



Communicable Diseases-1-1-1

community health nursing (Kenya Medical Training College)



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UNIT FOUR: COMMUNICABLE DISEASES

In this unit, you will explore the common communicable diseases found in this country. The unit is divided into eight sections. In section one and two you will cover patterns and principles of communicable diseases, respectively. In sections three to eight, you will examine specific communicable diseases classified according to their modes of transmission.

This eight units are as follows:

Section One: Patterns of Communicable Diseases

Section Two: Principles of Communicable Disease Control

Section Three: Contact Diseases

Section Four: Vector-borne Diseases

Section Five: Diseases Caused by Faecal-oral Contamination

Section Six: Airborne Diseases

Section Seven: Helminthic Diseases

Section Eight: Diseases of Contact With Animals or Animal Products

Unit Objectives

By the end of this unit you will be able to:

- Describe the pattern of communicable diseases in a community
- Explain the principles of communicable diseases
- Describe the causative factors of communicable diseases

- Explain the mode of transmission of communicable diseases
- Describe the management approaches for communicable diseases
- Describe the preventive measures for communicable diseases

SECTION 1: PATTERNS OF COMMUNICABLE DISEASES

Introduction

In unit three, you covered environmental health and learnt that although the environment can be a source of ill health, you can prevent many diseases and health problems through simple measures such as good personal hygiene and proper waste disposal. However, despite your best efforts, you can still contract diseases from different sources. The common cold is a good example. It takes just one person infected with the flu virus to spread it round to the others in the office or at home. A disease that is passed from one person to another person is called a communicable or transmissible disease. Transmissible diseases include: measles, HIV infection, tuberculosis, chickenpox, gonorrhoea,

scabies, malaria, cholera, and roundworms among others.

Communicable diseases are among the most important diseases in this country. They are important because:

- Many of them are common
- Some of them are very serious and cause death and disability
- Some of them cause widespread outbreaks of disease – epidemics
- Most of them can be prevented by fairly simple means

In this section you will cover disease patterns in the community, the meaning of host and infection, as well as the transmission cycle of communicable diseases.

Objectives

By the end of this section you will be able to:

- Describe the patterns of diseases in the community
- Describe the interaction between the host and the infecting agent
- Describe a typical transmission cycle

Patterns of Communicable Diseases in the Community

Different diseases are common in different places and at different times. To understand why this happens, you need to consider the disease causative organisms (the agents); the people they infect (the hosts); and the surroundings in which they live (the environment).

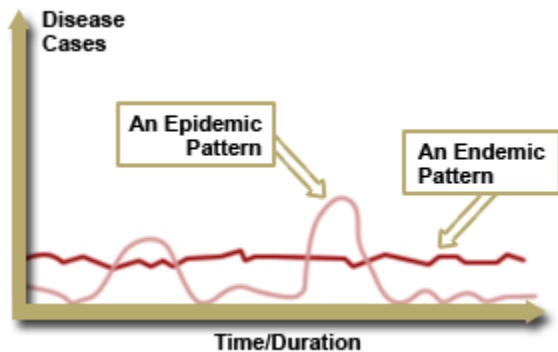
A delicate balance exists between the agent, the host and the environment and it can change in different ways. For instance, the agent needs a suitable environment in which to grow and multiply and thus be able to spread and infect other hosts. If the environment does not support the agent it dies or transforms to a dormant state. The host (person) is also affected by the environment. For example, a person may live in a hot, wet climate where there are many mosquitoes. However they can change this environment by draining swamps, clearing the vegetation and adding competing hosts such as animals. If the balance is shifted against the agent, the disease will be controlled and the number of cases will go down.

When the balance between the agent, the host and the environment is fairly constant, you tend to see approximately the same number of cases of the disease every month. When this happens the disease is said to be endemic. When the balance is shifted in favour of the agent (organism), for example, when many non-immune children have been born in an area since the last measles epidemic, a large number of cases of measles may occur in a short time. This is called an epidemic. Epidemic diseases occur during certain periods or seasons and cause sudden deaths and much suffering in the community.

An endemic disease can be termed as that which occurs in a given population at a constant rate over a period of several years. An epidemic

disease is that which occurs in a population at a higher rate than is usually the normal for that population over a given time interval.

Diagrammatically the endemic and epidemic disease patterns can be illustrated as shown on the right.



List down three epidemic diseases in Kenya.

Some Common Epidemic Diseases in Kenya

- Cholera
- Typhoid fever
- Highland malaria
- Acute bacterial meningitis

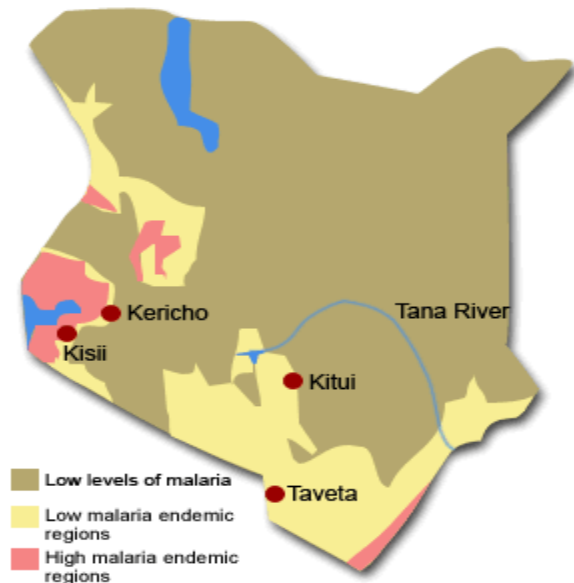
In Kenya, malaria is endemic in the lowlands, such as the Tana River basin, the coastal strip, and the Lake Victoria region. Schistosomiasis which is related to water use is endemic around the Lake Victoria region and the Mwea irrigation scheme. Leishmaniasis is endemic in Baringo,

along Tana River, and along the River Athi in Machakos.

In some parts of the country, some disease outbreaks occur only occasionally without a regular pattern. Such diseases are said to be sporadic in their occurrence.

Opposite is a map of Kenya illustrating the malaria patterns.

Now, look at what happens once an infecting organism enters a person's body and causes disease.



The Host and Infection

A person who is invaded by a disease-causing micro-organism is called a host. An infection occurs when this micro-organism begins to reproduce (multiply) and grow. When an organism infects a person, there are three possible stages to consider.

Incubation Period

The incubation period is the time between infection and the appearance of symptoms and signs of an illness. During the incubation period

the host does not realise that they have an infection until several days later when detectable symptoms and signs of the illness occur.

Sub-clinical Infection

At this stage, infection does not produce clear signs and symptoms. The host's immune system is trying to fight off the agent. In some cases, the organism is overcome by the host immune cells hence no signs and symptoms are felt and the infection process is terminated.

Clinical Infection

This is the period when the host develops detectable symptoms and signs of an illness. At this time the agent has multiplied within the host overcoming the host's immune system and has started causing abnormal functioning of some body cells and tissues. This produces overt signs and symptoms of the disease. It is important for you to understand these stages because people with symptoms are easier to identify as they come to your health facilities for treatment. People with sub-clinical infections do not always know they are infected and hence are a danger to other people. They are also difficult to detect in the general population without special tests. An individual who is suffering from a sub-clinical infection is also likely to infect others, as in the case of HIV infection which leads to AIDS after a long period. They are therefore known as carriers. An individual who develops a clinical or sub-clinical infection is said to be susceptible to the disease. A susceptible individual is one whose body lacks resistance to the disease. Resistance of the body to a disease occurs due to various immunity mechanisms.

Write down the two types of immunity found in our body.

Your answer should have included the following two types of immunity:

- Natural acquired immunity (passive and active)
- Artificially acquired immunity (passive and active)

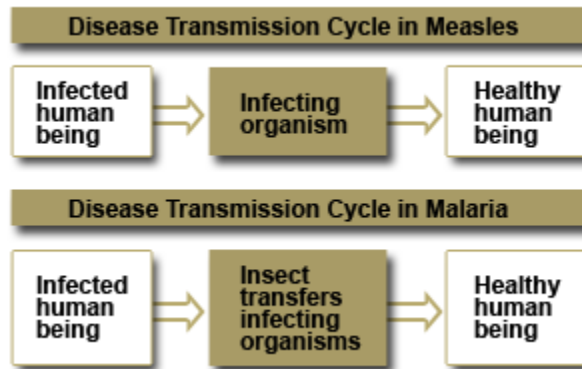
For more information on immunity, read module one, unit three part one, section one, on the organisation of the human body, dysfunctions and management.

The Disease Transmission Cycle

A disease transmission cycle is a series of steps that a disease-causing organism undergoes in its disease-causing process.

Disease-causing organisms are living things that need somewhere to live and reproduce. This may be within inanimate or animate environment (example in rodents, insect, or the human body), which is known as the reservoir of infection. The human being is the main reservoir of most of the communicable diseases that affect humanity. When an infection spreads to a new host, the place, animal or human from which the organism came from is called the source of the infection. The way in which an organism leaves the source (the infected host) and passes to a new susceptible host is called the route of transmission. Each disease-causing organism has particular routes which play a large part in how these organisms spread in the community. For example, some organisms are spread through water and food, while others are spread by vectors like mosquitoes and snails.

The diagram above illustrates the differences between the disease transmission cycle in measles, and in malaria where the causative organism passes from human being to mosquito and back to the human being.



Every transmission cycle is made up of three parts.

The Source

This is where the disease-causing organisms spreads from. It could be an infected person, animal, place, or object. The reservoir is the source of infection.

Transmission Route

The main routes of transmission are:

- Direct contact, for example sexual contact, contact with skin or mucous membranes
- Vectors
- Faecal-oral (ingesting contaminated food and water)
- Airborne

- Transplacental (mother to foetus)
- Blood contact (transfusion, surgery, injection)
- Contact with animals or their products

Susceptible Host

An individual who has low resistance to a particular disease is said to be a susceptible host for that disease. There are a number of factors which lower the body's resistance to a disease:

- Not having come in contact with the disease-causing organisms before and therefore not having any immunity to it. For example, passive immunity against measles is lost at the age of 6 - 12 months. Therefore if a child comes into contact with the measles virus after this age, they may develop the disease.
- Having a serious illness like AIDS which suppresses a person's immunity. People with AIDS have a high risk of developing tuberculosis.
- Malnutrition
- Certain drugs such as those used to treat cancer can lower a person's resistance to disease.

SECTION 2: PRINCIPLES OF COMMUNICABLE DISEASE CONTROL

Introduction

Welcome to section two. In this section you will look at the main methods (also known as principles) that are used to control the occurrence and spread of communicable diseases. It is a short but intense section which will give you the foundation you need to prevent and control the diseases you shall cover in subsequent sections

Objectives

By the end of this section you will be able to:

- Explain the principles of communicable disease control
- Describe the methods used to prevent communicable diseases
- Explain the role of the community members in prevention of communicable diseases

Methods of Communicable Disease Control

Communicable disease can be controlled and eradicated from the community. When thinking about the control of diseases it is always good to think of all the possible methods. In most cases one or two specific methods will have the greatest effect and should be the focus of your activity, in other cases some methods will be useless against the disease. The aim of control is to tip the balance against the agent.

The control and eradication of communicable diseases can be done by:

- Attacking the source of the disease-causing organism
- Interrupting the transmission route
- Protecting the susceptible host

You will now look at each method in turn.

Attacking the Source

There are various specific measures which can be used to control the spread of an infectious disease.

They include:

- Treating the infected person or animal with the appropriate antibiotics that destroy the disease causing-organism.
- Treating the carriers and sub-clinical cases after carrying out screening tests among suspected individuals or groups.
- Treating specific groups of persons who are at high risk of being infected (mass treatment). This is called chemoprophylaxis.
- Isolating those persons who are infected with highly infectious diseases such as ebola, marburg fever, lassa fever; so as to prevent the spread of the organism to other healthy people.
- Treating sick animals such as cattle suffering from brucellosis, immunising animals such as cows from anthrax, and dogs from rabies; killing sick animals such as rats to control plague and dogs to prevent rabies; separating humans from animals.
- Notifying the local health authorities immediately you suspect a patient is suffering from an infectious disease. Though this does not directly affect the source, it is an essential way of keeping watch on the number of new cases and thereby monitoring the effectiveness of the control programme.

All of the methods mentioned on the previous page are methods of controlling the reservoir - where an animal is the reservoir.

In summary you can state that the measures for attacking the source are:

- Treating the infected person/s
- Treating the carrier
- Mass treatment of persons at risk
- Isolating the infected person/s
- Treating the sick animal such as cows
- Immunising animals such as dogs and cattle
- Killing the animal reservoir such as rats
- Separating humans and animals

Interrupting the Transmission Cycle

A number of methods are used to interrupt the transmission cycle. Some of the measures were covered in detail in unit three of this module.

They include the following:

- Personal hygiene
- Environmental health
- Water and sanitation
- Vector control

- Good and adequate housing
- Effective food handling and adequate nutrition

Remember: A clean environment and good personal hygiene are the most important measures in the primary prevention of diseases.

Please review each one of these measures in unit three of this module so as to complete this section successfully. In addition to the measures covered in unit three, add sterilisation of medical equipment and the use of sterile surgical equipment. These methods are useful for interrupting the transmission of diseases such as, Human Immunodeficiency Virus (HIV) infection and hepatitis-B infection.

Protecting the Host

This is the third principle of controlling the spread of communicable disease in the community. Any person who is not yet infected by a specific disease-causing organism is known as a susceptible host. This is because they are at risk of contracting the infection. All susceptible hosts must be protected from contracting the infection.

Remember: The most effective way of controlling communicable diseases is to use a combination of methods: attacking the source of the infecting organism, interrupting the route of transmission, and protecting the susceptible host.

There are various specific and general measures for protecting the host.

Specific Measures

- Immunisation using vaccines such as the KEPI vaccine
- Chemoprophylaxis using for example:
 - Proguanil (PaludrineR) to suppress malaria parasites
 - Tetracycline during cholera outbreaks
 - Cotrimoxazole during plague outbreaks

General Measures

- Use of barriers such as bed nets, gowns, gloves to prevent insect bites (especially mosquitoes)
- Use of chemicals for example insect repellents to prevent mosquito bites
- Wearing shoes to prevent penetration by hookworms from the soil
- Adequate housing to reduce overcrowding

- Improved nutrition
- Adequate ventilation
- Health education

Other Control Measures

There are other useful measures that can be taken to control the spread of communicable disease. Among these is the notification of disease. Notification requires you to keep watch (surveillance) on the number of new cases of communicable diseases in your area of work and to immediately inform the local health authority when you come across a patient suffering from an infectious disease. One of the main reasons for notification is to help the health authorities take measures to confirm your suspicion and to control the spread of the disease. Notification of infectious communicable diseases is the responsibility of all health care workers. It is also a legal requirement according to the Public Health Act, Chapter (cap) 242, section eight of the laws of Kenya.

Remember: It is your responsibility to notify your local health authority immediately should you suspect the presence of an infectious disease.

List any six notifiable diseases found in Kenya.

Notifiable Diseases in Kenya

- **Plague**

- **Cholera**
- Measles
- Poliomyelitis
- Diphtheria
- Tuberculosis
- Anthrax
- Trypanosomiasis
- Typhoid fever
- Whooping cough
- Meningococcal meningitis
- Rabies

Yellow fever

The diseases in bold spread so quickly that they need international control measures. These diseases are reported by the Ministry of Health to the World Health Organisation (WHO).

Application of Communicable Disease Control Measures

The actual application of the control methods you have just seen can be undertaken by different groups of people and institutions at various levels. These include individuals and village level, dispensary and health centre level and the district and central government (Ministry of Health) level.

Remember: A successful communicable disease control program is the one that involves members of the community.

Control Measures at Individual and Village Level

At this level, each person and indeed every member of the village is responsible for:

- Completing the immunisation
- Personal and environmental hygiene
- Food hygiene and adequate nutrition
- Using bed nets and protective wear
- Abstaining from casual sex, being faithful to one sexual partner or using condoms
- Protecting water supply and using clean water
- Digging and using pit latrines

- Controlling vectors
- Healthy habits, for example not smoking, consuming alcohol and abuse of drugs

Control Measures at Dispensary and Health Centre Level

The health care workers should support and encourage their clients and community to establish and sustain community based disease control programs. In addition, the health care workers should:

- Increase immunisation coverage
- Participate in vector and reservoir control
- Emphasise water protection and purification
- Inspect food, markets and eating places
- Encourage sanitation and refuse disposal
- Promote health and prevent diseases using Information, Education and Communication (IEC)
- Notify diseases

Control Measures at District, Regional and National Level

At these higher levels, health care workers are responsible for:

- Vector control schemes
- Mass immunisation campaigns
- Mass treatment and chemoprophylaxis
- Mass media IEC programmes
- Health statistics registration
- Research on disease control methods
- Emergency, epidemiology and control teams
- Manpower training and continuing education for staff

SECTION 3: CONTACT DISEASES

Introduction

Welcome to the third section of this unit. From this section onwards, you will group communicable diseases according to their mode of transmission and then investigate their individual causes, mode of transmission,

diagnosis, treatment and preventive measures. You will start with contact diseases (also known as contagious diseases). This is a group of communicable diseases that are transmitted through direct or indirect contact between susceptible and infected persons.

Objectives

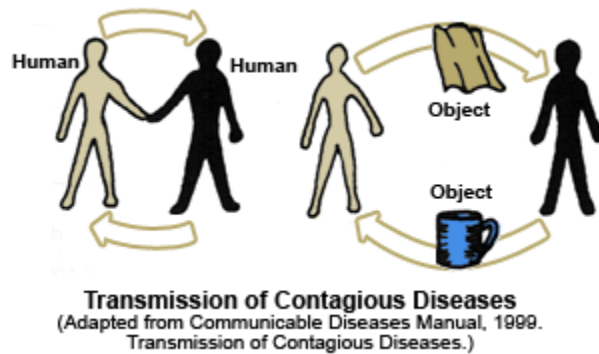
By the end of this section you will be able to:

- List factors that favour the transmission of contact (contagious) diseases
- Identify signs and symptoms of infections, namely; scabies, fungal skin infections and trachoma
- Describe the management of contagious diseases
- Describe the control measures of contagious diseases

Transmission of Contact Diseases

A large number of patients seen in your health facility, particularly school children, are suffering from contact diseases which are easily preventable. Contact diseases tend to occur in clusters within households, children's play groups, schools and workplaces. They are passed from one person to another either directly by skin-to-skin contact or indirectly by handling contaminated objects such as clothing,

bedding or combs. Such groups of infected people are known as clusters.



List four factors that increase the transmission of contact diseases.

Factors Increasing the Transmission of Contact Diseases.

- Close personal contact (for example: sexual intercourse)
- Inadequate housing leading to overcrowding
- Poor personal hygiene usually due to inadequate water supply
- High population density as in urban (slums) areas

Scabies

This is a parasitic infection of the superficial layer of the skin characterised by severe itching. It is caused by the female of an insect called *Sarcoptes Scabiei* (itch mite). The female mite burrows into the skin and makes a small tunnel. Within the tunnel, the insect deposits its eggs and faeces. The eggs hatch in four to five days and the larvae leave the mother's tunnel and bury themselves in the skin and in other places. The larvae do not make tunnels.

Mode of Transmission

Scabies is spread through direct close body contact, as in bed, or through contact between parents and children or among children playing together in schools. Transmission of scabies can also occur indirectly through clothing or bedclothes. Poor living conditions and poor hygiene promotes the spread of scabies.

Clinical Picture

The patient presents with intense itching, especially at night, and eczema-like signs. You will also find an itchy rash with typical distribution especially where the skin is curved (between fingers, elbows, buttocks, etc). Because it is very itchy, you might also find that the skin is torn

with scratches and thus secondary infection often follows.

There are a number of reasons why people with scabies do not seek early medical attention. The skin lesions may be so common that they are not considered to be a disease. Also people who suffer from leprosy or other diseases which interfere with normal sensation may not feel the itching caused by scabies.

Remember: Severe itching accompanied by typical distribution especially at the folds is suggestive of scabies

Management

The whole family should be treated together with the patient to prevent re-infection. The management of scabies is as follows:

- The patient should take a warm bath.
- Rub a handful of 10% Benzyl Benzoate Emulsion (BBE) all over the body.
- After 24 hours, the patient should bathe again and put on clean clothes.
- BBE does not kill the eggs of *Sarcoptes Scabiei* and therefore the treatment must be repeated after four to seven days to kill those larvae which have hatched since the first treatment.
- If itching is severe treat it symptomatically with calamine lotion.

Prevention of Scabies

The best way of preventing and treating scabies is good personal hygiene. Regular firm bathing, washing of clothes and frequent use of soap will control scabies.

Remember: All these activities require regular supply of water.

