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DEPARTMENT OF BIOLOGICAL SCIENCES

PRINCIPLES OF PARASITOLOGY

(BIO 302)

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PRINCIPLES OF PARASITOLOGY

INTRODUCTION

Parasitology is a science that deals with parasites. Parasite is an organism living temporarily or permanently in or on another organism (host) from which is physically or physiologically dependant upon other. A parasite could be unicellular, worm or an arthropod.

Features of Parasites

1. Smaller than their host

2. Outnumber the host

3. Short life span than their host, and

4. Have greater reproductive potential than their host.

Association of Organisms

Association between two organisms could be of the following types:

1. Mutualism; Mutual benefit is derived from the association.

2. Symbiosis: Permanent association between two different organisms, so dependant on each other, that their life part is impossible.

3. Commensalism: When the parasite benefited from the host while the host neither benefited nor harmed.

4. Parasitism: One organism lives at the expense of the other 9the host). The host usually suffers from the association.

Parasites can be Classified:

I. According to their habitat:

- Ectoparasites: parasites living on or affecting the skin surface of the host. E.g. lice, tick, etc.

- Endoparasites: Parasites living within the body of the host. E.g. *Leishmania species, Ascaris lumbricoides*, etc.

II. According to their dependence on the host:

- Permanent (obligate) parasites: The parasite depends completely upon its host for metabolites, shelter, and transportation. This parasite can not live outside its host. E.g. *Plasmodium species, Trichmonas vaginalis, etc.*
- Temporary (facultative) parasite: The parasite is capable of independent existence in addition to parasitic life. E.g. *Strongyloids stercolaris, Naegleria fowleri,* etc.

III. According to their Pathogenicity:

- Pathogenic parasites: It causes disease in the host. E.g., E. histolytica
- Non-Pathogenic (commensal) parasite:-The parasite derives food and protection from the host without causing harm to the host. E.g. *Entamoeba coli*
- Opportunistic parasites:- Parasites which cause mild disease in immunologically healthy individuals, but they cause severe disease in immuno-deficient hosts. E.g. *Pneumocystis carnii, Toxcoplasma gondii, Isospora belli*

HOSTS

Hosts are organism which harbors the parasite.

Types of Hosts

1. Definitive host:- This is either a host which harbors the adult stage of a parasite or most highly developed form of the parasite occurs; or sexually mature stages of a parasite and fertilization takes place in it, e.g., man is the definitive host of *Taenia saginata*. When the mature or most highly developed form is not obvious the definitive host is the mammalian host, e.g., human is the definitive host for trypanosomes that cause African trypanosomiasis.

2. Intermediate host:- Is a host harboring sexually immature or larval stage of a parasite and in which no fertilization takes place in it. E.g. Cow is the intermediate host for *Taenia saginata*

3. Amplifier host- Intermediate hosts in which parasites under go multiplication.

3. Reservoir host:- A wild or domestic animal which harbors a parasite and acts as sources of infection to humans.

4. Carrier host:- A host harboring and disseminating a parasite but exhibiting no clinical sign.

5. Accidental (Incidental) host:- Infection of a host other than the normal host species.

Sources of Exposure to Parasitic Infections

A. Contaminated soil with human excreta.
B. Contaminated water with Viable cysts, etc
C. Insufficiently cooked meat
D. Blood sucking arthropods
E. Animals

Host Susceptibility Factors

Host Factors:

1. Genetic constitution
2. Age
3. Sex
4. Level of immunity: natural and acquired immunity.
5. Nutrition (malnutrition or under nutrition)
6. Intensity and frequency of infections
7. Presence of co-existing disease or conditions which reduces immune response. e.g. Pregnancy, HIV
8. Life style and occupation

Parasite factors:

1. Strain of the parasite and adaptation to human host
2. Parasite load (number of parasites)
3. Site (s) occupied in the body
4. Metabolic process of the parasite, particularly the nature of any waste products or toxins produced by the parasite during its growth and reproduction.

PROTOZOAN PARASITES

Protozoa consists of a vast assemblage of single cell micro-organisms that are placed in the subkingdom, or phylum protozoa. They are made of a mass of protoplasm differentiated in to cytoplasm and nucleoplasm. The cytoplasm consists of ectoplasm and endoplasm. The ectoplasm function in protection, locomotion, ingestion of food, excretion, respiration. The endoplasm is concerned with metabolism. It contains the nucleus and many organelles. Reproduction and maintenance of life is performed by the nucleus. The protozoa of medical importance to humans include Amoebas, Flagellates, Ciliates, Coccidia, sporozoa and Microsporidia. Many protozoan species are not pathogenic.

Entamoeba histolytica

GEOGRAPHICAL DISTRIBUTION: Cosmopolitan distribution, mainly in the tropics and subtropics, and is mainly related to inadequate personal hygiene environmental sanitation, lack of safe water supply and poor socioeconomic situation.

HABITAT:

- Trophozoite: Large intestine, liver abscesses and other extraintestinal organs
- Cyst: found in the stools of chronic dysenteric patients and carriers.

MORPHOLOGY

Trophozoite:

- Size: 12 to 35μm.
- Shape: elongated form when actively motile and rounded form when at rest. Motility: Active, Progressive , directional amoeboid motility in fresh warm stool specimen.
- Pseudopodia: Finger like, broadly rounded end.
- Cytoplasm: Well differentiated into ectoplasm and endoplasm. May contain ingested host's red blood cells in dysenteric specimens
- Nucleus: Single nucleus, not visible in the motile form but in iodine stained smear clearly seen.
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Cyst:

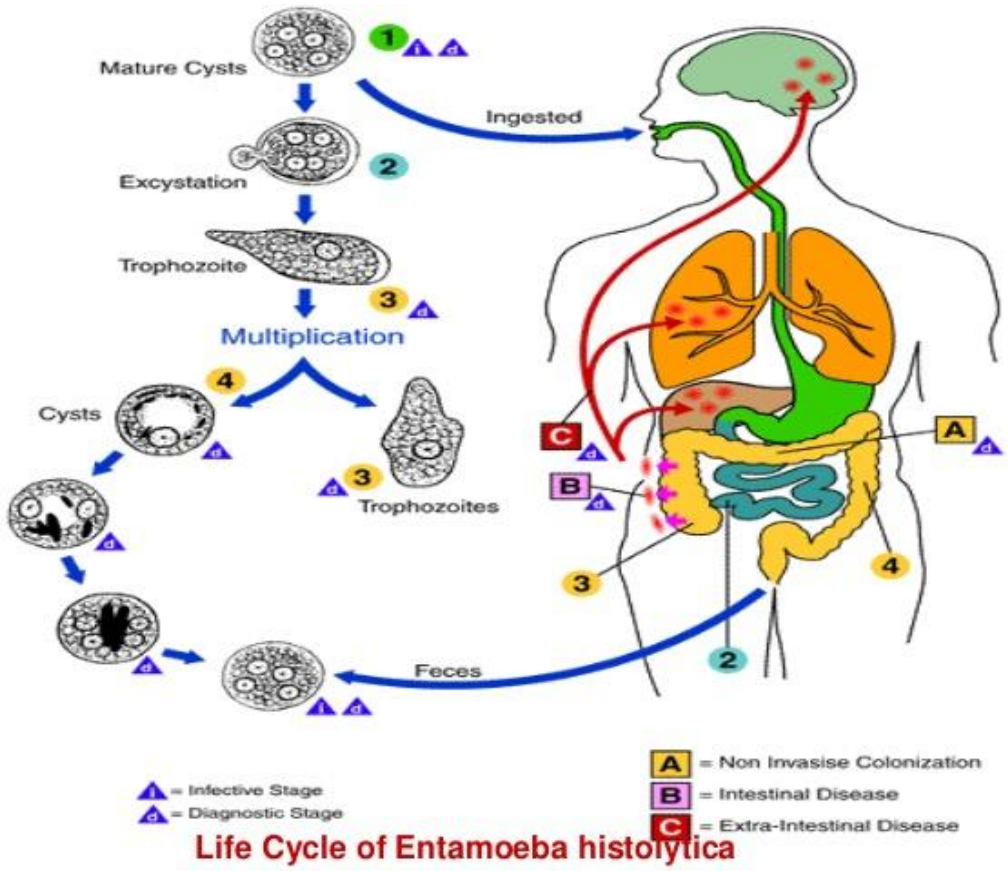
- Size: 12-15μm.
- Shape: spherical Nuclei: 1-4 nuclei Nuclear membrane: thin, regular and circular lined with fine chromatin granules internally , and small, compact central karyosome.
- Cytoplasm: Yellowish-gray and granular in iodine stained smear. Stored food: Sausage shaped chromatoidal bars with blunt ends and glycogen mass in immature cysts with one or two nuclei.

LIFE CYCLE:

Entamoeba histolytica requires a single host to complete its life cycle.

