Axcaris lumbicoides
- Stnonglyoides stercolaus
- Anklylostoma duodenale (hookworms)
- Nector americanus
- Trichuri xxxx
- Enterobius vermiculaus
- Filarial worms

2. Trematodes – flukes

They are leaf like or cylindrical:

- Schistoma mansoni
- Schistoma haematobium

3. Cestodes (tapeworms)

They are flat and segmented, includes;

- Taemia saginata
- Echinococcus (dog tapeworm. Hydrated disease)

NB: filarial disease and schistomasis are under infectious and vector-borne disease (IVBDs). The final host of all worms is man except dog tapeworm – for which man is an incidental intermediate host. The eggs of all the intestinal worm are excreted in stools. Sanitary disposal of feaces is the preventive measure of choice because it will control all the worm the diseases except hydtidiosis. However faecal disposal is the most difficult preventive measures to achieve because the co-operation of every member of the community including children is necessary. Building of latrines is of no use if health education is given in an attempt to change the attitude and behavior of people.

ASKARIS LUMBRICOIDES

Ascaris is a chronic nematode infection, an intestinal roundworm.

Importance and occurrence

- World wide
- Common in warm hot humid areas like coastal belt with poor sanitation which if persist largely because of indiserxxxxxx defecation by children.
- Children are more frequently and most heavily infected than adults because of their habits of putting all kinds of things in their mouth.

Epidemiology

- The roundworms is a large intestine worm. It lives in small intestines. A female may lay (produce) up to 20,000 eggs daily.
- The eggs are passed out in feaces and they must be embryonated in soil before they are infective.

- Embryonation takes 5-50 days and soil must be loose and not dry and available at temperatures of 15 degrees Celsius. The embryonated eggs can be carried away from defecated place into houses by feet, footwear or in dust by wind.
- When eggs are swallowed by human beings they batch in intestinal canal
- Vehicle of transmission- salads xxxxxxxx
- To reach maturity the larvae need to pass through the lungs.
- The larvae penetrate the intestinal wall and reach the liver via portal system, from the liver they are carried through the right side of the heart to the lungs.
- Here in the lungs they penetrate into the airways and pass via bronchioles, bronchi and trachea to the pharynx.
- There they are swallowed return to the GIT and then settle in the jejunum
- During the lung passage, eosinophilia develops. The eosinophilia is temporary if new infection occur

Signs and Symptoms

- Difficulty in breathing and jaundice, usually it may be symptomless or symptoms are not characteristic.
- Fever – migration of larvae through the lungs cause fever
- Coughing and sneezing
- Abdominal crumps – especially if the infections is high
- Eosinophilia – raised WBCs level
- Adult worms in stool or vomits

Complications

- Intestinal obstruction
- Volvulis
- Malnutrition
- Wandering of worms

Management

Investigations

- Stool for ova
- Chest x-ray – may show larvae migration
- Blood for full haemograms, show raised WBCs (eosinophilia)

Patient usually treated as an outpatient

Medical mx;

- Membedazole
- Pyrantel pamoate
- Larvamisole (ketrax) 3 stat
- Piperazine (antepar) 150mgs xxxxxxxxxxxxx

Intestinal obstruction and volualus – refer the patient for surgery – pass nasogansic tube

NB when obstruction is evident, avoid piperazine to prevent worm wandering

Prevention and Control

Feacal disposal

- Provision of adequate facilities for faeces disposal and health educate on their use
- Feaces use – discourage feaces uses for manuring. However, compositing for 6 months will kill ascaris then it can be used safely as fertilizer

Health education

- On use of toilets
- Washing hands before handling foods
- Not to eat foods dropped on the floor
- Treatment of infected person

NB: treatment and screening of individuals is of no use as long as feaces are not disposed safely

Action

- Inspect health centres and other health facilities by the health officers
- Pit latrines of schools, markets for their existence and cleanliness
- Ask committees to make materials available for constructing latrines
- Build a collaboration with community members a demonstration pit latrines in a suitable place to serve as an examples of proper construction
- Find out the community attitude for not using the latrines, local taboos
- Alleviate on changing any unhygienic traditional behavior.