Dermatomycosis

The term dermatomycosis means fungal infections of the skin and mucous membranes. Fungal skin infections are mainly a problem of personal appearance rather than illness, but it is

important to distinguish them from leprosy and syphilis. Also, fungal skin and mucous membrane infections are sometimes indicators of immunosuppression as occurs in AIDS, cancer and tuberculosis.

Mode of Transmission

Fungal infections are usually spread by direct and indirect contact. Genital infections such as vulvo-vaginitis may be spread during sexual intercourse.

Clinical Picture

A fungal infection typically produces a flat patch or shamba-like growth on the human skin. This patch may be found on the head, on dry exposed body skin, between the toes, or in moist places like the mouth or private parts. Each of these patches (depending on where they are found) looks slightly different and has a different name, but they are all fungi. Fungal infections can be divided into two groups: ringworms and candidiasis.



Ringworms

Ringworm manifestations are described in Latin after the areas of the body they commonly affect:

- Tinea capitis (ringworm of the scalp)
- Tinea corporis (ringworm of the body)
- Tinea pedis (ringworm of the foot)
- Tinea unguium (ringworm of the nails)
- Tinea versicolor or pityriasis

Characteristics of Ringworm Diseases

(Adapted from Communicable Diseases Manual, 1999, Characteristics of Fungal Skin Infections.)

Fungus	Risk Factors	Main Symptoms	Diagnosis	Treatment
Tinea Capitis	Children under 10	Brittle hair; areas of broken hair on scalp		Whitefield's ointment b.i.d. for 3 weeks
Tinea corporis	Excessive sweating; hot and humid areas	Ring-shaped lesions; central healing	Branching filaments crossing borders of cells	Keep dry and clean
Tinea pedis (athlete's foot)	AIDS	Scaling and cracking of skin between toes		Apply Whitefield's ointment daily
Tinea unguium	Nail injuries; corticosteroids	Thickening, discoloration or brittleness of nails		Surgical removal of nails plus Whitefield's ointment daily for 16 weeks
Tinea versicolor	AIDS; excessive sweating; hot humid areas	Superficial small round light coloured areas	Short filaments, round thick walled cells, budding	Personal hygiene

Candidiasis


Candidiasis which is also known as moniliasis or yeast infection manifests in the following ways:

- Oral thrush – patchy white dots which appear on the mucous membrane of the mouth

- Vulvo-vaginitis
- Balanitis
- Intertrigo

Characteristics of Candidiasis Diseases

(Adapted from Communicable Diseases Manual, 1999, Characteristics of Fungal Skin Infections.)

Fungus	Risk Factors	Main Symptoms	Diagnosis	Treatment
Oral thrush	Measles; Newborn; Vulvo-vaginitis of mother after antibiotics; AIDS	Pseudomembranes on mucous membranes		G.V paint
Vulvo-vaginitis	Pregnancy; Diabetes; AIDS; Post-antibiotics	Thick white vaginal discharge and itching		Nystatin vag. Tab, G.V. paint
Balanitis	Lack of personal hygiene (acquired by sexual intercourse)	Itching and redness		Hygiene, G.V paint
Intertrigo	Fatness; Diabetes; Humid areas (armpits, breast folds, between toes)	Intertriginous areas of redness (like eczema)	Long filaments, oval cells budding	G.V paint; (Diet in diabetes); Keep areas dry by exposure and talcum powder.

Trachoma

This is a chronic inflammation of the conjunctiva and the cornea of the eye. It is caused by an organism called Chlamydia trachomatis of the

Chlamydiae group. Other organisms of the Chlamydiae group cause non-gonococcal ophthalmia neonatorum, non-gonococcal urethritis, cervicitis and salpingitis. Trachoma is a major cause of blindness especially in those parts of East Africa where water is scarce, such as among the pastoralist communities who inhabit the drier grasslands.

Mode of Transmission

Trachoma is very common among communities living in dry areas where there is scarcity of water. Transmission of trachoma is by direct contact with the eye discharge of an infected person. Flies and fingers are important in the transmission of the disease. After infection, the disease progresses very slowly all the time destroying the cornea and the conjunctiva, eventually leading to permanent blindness in one or both eyes. The early stages of the diseases are the most infective and transmission is high among children.

Clinical Picture

Trachoma develops in four stages.

Stage 1: Early Trachoma

Initially the eyes are red and watery (as in ordinary conjunctivitis). After 30 or more days, follicles (small pinkish-grey lumps) form inside the upper eyelids. To see these you would have to turn back the lid. Usually there is a little pus in the eye, but if the pus is copious this may indicate a secondary infection by bacteria. Examination of scrapings from the conjunctiva in the laboratory show the cells with a characteristic dark object in the cytoplasm. The dark object is called an inclusion body and its presence in the cell helps to confirm the diagnosis of trachoma.

Stage 2: Pannus Formation

Normally, the cornea has no blood capillaries on it. But during this stage, many tiny blood vessels are found to be growing towards the edge of the cornea. These tiny blood vessels which grow in the cornea are called pannus. You can see the pannus by using an ordinary magnifying glass. Again, the presence of both the follicles and the pannus strongly suggests the diagnosis of trachoma.

Stage 3: Scarring of the Conjunctiva

After several years the follicles on the conjunctiva slowly begin to disappear leaving behind whitish scars on the conjunctiva. In the cornea, the small blood vessels degenerate. The vision becomes hazy and remains so for many years unless there is rupture of the cornea scars, in which case blindness occurs.

Stage 4: Entropion and Trichiasis Formation

The scars formed after the healing process, several years after the onset of the disease, are the ones that do the greatest damage. Due to this scarring, the scar tissue retracts (shortens), thereby causing the eyelids to become thick and to turn inwards. This is called entropion. As the thick, rough eyelids turn inwards, the eyelashes point inwards and rub against the cornea. This is called trichiasis. trichiasis adds to the damage already done to the eye and results in blindness

Remember: The combination of entropion and trichiasis completely destroy the cornea leading to blindness

Management

The drug of choice for the first three stages is 3% tetracycline topical eye ointment twice a day for five days every month for six months. Stage four of the disease with entropion must be treated surgically. It is essential to do this as soon as possible because every time the patient blinks it increases corneal damage. If it is not possible to perform surgery in your health facility, you can remove the in-turned eyelashes by pulling them out with sterile forceps. This should be done before you refer the patient. While entropion operations can be carried out at the health centre level, pannus and opacity of the cornea have to be done by eye specialists.

Prevention and Control of Trachoma

Trachoma is an example of diseases that are associated with lack of water in the community (water scarcity). The most effective way of controlling and eradicating trachoma is through supply of adequate water to the community. Regular bathing and washing of children's faces with water and soap should be encouraged. Other additional measures include:

- Where water is scarce, the community should be taught how to use the 'leaky tin' (a technique where water for hand washing is put in a container that has a

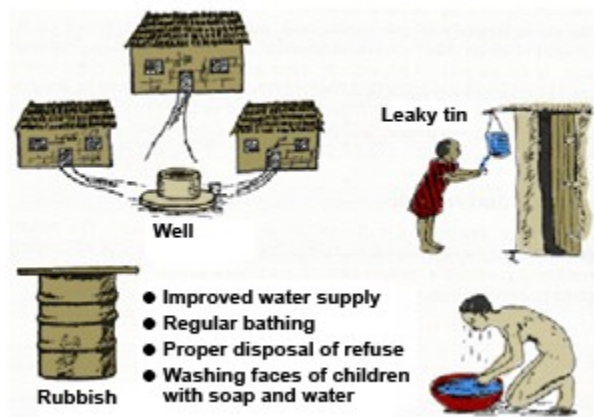
small hole near the bottom, the hole is closed with a piece of stick and when the sick is removed water trickles slowly. It minimises wastage of precious water in water scarce areas and improves personal hygiene) and be motivated to use it

- Reducing the fly population in the community through proper disposal of refuse and waste
- Early treatment of infected individuals
- Mass treatment especially of school children who live in trachoma endemic areas using 3%

tetracycline eye ointment twice a day for three

to five days each month for six months

Prevention and Control of Trachoma



SECTION 4: VECTOR-BORNE DISEASES

Introduction

In this section you will examine communicable diseases which are transmitted by invertebrate hosts, that is organisms without a backbone. This section is longer than the previous one due to the large number of important diseases which fall under this group. As usual you will look at each type of disease, its causative organisms, clinical features, management and prevention.

Objectives

By the end of this section you will be able to:

- List at least nine common vector-borne diseases
- Describe the clinical features of vector-borne diseases
- Describe the transmission cycle of vector-borne diseases
- Explain the management of vector-borne diseases
- Discuss the preventive measures of vector-borne diseases namely:
 - Malaria
 - Filariasis
 - Yellow fever
 - Trypanosomiasis
 - Schistosomiasis
 - Leishmaniasis
 - Plague
 - Relapsing fever
 - Onchocerciasis

Vector-borne Diseases

The organisms which cause vector-borne diseases usually undergo part of their development inside the vectors themselves. The time taken by the disease-causing organism to develop inside the vector is called the extrinsic incubation period. Although the housefly is an insect that is known to carry bacteria and chlamydia, it is not considered a vector. This is because it is merely a mechanical transmitter of disease; the organisms do not develop inside its body.

The majority of vectors are insects with the mosquito being the most common. This is because the mosquito is responsible for transmitting more diseases than any other vector. Insect vectors usually acquire disease organisms by sucking blood from infected persons. They then transmit the infection by depositing infected faeces or body fluids in skin cracks or abrasions. Most vectors have quite specific breeding, feeding and attacking behaviour. They therefore only thrive in areas where suitable conditions exist for their survival. As a result, vector-borne diseases tend to be present all the time (endemic) in a given geographical area or population. Many of the diseases transmitted by vectors can also become epidemic, especially when there are environmental or other changes leading to increased transmission. Some serious epidemics which have occurred in Africa have been as follows:

- Yellow fever: Ethiopia, Sudan, Nigeria, Ghana

- Trypanosomiasis: Uganda
- Kala Azar: Kenya, Sudan
- Plague: Uganda, Kenya, Tanzania
- Typhus fever: Burundi, Rwanda, Ethiopia

When communicable diseases are present in animals all the time, such as the case of yellow fever in monkeys and plague in rats, the disease is said to be enzootic (epidemic in animals).

Some Diseases and Their Vectors

Disease	Causative Organism	Vector
Malaria	Plasmodium falciparum Plasmodium ovale Plasmodium malariae Plasmodium vivax	Anopheles funestus (female mosquito) Anopheles gambiae (female mosquito)
Filariasis (elephantiasis)	Wuchereria bancrofti	Culex pipiens Culex quinquefasciatus Anopheles spp (female mosquito)
Yellow fever	Flavivirus	Aedes aegypti (mosquito)
Visceral Leishmaniasis (Kala Azar)	Leishmania donovani	Phlebotomus lutzomyia (Sandfly)
Trypanosomiasis (Sleeping sickness)	Trypanosoma gambiense Trypanosoma rhodesiense	Glossina palpalis Glossina morsitans (Tsetse fly)
Onchocerciasis (River blindness)	Onchocerca volvulus	Simulium damnosum (black fly)
Plague	Yersinia pestis	Xenopsylla cheopis (Rat flea)

Malaria

Malaria is an acute infection of the blood caused by protozoa of the genus plasmodium. For a

long time in Kenya, it was found mainly in humid low-lying areas of the coastal plains and the shores of Lake Victoria. However, these days it is also common in the highlands. Malaria is

directly or indirectly responsible for much ill-health and death, especially of children.

The vector responsible for the transmission of malaria is the anopheline mosquito (*Anopheles gambiae* and *Anopheles funestus*), which thrive in humid, warm climates where water is available. The parasites develop properly in the mosquito in places where the mean temperature is 16 - 32°C. The cooler the environmental temperature the longer it takes for the parasites to develop in the mosquito. The parasite takes about 35 - 36 days to develop at a mean daily temperature of 16°C, and nine days when the mean daily temperature is 30°C or above. Mosquitoes have an average life span of two to four months.

Remember: Treatment and prevention of malaria is one of the national public health 'high priority packages' in Kenya today.

Malaria Epidemiology

Malaria is caused by the plasmodium (parasite) that is transmitted to human beings by the bite of the infected female anopheline mosquito. There are four plasmodium species and any of them can cause malaria. They are:

- Plasmodium falciparum
- Plasmodium malariae
- Plasmodium ovale
- Plasmodium vivax

In Kenya 98% of malaria is caused by plasmodium falciparum. The other 2% of the cases are caused by plasmodium malariae and plasmodium ovale. Malaria caused by plasmodium vivax is very rare. Malaria due to plasmodium falciparum is usually the most severe form of malaria and is called malignant malaria. The mortality rate due to malaria is highest in children under five years of age. In Kenya, malaria occurs in two patterns:

Endemic Malaria

Endemic malaria (also called 'stable malaria') is transmitted all the year round. This type of malaria is found around Lake Victoria and the coastal region of Kenya. Endemic malaria causes severe infection in children under five years of age and in pregnant mothers. The mortality rate is high among infected children. After repeated bites by infected mosquitoes older children and adults develop partial immunity to malaria.

Remember: Endemic malaria is transmitted all the year and severely affects children under five years old and pregnant mothers.

Epidemic Malaria

Epidemic malaria (also called 'unstable malaria') occurs seasonally and affects people of all ages. Seasonal malaria occurs in Machakos, Embu, Kitui, Tharaka and Marigat in Baringo.

Another form of epidemic malaria occurs in the highlands and those areas bordering endemic zones. This type of malaria is called highland malaria and is seen seasonally and affects all people severely. Highland malaria epidemics have had high mortality rates. The areas in Kenya which have been affected by highland malaria include Kisii, Nyamira, Kericho, Turkana and Narok.

Remember: Epidemic malaria occurs seasonally and affects people of all ages.

Transmission and Life Cycle of Malaria

Malaria parasites develop in two cycles: The first cycle takes place in the mosquito and the other cycle in the infected human being. The first cycle which takes place in the mosquito is called the sexual cycle, while that which takes place in the human being is called the asexual cycle. You will now examine each transmission cycle starting with the asexual cycle.

Asexual Cycle

The asexual cycle of transmission takes place in the body of the infected human being and starts when the infected female anopheline mosquito bites a person.

Upon biting humans, the infected female mosquito injects sporozoites via its proboscis into the blood stream (1). The sporozoites circulate in the blood for about one hour and then they enter the liver cells (2). In 10 - 14 days the sporozoites develop into liver schizonts while still in the liver (3). The liver schizonts later burst releasing large numbers of merozoites. The merozoites leave the liver and enter the blood stream (4) where they penetrate the red blood cells.

Inside the red blood cells, the merozoites develop into trophozoites. The trophozoites then develop into erythrocytic schizonts.

These erythrocytic schizonts burst releasing a shower of merozoites, which invade fresh erythrocytes (5). Some of the released erythrocytic merozoites form male and female

The Disease Transmission Cycle of Malaria

The diagram illustrates the life cycle of the malaria parasite, divided into two main stages: the Mosquito stage and the Human stage.

Mosquito Stage:

- 8:** A mosquito is shown at the top, with a large arrow pointing down towards the human stage.
- 7:** A mosquito is shown ingesting a blood meal from a human, indicated by a large arrow pointing from the human stage to the mosquito.
- 6:** The parasite is shown as a small circle within the mosquito's body.
- 5:** The parasite is shown as a small circle within the mosquito's body, developing into a more complex form.
- 4:** The parasite is shown as a small circle within the mosquito's body, developing into a more complex form.
- 3:** The parasite is shown as a small circle within the mosquito's body, developing into a more complex form.
- 2:** The parasite is shown as a small circle within the mosquito's body, developing into a more complex form.
- 1:** The parasite is shown as a small circle within the mosquito's body, developing into a more complex form.

Human Stage:

- 1:** A human is shown at the top, with a large arrow pointing down towards the mosquito stage.
- 2:** A human is shown ingesting a blood meal from a mosquito, indicated by a large arrow pointing from the mosquito stage to the human.
- 3:** The parasite is shown as a small circle within the human's body, developing into a more complex form.
- 4:** The parasite is shown as a small circle within the human's body, developing into a more complex form.
- 5:** The parasite is shown as a small circle within the human's body, developing into a more complex form.
- 6:** The parasite is shown as a small circle within the human's body, developing into a more complex form.
- 7:** The parasite is shown as a small circle within the human's body, developing into a more complex form.
- 8:** The parasite is shown as a small circle within the human's body, developing into a more complex form.

The sexual cycle of the malaria parasite takes place in the body of the female anopheles mosquito. This cycle begins when the feeding mosquito sucks blood containing the male and female gametocytes. In the stomach of the mosquito, the male gametocytes mate with the female gametocytes. The fertilised gametocyte is called the ookinete (6). The ookinete stays in the stomach of a mosquito for about 12 - 18 hours after which it penetrates the stomach wall. Upon reaching the outer surface of the stomach wall, the ookinete changes into an oocyst (7). The oocysts grow rapidly and burst releasing large numbers of sporozoites into the body cavity of the mosquito. Many of the sporozoites move to the salivary glands of the mosquito (8) from where they are injected into the body of the next human being when the mosquito feeds.

The diagram is divided into two panels. The left panel, labeled 'Mosquito', shows the parasite's development within the mosquito's body. It starts with a mosquito at the top, followed by a large arrow pointing down to a cluster of cells. This cluster contains several numbered red circles: 8 (a small cell), 6 (a cell with a nucleus), 7 (a cell with a flagellum), and 5 (a cell with a flagellum). The right panel, labeled 'Human', shows the parasite's development within the human body. It starts with a mosquito at the top, followed by a large arrow pointing down to a cluster of cells. This cluster contains several numbered red circles: 1 (a small cell), 2 (a cell with a nucleus), 3 (a cell with a nucleus), 4 (a cell with a nucleus), and 5 (a cell with a flagellum). The diagram illustrates the complex life cycle of the parasite, involving both the mosquito and the human host.

The incubation period of malaria is about 10 - 14 days after the infection. The symptoms appear once the invaded erythrocytes rupture to release new merozoites. This stimulates the body's

- Headache and dizziness
- Joint pains
- Backache
- Fever and chills (high body temperature, rigors)
- Nausea and vomiting
- Diarrhoea
- Excessive sweating
- Jaundice
- Enlargement of spleen
- Convulsions
- Anaemia

This stage starts suddenly and lasts for fifteen minutes to one hour. The patient's body temperature rises and they shiver. During this stage, the infected erythrocytes rupture releasing merozoites in the blood circulation.

The hot stage last for two to six hours. The body temperature is high (40 - 41°C) with severe headache, nausea and vomiting. The skin is hot and dry.

The fever drops rapidly and the patient sweats profusely. This stage last for two to four hours.

Severe malaria can cause serious complications and is life threatening.

Mental disturbance appearing as acute psychosis, meningitis-like symptoms and coma.

- Clinical symptoms
- Laboratory examination of thick and thin peripheral blood films/slides (smears) which demonstrate the parasites (Trophozoites)

The treatment of malaria depends on whether the disease is complicated malaria or uncomplicated malaria (severe malaria).

Uncomplicated malaria is usually treated on an outpatient basis.

Dosage of tablets of sulfadoxine (500mg) plus pyramethamine (25mg) and paracetamol for all age groups.

Treatment of Uncomplicated Malaria

Age Group	Weight Range	Total SP Tablets as a Single Dose	Paracetamol Tablets (maximum of four doses in 24 hours)
< 3months	<5	$\frac{1}{4}$	$\frac{1}{4}$
3mo to 11 mo	5 to 9	$\frac{1}{2}$	$\frac{1}{4}$
12mo to 3yr	10 to 14	$\frac{3}{4}$	$\frac{1}{2}$
4yr to 6 yr	15 to 18	1	$\frac{1}{2}$
7 yr to 11yr	19 to 37	1 $\frac{1}{2}$	1
12 yr to 15 yr	38 to 49	2 $\frac{1}{2}$	1 $\frac{1}{2}$
16 yr and above	50 and above	3	2
Dose per Kg body weight		25mg/kg of Sulfa component	Paracetamol 15mg/kg

Patients presenting with coma, convulsions, respiratory distress, acute renal failure, jaundice, shock, hypoglycaemia, or acidosis due to

malaria should be admitted into the ward for complicated malaria.

Treatment of Complicated Malaria

Age Group	Expected Weight (Kg)	Number of tablets per dose given eight hourly	
		Quinine Sulphate 200mg	Quinine Bisulphate 300mg
< 3months	<5	$\frac{1}{4}$	$\frac{1}{4}$
3mo to 11 mo	5 to 9	$\frac{1}{2}$	$\frac{1}{2}$
12mo to 3yr	10 to 14	$\frac{3}{4}$	$\frac{3}{4}$
4yr to 5yr	15 to 18	1	1
6yr to 7yr	19 to 25	1 $\frac{1}{4}$	1 $\frac{1}{4}$
8yr to 12yr	26 to 37	1 $\frac{3}{4}$	1 $\frac{3}{4}$
13yr to 15yr	38 to 49	2 $\frac{1}{2}$	2 $\frac{1}{2}$
16 yr and above	50 and above	3	3

Intravenous quinine in dextrose is used in severe complicated Malaria where the patient presents with vomiting and coma.