- When mature tetra-nucleated cyst from contaminated food or drink or form hands contaminated with feces is ingested it excysts in the small intestine to produce metacystic trophozoite by a process of binary fission.
- The immature trophozoites migrate to the colon and grow to become mature trophozoite stage, multiply by binnary fission to invade the mucus membrane of the large intestine. Some times it can perforate the intestinal wall causing extra-intestinal amoebiasis.
- The trophozoite stage may pass with diarrhea or dysentery. After a period of growth and multiplication, encystment occurs in the large intestine.
- In the process of cyst formation, the trophozoite discharge undigested food appears spherical in shape and condense to become pre-cyst.
- The pre-cyst secrets cyst wall to form a mono-nucleated cyst which is followed by a nuclear division to produce a bi-nucleated and then a tetra-nucleated mature cyst.

Cyst and precyst will also pass in semi- formed or formed stool, where cyst is infective if it is ingested by any means of transmission.



(From Jeffrey HC and Leach RM. Atlas of Medical Helminthology and Protozoology, 1975.)

CLINICAL FEATURES AND PATHOLOGY

The outcome of infection may result in a carrier state, intestinal amebiasis, or exteraintestinal amebiasis. Diarrhoea, flatulence, and cramping are complaints of symptomatic patients. More severe disease is characterised by the passing of numerous bloody stools in a day. Systemic signs of infection (fever, leukocytosis, rigors) are present in patients with extraintestinal amebiasis. The liver is primarily involved, because trophozoites in the blood are removed from the blood by the portal veins. The right lobe is most commonly involved, thus pain over the liver with hepatomegaly and elevation of the diaphragm is observed

PREVENTION AND CONTROL

1. Cooking of food and vegetables

2. Hand washing after defecation and before eating 3. Safe water supply (treatment, boiling, filtration, etc.)

4. Control of mechanical vectors

5. Avoid use of night soil as a fertilizer proper sanitary disposal of faeces.

6. Treatment of infected individuals and health education.

LABORATORY DIAGNOSIS

Laboratory diagnosis of intestinal amoebiasis is based on:

- Examination of a fresh diarrheic or dysenteric faecal specimen or rectal scraping for motile amoebae using saline, or
- Examination of formed or semi-formed faeces for cyst stages. Such stool can be examined by direct saline and/or iodine smear, and Zinc sulphate floatation or centrifugal floatation method.

Giardia lamblia

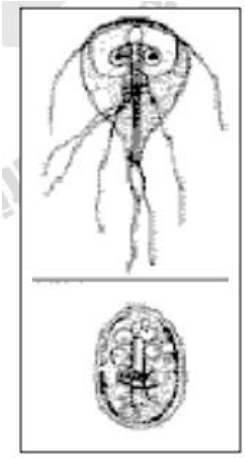
Also called *Giardia intestinalis* and *G.duodenale* Geographical **Distribution**: Cosmopolitan distribution in warm climate and is more prevalent in children than in adults. It is the most commonly diagnosed flagellate of the human intestinal tract. High prevalence occurs in young, malnourished children in large families, orphan asylums, and elementary schools.

Habitat: Upper parts of the small intestine mainly in the duodenum and jejunum.

Morphology:

Trophozoite:

- Size: 10-21 by 5-15µm Shape: pyriform (pear-shaped), i.e. rounded anteriorly and pointed posteriorly.
- Motility: Progressive, rapid, tumbling and spinning often linked to a "falling leaf" type of motility in fresh liquid stools.
- Bilaterally symmetrical Covex dorsal surface and a flattened ventral side.



Trophozoite and cyst of *G.lamblia*

(From Jeffrey HC and Leach RM. Atlas of Medical Helminthology and Protozoology, 1975.)

Cyst :

- Size: 8-12µm, oval shape with thick cyst wall. Finely granular cytoplasm clearly separated from cyst wall. 2-4 oval nuclei at one pole, each with small, central karyosome.

- Cytoplasm: clear when unstained; yellowish green or bluish in iodine solution.
- Fibril: thread-like remains of flagella; axonemes and parabasal bodies folded as S-shaped placed length wise in the center of the cyst.

LIFE CYCLE

It requires a single host to complete its cycle and reproduces by a simple longitudinal binary fission.

Cystingested→excystation→Trophozoite→binaryfission→Encystation→cyst in faeces

- Infection occurs by ingestion of mature tetranucleated cyst with contaminated food, drink, finger, etc.
- Following ingestion, the cyst excyst in the upper part of the small intestine to form flagellates.
- They become attached to the intestinal wall by a sucking disc and absorb nourishment through their body surface.
- They multiply by longitudinal binar fission and some of them are carried down the intestinal tract to undergo encystation.
- The trophozoites and infective cysts are excreted in the faeces.

CLINICAL FEATURE AND PATHOLOGY

Major symptoms includes duodenitis, excess secretion of mucus or malabsorption of fat (steatorrhoea), sugar and vitamins, dehydration, diarrhoea, weight loss, poor appetite, vomiting, lethargy bile passage obstruction

PREVENTION AND CONTROL

1. Improving personal, family and group sanitation and hygiene.

2. Avoid contamination of food, drink and hands with the faeces.

3. Safe water supply and latrine construction.

4. Treatment of infected individuals and health education.

LABORATORY DIAGNOSIS:

Finding the trophozoite and cyst stages in stool specimen. The stool is usually offensive, bulky, pale, mucoid (fatty), diarrheic (watery) but there is no blood in the stool.

Trichomonas vaginalis

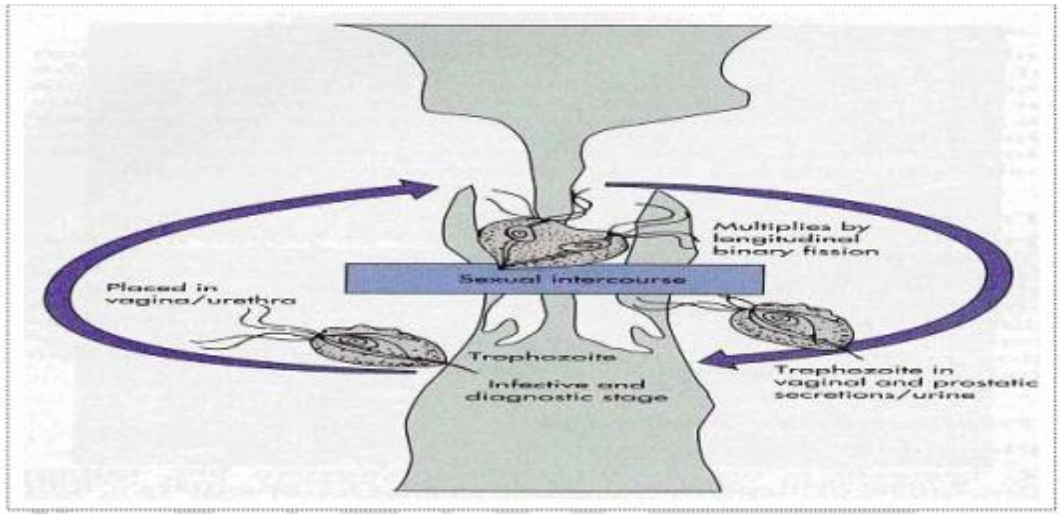
Geographical Distribution

World wide distribution and mainly common in the temperate region. **HABITAT**: In the genital tract of male and commonly in female, especially the vagina, cervix, urinary bladder, prostate and seminal vesicles.

MORPHOLOGY: Has trophozoite stage only.

- Trophozoite Size 15-25 by 5-12μm, is the largest Trichomonas.
- Shape: pyriform Moility: Jerky (on-spot), non-directional motility in fresh specimen.
- Short undulating membrane: extending along one third of the body.
- Nucleus: Single with uniformly distributed chromatin granules.
- Flagella: 4 anterior free flagella and one on the margin of the undulating membrane Axostyle may be split into several fibrils anteriorly. Less conspicuous cytostome and has marked parabasal body.

LIFE CYCLE: The trophozoite stage reproduces by longitudinal binary fission and mode of transmission is usually via sexual intercourse but also by communal bathing, sharing of washclothes, toilet equipment seats and mother to daughter during birth.



Life cycle of *T.vaginalis* (From Nasir NT. Review of Human Parasitology,2nd ed. Cairo: The Scientific Book Center.)

PATHOLOGY: Causes trichomoniasis.

Major symptoms are Vaginitis, urethritis, prostatitis, chaffing of vulva, cervical erosion, burning sensation, yellowish prulent discharge, reversiable sterility in male.

PREVENTION AND CONTROL

1. Personal hygiene and sanitation
2. 2. Simultaneous treatment of both partners.

LABORATORY DIAGNOSIS: Finding the trophozoites in unstained or stained preparation of vaginal or urethral discharges, urine sediment, vaginal swab, prostate secretions.