HOOKWORM (ANKYLOSTOMIASIS, SAFURA)

Definition:

It is a nematode infection of the intestines, hookworm infestation may be caused by;

- Ankylostoma duodenale
- Nector americanus

Importance and occurrence

About ¼ of world population is infected with hookworms. Common in warm, moist places (where sanitation is poor- with minimum temperature of 10 degrees Celsius). Many people harbor hookworms without any ill effect (hookworm carriers). Americanus duodenale is found in Mediterranean area, India, China and Japan. Nector americanus is found tropical areas of Africa, Asia and America. The nutrition status, the daily iron intake and the total worm load determine where carrier going to have ill effects from the parasite and become hookworm sufferer.

Hookworm anaemia is one of the main causes of anaemia in the community. Anaemia has a profound effect on the working capacity and the xxxx of wellbeing of an individual. The economic has caused by anemia is enormous but difficult to calculate.

Life cycle

There are two types of hookworms both of them nematodes.

Eggs of either hookworm are discharged (passed) in the stools and hatch in the soil after incubating for 1-2 days. In a few days the larvae are released and live in the soil (damp moist) these are not infective before they have changed to sheathed filariform stage (about 5 days)

The filariform larvae may attach themselves to grass or hide in the soil. And as soon as they are touched by something they attach themselves to it. Human beings become infected by the walking barefoot through a field contaminated by the infected human feaces. They penetrate the skin actively and they reach the lungs via lymphatic and venous systems. In the lungs penetrate into the alveoli through the bronchioles, bronchi and trachea to the larynx and pharynx. Xxxx they are swallowed and reach the duodenum 3-5 days after penetrating the skin. The larvae attach themselves by their mouths with hook like teeth to the small intestines and such. The blood and the cycle is complete within 40 days.

Signs and symptoms

Anaemia which is non deficiency is the main sign and it's due to sucking of blood by the worms and it responds well to used therapy. An itchy that raised rash (ground rash) develops where penetration took place.

NB: one N. americanus causes blood loss of xxxx . A worm load than 100 americanus. N. americanus always cause anemia. Hookworm is able to suck 0.5mls of blood everyday

1-25 worms – blood lost – 12.5 mls –mild

25-100 worms – blood lost – 50mls – moderate

100-500 worms – blood lost – 250mls – considerate

500-1000 worms – blood lost – 500mls –

When the larvae are in the lungs there xxxx syndrome – when larvae are penetrating causes a characterized by a dry cough.

Ground itch – on the place where the larvae penetrate the skin some enzymes papules is seen and itching develops most common in toes and on the back of the feet.

- Eosinophilia
- Dyspepoia
- Abdominal pains, distension and sometimes diarrhoea
- In mixed infection there's diarrhoea mixed with blood

Investigations

- Stool for ova
- Blood for fall haemogram eosinophilia
- More than 100 eggs in an ordinary faecal smear indicates a heavy infection

Management

- Not all hookworm infections will need treatment
- Re- infection is very likely if the community does not improve its way of feacal disposal
- Anaemia is treated non therapy fesox 200mg x 2/12, protein diet to replace lost proteins also non infection. Xxxxx severe causes blood transfusion may be needed
- When condition improves antilel- minthetic is given

  Pyrantel

Dewormers

1. **ICE** – tetrachloethylene 4mls orally on empty stomach

2. **Levamiscle**

Prevention and Control

- Wearing of shoes will prevent the infection
- Avoid working/walking in contaminated areas
- When handling suspicious materials wear gloves
- General control- public hygiene – provision of toilets and seen they are used
- For young ones- let the feaces be collected and disposed into pit latrines

NB: Never burry feaces – you will be incubating them

- Deworming campaigns or mass treatment.