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

Prevention and Control of Malaria

Chemoprophylaxis

Antimalarial chemoprophylaxis using oral proguanil (PaludrineR) may be given according to the national guidelines for diagnosis, treatment and prevention of malaria for health workers. Individuals who will benefit from chemoprophylaxis include:

- Patients with leukaemia (lowered immunity)
- Patients with sickle cell disease
- Patients with tropical splenomegally

- Non-immune visitors to malaria-endemic areas

Intermittent Preventive Treatment (IPT)

IPT is based on the assumption that the pregnant woman is infected with malaria. According to the Ministry of Health (MoH) guidelines, the drugs used for IPT are the ones that contain Sulfadoxine and Pyrimethamine (SP) such as FansidarR, MalaraxinR, FansidinR, MetakelfinR, OrodarR, and FalcidinR. The first single dose of three tablets of SP is given to the pregnant woman between 16 and 24 weeks of gestation; the second and last dose of three tablets of SP is given between 24 and 36 weeks of gestation. (MoH, 2002)

Vector Control

Actions to reduce mosquito-breeding areas include:

- Using insecticide-treated bed nets
- Using mosquito screens in houses
- Using chemical mosquito repellents
- Cleaning drainages and water disposal systems

- Clearing bushes and burying or burning rubbish heaps
- Use of larvicides and insecticides

Health Education

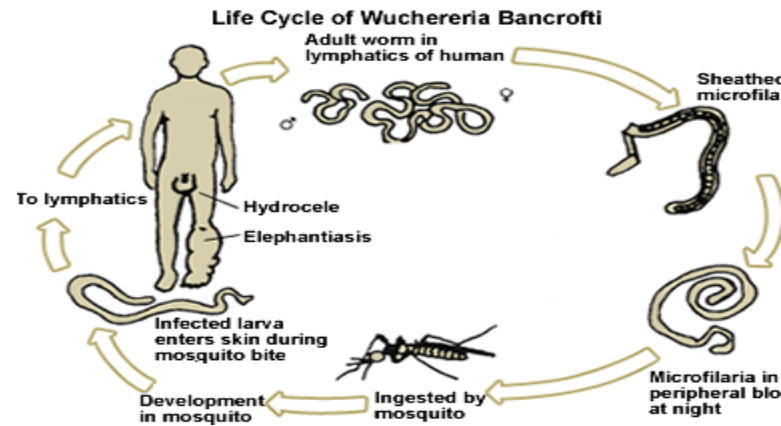
You should encourage community members to seek early diagnosis and prompt treatment for malaria and to use insecticide treated bed nets every night.

Filariasis (Elephantiasis)

This is a disfiguring disease caused by a tiny worm (nematode) called *wuchereria bancrofti*. It is mainly transmitted by mosquitoes; the *culex quinquefasciatus* found in heavily contaminated water especially in the urban areas and the *culex pipiens* and the *anopheles* mosquito in rural areas. These mosquitoes transmit the worm from person to person in the same way as malaria. The parasitic worm lives in the lymphatic system of the patient causing inflammation of the lymphatic vessels and lymph glands (lymphangitis, lymphadenitis), filarial fever, and eventually elephantiasis of the arms, legs and genitals. The disease is most frequent in the tropical coastal belts and the lake region.

Mode of Transmission

The microfilariae ingested by the feeding mosquito exsheath in the stomach and become first stage larva. They then penetrate the mosquito stomach wall and migrate to the thorax muscles where they moult twice and develop into the infective stage. Mature infective microfilariae migrate to the mouthparts of the mosquito. The extrinsic incubation period takes 10 -12 days.



Clinical Features

The presence of mature filarial worms in the lymphatic vessels triggers an inflammatory reaction in the walls of these vessels. When the worms die, more foreign proteins are released causing calcification of the lymphatic walls which eventually leads to obstruction of the flow of lymph fluid. If the obstruction of the lymph flow is extensive, chronic oedema develops in the affected areas of the body. Filariasis progresses through three stages.

Acute Phase

- Fever
- Eosinophilia
- Enlarged lymph nodes
- Inflamed lymph vessels (lymphangitis)

Sub Acute Phase

- Fever
- Eosinophilia (severe)
- Attacks of dyspnoea (asthma-like)
- Funiculitis (pain and swelling of the spermatic cord/s)
- Epididymitis
- Hydrocele
- Lymphadenitis (tender lymph nodes)

Chronic Phase

- Lymphoedema
- Elephantiasis
- Chyluria
- Hydrocele

Diagnosis

- Fluid aspirated from swollen lymph glands or from hydrocele can be examined under a microscope to show the microfilariae.
- Thick blood slides for microfilariae should be taken between 10:00pm and

2:00am. This is because microfilariae are not present in the peripheral blood during the day.

- Blood slides for microfilariae may be taken 45 minutes after administration of a provocative dose of diethylcarbamazine 100mg.

Management

The drug of choice for filariasis (adult worms and microfilariae) is diethylcarbamazine (DEC, hetrazan, benocide, notezine) 6mg/kg body weight daily in divided doses (150mg) eight hourly for 12 days for an adult.

Diethylcarbamazine may be combined with levamisole. This combination kills microfilariae and reduces the parasite worm count in the body more rapidly.

Prevention and Control

The prevention and control of filariasis includes:

- Anti-mosquito measures; the same as those used for control and prevention of malaria
- Use of larvicides such as polystyrene powder in the pit latrine
- Reduction of human-mosquito contact including the use of insecticide treated bed nets and screening of houses

Yellow Fever

Yellow fever is an acute viral disease transmitted to human being by the aedes aegypti mosquito. Yellow fever can spread rapidly, and case fatality rate may reach as high as 30% in non-immune populations. Yellow fever is a disease of forest monkeys (zoonoses) and is transmitted among them by the aedes africanus mosquito. Humans may be bitten outside the forest by mosquitoes which have acquired the disease from monkeys feeding on bananas and other agricultural plantations. In urban areas, yellow fever is transmitted by the aedes aegypti. Yellow fever is a disease of tropical African countries, especially in the rain forests.

Mode of Transmission

The mosquito becomes infected after feeding on the blood of an infected monkey or person on the third day of fever. The incubation period takes 18 days at a daily temperature of 18°C and four days at 37°C. The cycle takes four days

and once infected, the mosquito remains infected and infective for its entire life (about two to four months).

A person may also become infected with yellow fever through handling of blood from an infected individual in the first three days of the disease or handling infected monkeys in the early stages of viraemia. Laboratory staff may become infected when working on infected monkeys or infected mosquitoes.

Clinical Picture

The onset is sudden with the following signs and symptoms:

- Fever
- Headache
- Backache
- Nausea and vomiting
- A bleeding tendency (epistaxis, bleeding gums, haematemesis, malaena)
- Liver cell necrosis (in severe illness) resulting in jaundice
- Nephritis leading to albuminuria which may proceed to anuria and renal failure

Management

Yellow fever like most other viral haemorrhagic diseases has no specific drug for treatment. You only give supportive treatment and ensure that the patient is nursed in strict isolation using ordinary barrier nursing techniques. You also ensure that the patient has no further contact with the mosquitoes through the use of insecticide treated bed nets and ensuring that the room is well screened. This is to prevent further spread of the disease from the patient to other healthy people.

Prevention and Control

- Administering yellow fever vaccine to all travellers coming from or going to yellow fever endemic areas.
- Spraying aircraft coming in from yellow fever endemic areas with insecticides to kill imported mosquitoes, which may be infected.
- Isolating all persons who have been in contact with the infected persons. Such individuals should be quarantined in screened houses for seven days.
- Mass immunisation campaign for the community in areas infested with the aedes aegypti mosquito.

- Spraying of larvicides in all possible mosquito breeding places including water holding plants.

Trypanosomiasis (Sleeping Sickness)

Trypanosomiasis is a tropical disease caused by protozoa called *Trypanosoma brucei gambiense* (Tbg) and *Trypanosoma brucei rhodesiense* (Tbr). The important reservoir of Tbr in the wild is the bushbuck. Trypanosomiasis affects both humans and cattle and is invariably fatal over varying periods of time if not treated.

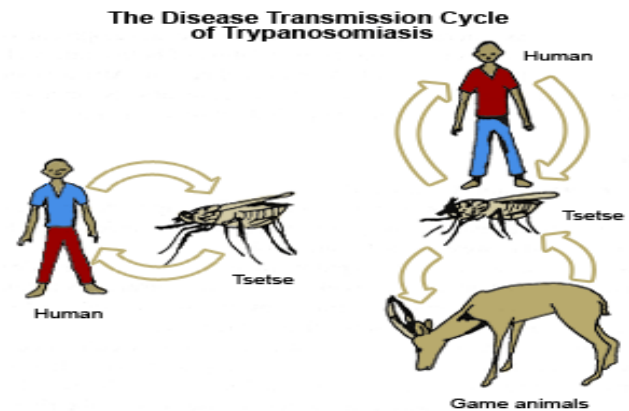
Trypanosoma brucei gambiense causes an acute, rapidly progressive illness with death from cardiac complications within several weeks or months. Reservoirs include antelope and pigs. Tbr is found in eastern Africa, now mostly in south-east Uganda.

Trypanosomiasis spreads very rapidly unless the source (the very first case) is identified early, isolated and treated properly. Trypanosomiasis is found in the same areas in Africa where yellow fever is found.

Mode of Transmission

Trypanosomiasis is transmitted by tsetse flies which live in areas of wooded vegetation. Tsetse flies are usually not found in flat plains, closely cultivated areas or areas densely inhabited by people. There are two important types of tsetse flies known to transmit the disease to humans. There is *Glossina palpalis*, a riverine type which breeds along rivers and lakes, and *Glossina morsitans* the woodland type which lives away from water. *Glossina palpalis* is the main vector of Tb gambiense, while *Glossina morsitans* is the main vector for Tb rhodesiense and it prefers to bite cattle and game but will also bite humans. Of the two types of tsetse flies, *Glossina palpalis* (which transmit Tb gambiense parasite) is the main vector in East Africa.

Tsetse flies become infected with sleeping sickness parasites when they take a blood meal from infected persons or animals. After a period of time, during which the trypanosomes undergo development changes, the fly is able to transmit the infection when it bites another susceptible animal or person.



Clinical Features

There is considerable variation in the clinical picture of African trypanosomiasis (AT). Within a few days of a tsetse bite, fever develops due to the invasion of the blood stream by the trypanosomes. The incubation period between the tsetse bite and the onset of fever varies from as short as a few hours following the chancre to several weeks. The early stages of trypanosomiasis are characterised by irregular episodes of fever with headaches, malaise, weight loss, muscle and joint pains, pruritus, anaemia, skin rash, and deep hyperaesthesia (Karandel's sign).

The clinical features of trypanosomiasis depend on the infecting parasite as follows:

- *Trypanosoma brucei gambiense* (Tbg) infection causes a slow chronic sleeping sickness, resulting in death from the disease in several months or years. Pigs, dogs and antelopes are the reservoirs.
- *Trypanosoma brucei rhodesiense* (Tbr) infection is acute and rapidly progressive unless prompt treatment is administered. The parasites damage the heart causing cardiac complications and death within several weeks or months. Pigs and antelopes are the reservoirs for Tb rhodesiense.

Trypanosomiasis presents in the following three stages:

Primary Stage (chancre stage)

Within a few days of the tsetse bite, a painful indurated erythematous nodule may appear at the site of the bite. This chancre may last for one to two weeks and then resolve spontaneously. The chancre occurs in 70% of cases in Europeans but is rare in Africans.

Blood Stage (systemic illness)

During this stage, the trypanosomes spread to the blood, lymph and lymph nodes. There is fever, which does not follow any typical pattern but recurs at intervals of days or weeks. After the fever resolves, the patient develops anaemia, debilitation and general body weakness. The spleen becomes enlarged as well as the lymph nodes. The cervical lymph nodes especially of the lower back of neck become visibly enlarged in 80% of patients - this is called Winterbottom's sign. The other signs and symptoms of trypanosomiasis include:

- Pruritic rash (beginning six to eight weeks after infection)
- Hepatosplenomegaly
- Poor appetite resulting in weight loss, debility, pitting oedema of face and lower legs
- Impotence and menstrual irregularities
- Heart failure

Cerebral Stage (Sleeping sickness stage)

This is the terminal stage of trypanosomiasis. During this stage of the disease, the parasites

invade the brain leading to mental deterioration and coma. Convulsions and localised signs such as hemiplegia and facial palsy may occur. Patients are very weak, they sleep during the day but are restless at night. As the disease progresses, the patients become severely ill and die if not treated.

Diagnosis

- Microscopic examination of the chancre fluid to demonstrate the trypanosomes
- Examination of blood (buffy coat) for trypanosomes
- Wet blood smear for microscopy
- Thick blood smear for microscopy
- Serological test (card agglutination test)
- Lymph node aspiration (microscopy)

Management

Day 1	Day 2	Day 3	Week 2	Week 3	Week 4
0.5ml	1.0ml	1.0ml	2.5ml*	3.5ml*	5.0ml*

important: The drugs used for the treatment of trypanosomes are highly toxic.

Common Side Effects of Trypanosomiasis Drugs

Drug	Toxicity
Suramin	Mild proteinuria, arthralgia, severe dermatitis, diarrhoea, nephritis
Pentamidine	Hypoglycaemia, nephritis, diabetes mellitus, injection abscess, collapse if injected intravenously
Melarsoprol	Jarisch-herxheimer reaction, arsenical encephalopathy, mortality up to 100%
Difluoromethyl ornithine	Diarrhoea, abdominal pain
Nitrofurazone	Haemolysis, neuropathy

Remember: The drugs used for the treatment of trypanosomes are highly toxic. As such the patient should be monitored carefully and the drugs administered very carefully.

Prevention and Control

The following measures are effective in the prevention and control of sleeping sickness.

- Chemoprophylaxis; IM pentamidine 250mg single dose protects against Tb gambiense infection for six months in those working in endemic bush land

areas such as wildlife personnel.

- Bush clearing (which may harm the environment) and establishment of agricultural settlement will in the long run destroy tsetse fly breeding areas.
- Use of baited flytraps which have an efficacy of 95% at reducing the tsetse fly population.

Schistosomiasis

This disease is commonly known as Bilharzia after Theodor Bilharz who discovered it in Cairo in 1861. The incidence of schistosomiasis is related to water use. Irrigation schemes or water projects for electricity provide the habitat for the

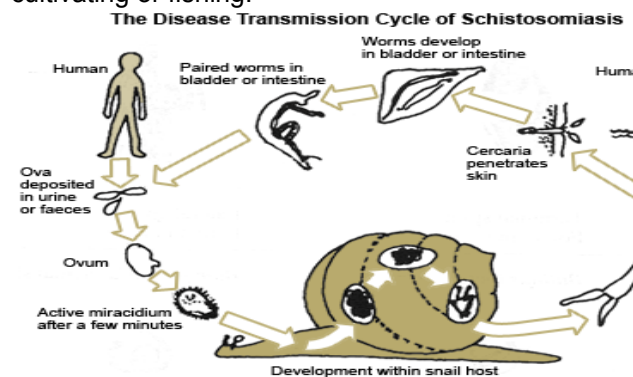
snail vectors. Up to 75% of schistosomiasis is transmitted by infected humans while 25% is said to be transmitted by dogs, cows, rats, and baboons. In East Africa, there are two types of schistosomiasis, both of which are named after the causative parasite. They are schistosoma mansoni and schistosoma haematobium.

Mode of Transmission

S. mansoni is spread in infected stool while *S. haematobium* is spread in infected urine. When the schistosome eggs in the urine enter a body of water such as a lake, dam, rice paddy or pond, they hatch into free-swimming larvae called miracidia. The intermediate host for *S. mansoni* is a vector snail of the genus *biomphalaria pfeifferi*; while for the *S. haematobium* it is *bulinus africanus*. The miracidia, after being shed from the ovum, must enter the appropriate snail host within 24 hours or die.

Transmission Cycle of Schistosomiasis

Inside the body of the snail host, it takes the miracidia four to seven days to develop and multiply into numerous infective cercariae. The snail sheds them in water where they can only live for 48 hours unless they infect a human. A human being becomes infected when they enter cercariae-infested water, such as when bathing, swimming, laundering, cultivating or fishing.



The cercariae penetrate the skin and enter the bloodstream from where they are carried to the

liver or bladder to develop into adult worms. Within four to six weeks, paired adults reach mesenteric and pelvic veins.

Clinical Features

Schistosomiasis as a disease develops in four stages, each of which is characterised by specific signs and symptoms.

Effects of Late Stage Schistosomiasis

Urinary Bladder:	<ul style="list-style-type: none"> • Obstruction to and dilation of ureters leading to hydronephrosis which may cause kidney failure • Pyelonephritis • Bladder polyps • Calcification of bladder • Cancer of bladder
Liver:	<ul style="list-style-type: none"> • Portal vein fibrosis leading to portal hypertension • Portal hypertension leading to oesophageal varices which may cause massive haematemesis • Caput medusae and ascites • Hepatomegally
Lungs:	<ul style="list-style-type: none"> • Pulmonary fibrosis leading to pulmonary hypertension, causing congestive heart failure
Bowel:	<ul style="list-style-type: none"> • Bowel fibrosis and glaucomas • Gastric varices • Haemorrhoids

Diagnosis

The diagnosis of schistosomiasis is confirmed by finding eggs in stools or urine during a microscopic examination. If this test is found to be negative, a colonic or urinary bladder biopsy can be done. Serological tests are also highly sensitive and yield specific results.

Management

The main aim of treatment is to kill the adult worms and to stop their egg-laying activity.

Drugs Used in the Oral Treatment of Schistosomiasis

Type of drug	Dose	Contraindications	Side effects	Remarks
Praziquantel (Biltricide, Distocide, Cysticide, Cesol, Cestox, Coestocide)	S. mansoni: 30mg/kg twice in one day S. haematobium 40mg/kg once (single dose)	None (drug of choice if liver diseased)	Mild giddiness	Expensive but equally effective for both types
Oxaminoquine (Vansil)	S. mansoni 15mg/kg 12 hourly (twice daily) for 2 days	Epilepsy	Occasional drowsiness, dizziness Or psychosis	Not effective for S. haematobium
Metrophonate (Bilarcil)	S. haematobium 7.5- 10 mg/kg body wt. given in 3 doses at intervals of 14 days; that is a total dose of 22.5 - 30mg/kg.	None	None if correct dose	Not effective for S. mansoni Cheap

Prevention and Control

The prevention of schistosomiasis can be achieved through the following measures:

- Prevention of ova-containing urine and stool from reaching the water by:
 - Digging and using pit latrines
 - Safe water supply
 - Treating the infected persons
- Attacking the intermediate host (the snail) using molluscicides such as copper sulphate which kills snails and their eggs.
- Avoiding contact with infested water by using protective clothing when laundering, cultivating, swimming and wading. Bathing should be done at home (storing water at home for three days will kill the cercariae).
- Conducting mass treatment campaigns for communities at risk using oral praziquantel, especially school-going children.

Leishmaniasis (Kala Azar)

This is an infection caused by a parasite of the leishmania group. The disease is also known as Kala Azar. There are three forms of leishmaniasis which are caused by different parasites.

The vector of leishmaniasis is the female sandfly (phlebotomus). The four types of sand flies are:

- *Phlebotomus martini*
- *Phlebotomus orientalis*
- *Phlebotomus longipes*
- *Phlebotomus pedifer*

In Kenya, the main vectors are *phlebotomus martini* which transmit the parasite *leishmania donovani*, responsible for visceral leishmaniasis. The species *P. orientalis* is common in Sudan while *P. longipes* and *P. pedifer* are commonly found in Ethiopian and Kenyan highlands. Together they transmit the parasite *leishmania aethiopica* which is responsible for cutaneous leishmaniasis.

Type of Leishmaniasis	Causitive Parasites
Visceral	<i>Leishmania donovani</i> <i>Leishmania infantum</i>
Cutaneous	<i>Leishmania tropica</i> <i>Leishmania aethiopica</i> <i>Leishmania mexicana</i>
Mucocutaneous	<i>Leishmania braziliensis</i>

Mode of Transmission

The zoonotic hosts of leishmaniasis are mainly dogs and rodents, although in some parts of Kenya humans have become the reservoir as well as host. The parasites of leishmaniasis are transmitted when the sandfly bites an infected person and ingests amastigotes. On reaching the sandfly's stomach, the amastigotes change into promastigotes. After four to seven days, they migrate to the foregut where they develop into infective promastigotes. The infective promastigotes are then conveyed in the saliva of the sandfly during feeding.