BLOOD FLAGELLATES/ HAEMOFLAGELATES

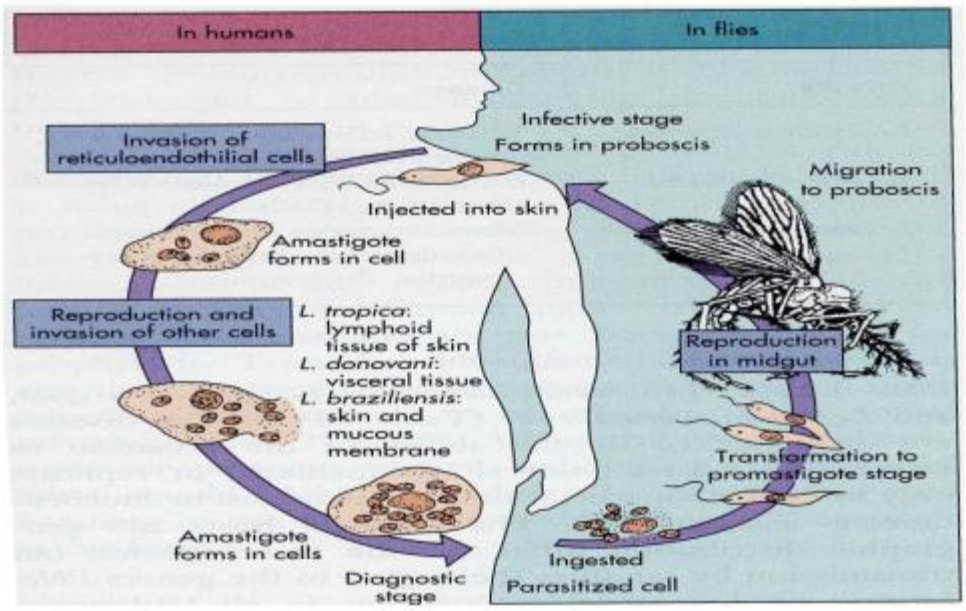
Leishmania Species

Clinical disease - Veseral leishmaniasis

- Cutaneous leishmaniasis

- Mucocutaneous leishmaniasis

The species of leishmania exist in two forms, amastigote (aflagellar) and promastigote (flagellated) in their life cycle. They are transmitted by certain species of sand flies (Phlebotomus & Lutzomyia)



Life cycle of *Leishmania species*

Visceral leishmaniasis

Leishmania donovani

Important features- the natural habitat of *L.donovani* in man is the reticuloendothelial system of the viscera, in which the amastigote multiplies by simple binary fission until the host cells are destroyed, whereupon new macrophages are parasitized. In the digestive tract of appropriate insects, the developmental cycle is also simple by longitudinal fission of promastigote forms. The amastigote stage appears as an ovoidal or

rounded body, measuring about 2-3μm in length; and the promastigotes are 15-25μm lengths by 1.5-3.5μm breadths.

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anaemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anaemia and lymphadenopathy also occurs. Increased production of globulin results in hyperglobulinemia, and reversal of the albumin-to-globulin ratio.

Epidemiology

L. donovani donovani, infection of the classic kala-azar ("black sickness") or dumdum fever type occurs in many parts of Asia, Africa and Southeast Asia. Kala-azar occurs in three distinct epidemiologic patterns. The vector is the *Phlebotomus,* sand fly.

Clinical features

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate & invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia, and emaciation occurs. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis, occurs.

Untreated visceral leishmaniasis is nearly always fatal as a result of secondary infection.

Laboratory diagnosis

• Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).

• The amastigotes appear as intracellular & extra cellular L. donovan (LD) bodies.

• Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.

• Serologic testing is also available.

Treatment

The drug of choice is sodium stibogluconate, a pentavalent antimonial compound.

Alternative approaches include the addition of allopurinol and the use of pentamidine or amphotercin B.

Prevention

• Prompt treatment of human infections and control of reservoir hosts.

• Protection from sand flies by screening and insect repellents.

Old World Cutaneous Leishmaniasis (Oriental sore)

Clinical disease

L.tropica minor - dry or urban cutaneous leishmaniasis *L.tropica major* - wet or rural cutaneous leishmaniasis *L.aethiopica* - cutaneous leishmaniasis

Important features

These are parasites of the skin found in endothelial cells of the capillaries of the infected site, nearby lymph nodes, within large mononuclear cells, in neutrophilic leukocytes, and free in the serum exuding from the ulcerative site. Metastasis to other site or invasion of the viscera is rare.

Pathogenesis

In neutrophilic leukocytes, phagocytosis is usually successful, but in macrophages the introduced parasites round up to form amastigote and multiply. In the early stage, the lesion is characterized by the proliferation of macrophages that contain numerous amastigotes. There is a variable infiltration of lymphocytes and plasma cell. The overlying epithelium shows acanthosis and hyperkeratosis, which is usually followed by necrosis and ulceration.

Epidemiology

Cutaneous leishmaniasis produced by *L.tropica* complex is present in many parts of Asia, Africa, Mediterranean Europe and the southern region of the former Soviet Union. The urban Cutaneous leishmaniasis is thought to be an anthroponosis while the rural cutaneous leishmaniasis is zoonosis with human infections occurring only sporadically. The reservoir hosts in *L. major* are rodents. *L.aethopica* is endemic in Ethiopia and Kenya. The disease is a zoonosis with rock & tree hyraxes serving as reservoir hosts. The vector for the old world cutaneous leishmaniasis is the Phlebotomus sand fly.

Clinical features

The first sign, a red papule, appears at the site of the fly's bite. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate. Gradually the ulcer becomes hard and

crusted and exudes a thin, serous material. At this stage, secondary bacterial infection may complicate the disease. In the case of the Ethiopian cutaneous leishmaniasis, there are similar developments of lesions, but they may also give rise to diffuse cutaneous leishmaniasis (DCL) in patients who produce little or no cell mediated immunity against the parasite. This leads to the formation of disfiguring nodules over the surface of the body.

Treatment

The drug of choice is sodium stibogluconate, with an alternative treatment of applying heat directly to the lesion. Treatment of *L.aethopica* remains to be a problem as there is no safe and effective drug.

Prevention

- Prompt treatment & eradication of ulcers
- Control of sand flies & reservoir hosts.

TRYPANOSOMIASIS

Etiologic agents

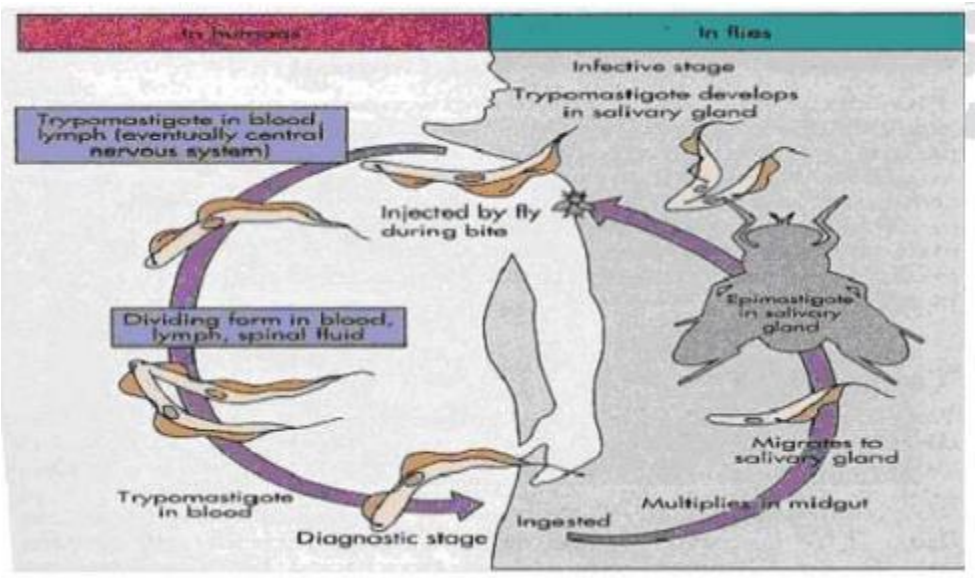
1. *Trypanosoma brucei complex* – African trypanosomiasis (sleeping sickness)
2. *Trypanosoma cruzi* – American trypanosomiasis (Chagas' disease)

Important features

These species may have amastigote, promastigote, epimastigote, and trypomastigote stages in their life cycle. In human trypanosomes of the African form, however, the amastigote and promastigote stages of development are absent. Typical trypanosome structure is an elongated spindle-shaped body that more or less tapers at both ends, a centrally situated nucleus, a kinetoplast posterior to nucleus, an undulating membrane arising from the kinetoplast and proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end.

African trypanosomiasis

Trypanosoma gambiense & *Trypanosoma rhodesiene* are causative agents of the African typanosomiasis, transmitted by insect bites. The vector for both is the tsetse fly.