TRICHURIASIS (WHIPWORM)

It's a nematode roundworm infection of the large intestines caused by trichivis trichiorra

Occurrence and Importance

- Common in hot, humid climate
- Occurs in most parts of the world mainly in subtropics and tropics where there's poor sanitation
- Common in regions where pigs are fed on root vegetables – France, USA

Mode of Transmission

Man acquires infection indirectly

Epidemiology

Infections results from eating raw or inadequately cooked, processed pork or pork products. L

argely occurs are marine animals. Any of these animals may contain a cyst form of the larvae (trichuriae)

Life cycle

Eggs are passed in the feaces, which require embryonation in soil, so anti-infection is not possible. When embryonated eggs are ingested they hatch – in stomach duodenum. These embed themselves from caecum to rectum. Depending on the worm load, when the load is embedded in the mucosa they produce cytolytic enzyme which dissolve in the body tissue. Production of cytolytic enzyme bring about inflammation of mucosa and cause bleeding. When very many, they loses intestinal mucosa and cause rectal prolapse. The eggs have very many strong cells and remain viable for seven years under favorable conditions.

NB: each larvae grows to about 4 ½ inches mature females produce about 15,000 eggs a day which are passed in stool.

Signs and Symptoms

- Eosinophilia is present only in heavy infection
- Mild infection are symptomless
- Abdominal discomfort
- Loss of weight
- Anemia
- Prolapse of the rectum especially a child or a woman in labour

Dx: Stool for Ova – the barrel-shaped eggs are visually visible in stool samples examined under microscope.

Management

Tabs membedazole (thiabendazole) minexx 500mgs/kg/day x 3/2

Prevention and Control

- Good personal hygiene
- Sanitary disposal of feaces
- Avoiding uncleaned vegetables
- Availability of latrines

ENTEROBIUS VERMICULARIS (PINWORM, THREADWORM, ENTEROBIASIS, SEATWORMS)

It's a benign intestinal disease with mild non-specific symptoms caused by enterobius vermicularis

Occurrence and Importance

Worldwide in occurrence in temperature regions and less common in intemperate zones. More common in school children and in learning institution. Usually a whole family may be infected with enterobius.

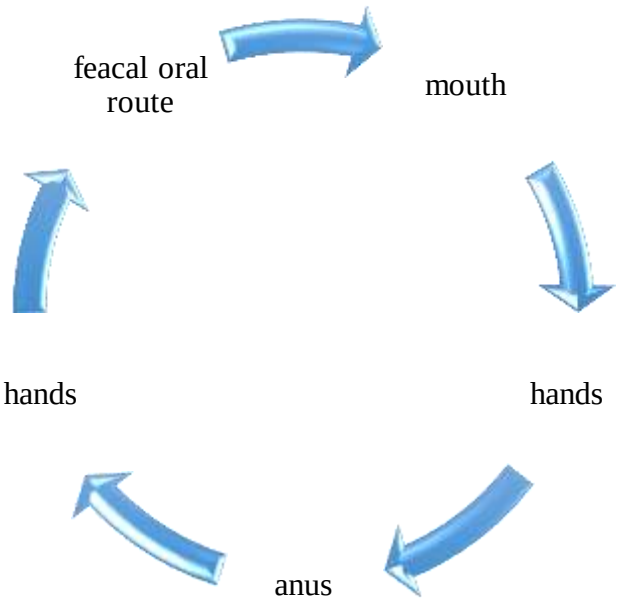
Epidemiology

Caused by enterobius vermicularis, which is a nematode (phylum) and in order of Rhabifidae and family of oxyuridae. Initial infection occurs by the feacal-oral route, infection is maintained by direct transfer of infective eggs from the anus to the mouth (xxxxxx) or indirect feacal oral contact