During feeding, the sandfly tears the host's tissue to feed on blood and at the same time deposits infective promastigotes at that site. From here the promastigotes enter the bloodstream and into the macrophages. On entering the macrophages, the parasites escape detection by the body's defences and are spread to various body tissues.

Visceral Leishmaniasis

Visceral leishmaniasis is found in many areas of the North Eastern region of Kenya in Machakos, Kitui, Masinga, Tseikuru (Mwingi), Makueni, Kibwezi, and Wajir.

Clinical Features of Visceral Leishmaniasis

Visceral leishmaniasis is characterised by fever, splenomegaly, hepatomegally accompanied by anaemia and weight loss. Visceral leishmaniasis has a rather long incubation period of four to ten months or longer, before definitive signs and symptoms manifest. Most of the patients (96%) are killed by secondary bacterial infections of the lesions.

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Cutaneous Leishmaniasis

Cutaneous leishmaniasis is found in West Pokot, Turkana, Baringo, Laikipia and Kerio valley. It is characterised by single or several painful chronic ulcers in those parts of the body exposed to sandfly bites, such as arms, legs or face. In the lower hotter areas of Kenya such as Baringo, the vector is the *P. orientalis*, while in the highlands of Kenya, the high altitude sandflies, *P. longipes* and *P. pedifer* are the vectors. *Phlebotomus longipes* bites human beings in their houses at night transmitting the parasite *leishmania aethiopica*, which is responsible for cutaneous leishmaniasis.

Clinical Picture of Cutaneous Leishmaniasis

In about two to eight weeks following a bite from an infected sand fly, a small itchy papule appears at the site of the bite. Over several weeks, the papule grows in size expanding to form a single indolent ulcer or multiple ulcers. The disease may be mistaken for leprosy. There may be enlargement of the local lymph nodes. The lesions begin to heal spontaneously two to twelve months later. Cutaneous leishmaniasis does not spread to other body organs.



Management of Cutaneous Leishmaniasis

Small lesions may be treated surgically by curettage or by freezing, using liquid carbon dioxide or by infiltrating them with 1 - 2ml sodium stibogluconate. Large disfiguring or multiple skin lesions are treated in the same way as for visceral leishmaniasis using IV or IM sodium stibogluconate 20mg/kg daily for 20 - 30 days. The drug of choice for visceral leishmaniasis caused by *leishmania aethiopica* is IM pentamidine isothianate 3 - 4mg/kg once or twice a week.

Mucocutaneous Leishmaniasis

This form of leishmaniasis occurs primarily in the tropics of South America. The disease begins with the same sores noted in localised cutaneous leishmaniasis. Sometimes these primary lesions heal, other times they spread and become larger. Some years after the first lesion is noted (and sometimes several years after that lesion has totally healed), new lesions appear in the mouth and nose, and occasionally in the area between the genitalia and the anus (the perineum). These new lesions are particularly destructive and painful. They erode underlying tissue and cartilage, frequently eating through the septum (the cartilage which separates the two nostrils). If the lesions spread to the roof of the mouth and the larynx (the part of the wind pipe which contains the vocal cords), they may prevent speech. Other symptoms include fever, weight loss, anaemia (low red blood cell count). There is always a large danger of bacteria infecting the already open sores.

Treatment is similar to that of cutaneous leishmaniasis. Prevention or early detection and appropriate treatment are preferred. Corrective surgery can be done where need arises.

Prevention and Control

Kala Azar can be prevented through:

- Use of insecticide treated curtains in homes (these have been used with success in Baringo district)
- Destruction of infected dogs and rodents
- Early diagnosis and treatment of the infected persons
- Health education for communities on preventive measures

Plague

This is a highly infectious disease caused by bacteria called *Yersinia pestis*. Plague is a disease of rodents, especially rats

and is spread from rat to rat by a rat flea called *Xenopsylla*

cheopis. Plague is a very rare but serious disease because

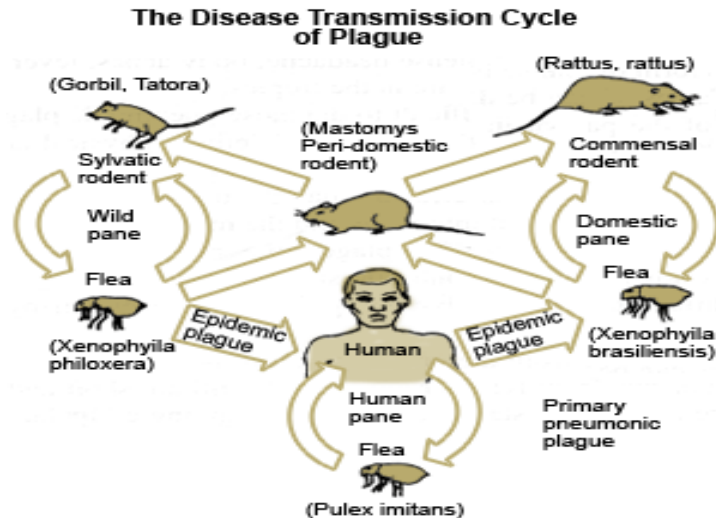
it can spread very rapidly unless the first case is recognised

early and appropriate action taken. It is also a serious disease

with a high mortality rate (case fatality rate in the absence of treatment can be as high as 60%).

Mode of Transmission

Plague occurs when infected wild rats, especially the sewer rat (*R. norvegicus*) die from the disease and their fleas look for substitute domestic rat (*Rattus italica*) hosts. The domestic rat becomes infected and after it dies the fleas start biting human beings. When the first human is infected, the disease causes bubonic plague. People working in the fields may also be bitten by fleas from the dead infected wild rats and develop bubonic plague.



Clinical Picture

Plague has three clinical presentations, bubonic, septicaemic and pneumonic.

Diagnosis

The diagnosis of plague can be confirmed by doing a microscopy (staining) of sputum or pus from the bubo to demonstrate the bacilli.

Remember: Early recognition of plague followed by correct action is a matter of life or death.

You must start treatment as soon as you confirm the diagnosis from clinical and laboratory findings. The plague bacillus (*Yersinia pestis*) is sensitive to most common antibiotics except penicillin. Drug treatment with any of the following antibiotics is effective:

- IM streptomycin 30mg/kg two to four times daily for ten days
- Oral or IV tetracycline 10mg/kg six hourly for ten days
- Oral cotrimoxazole two tabs twelve hourly for seven days
- Oral chloramphenicol 500mg six hourly for seven days

Remember: Plague is an internationally notifiable disease.

Management

Prevention and Control

The prevention and control of plague depends on the following measures:

- Early diagnosis and notification so that the patients are not moved or referred to the hospital
- Chemoprophylaxis of all contacts of the patients such as family, visitors and health care workers using tetracycline or cotrimoxazole
- Isolation of the infected and quarantine of the contacts for ten days
- Use of insecticides to kill fleas
- Eradication of rats, for example using rat poison
- Vaccination during epidemics using an anti-plague vaccine
- Health education for communities on preventive measures

Relapsing Fever

This is an acute infectious bacterial disease which is characterised by alternating febrile periods. It is also known as Recurrent fever, Spirillum, Tick fever, or Tick Bite fever. It is transmitted by ticks and lice. There are two types of relapsing fever, namely:

- Louse-borne relapsing fever
- Tick-borne relapsing fever

The louse-borne relapsing fever is spread by the human head louse, *pediculus capitis*, and the body louse, *pediculus corporis*. They transmit spirochaetes of the genus *Borrelia recurrentis*. The tick-borne relapsing fever is transmitted by soft ticks (*Ornithodoros moubata*) which live in cracks and crevices of walls and floors. They transmit spirochaetes of the genus *Borrelia duttoni*, which cause tick-borne relapsing fever. Children, visitors and pregnant women travelling to endemic areas are more susceptible to the disease. Adults in endemic areas are semi-immune to relapsing fever.

Mode of Transmission

The disease is transmitted from person to person by the bite of the head louse, body louse or soft tick.

Louse-borne

The human louse transmits louse-borne relapsing fever from person to person. When the louse feeds on the blood of an infected person, it takes up the bacteria. The bacteria multiply within the body of the louse (but these spirochaetes are not found in the saliva or the excreta of the louse). The infection is transmitted to another person only when the louse is crushed on the body surface near a bite wound. The offspring of an infected louse does not carry the spirochaetes. Epidemics of louse-borne relapsing fever are associated with times of war and famine when refugees are crowded together in unsanitary conditions, which promote infestation with human body lice.

Tick-borne

Tick-borne relapsing fever is transmitted when a tick sucks blood from an infected person. The spirochaetes are taken up and multiply in the tick's body. In seven days, the spirochaetes appear in the tick's salivary glands and the coxal fluid ready to be transmitted to a new host. The organisms can either be injected directly when the tick feeds on the host, or they can infect a new host by penetrating intact mucous membranes (for example in laboratory infections). Unlike in louse-borne fever where the offspring does not carry the organism, in tick-borne fever the *Borrelia duttoni* organisms pass into the ovary of the tick, thus automatically infecting the offspring of the ticks (vertical transmission). In this way, a house once inhabited by infected ticks will remain dangerous for up to ten years. In an infected pregnant woman, the spirochaete can cross the placenta to the foetus resulting either in abortion, stillbirth, premature delivery, or congenital infection in the newborn.

Clinical Features

The patient presents with sudden onset of fever which ranges between 39.5°C - 40.5°C. There is rapid pulse, headache, aching joints, vomiting and infected conjunctiva. Often there is potential rash, epistaxis, and herpes labialis. After five to seven days, the temperature drops by crisis. In about 60% of the patients, a less severe relapse of the symptoms occurs five to ten days after the first attack. A second relapse may occur in about 25% of the patients. In untreated cases, there may be up to ten relapses. The fever and clinical symptoms become less severe each time after the relapse. Relapsing fever has a high mortality rate of 40%. Common complications of relapsing fever include meningitis, iritis, optic nerve atrophy (blindness), myocarditis and liver failure bleeding.

Diagnosis

You can confirm relapsing fever by doing a microscopic examination of a thick blood smear for the spirochaetes.

Management

Treatment should eradicate the spirochaete from the body without eliciting Jarisch-Herxheimer reaction. Some deaths occur after starting treatment as a result of a severe Jarisch-Herxheimer reaction. The antibiotics suddenly kill a large number of spirochaetes which release toxins into the circulation causing

the patient to collapse. This reaction is characterised by chills, rapid breathing, elevated temperature (40 – 42°C), confusion, delirium, and sometimes convulsions and coma. The patient then develops very severe hypotension, and may go into heart failure. This complication is however not seen in tick-borne infections. Patients must be nursed flat, given adequate fluids and be confined to bed for at least 24 hours.

The treatment of relapsing fever is IM procaine penicillin 400,000 units stat, followed the next day by oral tetracycline 500mg six hourly for five to seven days. An alternative to tetracycline is oral doxycycline 200mg once (single dose).

Remember: Tetracycline should not be given to children and pregnant women because it discolours the teeth permanently and also causes premature calcification of bones.

Prevention and Control

Louse-borne

To eradicate lice you should advise the patient to do the following:

- Improve their personal hygiene
- Use insecticides to kill lice, for example malathion powder
- Boil clothes to kill lice and eggs (delousing)

Onchocerciasis

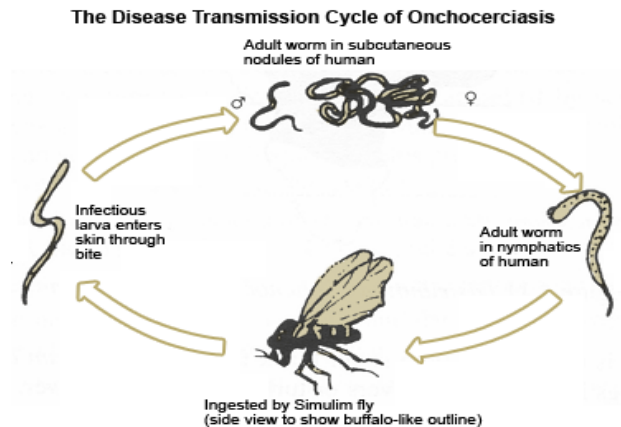
Onchocerciasis is a chronic disease caused by a filarial worm called onchocerca volvulus. It lives in the subcutaneous and connective tissue of the infected person. It manifests mainly as skin nodules on bony surfaces, and causes eye lesions which result in blindness. That is why it is also known as river blindness. The vector for *O. volvulus* is the female black fly of the genus *simulium*. In western African countries where the disease is more prevalent, the vector is *simulium damnosum*, while in East Africa the vector is *simulium neavei*. The disease is found in western Uganda, southern Sudan, and eastern Democratic Republic of the Congo (DRC). Blackflies are able to travel up to 80km in a day.

The *simulium* fly breeds in fast running well aerated rivers or turbulent areas of a river such as at the waterfalls and rapids. The eggs of the *simulium* fly are able to develop into larvae only in water that is rich in oxygen, such as fast flowing rivers. Larvae are attached to submerged plants, rocks and living crabs. The female *O. volvulus* worm is only about 0.3mm in diameter but can be as long as half a meter (50cm) long. The male is about 0.2mm in diameter and 4 - 13cm long.

Mode of Transmission

River blindness is spread from person to person by the bite of an infected blackfly. Black flies feed during the day both inside and outside houses. They usually bite early in the morning or late in the evening. The blackfly takes up microfilariae when it sucks the blood of an infected person. Once in the stomach, the microfilariae penetrate the stomach wall and travel to the thoracic muscles where they develop further for about seven days. They then move to the head of the fly ready to be transmitted to the next susceptible person when the fly feeds.

When the fly bites again, it injects the larvae of *O. volvulus* into the skin of the healthy host. The larvae mature in the human subcutaneous tissue into adult worms in about one to three years.



Clinical Features

After the adult *O. volvulus* has lived in the body of an infected person for about one year, it begins to give birth to microfilariae. One adult female worm can produce up to one million microfilariae every year. The microfilariae of *O. volvulus* have a strong liking for the skin and eyes of the infected host. Adult worms live up to 17 years in nodules in the subcutaneous and connective tissue. Most nodules are found on the bony skin surface such as the elbow, skull, ribs, iliac crests, and shoulder scapula. The disease has four different clinical presentations:

Severe Itching

This is one of the early symptoms and mainly affects the buttocks. The severe itching is often accompanied by skin depigmentation giving rise to a 'leopard skin'.

Skin Nodules

These are caused by the adult worms which you saw earlier like to live in the skin. They contain adult worms and are painless, rubbery, and firm; ranging in diameter from 3mm - 3cm.

Dermatitis

This is caused by a reaction to the presence of microfilariae in the epidermis and manifests as itchy papules and macules. Later, the skin becomes loose, scaly, atrophic and depigmented.

Blindness

This is caused by the presence of microfilariae in the cornea and the anterior chamber of the eye. It starts with oedema of conjunctiva; then corneal spots and a pannus begin to develop. Finally cataracts, iritis, sclerosing keratitis, and glaucoma develop leading to blindness. You can differentiate between trachoma and river blindness because in river blindness the pannus start at the lower limbus, while in trachoma it affects the upper limbus.

Diagnosis

The diagnosis is made by examining skin snips from the thighs, buttocks and iliac crests under a microscope for microfilariae.

Treatment

Onchocerciasis is not a fatal disease. If the patient has no serious complaints and is likely to be re-infected, there is no urgency for treatment, since the traditional drugs used have been known to

cause severe reactions. However, the following groups of patients do need treatment:

- Patient with eye lesions
- Patients with severe skin lesions
- Patients with heavy infections

Two types of treatment are used in the management of this disease. The first one is to kill the microfilariae. Give the patient oral

Ivermectine (mectizan) 150 microgram/kg single dose repeated once every six to twelve months. The second type of treatment is aimed at killing or removing the adult worms by surgical resection of the nodules.

Prevention and Control

The following measures have been found to be useful in preventing onchocerciasis:

- Addition of insecticide to the water of rivers known to be breeding places of the simulium fly
- Wearing of long clothing which covers most of the body
- Moving the whole community away from sites near where black flies breed
- Treating infected people with microfilaricides
- Mass treatment of communities using ivermectine

SECTION 5: DISEASES CAUSED BY FAECAL-ORAL CONTAMINATION

Introduction

Diseases caused by faecal-oral contamination are those whose causative organisms are excreted in the stool of an infected person and then, by various ways, enter the mouth of a susceptible person.

Water that looks clean to the eye may be dangerously polluted. Contaminated food may look, smell and taste delicious and yet harbour dangerous organisms. Food and water transmits diseases if contaminated by infected hands, soil, flies, animals, animal products or polluted water. Flies transmit diseases by vomiting on food or by carrying pathogens from faeces and transferring them to food. Indeed, most of the primary diarrhoeal diseases are caused by direct contamination of food or water by faeces, through flies and fingers.



Objectives

By the end of this section you will be able to:

- List eight common diseases spread by the faecal-oral route
- Describe the methods used to interrupt the transmission cycle of faecal-oral transmitted diseases
- Describe the clinical features, of faecal-oral route transmitted diseases
- Describe the management of faecal-oral route transmitted diseases.

Now move on to look at the diseases one by one starting with enteric fevers.

Enteric Fevers

Enteric fevers include typhoid fever and paratyphoid A and B fevers. Typhoid fever is an infectious disease characterised by high continuous fever, malaise and involvement of lymphoid tissue and spleen. Diarrhoea is not a common symptom in typhoid fever.

Paratyphoid fever may present like typhoid fever, but in most cases it presents as gastroenteritis or transient diarrhoea. Both are mainly spread by the faecal-oral route through contaminated food, water and milk. Flies are also important in the transmission of enteric fevers.

You will now consider each type of disease in turn.



Typhoid Fever

This is an infectious bacterial disease caused by salmonella typhi.

Typhoid fever is endemic in many regions of East Africa, although epidemic outbreaks have occurred when a source of water or food used by many people has been contaminated.

The disease has a case fatality rate of 3% with treatment and 10% without adequate antibiotic treatment. Human beings are the only known reservoir and host.

Clinical Features

The incubation period of typhoid fever is 7 - 21 days. The disease has a gradual onset which progresses through the following four stages.

First Week

During the first week and early in the disease, the patient has severe headache, malaise, loss of appetite, body pains and aches and a tendency to nose-bleed.

The body temperature rises day by day or in steps to 39.5°C or higher. Most patients cough because they develop bronchitis and may also complain of constipation.

Second Week

In the second week, temperature continues to rise, but the pulse rate is slower than would be expected for that temperature. There is swelling of lymphoid tissue in the intestines as well as Peyer's patches, necrosis and ulcers, which cause the abdomen to become distended and tender.

The high temperature and toxæmia causes mental confusion and disorientation in the patient. Half the patients may develop greenish watery ('pea-soup') diarrhoea and broncho-pneumonia.

Third Week

Body temperature decreases step by step and the patient improves slowly. If there is no improvement, the Peyer's patches in the intestines perforate and toxæmia increases.

The patient becomes delirious and incontinent of urine and stool, muscles twitch and coma may precede death.

Fourth Week

For the patients who do not suffer the serious complications of the third week, the fourth week is a period of convalescence.

The temperature drops back to normal and the patient recovers gradually.

Diagnosis

The best way to diagnose typhoid fever is through a blood culture. This may be positive during the first week and for a variable period after this. Stool and urine cultures can also be made although they are only positive after the first week. Other tests which are undertaken include:

- Widal test during the first and second week, that is indicative of high and rising titres
- [WBC](#) count which indicates low levels (leucopenia) with raised lymphocyte count
- Stool to check for presence of occult blood which is found in 100% of the cases

How reliable is the Widal test in typhoid diagnosis?

Although the Widal test is still very useful, especially when two tests are performed four to five days apart after the end of the first week, its interpretation is full of difficulties especially in endemic areas and in people who have had the typhoid vaccine. That is why it is a good idea to also carry out one of the other laboratory tests.

Treatment

The treatment of typhoid fever includes the following:

- Fluid replacement due to diarrhoea
- Oral norfloxacin 400mg 12 hourly for 10 - 14 days
- Oral ciprofloxacin 500mg bd. for 14 days
- Oral corticosteroids to prevent Jarisch-Herxheimer's reaction
- Patient should be isolated in fly-proof room
- Contaminated articles should be disposed by incineration
- Stools and urine should be disposed of in a pit latrine or septic tank
- Surgical treatment for perforated bowels

Note:

When treatment is started early it is not usually necessary to refer typhoid patients.