Life cycle of *Trypanosoma brucei*

Pathogenesis

The trypomastigotes spread from the skin through the blood to the lymph node and the brain. The typical somnolence (sleeping sickness) usually progresses to coma as a result of demyelinating encephalitis. In acute form, cyclical fever spike (approximately every 2 weeks)

occurs that is related to antigenic variation. As antibody mediated agglutination and lysis of the trypomastigotes occurs, the fever subsides. With a few remains of antigenic variants new fever spike occurs and the cycle repeats itself over a long period.

Epidemiology

T.burcei gambiense is limited to tropical west and central Africa, correlating with the range of the tsetse fly vector. The tsetse flies transmitting *T.b. gambiense* prefer shaded stream banks for reproduction and proximity to human dwellings. People who work in such areas are at greatest risk of infection. An animal reservoir has not been proved for this infection.

T.burcei rhodeseinse is found primarily in East Africa, especially the cattle-raising countries, where tsetse flies breed in the brush rather than along stream banks. *T.b. rhodeseines* also differs from *T.b. gambiense* in that domestic animal hosts (cattle and sheep) and wild game animals act as reservoir hosts. This transmission and vector cycle makes the organism more difficult to control than *T.b. gambiense*.

Clinical features

Although both species cause sleeping sickness, the progress of the disease is different. *T.gambiense* induced disease runs a low-grade chronic course over a few years. One of the earliest signs of disease is an occasional ulcer at the site of the fly bite. As reproduction of organisms continues, the lymph nodes are invaded, and fever, myalgia, arthralgia, and lymph node enlargement results. Swelling of the posterior cervical lymph nodes is characteristic of Gambian sleeping sickness and is called winterbottom's sign.

Chronic disease progresses to CNS involvement with lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration. In the final stages, convulsions, hemiplegia, and incontinence occur. The patient becomes difficult to arouse or obtain a response from, eventually progressing to a comatose state. Death is the result of CNS damage and other infections, such as pneumonia.

In *T.rhodesiense*, the disease caused is a more acute, rapidly progressive disease that is usually fatal. This more virulent organism also develops in greater numbers in the blood. Lymphadenopathy is uncommon, and early in the infection, CNS invasion occurs, resulting in lethargy, anorexia, and mental disturbance. The chronic stages described for *T.gambiense* are not

often seen, because in addition to rapid CNS disease, the organism produces kidney damage & myocarditis, leading to death.

Laboratory diagnosis

Examination of thin and thick films, in concentrated anticoagulated blood preparations, and in aspiration from lymph nodes and concentrated spinal fluid. Methods for concentrating parasites in blood may be helpful approaches including centrifugation of heparinized samples and an ion–exchange chromatography.

Levels of parasitosis vary widely, and several attempts to visualize the organism over a number of days may be necessary.

Treatment

The same treatment protocol is applied for these parasites. For the acute stages of the disease the drug of choice is suramin with pentamidine as an alternative. In chronic disease with CNS involvement, the drug of choice is melarsoprol. Alternatives include trypars amide combined with suramin.

Prevention

• Control of breeding sites of tsetse flies and use of insecticides.

• Treatment of human cases to reduce transmission to flies.

• Avoiding insect bite by wearing protective clothing & use of screen,
bed netting and insect repellants.

HAEMOSPORIDIA (THE MALARIA PARASITES)

Malaria is the most important of all protozoan disease; it annually infects over 250 million individuals and is a leading cause of illness and death in the developing world. In many endemic areas it is becoming increasingly difficult to control because of Anopheline mosquito vector and the parasite to develop resistance to various eradication and treatment options.

GENERAL CHARACTERISTICS

1. Intracellular obligate parasites.

2. Man is intermediate host.

3. Female Anopheles mosquitoes are the definitive hosts.

4. Those species which infect human being are *P.vivax, P.falciparum, P.malariae and P.ovale*

5. Has no animal reservoir host except *P.malariae* in which monkeys are the reservoir hosts

6. Infective stage to man from the insect vector is sporozoites and to the insect vector from man is gametocytes.

GEOGRAPHICAL DISTRIBUTION

Malaria is endemic in 91 countries with about 40% of the world population is at risk. Plasmodium falciparum is the most prevalent species in the hotter and more humid regions of the world. *P.vivax* is the most widely distributed in the temperate, subtropics and some parts of the tropics. Unlike the other species, it is more common and well adapted to the temperate region than in the tropics. *P.malariae* has much lower prevalence than *P.vivax*, *P.falciparum* and *P.ovale*. It is confined mainly to tropical Africa. Also it is found in South America and South west Asia. Infection rates in Ethiopia are about 60%, Parasitology 94 40%, 1% and less than 1% for *P.falciparum , P.vivax, P.malariae and P.ovale*, respectively.

HABITAT:

The parasite enters the blood and carried to the parenchyma cells of liver, where they multiply enormously. This is called the pre-erythrocytic or tissue phase. By rupture of the infected cells

they enter the RBCs, the erythrocytic phase (Schizogony) and reach all the organs of the blood via the circulating blood, producing parasitaemia.

MORPHOLOGY

There are sequential developmental stages with distinct morphological features that helps for species identification in Romanowsky stains of Peripheral blood films.

P.falciparum

Young Trophozoite (Ring forms) Stage frequently found in blood film. Size: - Small rings, 0.15-0.5 diameters of RBCs which is unaltered in size.

- Shape: small fine pale blue ring
- Chromatin: 1 or 2 small red dots. Often with double chromatin dot May lie on red cell membrane (accole forms)
- Pigment: absent

Mature Trophozoite Stage rarely seen in peripheral blood RBC unaltered in size, sometimes stippled, pale

Shape: compact thin blue ring , comma or exclamation mark shaped Chromatin: 1 or 2 red dots

Pigment: black or dense brown mass Schizont RBC unaltered in size , sometimes stippled, pale.

Size: parasite about 0.6RBC

Merozoites: 8-32; average 24

Pigment: clumped black Not usually seen in peripheral blood Gametocytes RBC is distorted. Fairly frequently found

Size: larger than red cell Shape: crescent or banana or kidney or sickle shaped Rounded forms may be seen if film dries slowly Stippling Maurer's cleft Infected red blood cells All age group of red blood cells are infected. Often contain several parasites

Density: often high density of parasites

GENERAL LIFE CYCLE OF MALARIA PARASITES

Malaria parasites require two hosts to complete their life cycle.

Female Anopheles mosquitoes as the definitive host, where sexual reproduction (sporogony) takes place and

Human being as the intermediate host, where the asexual reproduction (schizogony) takes place.

- Sporozoites from infected female Anopheles mosquito are injected with the saliva into the blood circulation of man when the vector takes a blood meal.
- After circulating in the blood stream for not more than one hour, the sporozoites enter into the liver cells. - Two cycles occur in man, in the liver as exo-erythrocytic schizogonic reproduction and in the red blood cells as erythrocytic schizogolnic reproduction.
- In the liver the parasites multiply and develop into schizonts.
- When mature, the schizont in the liver cell rupture releasing large number of merozoites.
- The merozoites enter the red blood cells and develop to trophozoite stage.
- The trophozoite feeds on haemoglobin and forms malaria pigment (haemozoin).
- The trophozoite stage develops into schizontes. - In the schizont nuclear division takes place to produce large number of merozoites that are released from the schizont to invades new red blood cells.
- After several erythrocytic schizogonic reproductions, the merozoites develop into gametocytes.

To continue the life cycle, the gametocytes are ingested by a female Anopheles mosquito while taking a blood meal.

- In the stomach of the mosquito, the male and female gametocytes undergo fertilization and produces a zygote.
- The zygote develops into a motile ookinete which penetrates the stomach wall of the mosquito to form an oocyst. Inside the oocyst large numbers of sporozoites are formed.
- The oocyst ruptures releasing the sporozoites that also enter into the salivary gland to be transmitted to another individual when the insect takes a blood meal.

MODES OF TRANSMISSIONS

1) Bite of infected female Anopheles mosquitoes
2) Blood transfusion causes only erytrocytic infection.
3) Congenital / transplacental

CLINICAL FEATURE OF MALARIA

P.falciparum causes malignant Tertian or subtertian malaria. Major symptom in malaria fever usually occurs in three stages:

1. Cold stage: Rigor, headache, coldness and shivering
2. Fever stage: rise in temperature, up to 40^0C, severe headache, back and joint pain, vomiting, diarrhea
3. Sweating stage: perspiration, temperature falls, headache and pain relived until the next rigor.

The malaria fever is due to the rupture of the infected red blood cells containing mature schizonte stage releasing malaria pigment, toxins, metabolic by products, debris of red blood cells and merozoites that can infect other red blood cells

Factors That Provide Protection against Malaria Infections are:

1) Glucose-6-phosphate dehydrogenase deficiency
2) Sickle cell anemia
3) Ovalocytosis and
4) Adenosine tri-phosphate deficiency in non-immune black males provides protection against *P.falciparum* infection

PREVENTION AND CONTROL

This can be become difficult as a result of resistance of the parasites to the drugs and failure of control measures. Besides this population movement, climatic changes and economic problems are also considered currently as factors that related with malaria spread. However, the following are some of the measures to be taken as prevention and control measures:

1) Avoid mosquito bites by - Selecting healthy sites for houses and screening windows and doors with mosquito net. - Using mosquito bed nets - Wearing protective clothes such as long trousers - Using mosquito repellents.