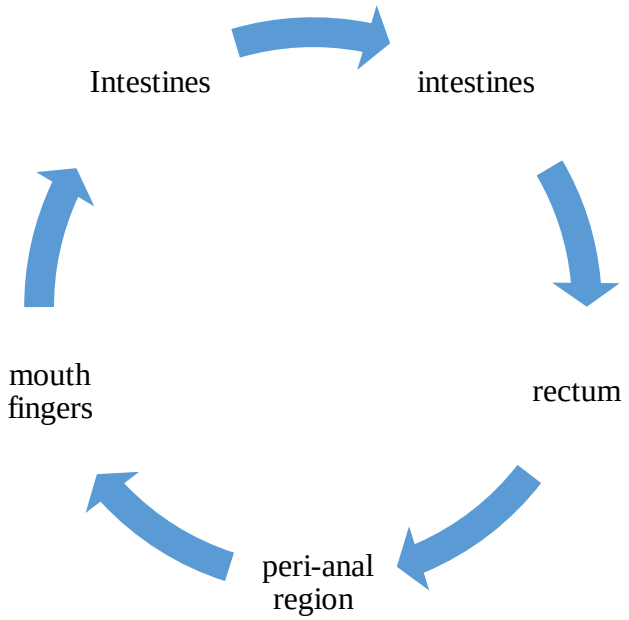
through clothing, bedding, food or other articles. Airborne infection through inhalation of dust containing eggs and subsequent swallowing them. Auto infection is easily established.

Life cycle

Man acquire infection through ingestion of the eggs. In duodenum the eggs hatch. They have to move in small intestines before they settle in large intestines. In large intestines they are visually found in caecum. When mature, females are fertilized by males which die immediately and female develops eggs. When females are about to lay eggs they migrate to the anus –peri-anal region and under relative lower temperature the uterus burst releasing so many eggs. The eggs are partially embryonated 3 hours they complete embryonation and are infective. These worms can infect many within a short time especially in crowded places such as large families and schools. When the females empty along the peri-anal region they produce a lot of itching around the anal area – especially in small children by producing chemicals which are xxxx to peri-anal region. At times these eggs hatch in perianal region and the larvae migrate back to the rectum- retro infection.



Life cycle of enterobius vermicularis



Symptomology

- Xxxxx xxxx (itching)
- Xxxxx valval – can migrate to f.tubes
- Scratching effect
- Disturbance of sleep

Lab Investigation

Not easy

- Sotch take method – a seal tape adhesion take over the anus in the early morning
- Stick tape onto the slide and examined under microscope

Management

- Membedazole
- Parantel } single dose
- The whole family must be treated

Prevention and Control

- Personal hygiene – bathing and hand washing, cutting nails short, clean under clothes,night clothes and bed clothes
- Avoid overcrowding
- Proper feaces disposal
- Treating the whole family to avoid re-infection
- Health education on above

HYDATIDOSIS (ECHINOCOCOSIS, HYDATID CYST)

Disease caused by the cysts of the dog tapeworm

Mode of transmission – feacal oral route

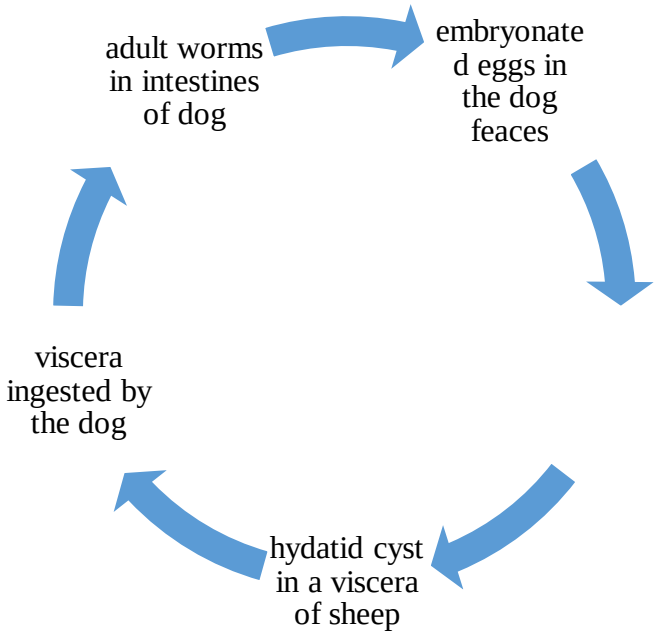
Importance and Occurrence – common in East Africa especially Kenya (Turkana)

Epidemiology

Dogs and other carnivores are the final host of the dog tapeworm. Eggs are passed in dog feaces and ingested by sheep and goats. The eggs hatch in the sheep intestines and larvae penetrate the intestinal wall to form cyst with many daughter cyst in the Liver. These cysts are the infective

stage for the dog and develop into mature worms when they are ingested similar to the cow man Cycle in haemasis but in taemasis man is the final host. Man becomes infected with cyst when he/she accidentally swallows the eggs from dog feaces.