Prevention and Control

The prevention and control of typhoid fever is similar to that of many diarrhoea diseases. It includes:

- Identification of the carriers especially those who work as food handlers and treat them promptly
- Administration of typhoid vaccine
- Safe water supply
- Improvement in food hygiene

Paratyphoid Fever

This is the second type of enteric fever which was mentioned earlier. It is caused by bacteria known as salmonella paratyphi types A, B and C. The disease runs a milder course than typhoid fever and also has enlargement of the spleen, bloodstained diarrhoea and swelling of the Peyer's patches.

Treatment

The treatment of paratyphoid fever is as follows:

- Intravenous fluid if diarrhoea is severe
- Oral rehydration if diarrhoea is mild
- Oral cotrimoxazole two tablets bd. for five to seven days

Prevention and Control

The prevention and control measures are similar to those that were covered under typhoid fever.

Cholera

Cholera is an intestinal disease which is characterised by sudden onset of profuse watery stools and vomiting, leading to severe dehydration, acidosis and circulatory collapse.

Epidemiology of Cholera

It is caused by a small comma-shaped motile organism called vibrio cholerae. There are about four sub-strains of the cholera vibrio, namely, El Tor, Ogawa, Luaba and Hikojima. The El Tor sub-strain causes cholera epidemics in East Africa.

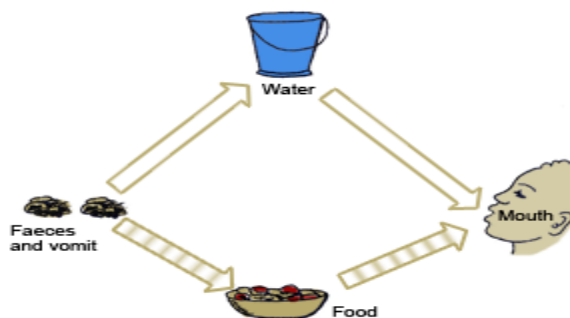
Cholera is transmitted through the faecal-oral route, mostly by water which has been contaminated with faecal matter. The vibrios are very sensitive to the hydrochloric acid found in the human stomach, and so a large number of organisms must be ingested for infection to occur.

Cholera occurs in all parts of the world where the living conditions are unsanitary.

The human being is the reservoir and host. Vibrios prefer brackish (salty) water. In seawater, the organisms can live even longer multiplying in crabs and shrimps. Vibrios also multiply in certain foods such as milk and boiled rice.

What is the role and importance of carriers?

The reservoir of infection in cholera is formed mainly by the carriers. For every clinical case of cholera there may be 50 - 100 asymptomatic carriers. Although the carriers excrete a smaller number of vibrios than the patients, they form the greatest danger to the community because of their sheer number and freedom of movement.



Clinical Features

Cholera has a short incubation period of two to three days. The vibrios remain in the digestive tract from where they cause water loss and electrolyte imbalance.

What signs and symptoms would lead you to suspect cholera?

Unlike typhoid, cholera is not a systemic infection and therefore fever is generally low or absent. Cholera progresses through the following three stages.

First Stage

This stage lasts for 3 - 12 hours. During this stage profuse watery stool is passed by the patient until faecal matter disappears. The stool becomes almost clear fluid with flakes of mucus, giving it the classical rice-water stool appearance. Vomiting follows diarrhoea. Initially the patient vomits food but soon after only clear fluid or rice-water is vomited. The patient develops severe cramps in the abdomen and limbs due to electrolyte loss.

Second Stage

The patient becomes severely dehydrated, the skin is cold, dry and inelastic. Blood pressure drops severely, and it may not be recordable. The pulse becomes weak and rapid, urine production ceases, patient collapses and may go into irreversible shock.

Third Stage

This is the stage of recovery. Some patients recover spontaneously or with treatment. The general condition rapidly improves, diarrhoea becomes less profuse and the patient is able to take oral fluids.

Diagnosis

Cholera should be suspected in any outbreak of diarrhoeal diseases. The diagnosis is made on clinical grounds and also through laboratory isolation of vibrio cholerae from a rectal swab, stool or vomitus specimen.

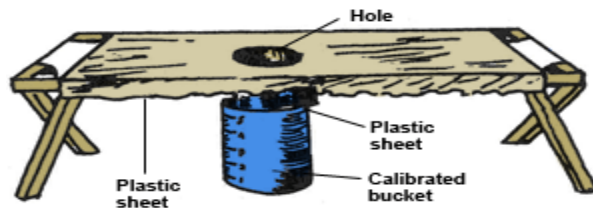
Management

The management of cholera is easily done at health centre level and so there is no need to refer patients to hospital. This is because the main cause of death in cholera patients is dehydration. This can occur very quickly and early in an outbreak before the urgency of treatment is recognised. Therefore early rehydration is the most important part of management.

Other measures taken in the management of cholera include the following:

- The immediate notification of district medical officer. Cholera is an internationally notifiable disease.
- Admission of patients in temporary treatment centres, such as school or church and the treatment of patients on 'cholera beds', that is beds with a central hole through which the stools can pass into a bucket and measured.
- Barrier nursing and patient isolation should be practiced to prevent spread of the disease.
- Disinfection of hospital equipment and proper disposal of stool and vomitus into a pit latrine or septic tanks.

Now move on to see more measures that are taken in the management of cholera.



Measures taken in the management of cholera also include the following:

- Immediate introduction of intravenous fluids to correct the severe fluid and electrolyte loss. If this is started in time it can save many cholera cases. As soon as a patient is able to drink, Oral Rehydration Solution (ORS) should be given in water at a rate of 200 - 300ml per hour.
- Intravenous fluids for patients who are in shock or too weak to drink.
- Oral tetracycline, 500mg six hourly for five days. This speeds up recovery and prevents convalescent carrier state.
- Oral cotrimoxazole, two tablets 12 hourly for three days can also be used effectively.

Remember:

Rehydration will save almost all cholera cases.

Prevention and Control

The following measures are useful in the prevention and control of cholera.

- Surveillance: early detection is central to the success of cholera control because it enables immediate action to be taken as soon as there is an outbreak of the disease. Surveillance leads to immediate notification of an outbreak.
- Provision of clean safe water to the community can easily control cholera because it is mainly a water borne disease.
- Teaching and demonstrating to members of the community cheap and effective methods of purifying water at their home.
- Foods which can transmit cholera such as milk, should be pasteurised or boiled; raw or uncooked food should be avoided or washed in safe water, foods should be protected from flies and markets inspected.

Now move on to see more measures that are useful in the prevention and control of cholera.

Measures that are useful in the prevention and control of cholera also include.

- Encouraging the digging and use of pit latrines.
- Provision of chemoprophylaxis to all contacts of the patients including family, friends and visitors using oral tetracycline.
- Administering cholera vaccine to health care workers in contact with the patients during the epidemics.
- Enrolling the assistance of formal and informal community leaders to address negative cultures and customs that contribute to the spread of cholera. Such communities should be targeted with information, education and communication messages.

Remember:

Cholera is an internationally notifiable disease.

Bacillary Dysentery (Shigellosis)

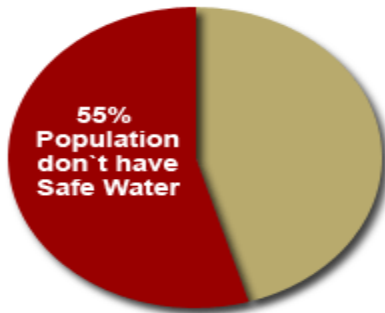
Bacillary dysentery, also known as shigellosis, is an acute bacterial disease of the intestines. It is common especially in areas where the standards of hygiene are low, particularly, where there is scarcity of safe water, improper human excreta disposal, large population of flies and child malnutrition. Once again humans are the only known reservoir.

It is caused by a non-motile gram-negative bacilli of the genus shigella spp. The organisms responsible for outbreaks are:

- *Shigella sonnei*
- *Shigella dysenteriae*
- *Shigella flexneri*
- *Shigella boydii*

However, the first three organisms are the most common causes of outbreaks.

Safe Water



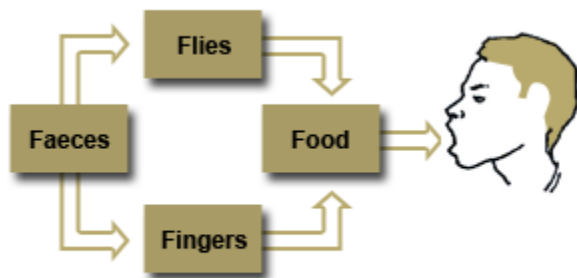
Mode of Transmission

The mode of transmission of the disease is the faecal-oral route. The organisms are transmitted directly through flies or contaminated hands. Indirect transmission may also occur through dishes which are poorly washed. The shigella multiply in food which when ingested causes dysentery.

Clinical Features

The disease has a short incubation period of one to four days. The onset is sudden with fever, headache, diarrhoea with streaks of blood, and colicky abdominal pains.

After a few motions (usually in a few hours) diarrhoea stops and is followed by severe colicky abdominal pain known as dysenteric syndrome, and painful contractions of the sphincter ani which produce an irresistible urge to defecate (tenesmus). When the patient goes to the toilet they pass small amounts of purulent mucus and blood. Vomiting may also occur. Toxins produced by the shigella on the wall of the colon may be absorbed into the blood stream resulting in toxæmia. Toxaemia causes high fever and rapid pulse. Dehydration is also common and dangerous as it may cause muscular cramps, oliguria and shock. In infants, rectal prolapse may occur as well as convulsions.



The 4 F Connection

Diagnosis

The following laboratory examinations are undertaken:

- Stool examination which shows the presence of blood and mucus
- Stool microscopy which shows presence of large numbers of white blood cells and erythrocytes
- Stool culture for shigella spp.

Management

Mild bacillary dysentery is self-limiting and all it requires is prevention or treatment of dehydration. However, in the case of severe infection, you will need to combine rehydration with antibiotics as follows:

- Antibiotics: oral ciprofloxacin 500mg 12 hourly for five to seven days
- Analgesics for colic such as codeine phosphate and loperamide, belladonna, or paracetamol
- Rehydration due to diarrhoea and fluid loss. Oral rehydration using ORS in water is always useful as an aid to parenteral rehydration. It also carries less danger of disturbing electrolyte balance. However, intravenous fluid should be given to the very ill who cannot take anything orally.

Prevention and Control

The prevention and control of bacillary dysentery depends on stopping the faecal-oral transmission through the following ways:

- Safe water supply
- Improvement in personal hygiene
- Digging and use of pit latrines
- Practising food hygiene
- Giving health education that emphasises environmental hygiene and breastfeeding
- Inspection of public eating places, markets, boarding schools and camps

Giardiasis

This is an infection of the small intestines by protozoa called giardia lamblia. The disease may be mild (asymptomatic) in some individuals, while in others it may cause diarrhoea, malabsorption of digested nutrients and weight loss.

Giardiasis is found in all the countries of the world, but it is more common in developing countries such as Kenya, where the water supply may be contaminated by human faeces or sewerage.

Mode of Transmission

Often, the disease is spread from person to person, especially within families by asymptomatic carriers. Cysts which are excreted in the stool of an infected person remain infectious for up to three months in cold water or four days if the temperature is 37°C. As soon as the cysts are ingested by a human being, they are activated by the hydrochloric acid in the stomach. Trophozoites emerge and adhere to the wall of the upper portion of the small intestine. Here they begin to multiply and in about 10 - 14 days, the symptoms manifest.

Clinical Features

Acute giardiasis is characterised by sudden onset of nausea, loss of appetite, abdominal distension (bloating sensation), prominent bowel sounds, and diarrhoea with frequent, frothy, yellowish stools with offensive odour. Fatigue, lethargy and weight loss often occur.

After about three weeks the symptoms reduce in severity and for many of the patients, this is the beginning of spontaneous recovery. Some patients however, remain symptomatic and continue to lose weight because of ongoing malabsorption of nutrients, mostly fat, vitamin B12 and lactose. The disease may persist for months or years

Diagnosis

Diagnosis of giardiasis is often difficult to establish because stool examination rarely reveals motile trophozoites. However, approximately 60% of samples will show cysts. The diagnosis is therefore made through the following ways:

- Stool microscopy to show cysts (three separate stool specimens should be collected to increase sensitivity of the test)
- Serology (giardia antigens can be detected in stools) - immunological test

Management

Any one of the following three alternative treatments is effective enough to clear the infection:

- Oral tinidazole 50mg/kg body weight single dose
- Oral metronidazole 2g single dose. Repeat the dose after ten days to increase the cure rate
- Oral metronidazole 250mg eight hourly for seven days

Prevention and Control

The cysts of giardia lamblia are not affected by chlorine treatment of water or by iodine. However, they are highly susceptible to heat, therefore, the following preventive measures are important:

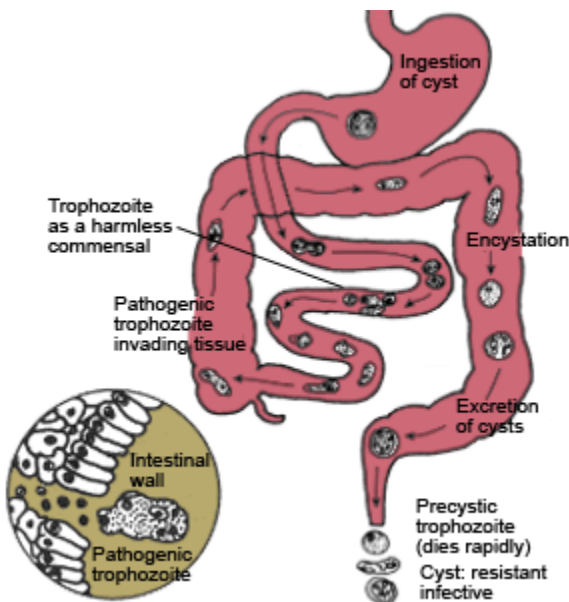
- Cooking food and boiling drinking water to kill the cysts
- Advise people to avoid eating raw salads, ice cream, unpeeled fruit and ice cubes in endemic areas
- Use of sand filters is also effective in protecting water from giardia cysts
- Tracing and treatment of healthy human carriers

Amoebiasis

This is a protozoal infection mainly of the intestinal mucous membrane in humans caused by *entamoeba histolytica*. The disease is found in all parts of the world but more common where sanitary conditions are poor. Amoebiasis can occur in families or spread through institutions but usually does not occur in epidemics. It can be endemic in a population in which many individuals are asymptomatic cyst-passers with only a few getting the disease.

Mode of Transmission

Cysts are passed from person to person by the faecal-oral route, by fingers soiled with faeces either directly into the mouth or via food. Infections may also occur from drinking contaminated water. Amoebiasis can occasionally spread from the bowels to other organs of the body, especially to the liver leading to amoebic liver disease.



Pathogenesis

Once the cysts are ingested, the emerging trophozoites take up residence in the intestinal mucosa.

The organisms multiply in the mucosa (causing the formation of bottle-shaped ulcers each 1-2cm in diameter). Too many such ulcers may cover the large intestine. Some of the ulcers may become perforated leading to severe peritonitis with shock. In the small intestines, the *entamoeba histolytica* may pass through the mucous membrane and enter the liver. After a variable incubation period a liver abscess may form.

Clinical Features

The signs and symptoms of amoebiasis include:

- Colicky abdominal pain
- Watery foul smelling diarrhoea containing blood-streaked mucus
- There may be a hard large tender abdominal mass (amoebic)

Remember:

Amoebic infection is usually asymptomatic.

Diagnosis

This is reached by doing a stool microscopy for cysts of *entamoeba histolytica*.

Management

No treatment is necessary for asymptomatic patients as in time they clear the infection. However, for invasive disease either one of the following treatments is effective:

- Oral metronidazole 800mg eight hourly for five to seven days
- Oral diloxamide furoate 500mg eight hourly for ten days

In hepatic amoebiasis, oral metronidazole is very effective.

A three day course of 1.4g - 2.4g a day will treat the disease.

Extra-intestinal Amoebic Disease

The most common site for extra-intestinal amoebiasis is the liver where it forms a liver abscess. Other secondary sites include lungs and skin leading to:

- Amoebic infection of the skin
- Amoebic balanitis
- Amoebic lung abscess
- Amoebic brain abscess

Prevention and Control

Who is responsible for the spread of amoebic dysentery?

It is the cysts-passers who are usually asymptomatic. Therefore in order to prevent and control this disease, you need to do the following:

- Advise people to boil drinking water (chlorination does not kill the cysts)
- Search for and treat carriers among food handlers
- Commence a campaign for more latrines in an area with endemic amoebiasis
- Conduct community campaigns about good personal hygiene practices, such as regular hand washing

Viral Hepatitis

This is an acute viral disease which mainly affects the liver, causing inflammation of the liver cells (hepatocellular inflammation) followed by jaundice. The disease is found in all the countries of the world.

There are five types of viruses which cause hepatitis. These are:

- Hepatitis A Virus (HAV)

- Hepatitis B Virus (HBV)
- Hepatitis C Virus (HCV)
- Hepatitis D Virus (HDV)
- Hepatitis E Virus (HEV)

Hepatitis A virus causes infectious hepatitis and is the most infectious of these viruses, while hepatitis B virus causes serum hepatitis (also called epidemic hepatitis). The hepatitis B virus causes chronic active infection of the liver (hepatitis) which may be followed some ten years later by liver cirrhosis (in 10 - 20% of the patients). In some of the patients who develop cirrhosis, the disease progresses to liver cancer (hepatocellular carcinoma). Hepatitis B infection occurs in about 1 - 3% of the human beings, but the incidence may be higher in patients undergoing kidney dialysis and in cancer wards (due to repeated blood transfusions), and among children because of close personal contact. It is difficult to tell the difference between infectious hepatitis and serum hepatitis on clinical observations alone.

Mode of Transmission

Hepatitis A and E infections are transmitted through faecal-oral route and are both called infectious hepatitis. Hepatitis B, C and D are transmitted through blood products and close personal contacts and are called serum hepatitis. Serum hepatitis may cause chronic liver infection and liver cirrhosis.

The human being is the only known reservoir and host of viral hepatitis. The disease is transmitted from the infected person to the susceptible host through faeces, contaminated food and objects, blood, blood serum and other body fluids.

In the case of hepatitis B infection, transmission takes place through two main routes:

- Percutaneous route - this is through injections and transfusion of blood and blood products.
- Non-percutaneous routes - these include close personal contact for example, kissing and sexual intercourse, from mother to foetus through placenta or to baby during delivery.

Clinical Features

All types of hepatitis infections are characterised by a similar clinical picture.

The incubation period is one to four weeks in the case of hepatitis A and 12 weeks or longer in the case of hepatitis B. Hepatitis infections manifest in two phases.

Pre-icteric Phase (no jaundice)

- Fever of sudden onset
- Malaise
- Loss of appetite
- Nausea
- Abdominal discomfort

Icteric Phase

- Appearance of jaundice
- Enlarged tender liver (hepatomegaly)
- Extreme tiredness and myalgia
- Feelings of deep sadness (depression)
- Pale stools
- Dark urine (contains bilirubin)

Those At Risk of Hepatitis

Reservoirs of hepatitis B virus include sexually promiscuous individuals, spouses of acutely infected persons, health care workers exposed to blood, family members of chronically infected persons, anyone who requires repeated blood transfusions.

Diagnosis

How would you diagnose viral hepatitis in a patient?

The urine of a person suffering from viral hepatitis is dark and contains bilirubin, while the stool is usually pale. In the blood, both direct and indirect bilirubin levels are raised. In the case of hepatitis B, diagnosis is made by detecting various immunological markers in the blood. The most important is the hepatitis B surface antigen (HBsAg) which is present when the virus is in the blood in the acute stage and in the chronic carrier state.

Management

No specific treatment is available for both hepatitis A and B.

The patient should be given symptomatic treatment together with diet and bed rest at home to prevent the spread of the disease. If admission is indicated for one reason or the other, you should ensure that the patient is isolated and extra precautions taken during handling and disposal of excreta. Since alcohol increases the risk of cirrhosis, you should advise the patient to avoid alcohol for at least six months.

Prevention and Control

Just like in the other diseases that have been covered, improvement of environmental sanitation will prevent the transmission of hepatitis A.

Other control measures include:

- Isolating patients suffering viral hepatitis
- Administration of hepatitis vaccine
- Screening blood for hepatitis B surface antigen before giving it for transfusion and excluding all donors with a history of jaundice
- Effective sterilisation and high level disinfection of all instruments, needles and syringes

Bacterial Food Poisoning

Food poisoning is a sudden, acute and sometimes life threatening illness which follows ingestion of contaminated food, drink or water. The major causes of food poisoning include intoxication with chemicals, toxins produced by bacterial growth, and a variety of organic substances such as poisonous plants and mushrooms. Food poisoning occurs in small outbreaks and mortality is usually low. In this country, you often hear of severe cases of food poisoning caused by consumption of cheap alcoholic drinks ('Kumi Kumi'). It is also suspected that some cases diagnosed as gastroenteritis in health facilities are actually caused by food poisoning.