2) Destroy adult mosquitoes by - Indoor residual regular effective spraying.

3) Preventing breeding of mosquitoes by - Altering the habitat to discourage breeding - Flooding or flushing of breeding places - Drainage to remove surface water, filling in ponds, pot holes, etc. - Spraying breeding places with effective chemicals particularly with larvicides

4) Using drugs to - Prompt diagnosis and treatment of malaria cases - Prevent infections using chemoprophilaxis, especially in nonimmune persons visiting or going to malarious areas or in persons with reduced immunity such as pregnant women.

5) Health education.

6) Blood screening for malaria before providing for those who need blood.

LABORATORY DIAGNOSIS

1) Malaria parasites are detected in thin or thick blood films stained by wright's stain, Giemsa stain, leishman stain or Field stain. Take blood films when the patient feels febrile because the parasites are usually most numerous in the blood towards the end of an attack of fever. Always collect the blood before anti-malarial drugs are taken. Field stain is recommended for smears stained straight away and Giemsa stain for smears to be stained after a few days.

2) Using a rapid immunodiagnostic tests such as ParaSight F, ICT malaria Pf / Pv and, OptiMALr.

3) ELISA

4) PCR

HELMINTHS (WORMS)

Helminthes are trophoblastic metazoa (multi-cellular organisms). Helminthes are among the common parasitic causes of human suffering. They are the cause of high morbidity and mortality of people worldwide. They cause different diseases in humans, but few helminthic infections cause life- threatening diseases. They cause anemia and malnutrition. In children they cause a reduction in academic performance. Helminthes also cause economic loss as a result of infections of domestic animals. There is age dependent distribution of infections from geo-helminthes and schistosomes. As a result of predisposing behavioral and immunological status, children disproportionately carry the burden of schistosomes and geo-helminthes.

The sources of the parasites are different. Exposure of humans to the parasites may occur in one of the following ways:

1. Contaminated soil (Geo-helminthes), water (cercariae of blood flukes) and food (Taenia in raw meat).
2. Blood sucking insects or arthropods (as in filarial worms).
3. Domestic or wild animals harboring the parasite (as in echinococcus in dogs).
4. Person to person (as in *Enterobius vermicularis*, *Hymenolopis nana*).
5. Oneself (auto-infection) as in *Enterobius vermicularis.*

They enter the body through different routes including: mouth, skin and the respiratory tract by means of inhalation of airborne eggs.

The helminthes are classified into three major groups. These are:

1. Trematodes (Flukes)
2. Nematodes (Round worms)
3. Cestodes (Tape worms)

The Trematodes and Cestodes are groups of flat worms.

MEDICALLY IMPORTANT TREMATODES (FLUKES)

INTRODUCTION

Trematodes belong to the phylum platyhelminthes. They are found in a wide range of habitats. The great majority inhabit the alimentary canal, liver, bile duct, ureter and bladder of vertebrate animals.

According to the sites they inhabit, there are four groups of flukes. These are: Blood flukes, Intestinal flukes, Liver flukes, and Lung flukes

BLOOD FLUKES

These are flukes that reside mainly in the blood vessels of various organs and the schistosomes are the prototype and the commonest flukes in our country.

SCHISTOSOMIASIS (BILHARZIASIS)

It is estimated that about 600 million people in 79 countries suffer from schistosomiasis (Bilharziasis). The schistosomes cause intestinal, hepatosplenic, pulmonary, urogenital, cerebral and other forms of schistosomiasis. Schistosome is the only fluke with separate sexes. The female worm lies in the gynecophoral canal of the male. This condition is important for transportation. There are five medically important species:

1. *Schistosoma mansoni*: causes intestinal schistosomiasis.
2. *Schistosoma haematobium*: causes vesical (urinary) schistosomiasis.
3. *Schistosoma japonicum*: causes intestinal schistosomiasis.
4. *Schistosoma intercalatum*: causes intestinal schistosomiasis.
5. *Schistosoma mekongi*: causes intestinal schistosomiasis. This seems to cause milder disease in man. It causes disease in other vertebrate hosts.

SCHISTOSOMA MANSONI

Habitat - This species lives in the veins of the intestine.

Geographical distribution: It is found in Africa, South America, Middle East (some Arab countries) etc. Stream and lake-based transmission is common. The snail hosts that harbor *S. mansoni* are the genera: Biomphalaria (*B. glabrata*) and Trobicorbis. These have oval shells.

Morphology

Male: The male ranges in size from 1-1.4 cm in length and the body is covered by coarse tubercles. It has 6-9 testes

Female: The female is 1.5-2.0 cm in length. The ovary is present in the anterior third and Vitelline glands occupy the posterior two-thirds. It lays about 100-300 eggs daily. The uterus is short containing few ova.

URINARY SCISTOSOMIASIS Etiology - *Schistosoma haematobium*

Habitat - The worm lives in the veins of the bladder of humans.

The peak prevalence is the 10-14 year age group. The snail hosts that harbor *S. haematobium* are the genera *Bulinus (Bulinus africanus, B. truncatus*) and Physopsis.

Male: The male ranges in size from 1-1.5 cm in length. The body is covered by fine tubercles. It has 4-5 testes.

Female: The female ranges in size from 2-2.5 cm in length. The ovary is present in the posterior third. Vitelline glands occupy the posterior thirds. Uterus is long containing many ova. It lays about 20-200 eggs daily

SCHISTOSOMA JAPONICUM

The female adult worm lays about 500-3500 eggs daily. The eggs are ovoid, bearing only a minute lateral spine or a small knob postero-laterally. It is found in Japan, China, and Philippines, etc.

SCHISTOSOMA INTERCALATUM

This is the rarest and least pathogenic schistosome that matures in man. It is found in Western and Central Africa. The daily egg output is about 300. The eggs have a terminal spine.

LIFE CYCLE OF SCHISTOSOMES

Adult worms reside in pairs: the female lying in the gynecophoral canal of the male. After fertilization, eggs are passed into the venules. A larval form – the miracidium - develops within the egg. Its lytic enzymes and the contraction of the venule rupture the wall of the venule liberating the egg into the perivascular tissues of the intestine (*S. mansoni*) or urinary bladder (*S. haematobium*). The eggs pass into the lumens and organs and are evacuated in the feces (*S. mansoni*) or the urine (*S. haematobium*). On contact with fresh water the miracidia hatch from the eggs and swim about until they find the appropriate snail, which they penetrate. After two generations of sporocyst development and multiplication within the snail, the fork-tailed cercariae emerge. Infection to man takes place during bathing or swimming. The cercariae penetrate the skin, are carried into the systemic circulation and pass through to the portal vessels. Within the intrahepatic portion of the portal system, the worms feed and grow to maturity.

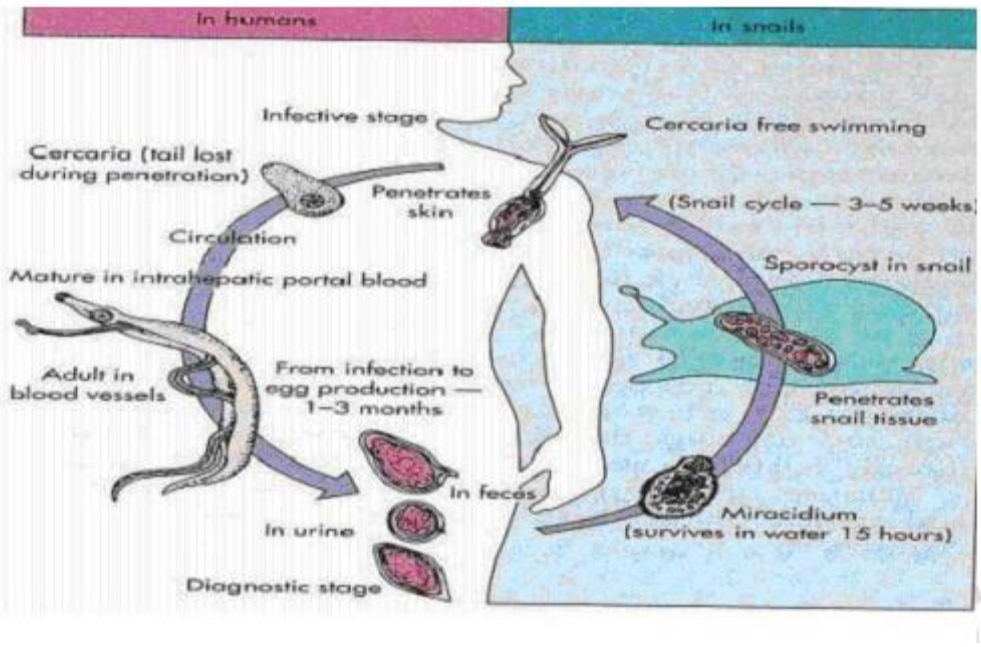


Figure 1.1. Life cycle of schistosomes

Symptoms and complications

Patients infected with *S. haematobium* suffer from terminal haematuria and painful micturition. There is inflammation of the urinary bladder (cystitis), and enlargement of spleen and liver.