Life Cycle of Echinococous Grambosis



Signs and Symptoms

In sheep slowly growing cysts develop usually in the liver or any other body tissue and the lungs which are the next commonest site. With the liver enlargement the disease suggest liver abscess.

Management

Usually the cyst is punctured. Puncturing provokes severe anaphylactic shock (response). Chemotherapy is not xxxx.

Prevention and Control

- Meat inspection intensification
- Condemning inspected meat by health officers
- Deworming dogs regularly
- Health education especially children in endemic areas on dangers of close contact with dogs (licking)

Prevention and Control

- Meat inspection by qualified veterinary officer
- The owner is instructed not to sell the meat to customers once the meat is condemned
- Proper cooking even after the meat has been inspected
- Proper disposal of human feaces by use of pit latrines
- Men fence their grazing field to keep away the trespassers who help themselves on grass.

STRONGYLOIDIASIS

Definition: Is an infection by strongyloidiasis steicoralis, a nematode worm. The female adult worms live in the mucosa of duodenum and jejunum. Most infections are without signs and symptoms.

Importance and Occurrence

Occurs in warm moist places where sanitation is poor. Found in tropical areas of Africa, Asia and America.

Epidemiology

Strongyloides stercoralis resembles hookworms in appearance of adult eggs and larvae. In strongyloides infections larvae are found in the stool. These larvae may develop either in free-living adults which produce the next generation infective stage outside the body or the larvae may develop directly into infective filase form larvae which penetrates the skin. Because of direct development into infective stage into immune-infectious is common. Even within the bowel the larvae may become infective and penetrate the bowel way. This is called **Endogenous reinfection.** If these feaces are passed in grazing areas they are ingested by cattle and camels. The cow ingest the whole segment or eggs, when ingested they into intestines where the eggs hatch out – hexacanthxx embryophore stage. When these larvae mature they have capacity of burrowing in mucosa and blood capillaries. In the blood the eggs are carried to all parts of the body and end up settling in striated muscles – masseters, tongue, diaphragm and muscles of the fore legs

Signs and Symptoms

NB In most people the disease causes no symptoms

- Upper abdominal pains
- Diarrhoea
- Weight loss
- Feeling of a piece of the worm move out through the anus
- Presence of worms in the feaces

Treatment

- Niclosamide / yomesan
- Praziquantel often

- Checking of stool – 3months+ 6months

TAENIASIS (TAPEWORM)

Thus:

TAENIA SAGINATA- BEEF TAPEWORM

TAENIA SOLIUM – PORK TAPEWORM

They belong to lestode group.

Occurrence and Importance

Taemias is common in all countries where beef is consumed raw or only lightly cooked. Effects on patients are mild. Tapeworms are segmented and each segment is a complete unit and can detach and fall. Male and female are found in one i.e they are hermaphrodite. When they are mature the eggs are produced and are already fertilized. Mostly affect men and women.

Life Cycle

Man is a definitive host – harbors adult stage of the worm. Cattle and camel are intermediate hosts. Man acquires infections through ingestion of raw meat/half cooked meat containing of state of the worm called cysticercus boris. When this gets into the intestines of man the proscolegc invaginates into the walls of the ileum and small intestines and grow to a mature worm within 6-8 weeks. When mature the terminal contain mature fertilized eggs and passed into feaces. Sometimes these segments can clip out of the bowel and move down the legs.

Signs and Symptoms

- Radiating pain in the xxxxx

- Malabsorption
- Diarrhoea
- Rash in a linear pattern (sensitivity reactions due to continuous reinfection

Management

- Thiabendazole (mintezol) 25mg/kg/day in divided doses x 3/7
- Levamisole (ketrax)

Prevention and Control – see hookworm infections