There are two common types of bacterial food poisoning found in communities.

These are:

- Staphylococcal food poisoning
- *Clostridium botulinum* food poisoning

Staphylococcal Food Poisoning

This type of poisoning is caused by contamination of food (for example, with pus from a septic finger) by an infected person). The staphylococci in the pus multiply and produce toxins when the food is allowed to stand for several hours before being served. Although the bacteria itself is harmless if ingested, the toxins it produces are very poisonous. Following ingestion of the toxin-contaminated food, there is sudden severe abdominal cramping, nausea, vomiting, diarrhoea, headache and excessive salivation.

Diagnosis

This disease is usually recognised when people who have shared food all fall sick within a short time.

***Clostridium Botulinum* Food Poisoning**

Botulinum poisoning occurs when food contaminated with botulinum spores (from the soil) is kept warm and in tightly covered containers for many hours. The organisms multiply in warm anaerobic (low oxygen) environments especially in protein-rich foods.

When such food is contaminated, *clostridium botulinum* multiplies and starts producing toxins. The contaminated food may appear spoiled (greenish) and emit an offensive odour. Once a person eats this food, they may suffer a mild illness that requires no medical treatment or a rapidly fatal illness terminating in death within

24 hours.

The symptoms of botulism begin to manifest

12 - 36 hours after ingestion of toxin contaminated food. The patient presents with the following signs and symptoms:

- Nausea and vomiting
- Dizziness and tinnitus
- Seeing double images (diplopia)
- Inability to speak clearly (dysphasia)
- Difficulty swallowing (dysphagia)
- Difficulty breathing (dyspnoea)
- Muscle weakness (neck, limbs, respiratory)
- Death may occur from sudden respiratory paralysis and airway obstruction

Diagnosis

Diagnosis is difficult when only one person is affected because the signs of botulism are similar to those of acute polio, myasthenia gravis and Guillain-Barre syndrome. However, diagnosis can be made when a group of people who had consumed the same food (especially tinned or canned foods) suffers from the same neurological symptoms without mental confusion or loss of awareness.

Management

The main cause of death in botulism is respiratory failure.

The patient therefore must be managed in a high-dependence unit.

A tracheostomy is performed and mechanical respirator used. Cleansing enemas are administered to remove unabsorbed toxin from the colon and botulinum autotoxin serum is given and repeated after two to four hours.

Prevention and Control

What points would you emphasise when giving a health education talk on prevention of bacterial food poisoning?

Your list should include the following

preventative measures:

- Health education to encourage people to serve meals immediately they are prepared in order to prevent growth of organisms, such as staphylococci
- Keeping food covered to keep off dust and rodents
- Thorough reheating of left over foods (to kill toxins food must be heated to over 140°C)
- Excluding persons with skin infections from food handling
- Refrigerating cooked food
- Keeping the kitchen and cooling areas clean

SECTION 6: AIRBORNE DISEASES

Introduction

In this section you are going to learn about those communicable diseases whose main route of transmission is the air you breathe. That is, the organisms which cause these diseases enter the body through the respiratory tract. Most respiratory tract infections are airborne diseases. In module one unit four on paediatric nursing, you covered quite a number of respiratory tract diseases. Can you remember which ones were covered?

List the respiratory tract diseases you learnt in the unit on paediatric nursing.

In module one unit four on paediatric nursing, you covered Acute Respiratory Infections (ARI), streptococcal sore throat, Acute Laryngo-Tracheal Bronchitis (ALTB) and pneumonia.

In this section you will look at other airborne diseases, namely: influenza, measles, whooping cough, mumps, chickenpox, meningococcal meningitis, tuberculosis and leprosy.

Objectives

By the end of this section you will be able to:

- List at least eight common airborne diseases
- Describe the methods used to interrupt transmission cycles of airborne diseases
- Describe the clinical features of airborne disease
- Describe the management of airborne diseases
- Explain the preventive measures of airborne diseases namely: influenza, measles, whooping cough, mumps, chickenpox, meningococcal meningitis, tuberculosis and leprosy

Airborne Diseases

Airborne diseases have remained a major public health challenge in Eastern Africa. As mentioned in the introduction to this section, the organisms which cause these diseases enter the body through the respiratory tract.

When a patient or carrier of pathogens talks, coughs, laughs or sneezes, droplets of fluid are discharged into the air. The smallest of these droplets remain in the air for some time and may be inhaled by a new host. The bigger droplets fall to the ground and mix with the dust.

Some organisms survive the drying conditions and may be inhaled with the dust. Once they get into the body, they may affect the immediate organs involved in respiration, for example, nose and lungs, or they may pass through and spread to the blood or other distant organs like the brain or middle ear.

Overcrowded conditions such as congested houses, classrooms and public transport vehicles (matatus, buses and commuter trains), make the spread of these diseases very easy. Therefore, good ventilation

and good manners such as covering one's mouth when sneezing or coughing can go a long way to reduce transmission of these diseases.

Influenza

This is an acute viral infection of the respiratory tract caused by any one of the three strains of the influenza viruses, types A, B and C. Influenza occurs in all countries of the world. It has a high attack rate with high mortality rates, especially among the elderly and those suffering from chronic illness such as diabetes, kidney and heart disease.

Influenza viruses are also found in domestic animals (dogs, horses, pigs, ducks and chicken) and wild birds. Influenza spreads rapidly. Mortality is caused by secondary bacterial infections of the respiratory tract.

Mode of Transmission

The viruses are transmitted through secretions from the respiratory tract of an infected person. A susceptible host may be infected by:

- Direct contact with secretions from an infected person
- Inhaling droplets secreted when an infected person sneezes, coughs or talks
- Handling contaminated handkerchiefs and other articles belonging to an infected person

Clinical Features

The signs and symptoms of influenza include the following:

- Sudden onset of fever (39°C - 40°C)
- Malaise and prostration
- Sore throat
- Coughing
- Running nose (rhinorrhoea)
- Headache
- Muscle pain (myalgia)
- Nausea and vomiting
- Abdominal pain
- Diarrhoea

Complications

Some of the common complications of influenza are:

- Pneumonia
- Chronic bronchitis
- Myocarditis
- Meningitis

Management

As with many viral diseases, there is no specific treatment. You should prescribe bed rest and give paracetamol to relieve pain and fever. Prophylactic broad spectrum antibiotics may also be prescribed to prevent secondary bacterial infections.

Prevention and Control

Since the infective particles are spread by droplets from patients or carriers, an important part of the control of this disease is based on preventing droplets from being inhaled by others. That is, people must not inhale 'second-hand' air.

This can be achieved through the following measures:

- Avoiding overcrowded places especially where ventilation is poor

- Immunisation using anti-influenza vaccine about once every year
- Avoiding close contact with an infected person or handling the patient's personal articles, such as handkerchiefs
- Covering one's mouth when coughing or sneezing

Measles (Morbili, Rubeola)

This is an acute and highly contagious disease that mainly affects children. Measles is a major cause of child mortality in less developed countries such as Kenya. Together with pneumonia it accounts for about a quarter of all deaths occurring in hospitals in Eastern Africa.

Non-immunised and malnourished children under the age of three years are at high risk of contracting measles. The severity of measles is related to the viral load one gets from the source. That is, children who live in overcrowded dwellings and who are in close contact with the index case for the whole infective period obtain a high dose of the virus. Such children develop severe measles with high case fatality rates.

Mode of Transmission

The measles virus spreads through invisible droplets secreted from the respiratory tract of an infected person. Measles spreads very easily and fast. The virus infects the skin and the layer of cells that line the lungs, gastrointestinal tract, eyes, mouth and throat. In addition, the measles virus weakens the child's immune system for many weeks after the onset of the illness, leaving the child at risk of other infections.

Clinical Features

The clinical features of measles depends on the nutritional status of the affected child. The skin rash of measles is characteristic and is said to 'match' from one region of the body to another in a systematic way. It begins on the face and neck, then spreads to the chest and abdomen after 24 hours. On the third day, the rash spreads to the arms and lower limbs. Depending on the nutritional status of a child, measles can either be complicated or uncomplicated.

Uncomplicated measles generally occurs in well-nourished or slightly underweight children. It presents with the following signs and symptoms:

- Fever
- Conjunctivitis
- Rhinitis
- Coughing
- Koplik's spots
- Stomatitis
- Skin rash

All these may disappear after a few days with or without treatment.

Complicated measles occurs in malnourished children and those who are underweight. It presents with the following signs and symptoms:

- Nasal flaring
- Rapid respiration (pneumonia)
- Dyspnea
- Hoarse voice (laryngitis)
- Barking cough
- Inspiratory stridor
- Skin rash
- Loss of interest to feed
- Vomiting (this causes malnutrition)
- Diarrhoea (gastroenteritis)
- Dryness of eyes, hazy cornea (keratitis)
- Photophobia (encephalitis)

- Convulsions
- Ear discharge (otitis media)

Diagnosis

The diagnosis of measles is usually based on the following signs and symptoms:

- WHO Diagnostic criteria:
 - Rash of three or more days
 - Fever of 38°C or higher
 - Presence of 3Cs: coryza, cough and conjunctivitis
- Febrile xanthema in which there are red eyes and a cough
- Typical skin rash ('matching' skin rash)
- Koplik's spots

Management

Uncomplicated measles is usually treated on an outpatient basis. You should advise the mother to give the child adequate fluids, a light nutritious diet, and paracetamol for pain and fever.

Give a single dose of vitamin A 200,000iu in order to speed up recovery from measles and prevent the development of complications. Also, advise the mother to bring the child to the clinic everyday for follow up.

Remember:

Weigh all children suffering from measles.

In the case of complicated measles, you should admit the child to hospital and give them a balanced diet to improve their nutritional status.

List five complications of measles that you have come across.

You should watch out for the following complications and treat them accordingly.

- Convulsions: give anti-convulsants
- Gastroenteritis: give oral rehydration
- Xerophthalmia: give vitamin A 200,000 units
- Meningitis, pneumonia, conjunctivitis, otitis media: give broad spectrum antibiotics
- Fever: give antipyretics and apply fever reduction measures such as tepid sponging

Prevention and Control

The only successful method of preventing measles and its serious complications is immunisation. This should be given to all children from the age of nine months; both the healthy and the sick who have not been previously immunised.

Whooping Cough (Pertussis)

This is an acute infectious disease of the respiratory tract caused by bacteria of the genus *bordetella* called *bordetella pertussis*. Whooping cough is also known as pertussis.

The disease causes production of very sticky mucus that blocks the lumen of the bronchioles. This leads to a persistent cough in an attempt to get rid of the mucus. Usually, the cough occurs after feeding thereby causing the child to vomit. This robs the child of the little breast milk or food they may have eaten thus causing them to be malnourished. Mortality from whooping cough is highest in children aged one year or less.

Mode of Transmission

It is spread by droplets from secretions of the upper respiratory tract. The disease can also be spread by direct contact with freshly contaminated objects.

Clinical Features

The incubation period of the disease ranges from six to ten days after infection, after which the clinical features appear. In babies aged three months or less, there is no 'whoop' experienced during coughing. As such the diagnosis may be missed.

The characteristic 'whoop' is seen in children over three months of age.

Whooping cough progresses through three stages as follows.

Catarrhal Stage

This stage begins after the incubation period and lasts for one to two weeks. The patient has slight fever and a cough that is troublesome especially at night. The cough often ends with vomiting. Gradually the cough becomes paroxysmal in character with a running nose.

Paroxysmal Stage

During this stage, the fever and the running nose disappear but the cough becomes more troublesome. The cough occurs in paroxysms. The child coughs with his mouth open and tongue protruding out. This severe persistent cough causes cyanosis, protrusion of eyeballs, congestion of face and neck veins, sweating, and exhaustion. The patient may vomit suddenly, pass urine or stool, bleed from the nose, bite their tongue or suffer convulsions.

Convalescent Stage

Most patients improve gradually within one to three weeks, but some patients may continue to have paroxysms of coughing for months.

If whooping cough is not treated it can lead to a number of complications:

- Inguinal hernia
- Broncho-pneumonia
- Collapse of the lung (atelectasis)
- Convulsions
- Rectal prolapse
- Sub-conjunctival haemorrhage
- Pneumothorax
- Surgical emphysema
- Retinal detachment (which may lead to blindness)

Diagnosis

Diagnosis of whooping cough is made through a postnasal swab for culture and sensitivity of *bordetella pertussis* and clinical symptoms (paroxysmal cough with a 'whoop').

Remember:

Young babies do not 'whoop'.

Management

The management of whooping cough requires supportive treatment such as good nutrition, plenty of fluid intake and avoidance of factors which provoke coughing. Broad-spectrum antibiotics are also given to kill the pertussis organisms. However, antibiotic therapy does not shorten the paroxysmal stage of the disease. You should also avoid giving sedatives and cough suppressants because they may make the illness worse.

Prevention and Control

Just like in the case of measles, the only way to control whooping cough is by high immunisation coverage. To prevent whooping cough three doses of the pentavalent vaccine, starting at the age of six weeks is currently being administered. It is given at intervals of four weeks.

Why is the administration of the vaccine started so early?

This is because very little or no passive immunity is inherited from the mother, yet it is in the first three months of life that whooping cough has a high mortality rate.

Mumps (Epidemic Parotitis)

This is an acute viral disease, which usually affects school aged children and is characterised by fever and painful swelling of the salivary glands.

Mumps is not a major cause of death, but if contracted after puberty it can cause infertility due to its effect on the testis and ovaries. Other rare complications of mumps include; meningitis, encephalitis, pancreatitis, thyroiditis and unilateral deafness.

Mode of Transmission

The virus is transmitted by droplets and by direct contact with the saliva of an infected person, or indirectly through freshly contaminated articles.

The incubation period is 17 - 19 days.

Clinical Features

The general signs and symptoms of mumps are:

- Headache
- Sore throat
- Fever (pyrexia)
- Difficulty in swallowing
- Swelling and tenderness of salivary glands

There is no rash and the fever and swelling disappear after a few days. However, in some patients complications do develop.

Orchitis

This is a very common complication which occurs in about 20% of post-pubertal males. Orchitis is usually unilateral. The fever returns and the testis become swollen and painful. The affected testis may atrophy leading to infertility. In girls, oophoritis may develop. It is less common than orchitis. The child complains of severe lower abdominal pain and vomiting.

Pancreatitis

Pancreatitis may occur but it is not common. It presents with severe upper abdominal pain, fever and vomiting.

Meningitis

This is a common complication of mumps. It presents with fever, headache, vomiting, neck rigidity and spinal rigidity. The condition resolves spontaneously.

Encephalitis

This is rare and may occur with or without meningitis. The patient presents with disturbed behaviour, drowsiness, convulsions, and coma. Mumps encephalitis is a serious condition, and has a mortality rate of 2%.

Management

The treatment of mumps is supportive and includes:

- Analgesics/antipyretics
- Nutritious fluid diet
- Regular mouth washes
- Bed rest preferably at home
- Scrotal support for orchitis
- Corticosteroid therapy to reduce swelling and pain of orchitis (oral prednisolone 40mg od. for four days)

Prevention and Control

It can be prevented by the administration of live attenuated mumps vaccines where it is available. This live attenuated vaccine is combined with measles and rubella.

Chickenpox (Varicella)

This is a mild viral disease characterised mainly by a skin rash. It mainly affects children under ten years of age and its case fatality is very low. The causative organism is the Varicella-Zoster Virus (VZV). Chickenpox is highly contagious. An adult person who becomes infected suffers a severe form of the illness. Once a person develops chickenpox they develop immunity against the disease. However, the virus stays within the body and may reappear as herpes zoster (shingles) when the immunity of a person is weakened, for example in AIDS and diabetes.

Mode of Transmission

The virus is spread by droplets from the upper respiratory tract or from the discharges of ruptured lesions on the skin.

The incubation period is 14 - 21 days.

Clinical Features

The disease begins with mild fever, sore throat and a sore palate. After two days, a characteristic rash appears on the trunk, and within a few hours, the rash spreads to the face, axilla, and scalp, and sometimes to the arms and legs. The rash vesicles are superficial. The infection usually clears spontaneously and the vesicles usually collapse and dry after three to four days, leaving skin spots but no scars. The spots clear after a while. Complications usually do not occur.

Management

The treatment of chickenpox is symptomatic. Give the patient calamine lotion to relieve itching. A local antiseptic can also be given for infected skin lesions for example chlorhexidine (hibiscrub, hibitane).

Prevention and Control

Chickenpox is a self-limiting non-fatal disease. Healthy school children should be kept off school if there is an outbreak among schoolmates.

Meningococcal Meningitis (Epidemic Meningitis)

This is an acute and dangerous bacterial disease, which occurs sporadically and in epidemics. The causative bacterium is the neisseria meningitides, also known as meningococcus. There are two types of meningitis.

The first type known as meningococcal meningitis is spread by droplets from one person to another and may cause epidemics in crowded institutions such as army barracks, boarding schools, prisons and camps.

The second type is caused by a variety of other organisms usually occurring as a complication of some other disease in the body, or by direct extension from neighbouring structures such as the middle ear (otitis media). This type of meningitis occurs one case at a time, that is, it is sporadic.

Mode of Transmission

About 20 - 25% of people may be healthy carriers of the meningococcus and the other organisms which cause meningitis, such as, *haemophilus influenzae* type B and *streptococcus pneumoniae* (pneumococci). Transmission of the *neisseria meningitides* occurs by direct contact and by droplets from nasal and throat discharges of infected persons.

Clinical Features

When a susceptible host is infected the organism causes blood poisoning (septicaemia) and pyogenic meningitis. The onset is sudden with the following signs and symptoms:

- Severe headache and neck rigidity
- Fever and rigors
- Pain in the back and limbs
- Irritability and confusion
- Drowsiness and coma
- Positive Kernig's and Brudzink's signs
- On lumbar puncture, Cerebral Spinal Fluid (CSF) is under pressure and contains high levels of White Blood Cells (WBCs), has raised protein and lowered glucose
- Petechial haemorrhages
- Circulatory collapse (Waterhouse-Friderichson syndrome)

Diagnosis

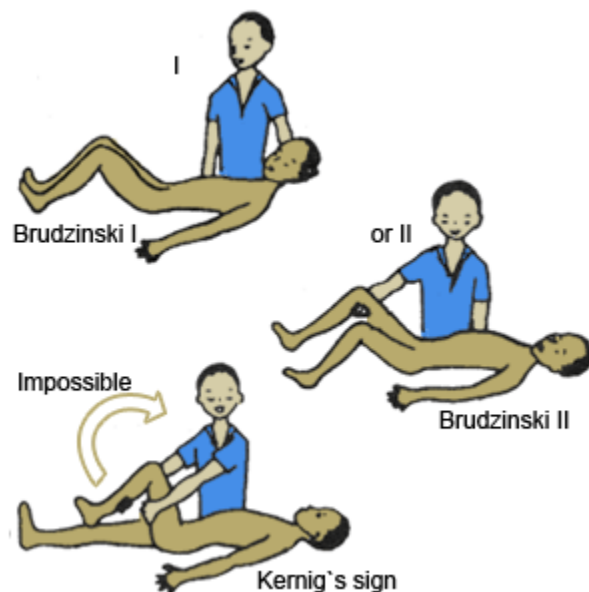
The following tests are useful to confirm a diagnosis:

- Lumbar puncture
- Positive Kernig's sign
- Positive Brudzink's 1 and 2 signs
- Blood culture for *neisseria meningitides*

Management

The patient must be admitted in the hospital and antibiotic therapy started immediately. Either one of the following drugs can be given:

- IM crystalline penicillin (benzyl penicillin) six mega units stat followed by three mega units every six hours
- IM chloramphenicol 500mg every six hours
- IM cefotaxime



Prevention and Control

The prevention of meningitis follows the same principles that were covered in the other airborne diseases. They include:

- Improvement in housing: adequate space, good ventilation
- Proper disposal of respiratory secretions
- Health education to avoid overcrowding in poorly ventilated houses
- Isolation of the suspected patients
- Notifying the District Medical Officer of Health
- Immunisation during epidemics using meningitis A and C vaccine
- Chemoprophylaxis (single dose of oral floxacillin 500mg or rifampicin) for all household and other contacts of the patient including the health care workers
- Use of gowns, gloves and masks while caring for these patients

Tuberculosis (Koch's Disease)

Tuberculosis (also known as Koch's disease) is a chronic bacterial infection caused by bacteria that belongs to the family of mycobacterium. These are *mycobacterium tuberculosis*, *mycobacterium bovis* and *mycobacterium avium*. The lungs are affected in most cases, but other organs such as the skin, bones, brain, lymph nodes, and intestine are also affected (extra-pulmonary tuberculosis).