Patients infected with *S. mansoni* suffer from cercarial dermatitis (swimmers itch) and dysentery (mucus and blood in stool with tenesmus) as well as enlargements of the spleen and liver.

S. haematobium causes squamous cell carcinoma in the bladder.

Laboratory Diagnosis *S. mansoni*

♦ Microscopic examination of the stool for eggs after concentration by sedimentation method. The egg has characteristic lateral spine.

♦ Rectal snip

S. haematobium:

♦ Examination of the urine after allowing it to sediment in a conical urinalysis glass. A drop from the sediment is taken and examined for eggs. Egg has terminal spine.

♦ Biopsy from bladder

Figure 1.2. Eggs of *S. mansoni* and *S. haematobium*

TREATMENT:

Praziquantel: single oral dose of 40 mg/kg divided into two doses.

PREVENTION:

1. Health education:

A. On use of clean latrines and safe water supply

B. Avoid urination and defecation in canals, avoid contact with canal water

2. Snail control:

A. Physical methods:

i. Periodic clearance of canals from vegetations.

ii. Manual removal of snails and their destruction.

B. Biological methods: Use of natural enemies to the snails such as

C. Chemical methods: Molluscides are applied in the canals to kill the snails. e.g. Endod

1.2. INTESTINAL FLUKES

♦ ***Fasciolopsis buski***: These giant intestinal flukes (2-7.5 cm in length) are found in some Asian countries.

♦ **Heterophyids**: Minute flukes acquired by ingestion of raw fresh water fish. They are found in Asian countries.

LIVER FLUKES

♦ ***Clonorchis sinensis***: Chinese liver fluke - adult worms live in bile ducts.

♦ ***Faciola hepatica***: Sheep liver fluke - is a common parasite, cosmopolitan in distribution. It is large (3 cm in length). Adult worms reside in the large biliary passages and gall bladder.

♦ Other: ***Faciola gigantica:*** lives in the liver of cattle. Human infections are very rare.

1.4. LUNG FLUKES

At least eight different species of lung flukes, all belonging to the genus Paragonimus, are known to infect man. *Paragonimus westermani*, best known species, affects man causing paragonimiasis (lung disease). It is found in Asia (China, India, Indonesia, Malaya etc) and some African countries.

NEMATODES (ROUND WORMS)

All the important human parasites of the Phylum Nemathelminthes
(Aschelminthes) belong to the Class Nematoda.

GENERAL CHARACTERISTICS OF NEMATODES

They are un-segmented, elongated and cylindrical. They have separate sexes with separate appearances. They have a tough protective covering or cuticle. They have a complete digestive tract with both oral and anal openings. The nematodes are free living (Majority) or parasites of humans, plants or animals.

The parasitic nematodes:

The nematodes are generally light cream-white colored. Their life cycle includes: egg, larvae and adult.

The parasitic nematodes are divided into:

1. **Intestinal nematodes**
2. **Intestinal nematodes with tissue stage**

A. *Ascaris lumbricoides*

B. Hookworms

C. *Strongyloides stercoralis*

Intestinal nematodes without tissue stage

A. *Enterobius vermicularis*

B. *Trichuris trichuira*.

2. Tissue and blood dwelling nematodes

2.1. Filarial worms

2.2. *Dracunculus medinensis*

2.3. Trichinella

2.4. Larva migrans.

INTESTINAL NEMATODES WITH TISSUE STAGE

ASCARIS LUMBRICOIDES

These are common roundworms infecting more than 700 million people worldwide.

Morphology:

Male adult worm measures 15-20 cm in length. The posterior end is curved ventrally. The female worm measures 20-40 cm in length. Its posterior end is straight.

Infective stage and modes of infection:

The egg containing larva when ingested with contaminated raw vegetables causes ascariasis.

Life cycle:

Ingested eggs hatch in the duodenum. The larvae penetrate the intestinal wall and circulate in the blood. From the heart they migrate to the lungs, ascend to the trachea, descend to the esophagus

and finally reach the small intestine to become adult. The female pass immature eggs which pass to the soil and mature in 2 weeks.

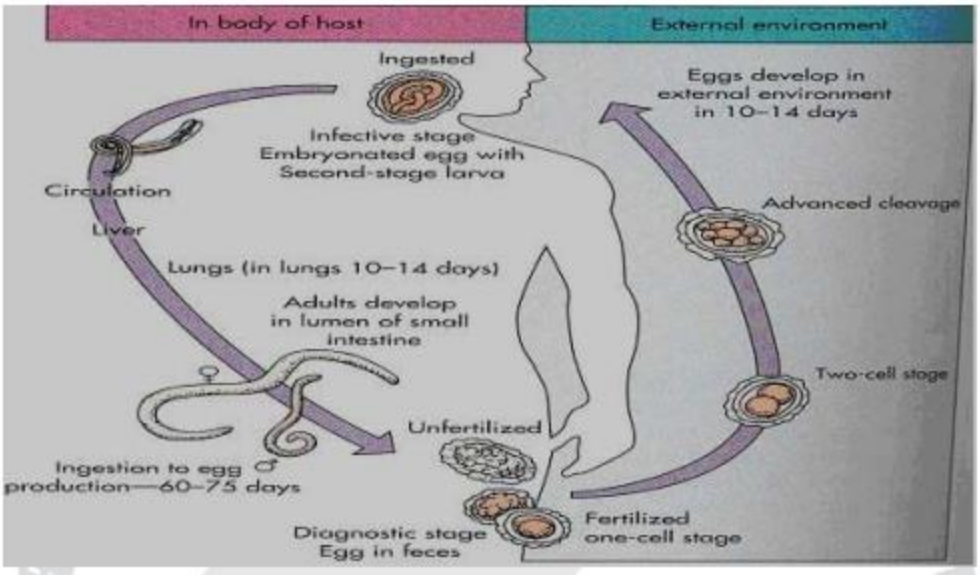


Figure 1.3. Life cycle of *Ascaris lumbricoides*

Pathogenecity and clinical features

Adult worms in the intestine cause abdominal pain and may cause intestinal obstruction especially in children. Larvae in the lungs may cause inflammation of the lungs (Loeffler's syndrome) – pneumonia-like symptoms.

Diagnosis

1. Examination of stool for eggs by direct saline smear method. The egg is ovoidal, 75x60 microns, covered by albuminous mamillatins.
2. Demonstration of adult worms

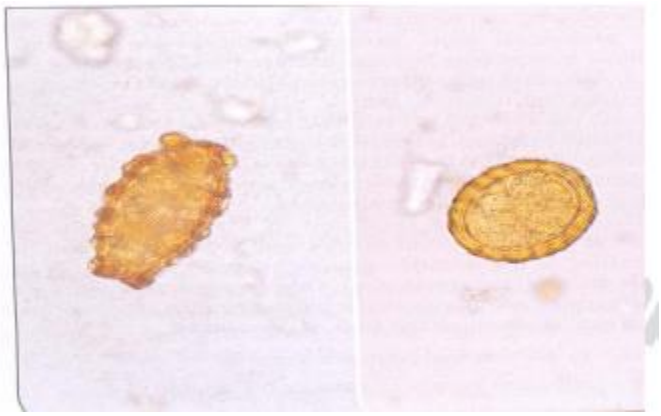


Figure1.4. Egg of *Ascaris lumbricoides*

Treatment

Mebendazole, Albendazole and Piperazine

HOOK WORMS

There are two species of hookworm:

1. Ancylostoma duodenale

2. *Necator americanus*

The adults are found in the small intestines of man. Mixed infection is common.

Ancylostoma duodenale:

Grayish-white in color. The body is slightly ventrally curved. The anterior end follows the body curvature. The buccal cavity is provided ventrally with pairs of teeth and dorsally with a notched dental plate.

Distribution: This species is found in the northern part of the world including China, Japan, Europe, North Africa and Ethiopia.

Morphology

Male: The male measures 10 cm in length. The posterior end is broadened into a membraneous copulatory bursa that is provided with two long spicules.

Female: The female measures 12 cm in length. The posterior end is straight.

Necator americanus

This species, so called American hookworm, is found in predominantly the tropics. The anterior end is hooked against the body curvature. The mouth is provided ventrally and dorsally with cutting plate.

Morphology

Male: The male measures 8 cm in length. The posterior end is broadened into a membraneous copulatory bursa, which is provided with two long spicules fused distally.

Female: The female measures 10 cm in length. The posterior end is straight **Infective stage and methods of infection:**

The filariform larva infects by skin penetration.