Tuberculosis is found in all parts of the world and affects all age groups and sexes equally. If untreated, tuberculosis causes death and severe disability. After many years of immunising children against tuberculosis with the BCG vaccine, tuberculosis was almost controlled. However, the HIV infection which can lead to AIDS has led to the resurgence of tuberculosis. The cases of AIDS related tuberculosis have risen and continue to rise. The prevalence of tuberculosis increases as social and economic status decrease.

Mode of Transmission

Tuberculosis is spread from an infected person to a healthy susceptible host by droplet infection. This happens when a person with pulmonary TB coughs out heavily infected sputum into the air. Though many people may thus become exposed to TB infection, only a few will progress to develop actual disease. This is because the majority have acquired active natural immunity to the disease. However if this immunity is depressed by for example, age or HIV infection, tuberculosis may flare up again and cause obvious clinical disease.

As mentioned earlier, there are three organisms that cause tuberculosis.

Mycobacterium Tuberculosis

This is the main cause of pulmonary tuberculosis and extra-pulmonary tuberculosis.

Mycobacterium Bovis

This causes disease in cattle and is spread to humans through infected milk. It also causes extra-pulmonary tuberculosis.

Mycobacterium Avium

This causes disease in birds. Bird droppings spread pulmonary tuberculosis in individuals whose immunity is depressed (opportunistic mycobacterium).

Types of Tuberculosis

List down three types of tuberculosis?

Clinical Features

The clinical features of tuberculosis can be divided according to the early and late signs and symptoms.

Early Signs and Symptoms of Tuberculosis

- Productive cough lasting three or more weeks
- Night sweats
- Unexplained weight loss
- Loss of appetite
- Fatigue
- Evening fever (pyrexia)
- Positive tuberculin test

Late Signs and Symptoms of Tuberculosis

- Coughing blood stained sputum (haemoptysis)
- Difficulty breathing
- Enlargement of lymph nodes
- Extreme loss of weight
- Signs and symptoms of other body organs affected for example meningitis, pleurisy, pericarditis, peritonitis and pleural effusion

Remember:

TB bacteria are shed in the air in droplets whenever a patient coughs, sneezes, talks or even breathes.

How to Diagnose Tuberculosis?

The best way to diagnose tuberculosis is by means of a direct sputum smear examination (Acid Fast Bacilli - AFB test) in the laboratory.

If possible, at least three early morning specimens must be examined within two days.

Other tests include skin tests such as the Mantoux test and chest x-rays.

Management

The Kenya National Leprosy and Tuberculosis Programme (NLTP) coordinate the treatment of tuberculosis and leprosy. The NLTP, which is a Ministry of Health project, has developed the treatment guidelines for these two diseases. Treatment regimen for tuberculosis depends on the type of tuberculosis as well as the age of patient.

The drugs used for the treatment of tuberculosis are abbreviated as follows:

S-streptomycin

E-ethambutol(plain400mgtablet)

H - isoniazid (150g combined with ethambutol 400mg,tablet)

R-rifampicin(tabletorcapsule)

Z-pyrazinamide(500mgtablet)

rifater (rhz): a combination of rifampicin 120mg, isoniazid 50mg and pyrazinamide 300mg

rifinah (rh): a combination of rifampicin 150mg andisoniazid100mg

ethizide: a combination of ethambutol 400mg and isoniazid 150mg

The following gives guidelines on dosages for anti-tuberculosis drugs as set down in the National Leprosy and Tuberculosis Programme (NLTP), Diagnostic Flow Chart for Pulmonary Tuberculosis.

Dosage for Anti-Tuberculosis Drugs

(Ministry of Health (2002), National Leprosy and Tuberculosis Programme (NLTP), Diagnostic Flow Chart for Pulmonary Tuberculosis.)

The following gives guidelines on dosages for anti-tuberculosis drugs as set down in the National Leprosy and Tuberculosis Programme (NLTP), Diagnostic Flow Chart for Pulmonary Tuberculosis.

Dosage for adults

Drug	Patient's Body Weight	Dose
RHZ(rifater)	30 to 39kg	2 Tablets
	40 to 49kg	3 Tablets
	Above 50kg	4 Tablets
RH (rifinah)	Under 50kg	3 Tablets
	Over 50kg	4 Tablets
E (ethambutol)*	30 to 39kg	1 1/2 Tablets
	40 to 49kg	2 Tablets
	50 to 59kg	2 1/2 Tablets
	60 and above	3 Tablets
S (streptomycin)	30 to 49kg	¾ gm
	50 and above	1 gm
	Not over 40 year's old and under 60kg body weight	Reduce dose by ¼ gm

**Children are not treated with ethambutol because it is known to impair vision and small children would not complain if affected.*

Dosage for children

Drug	Patient's Body Weight	Dose
RHZ(rifater)	30 to 39kg	2 Tablets
	40 to 49kg	3 Tablets
	Above 50kg	4 Tablets
RH (rifinah)	Under 50kg	3 Tablets
	Over 50kg	4 Tablets
E (ethambutol)*	30 to 39kg	1 1/2 Tablets
	40 to 49kg	2 Tablets
	50 to 59kg	2 1/2 Tablets
	60 and above	3 Tablets
S (streptomycin)	30 to 49kg	¾ gm
	50 and above	1 gm
	Not over 40 year's old and under 60kg body weight	Reduce dose by ¼ gm

Drug	Patient's Body Weight	Dose
RHZ (rifinah)	5 to 10kg	½ Tablet
	10 to 20kg	1 Tablet
	20 to 30kg	2 Tablet
RH (rifinah)	4 to 10kg	½ Tablet
	10 to 20kg	1 Tablet
	20 to 30kg	2 Tablets

First Regimen

(For AFB smear positive or very sick patients.)

- **2ERHZ/6EH**
 - Intensive therapy phase > 2ERHZ
 - Continuation therapy phase >6EH

Second Regimen

(For AFB smear negative and extra-pulmonary TB, and not severely ill patients.)

- **2RHZ/6EH**
 - Intensive Treatment phase > 2RHZ
 - Continuation treatment phase >6EH

Third Regimen

(For defaulters and drug resistant cases.)

For re-treatment of resistant tuberculosis and treatment defaulters and opportunistic mycobacterium infection as in AIDS associated TB.

- **Intensive Treatment Phase**
 - IM streptomycin daily for two months, and
 - Oral rifater daily for one month, and oral ethambutol daily for two months
- **Continuation Treatment Phase I**
 - Oral rifater daily for one month, and oral ethambutol daily for one month
- **Continuation Treatment Phase II**
 - Oral rifinah daily for five months and oral ethambutol daily for five months

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

The aim of tuberculosis treatment is to kill the mycobacteria as efficiently as possible and within the shortest possible time. That is why the [WHO](#) came up with a TB treatment strategy known as DOTs (Directly Observed Treatment short course).

Remember:

For the DOTs strategy to succeed the government must be committed to the programme.

When using the [DOTS](#) strategy, you must adhere to the following rules:

- Follow the national treatment guidelines
- Ensure that there is an adequate supply of anti-TB drugs
- Ensure each patient is on the correct treatment regimen
- Administer the initial (intensive) phase of treatment under supervision
- Encourage all patients to attend the TB clinic regularly during the continuation treatment phase

- Promptly trace defaulters
- Maintain accurate records on patient personal data and clinic attendance

Common Complications of Tuberculosis

The following are some of the common complications of tuberculosis:

- Severe haemoptysis
- Respiratory failure
- Meningitis
- Kidney failure
- Pleural effusion
- Pericardial effusion
- Potts disease (collapse of the backbone)

Prevention and Control

The following measures are important in the prevention and control of tuberculosis.

- Immunising the newborn babies with BCG vaccine
- Case finding and treatment (completing treatment)
- Health education to the patients so that they can stop spitting carelessly
- Encourage them to use a sputum mug
- Health education to the community members to avoid overcrowding and to improve ventilation in their houses
- Drinking only pasteurised or boiled milk

Remember:

To eliminate TB, find the people who have infectious TB and cure them so that they do not continue to spread the infection.

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 tuberculosis, 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 the infected individual.

In some communities patients suffering from leprosy are discriminated against or stigmatised due to ignorance and unfounded traditional beliefs. This causes a lot of distress and misery to those infected and their families. In Kenya, leprosy has almost been eradicated except for a few endemic areas in the Coast, 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 five to eight years. Just like tuberculosis, 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 common among family members of the infected.

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

- Presence of many untreated cases
- Overcrowding in living houses
- Presence of susceptible new comers in a leprosy endemic area

- Hiding patient with leprosy and starting treatment late

Classification (Types) of Leprosy

Broadly speaking, there are two forms of leprosy: the tuberculoid form and the lepromatous form.

Pauci-Bacillary Leprosy (PBL), also called **tuberculoid leprosy** is characterised by:

- Absence or presence of very few of bacilli in the skin smears or skin biopsy (skin smear is negative)
- Skin patches 1 - 5cm
- Reaction type I
- Nerve involvement/damages affects one or more peripheral nerves
- Disability and deformities are common as a result of irreversible nerve damage and most are disfiguring

Multi-Bacillary Leprosy (MBL), also called **lepromatous leprosy**, is characterised by:

- Presence of numerous bacilli in most tissues of the body, except brain and spinal cord
- Skin patches six or more cm
- Skin smears 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 anaesthesia. The patient may walk on an injured foot without realising 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 (lagophthalmos) 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 the 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 hands 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 I or reversal reaction and type II or erythema nodosum leprosum.

Type I Reaction (Reversal Action)

Type I reaction (reversal action) 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 causes 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 Nodosum Leprosum)

This appears six 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 or physical stress
- Menstruation, pregnancy, abortion, puberty and childbirth
- Using drugs containing iodine
- BCG vaccination
- Osteomyelitis
- Septic wounds

Remember:

Drugs for leprosy do not cause reactions and therefore should not be stopped.

Late Deformities of Leprosy

The following are the late deformities of leprosy:

- Paralytic deformities including claw hand, claw fingers, wrist drop, foot 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 and skin scrap, nasal smears for leprosy bacilli
- Chemical tests: histamine test, lepromin test

Management

The aim of leprosy treatment is to prevent nerve damage, deformity, blindness and defaulting. The National Leprosy and Tuberculosis Programme (NLTP) in Kenya uses the WHO recommended multiple drug therapy for the treatment of the two classes of leprosy.

Pauci-Bacillary (Tuberculoid) Leprosy (PBL)

This type of leprosy is treated for six months as shown in the table below.

Six months treatment for pauci-bacillary leprosy for all ages*.

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

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

Multi-Bacillary Leprosy (MBL)

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

Six months treatment for Multi-Bacillary Leprosy for all ages*

	0-5 years	6-14 years	Over 14 years
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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)

Having looked at drug therapy you will 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 advise the patient to do the following:

- Wear protective footwear
- Wear heatproof gloves when working and handling hot objects
- Inspect the feet and legs regularly for swelling, cracks, bruises, injuries, dryness - a small mirror can be used to inspect the soles of feet
- Soak feet for 20 minutes twice daily in salty water, then rub oil on the skin to keep it moist and prevent cracks
- Remove grit from inside the shoes

Eye Care

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

- Wear sun glasses
- Check the eye daily in front of a mirror for inflammation and foreign bodies
- Cover the eyes with pads at night
- Avoid rubbing the insensitive eyes

Exercises

It is 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 paralysed muscles, passive exercises help to loosen the stiff joints and lengthen the skin. The exercises should be done for five to ten minutes daily on a regular basis.

Prevention and Control

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

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

SECTION 7: HELMINTHIC DISEASES

Introduction

In this section you will look at helminthic diseases or diseases caused by worms. There is a wide variety of worms that can, like viruses and bacteria, get into the body of a human being.

Sometimes they present without causing any symptoms, sometimes they cause disease. Some only infect mankind and have a simple life cycle, entering the body through the mouth, living in the gut and leaving in the stools. Others have more complicated life cycles, entering the body through the skin, living in different organs, and having intermediate hosts for transmission.

In this section you will consider the common intestinal worms that fall under two groups, that is nematodes and flatworms.

Objectives

By the end of this section you will be able to:

- List at least six common intestinal worms
- Describe the mode of transmission of helminths namely; threadworm, whipworm, roundworm, tapeworm and hookworm
- Describe the clinical features of helminthic infections
- Describe the management of helminthic infections
- Explain the preventive measures of helminthic infections

Helminthic Diseases

Helminthic diseases are still a very common problem in Kenya, despite the fact that it is known how to prevent and treat them. They are common in low income areas such as slum settlements due to lack of proper facilities for human waste disposal as well as poor attitudes.

The other factors which promote the spread of some helminths are:

- Moist warm soil in the case of hookworms
- Cattle keeping areas in the case of tapeworms
- Lack of latrines in the case of roundworms
- Unwashed hands in the case of threadworm

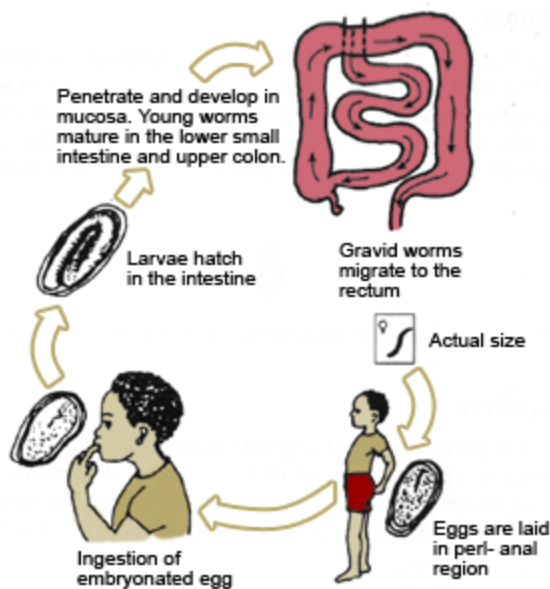
Helminthic diseases can be categorised into two groups: nematodes and flatworms.

Nematodes (Cylindrical-Shaped Worms)

This group is made up of cylinder shaped worms and includes threadworms, whipworms, and roundworms.

Threadworm or Pinworm

The threadworm is caused by *enterobius vermicularis*. It has a worldwide distribution and mainly affects school aged children, especially in boarding schools. The children reinfect themselves when they scratch their anus and then transfer the eggs on their fingers to the mouth.



Mode of Transmission

Infection with *enterobius vermicularis* is maintained by direct transfer of infective eggs from the anus to the mouth (auto infection) or indirect contact through clothing, bedding, food and other articles. After ingestion, the eggs hatch in the stomach and small intestine. The worms mature in the lower small intestine and upper colon and then they migrate to the rectum where they discharge eggs on the perianal skin, especially during the night. This causes itching and consequently scratching. The graphic illustrates the life cycle of the pinworm.

Clinical Features

List four clinical features of pinworm infestation.

Your list should include the following signs and symptoms of pinworm infestation:

- Mainly pruritus ani leading to intense scratching of the perianal region
- Disturbed sleep
- Restlessness
- Loss of appetite and weight loss

Diagnosis

Diagnosis is mainly made by a laboratory examination of stool microscopy for ova and cyst.

Management

You should treat the whole family with mebendazole 100mg given as a single dose. During treatment you should impress on the patient the importance of avoiding auto-infection.

Prevention and Control

The prevention and control of this disease lies in improved personal hygiene and proper disposal of faeces. You should give health education on the importance of bathing and hand washing, keeping nails short, and how to prevent reinfection.

Whipworm

This infestation is called trichuriasis because it is caused by an intestinal worm called *Trichuris trichiura*. The worm infects the large intestine and infestation is usually asymptomatic.

Mode of Transmission

The transmission of trichuriasis is indirect, as the eggs passed in the faeces require embryonation in soil. Therefore unlike the threadworm, auto-infection is not possible.

When the embryonated eggs are ingested, they hatch and eventually the mature worms attach themselves to the mucosa of caecum and colon. They are mainly transmitted through food that is contaminated by soil or dirty fingers.

Clinical Features

Often, mild infections are asymptomatic, but heavy infections may result in abdominal discomfort, bloody diarrhoea, loss of weight and prolapse of rectum.

Diagnosis

Diagnosis is made by examining a stool sample microscopically for ova and cyst.

Management

You can eliminate this infection by giving oral mebendazole 100mg 12 hourly for three days.

Prevention and Control

Just like the threadworm, the prevention of trichuriasis can be achieved through good personal hygiene and proper disposal of faeces.

Roundworm (Ascariasis)

This disease is caused by *Ascaris lumbricoides*, which infects the small intestine.

Ascaris is a large intestinal parasite which often infects children because of their habit of putting all kinds of things in their mouth.

It is one of the commonest and most widespread infections of the small intestine. The worms may multiply in large numbers in the intestinal lumen and cause intestinal obstruction at the ileocaecal valve.

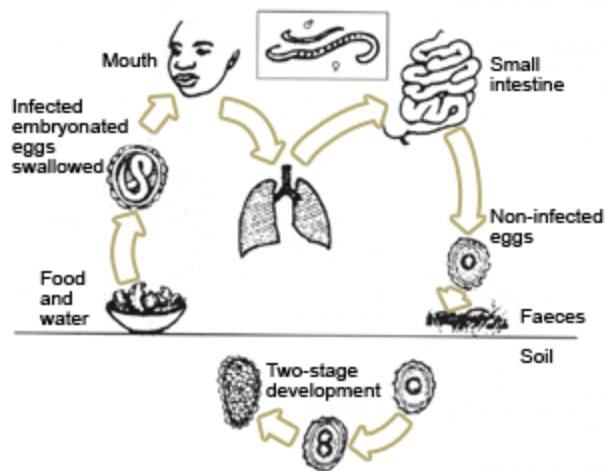
The worms also contribute to severe malnutrition and vitamin A deficiency, and may wander out of the intestinal lumen into the peritoneal cavity.

Mode of Transmission

Ascariasis is a soil transmitted parasite. Once the eggs are passed out in faeces, they require embryonation in the soil before they can become infective. This takes 8-50 days. Embryonated eggs can be carried away from the contaminated place into houses by feet, footwear or in dust by the wind. Human beings may ingest the eggs as they eat or drink using contaminated hands and utensils, or through eating raw contaminated foods like fruit. Once the eggs are ingested by a human being they hatch into worms.

In order to reach maturity, the larvae need to pass through the lungs and trachea to the pharynx. Once in the pharynx they are swallowed and return to the gastrointestinal tract where they can live for about a year.

The graphic illustrates the life cycle of *Ascaris lumbricoides*.



Clinical Features

Infection with a few ascaris is usually asymptomatic and if symptoms are present, they are not characteristic.

There may be vague abdominal discomfort or occasionally the worm may leave the body in vomitus or stool. Also during the stage of larval migration through the lungs there may be temporary symptoms of pneumonitis (cough).

Diagnosis

Diagnosis is by stool microscopy which should show ascaris ova and cyst.

Management

Any one of the following drug treatments is useful in the management of ascariasis.

- Oral mebendazole 100mg 12 hourly for three days
- Oral levamisole (3 tabs or 5mg/kg body wt) single dose
- Oral piperazine 150mg/kg body wt single dose

Note: For intestinal obstruction, surgical operation is indicated.

Prevention and Control

The prevention and control of ascariasis involves the following measures:

- Improved environmental sanitation such as proper excreta disposal, clean supply of water
- Discouraging the use of raw (fresh) human faeces for manure (Composting for six months kills the ascaris eggs)
- Washing of fruit and vegetables before eating
- Use of drying racks for utensils so that they do not come into contact with soil and dust
- Washing hands after opening bowels
- Washing hands before handling food

Hookworm (Ancylostomiasis)

This is an infection of the small intestine by a blood-sucking worm called *Ancylostoma duodenale* or *necator americanus*.

In East Africa, *necator americanus* is the cause of the disease.

The worm causes severe iron deficiency anaemia and protein loss.

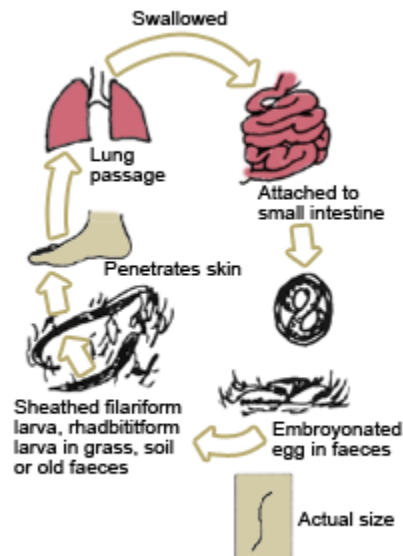
Each adult *necator americanus* worm causes a daily loss of 0.03ml of blood from the patient. In many infected individuals the disease is asymptomatic because the hookworm load is light.

Mode of Transmission

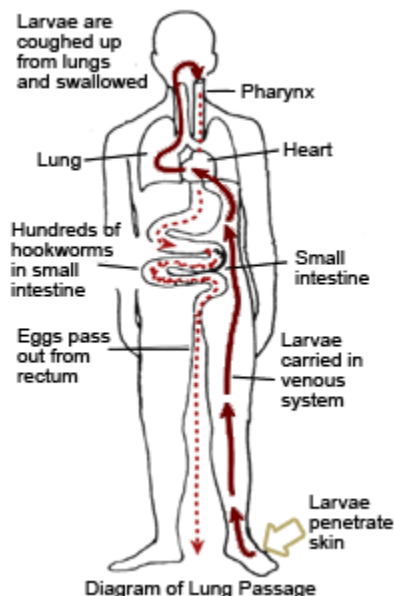
Hookworm eggs are embryonated by the time they are passed out with faeces. Indeed, when the faeces stand for a long time before examination the free larvae can be found.

When an infected person passes faeces in the soil, the larvae bury themselves in the moist damp soil.

The larvae are called rhabditiform and only become infective after five days, when they change into the sheathed filariform stage.



As soon as the filariform larvae come into contact with a human leg or foot, they penetrate actively through the skin and reach the lungs via the venous system and the right side of the heart. Once in the lungs they penetrate the alveoli and are carried to the larynx and pharynx, from here they are swallowed into the stomach. When they reach the stomach they attach themselves to the wall of the abdomen with hook-like teeth and start to suck blood from the patient's body.



Clinical Features

How would you diagnose a hookworm infection?

In most of the cases, hookworm infestation tends to be asymptomatic. However the following signs and symptoms are indicative of hookworm infestation:

- Itching of the skin at the site of entry (local irritation)
- Anaemia (due to haemorrhage), pallor
- Weakness, puffy face, malnutrition
- Flatulence, constipation

- Pain in abdomen
- Some little blood in stool

Diagnosis

Diagnosis of hookworm infestation is made by stool microscopy which should show ova and cysts and in some cases occult blood. More than 100 eggs in an ordinary faecal smear indicate heavy infection.

Management

The following drugs are commonly used in the treatment of hookworm infections:

- Levamisole 25mg/kg body weight as a single dose
- Mebendazole 100mg bd. for three days
- Albendazole 400mg stat

Flatworms

This group is made up of flat or segmented worms, their intermediate hosts are mainly animals, such as cattle, pigs and dogs.

You will look at two worm diseases under this group, namely tapeworms and hydatidosis.

Tapeworm (Taeniasis)

There are various types of tapeworms, but in human beings the infestations are commonly caused by:

- Taenia saginata or beef tapeworm (commonest infection)
- Taenia solium or pork tapeworm

You will now consider each type in turn.

Taenia Saginata or Beef Tapeworm

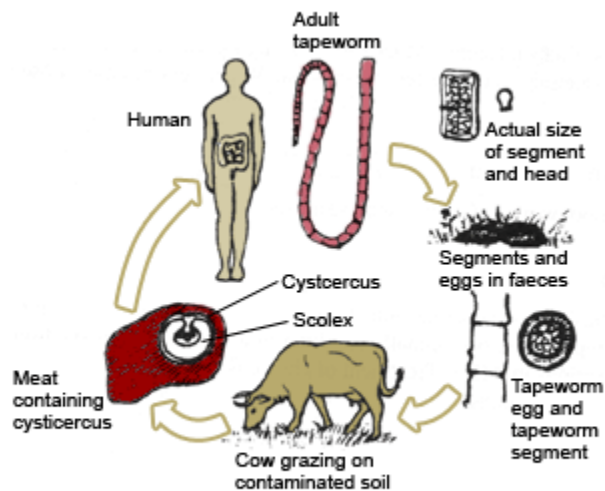
Infection with the beef tapeworm is common in areas where beef is eaten raw or lightly cooked.

Mode of Transmission

The eggs of adult tapeworms living in the small intestines of human beings are passed in the stools.

They are then ingested by cows as they feed on contaminated grass. Once in the gastrointestinal tract of the cow, the embryos hatch and penetrate the bowel wall and are carried via the bloodstream to striated muscles. Here the larvae grow and form infective cysts called cysticerci.

When human beings ingest cow meat containing these cysts, the cysts are dissolved by the gastric acid in the stomach to release embryos.



Clinical Features

Most tapeworm infections caused by *taenia saginata* do not cause any signs or symptoms. However, some people may complain of loss of weight, abdominal discomfort and itching around the anus (pruritis ani).

Diagnosis

Diagnosis of tapeworm infestation can be made by the presence in the stool of segments or eggs. The eggs are not laid singly and appear only accidentally in the stools.

Management

Drug treatment with oral niclosamide is effective. The dose is 1gm chewed and swallowed with water followed one hour later with 1gm (a total of 2gm).

Taenia Solium (Pork Tapeworm)

This disease occurs when a person ingests pork infected with the *taenia solium* larvae. Whereas in the beef tapeworm the embryo attaches itself to the wall of the small bowel and grows into an adult worm, the pork tapeworm behaves differently.

The embryo penetrates the intestinal wall of the human as it does the pig, and it is carried to organs like striated muscle or the brain. This can cause serious problems such as epilepsy and death.

Clinical Features

Taenia solium is a dangerous worm and the signs and symptoms depend on the organ it has invaded as follows:

- In the brain it causes epilepsy
- In the skeletal muscles it causes myositis (severe pain), which may make movement temporarily impossible
- In the laryngeal muscles it causes difficulty in speaking

- In the myocardium it causes (myocarditis), heart failure or cardiac arrest
- In the eyeball it can cause unilateral or bilateral blindness

Diagnosis

Diagnosis of taenia solium infections can be made by doing the following tests:

- Biopsy examination of the infected tissue
- X-ray examination to locate the calcified cysticercus
- Stool microscopy for ova and cyst

Management

The management involves both the surgical removal of calcified cysticercus where possible as well as drug treatment with niclosamide 2gm.

The dose is 1gm chewed and washed down with water followed one hour later by 1gm.

What measures would you recommend for the prevention and control of taeniasis?

The prevention and control of taeniasis can be achieved through the following simple measures:

- Proper disposal of human faeces in toilets instead of in the field and within reach of cattle and pigs
- Ensuring that beef, pork and fish are thoroughly cooked
- Eating only meats that have been inspected
- Burying in deep pits or incinerating the carcasses of heavily infected cattle and pigs
- Washing hands thoroughly after handling carcasses and raw meat
- Early diagnosis and treatment of infected persons

Hydatidosis (Hydatid Disease)

The hydatidosis disease is actually a disease of dogs (zoonotic).

Human beings become infected only by accident. Nevertheless, the disease is a serious problem among the Turkana community of northern Kenya.

It is also known as echinococcosis or hydatid disease.

Mode of Transmission

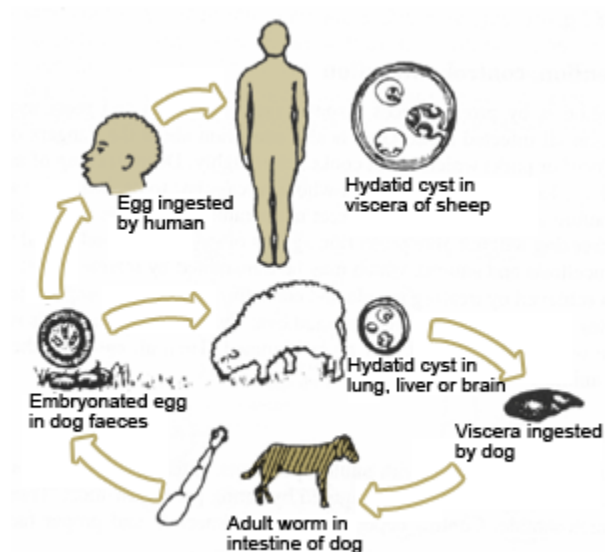
Hydatidosis is caused by the cysts of the dog tapeworm known as Echinococcus granulosus.

Dogs and other carnivores such as jackals and lions are the hosts of the dog tapeworm.

The eggs are passed in the faeces of an infected dog and ingested by domesticated animals such as sheep, goats, cattle, camels, donkeys, and wild antelopes. The eggs hatch in the animal's intestine and penetrate through the intestinal wall to the portal circulation. They are then carried to the liver and lungs where they form many cysts.

When a dog eats the diseased animal it becomes infected with these cysts, which then proceed to develop into mature worms.

Human beings become infected when they accidentally ingest eggs from dog faeces. The larvae migrate from the intestine to the liver or lungs causing cysts. The larvae can also cause cysts in other tissue in the body.



Clinical Features

In the liver, the cyst grows slowly over time thereby enlarging the liver. The abdomen may also become grossly distended.

Diagnosis

This is done through a chest x-ray or an abdominal ultrasound investigation. A serological test can also be done to assist in making the diagnosis.

Management

The treatment of hydatid disease can either be medical or surgical.

The medical treatment is as follows:

- Oral albendazole 20mg/kg in divided doses twice daily for 30 days (The cure rate with this treatment is 20%). The treatment can arrest the growth of the cyst and reduce its size
- PAIR (Puncture, Aspiration, Instillation of 95% alcohol and Re-aspiration). This is the treatment for the liver or spleen. The ultrasound machine is used to guide the PAIR procedure. This treatment is very effective and has a high cure rate

The surgical treatment is known as endocystectomy. It is the surgical removal of the cysts contents, especially those cysts that are easily accessible like abdominal cysts.

Prevention and Control

The prevention and control of the hydatid disease can be achieved by eradicating stray dogs and deworming them. Deworming should be done every six weeks with praziquantel.

You should also provide health education on the dangers of close contact with dogs (licking), especially among children. Also, infected meat should not be fed to dogs.

SECTION 8: DISEASES FROM CONTACT WITH ANIMALS OR ANIMAL PRODUCTS (ZOOONIC DISEASES)

Introduction

In this section you will look at infectious diseases which are transmitted between animals and humans.

Objectives

By the end of this section you will be able to:

- List three diseases transmitted through contact with animals or animal products
- Describe the management of zoonotic diseases namely; anthrax, rabies and brucellosis
- Describe the control measures of zoonotic diseases

Diseases from Contact with Animals

What do you call diseases that are transmitted between animals and humans?

Diseases that are transmitted between infected vertebrate animals (animals with a backbone) and humans are called zoonotic.

In some of these diseases, humans are usually the last in the transmission cycle or the final host as in the case of hydatidosis, unless of course the person's body is eaten by a predator.

Similarly in other diseases like rabies and brucellosis, the disease transmission ends with mankind, though possibilities of further transmission can occur if for example, a rabid patient bites another person, or a patient with brucellosis accidentally transmits it to another person.

Zoonoses are transmitted between animals and humans through the following means:

Vectors

These include:

- The rat flea which transmits plague among rats and other rodents
- The tsetse fly which transmits trypanosomiasis among game animals and nagana in cattle
- Mosquitoes which transmits yellow fever among monkeys

Ingestion of Contaminated Material

Ingestion of meat or dairy products from sick animals, leading to diseases such as:

- Anthrax (meat from cattle and game animals)
- Brucellosis (milk from infected cattle)
- Taeniasis (milk and meat from infected cattle and pigs)

Animal Bites

Bites, resulting in diseases such as:

- Rabies (from rabid domestic and wild dogs or foxes)

Direct Contact with Infected Animal

Close contact resulting in diseases such as:

- Hydatidosis (close contact with infected domestic dogs or other carnivores)
- Cutaneous anthrax (contact with infected cattle or their products)

In this section you will cover anthrax, rabies and brucellosis, looking at their mode of transmission, clinical picture, diagnosis, management and prevention.

Anthrax

Anthrax is an acute bacterial disease of herbivores (plant eating animals). However, it occasionally also infects human beings especially those who process hides, skins and wool or work in slaughterhouses. Anthrax is caused by a rod shaped bacteria (bacilli) called bacillus anthracis.

The disease can occur in large numbers among cattle (epizootic), especially during drought and flooding when they are moved from one place to another. In humans, this infection takes various forms depending on the route of entry.

There is anthrax of the skin which affects people who handle cattle, anthrax of the lungs which occurs in people working with infected wool; and anthrax of the bowels which affects families who eat the meat of dead animals.

The type of disease caused depends on the route of entry of the bacillus or its spores. In animals, anthrax causes a fever which is followed by septicaemia and death. Vultures, which feed on the dead animal can spread the spores.

Mode of Transmission

The bacillus anthracis forms spores when exposed to the air.

The spores can survive for years in the soil even under harsh weather conditions. The spores enter the animals orally (through the mouth or ingestion).

The body of a sick or dead animal contains millions of anthrax bacilli. These bacilli are shed through animal urine, droppings, saliva milk and blood.

If any of these body fluids are touched or the meat of an infected animal eaten, a person becomes infected with anthrax.

Clinical Features

The clinical features depend on the route of entry of the anthrax bacillus.

Skin or cutaneous anthrax presents with a malignant pustule with a black necrotic centre. The wound is usually painless and has swollen edges. Skin anthrax has low mortality.

Respiratory tract anthrax on the other hand has a high mortality rate and presents with severe respiratory distress and shock.

Digestive tract anthrax is characterised by fever, sepsis, watery diarrhoea and vomiting.

Diagnosis

The diagnosis of anthrax is made by taking a specimen (fluid from vesicles, sputum or stool) for a culture to confirm gram-positive rods.

Management

Bacillus anthracis responds to penicillin and most other antibiotics.

Patients with anthrax of the respiratory tract need respiratory support and oxygen therapy in a high dependence care unit.

Those with anthrax of the digestive tract may need fluid replacement due to diarrhoea and vomiting.

Prevention and Control

Although the main responsibility for the prevention and control of anthrax falls on the veterinary department, you as a health worker also have a role to play.

You should ensure that all meat offered for sale is inspected and educate the community on proper disposal of all infected animals. The carcasses must be burnt or buried two meters deep in the ground in calcium oxide powder (quick lime).

Other measures include annual vaccination of cows at risk, proper disinfection of hides and skins, and vaccination of members of the community who are at risk of getting anthrax.

Rabies

Rabies is a serious viral disease of canines which is incidentally transmitted to humans by the bite of a rabid animal.

It is caused by a virus known as lassa virus type I. The disease is of public health importance because it has a case fatality rate of 100%. If a patient is not treated immediately after the bite, once the clinical signs appear it is too late.

Rabies is found all over the world and in canines. It occurs all the time and in great numbers (enzootic and epizootic). In human beings, rabies is a zoonotic disease, and humans usually do not transmit it any further.

The main reservoirs of lassa virus type I are felines, hyenas, and mongoose.

Mode of Transmission

The rabies virus is transmitted to humans through the saliva of an infected animal such as a dog or cat. This happens when humans get bitten by a rabid animal or when its saliva comes into contact with the mucous membranes or open wound of a person.

The main reservoirs of the disease are wild animals such as mongooses, jackals and hyenas. These wild animals infect domestic animals including cattle, donkeys and horses, which in turn infect mankind. In North and South America, rabid bats have been known to infect humans. All warm blooded animals are susceptible to rabies.

Clinical Features

The incubation period of rabies ranges from two weeks to a year, with an average of two to three months. The length of the incubation period is influenced by the following factors:

- The size of the bite - the deeper the bite the shorter the incubation period
- Distance of the wound from the brain - the nearer the wound is to the brain the shorter the incubation period
- Type of wound - if the wound is big with extensive tissue damage the shorter the incubation period

Write down three symptoms of rabies infection.

The earliest symptoms usually consist of increasingly severe pain in the bite wound, depression, irritability, nausea, sore throat, headache and loss of appetite.

Later, two clinical presentations emerge:

Furious rabies whereby the infected person develops convulsions, intense fear of death and irrational excitement, which alternates with periods of alertness and calmness. The patient is also unable to tolerate noise, bright light and cold draught (aerophobia - fear of cold air). There is increased reflexes, muscle spasms, excessive sweating, dilatation of pupils, excessive salivation and lacrimation. The patient develops intense hydrophobia (fear of water) because of the intense pain experienced when swallowing water due to spasms of the pharyngeal muscles. This stage is also known as the 'furious' rabies stage and it lasts for two to three days and sometimes for five to six days. Death usually occurs due to cardiac or respiratory failure during a convulsion.

The next stage is the paralytic rabies stage which is characterised by paralysis of muscles causing paraplegia, quadriplegia and coma. Patients who reach this stage do not survive for more than a week.

Diagnosis

Diagnosis of rabies is made if a person is bitten by a dog with abnormal behaviour and without any provocation. In addition the presence of negri bodies in the brain of a suspected animal should confirm the disease.

Management

There is no cure for rabies once the disease has started. It is however possible to prevent it from reaching that stage by doing the following:

Post Bite Prophylaxis

Immediately someone is bitten you should give first aid treatment of the bite with the aim of removing as much virus as possible. This involves immediate flushing of the wounds and scratches preferably with running water and washing the surrounding skin with a lot of soap and water. Puncture wounds should be irrigated with a sterile catheter using methylated spirit and povidone. Iodine is also virucidal and may be used to clean the wound.

Bite wounds should not be sutured immediately to prevent more traumas from the suturing needle, which will increase the areas for viral entry into the body tissue. Suturing may be done 24 to 48 hours after the bite using very few sutures under the cover of anti-rabies serum locally.

Anti-Rabies Vaccine

This is a very safe and effective treatment following a rabid animal bite.

The vaccine HDCV (Human Diploid cells tissue Culture Vaccine) is administered in six doses sub-cutaneously as follows:

1ml immediately after exposure (day 0), 1ml on day 3, 1ml on day 7, 1ml on day 14, 1ml on day 30, 1ml on day 90.

Other Drugs

In order to prevent wound infection and tetanus you should give the patient broad spectrum antibiotics.

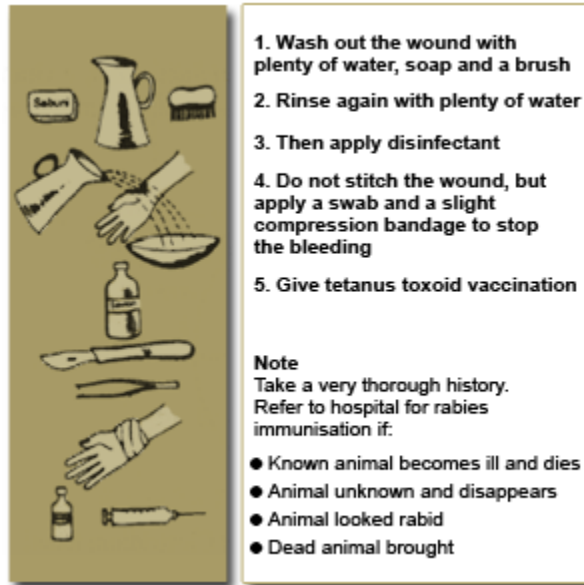
Note: The animal, which inflicted the bite, should be quarantine and observed for ten days from the day of the bite.

If it shows signs of rabies it should be killed and its head removed and sent under refrigeration for rabies examination.

Prevention and Control

Rabies is a notifiable disease. It is very important to give immediate first aid to a person who has been bitten by a suspect animal.

In addition, you should educate the community members on the importance of immunising their domestic dogs and cats every three years and eliminating all stray dogs and cats.



Brucellosis

Brucellosis is a zoonotic disease or disease of animals. It is caused by a bacteria called brucella melitensis in goats, sheep and camels, brucella abortus in cattle and brucella suis in pigs. All these bacteria however can be transmitted to mankind causing brucellosis.

Distribution

Brucellosis has a worldwide distribution, predominantly in rural areas among pastoral communities. It is also an occupational health hazard of farmers, veterinarians, abattoir workers and butchers.

Transmission

Brucellosis is transmitted through ingestion of unpasteurised milk or milk products such as cheese. It can also be transmitted by contact with blood, urine, tissues, through splashing of amniotic fluid or milk on the conjunctiva and blood transfusion.

Clinical Presentation

The incubation period takes about two to four weeks. Initially the signs and symptoms are non-specific and include the following:

- Headaches
- Fever
- Weakness
- Anorexia
- Rigors
- Night sweats
- Constipation

Patients may also complain of pain in the large joints like the hips and knees although any other joint may be affected. Hepatomegally, splenomegally and lymphadenopathy may also be present. If untreated, the disease can continue for many months and the patients may become depressed.

Diagnosis

A serological diagnosis of brucellosis can be made by doing an agglutination test in dilutions. A level of 1:160 or above is associated with the infection.

Blood cultures rarely give positive results but a bone marrow aspirate culture gives better yields of up to 90%. Full haemogram - normochromic, normocytic anaemia, neutropenia and lymphocytosis is common.

Treatment

The treatment of brucellosis is doxycycline 200mg daily for 14 - 21 days and cotrimoxazole tabs 2 bd. for 14 - 21 days.

Prevention

You should educate the community and especially farmers on the importance of boiling or pasteurising milk.

Animal handlers and those at special risk should be advised to take extra precautions.