Life cycle

Adult male and female worms live in the small intestine. The female lays eggs (oval, 60x40 microns), which contain immature embryo in the 4 cell stage. When the eggs pass in the stool to the soil and under favorable conditions of temperature, moisture and oxygen, they hatch into larvae, which molt twice and become infective. When the filariform larvae penetrate the skin, they circulate in the blood, reach the lungs, ascend to the trachea, descend to esophagus to reach the small intestine and become adults.

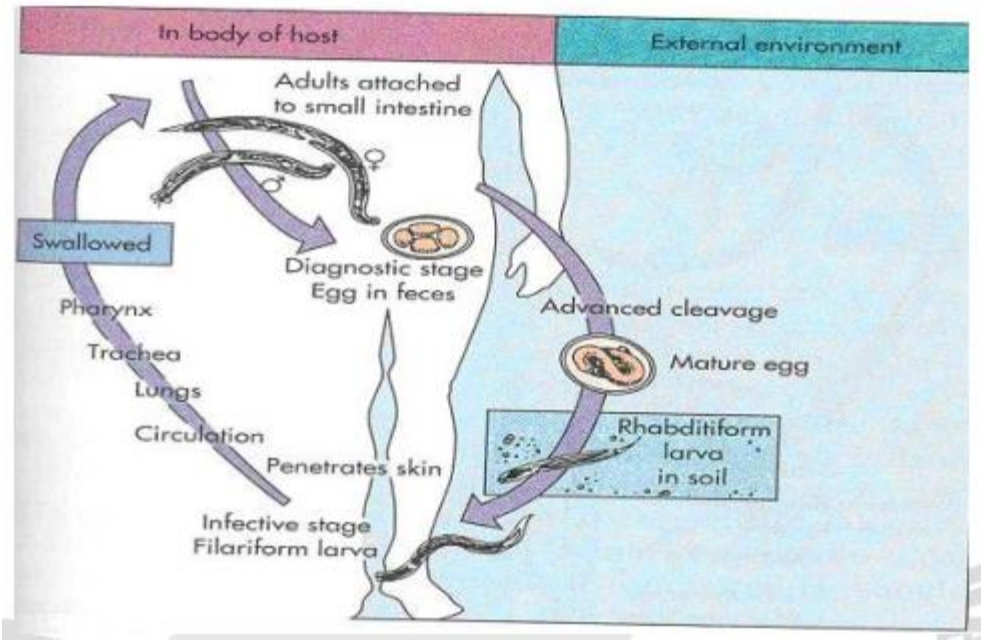


Figure1.5. Life cycle of hookworms

Pathogenecity

Adult worms in the intestine feed on blood causing iron deficiency anemia. The larvae may cause inflammation of the lungs.

Diagnosis: Examination of stool by direct saline smear to detect the eggs.

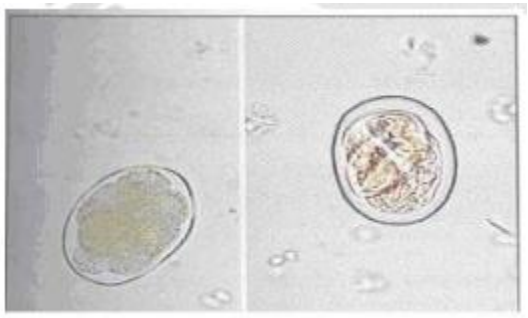


Figure1.6. Egg of hookworm

Treatment

Mebendazole: 1 tab 2x daily for 3 days.

LARVA MIGRANS

There are three types of larva migrans:

a. Cutaneous larva migrans (Creeping eruption)

Various animals harbor hookworms. Two species of dogs and cats are important.

1. *Ancylostoma braziliens*: infects both dogs and cats.
2. *Ancylostoma caninum*: infects only dogs.

Both of these are common in the tropics and subtropical regions where human hookworms can best complete their life cycles. If man comes in contact with infective larvae, penetration of the skin may take place; but the larvae are then unable to complete their migratory cycle. Trapped larvae may survive for weeks or even months, migrating through the subcutaneous tissues. They may evoke a fairly severe reaction - pruritus and dermatitis. The dermatitis leads to scratching and then bacterial superinfection.

Treatment

Thiabendazole: Applied topically.

b. Visceral larva migrans

A syndrome caused by the migration of parasitic larvae in the viscera of a host for months or years. It may be caused by transient larval migration in the life cycles of several parasites such as hookworm, *Ascaris lumbricoides, T. spiralis, S. strecoralis* and other filarial worms.

Toxocariasis

This is a kind of visceral larva migrans caused by ♦ *Toxocara canis* (Dog ascarid) and

♦ *Toxocara catis (*Cat ascarid).

These cause persistent larval migration and thus the visceral larva migrans is called toxocariasis.

Morphology

♦ The larvae of *Toxocara canis* and *Toxocara catis* measure about 400 μm in length.

♦ The life cycle of these parasites in their respective hosts is similar to that of *A. lumbricoides* in humans.

Epidemiology

Visceral larva migrans is cosmopolitan in distribution.

Transmission:

Ingestion of eggs of Toxocara species in contaminated food or soil or direct contact with infected patients. Children are more at risk.

Clinical features:

♦ Majority are asymptomatic.

♦ Eosinophilia

♦ Cerebral, myocardial and pulmonary involvement may cause death.

Diagnosis - Identification of larvae in tissue.

Treatment - Thiabendazole: 25 mg/kg twice daily for 5 days.

C. Intestinal larva migrans

This is an extremely rare kind of larva migrans

STRONGYLOIDES STERCORALIS

The worms may be present as parasitic in the host or free living in the soil.

Morphology:

Male: The male measures1 mm in length with curved posterior end and carries two spicules

Female: The female measures 2.5 mm in length with straight posterior end.

Infection: follows skin penetration by filariform larvae.

Life cycle

Adult male and female worms live in the small intestine. After fertilization, the female penetrates the mucosa of the small intestine and lay eggs in the submucosa. The eggs hatch and the larvae penetrate the mucosa back to the lumen. If the environmental conditions are favorable, the larvae

will come out with the stool to the soil. They transform into adults, which lay eggs, and hatching larvae get transformed to adults and so on. If the environmental conditions are not favorable, the larvae in the stool will moult and transform into infective filariform larvae, which pierce the intestine (auto-infection). Larvae penetrating the skin from the soil or by autoinfection are carried by the blood to the lungs, ascend to the trachea, descend to the esophagus and mature in the small intestine.

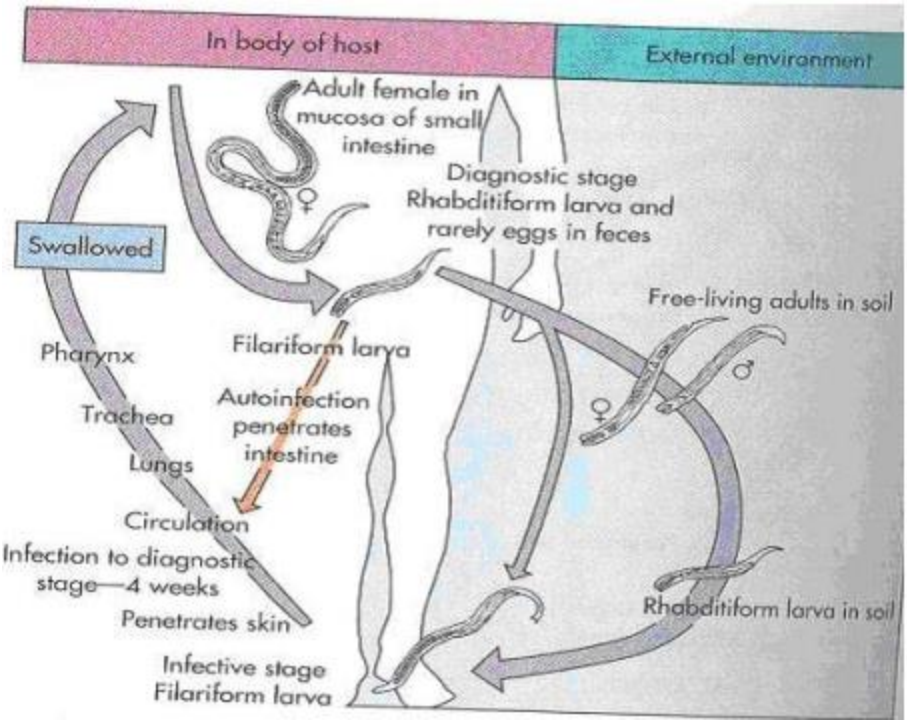


Figure 1.7. Life cycle of Strongyloides *stercoralis*

Clinical presentation

The patient complains of mucoid diarrhea. Larvae in the lungs may cause pneumonia.

Disseminated strongyloidiasis:

Multiplicity of symptoms are present due to the injury of other organs by the migrating larvae. Organs such as liver, heart adrenals, pancreas, kidneys, and CNS, etc. may be affected. This is usually seen in immunocompromized individuals.

Diagnosis - Detection of rhabditiform larvae of strongyloides in stool.

Figure 1.8. Larva of *S. stercoralis*

Treatment:

Thiabendazole: 25 mg/kg twice daily for 3 days.

INTESTINAL NEMATODES WITHOUT TISSUE STAGE

ENTEROBIUS VERMICULARIS (PIN WORM OR THREAD WORM)

Enterobius vermicularis is a small white worm with thread-like appearance. The worm causes enterobiasis. Infection is common in children.

Morphology

Male: The male measures 5 cm in length. The posterior end is curved and carries a single copulatory spicule.

Female: The female measures 13 cm in length. The posterior end is straight.

Infective stage: Infection is by ingestion of eggs containing larvae with contaminated raw vegetables.

Mode of infection

• By direct infection from a patient (Fecal-oral route).

• Autoinfection: the eggs are infective as soon as they are passed by the female worm. If the hands of the patient get contaminated with these eggs, he/she will infect him/herself again and again.

• Aerosol inhalation from contaminated sheets and dust.

Life cycle

Adult worm lives in the large intestine. After fertilization, the male dies and the female moves out through the anus to glue its eggs on the peri-anal skin. This takes place by night. The egg is

50x25 microns, plano-convex and contains larva. When the eggs are swallowed, they hatch in the small intestine and the larvae migrate to the large intestine to become adult.

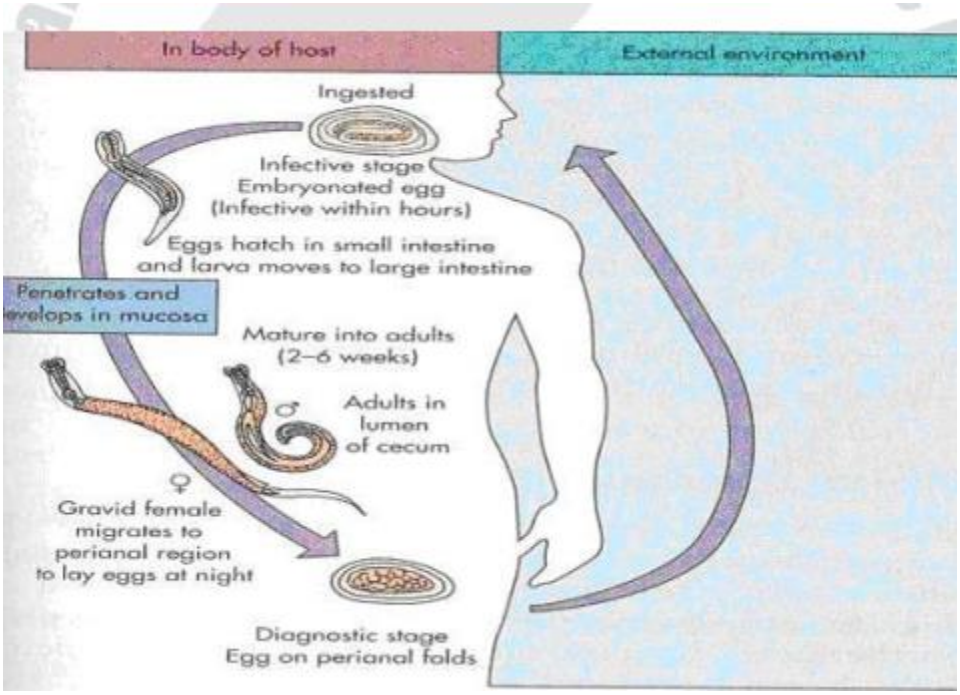


Figure 1.9. Life cycle of E. *vermicularis*

Clinical presentation

The migration of the worms causes allergic reactions around the anus and during night it causes nocturnal itching (pruritus ani) and enuresis. The worms may obstruct the appendix causing appendicitis.

Diagnosis

♦ Eggs in stool: Examination of the stool by direct saline smear to detect the egg: this is positive in about 5% of cases because the eggs are glued to the peri-anal skin.

♦ Peri-anal swab: The peri-anal region is swabbed with a piece of adhesive tape (cellotape) hold over a tongue depressor. The adhesive tape is placed on a glass slide and examined for eggs. The swab should be done in the early morning before bathing and defecation.

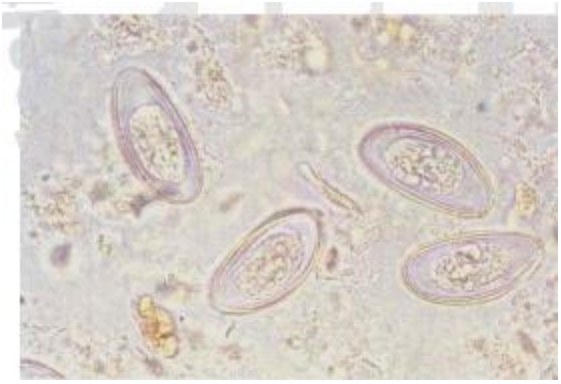


Figure 1.10. Egg of *E. vermicularis*

Treatment

Mebendazole; Piperazine.

TRICHURIS TRICHIURA (WHIP WORM)

The worm is divided into a thin whip-like anterior part measuring 3/5 of the worm and a thick fleshy posterior part of 2/5 the length.

Male: The male measures 3-4.5 cm in length. Its posterior end is coiled and possesses a single cubicle.

Female: The female measures 4-5 cm in length. Its posterior end is straight **Infective stage and mode of infection**

Infection is by ingestion of eggs containing larvae with contaminated raw vegetables.

Life cycle:

Ingested eggs hatch in the small intestine and the larvae migrate to the large intestine to become adult. After mating, the female lays immature eggs, which pass with the stool to the soil and mature in 2 weeks.

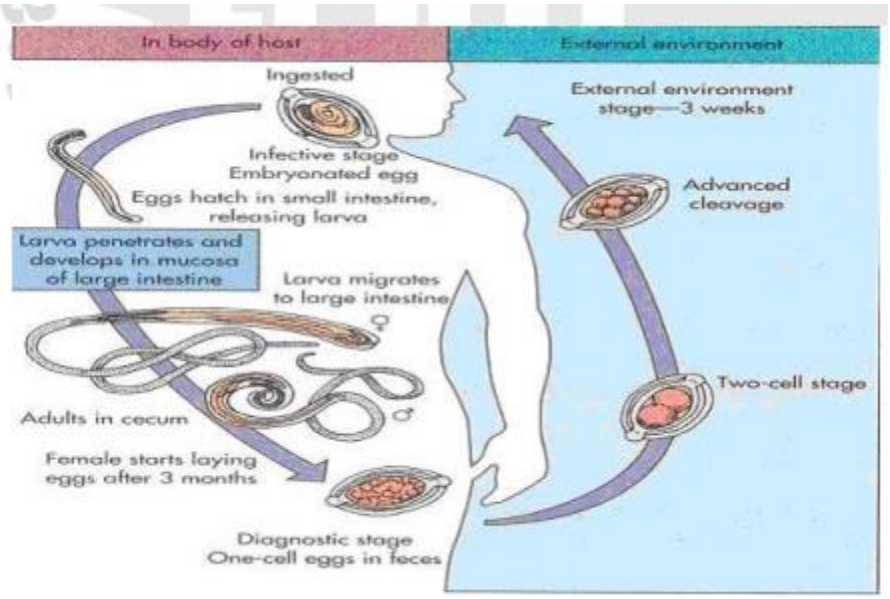


Figure 1.11. Life cycle of *Trichuris trichiura*

Symptoms

The patient complains of dysentery (blood and mucus in stool together with tenesmus). Rectal prolapse is also possible.

Diagnosis

Finding of characteristic eggs. The egg of trichuris is barrel-shaped, 50x25 microns. The shell is thick with a one mucoid plug at each pole.

Figure 1.12. Egg of *Trichuris trichiura*

Treatment

Mebendazole: 1 tablet twice daily for 2 days.

TISSUE NEMATODES

This group includes the filarial worms, the guinea worm (*Dranculuculus medinensis)* and *Trichinella spiralis.*

FILARIAL WORMS

The filarial worms have complex life cycles involving a developmental stage in an insect vector. They require an arthropod vector for their transmission. The worms inhabit either the lymphatic system or the subcutaneous tissues of man. The female worm gives rise to a young worm called microfilaria. The microfilariae, when taken by the arthropod intermediate host during biting, develop into filariform larvae, which are the infective stages. Humans get infected when bitten by the infected arthropod intermediate host.

Wuchereria bancrofti

This is a parasite of lymph nodes and lymphatic vessels- causing lymphatic filariasis. This filarial worm is transmitted by the bite of various species of mosquitoes. It is believed that over 100 million people are infected. The microfilariae are nocturnal – seen in greatest numbers in peripheral blood in the night between 10 PM -2 AM. The physiological basis of this nocturnal periodicity is not understood.

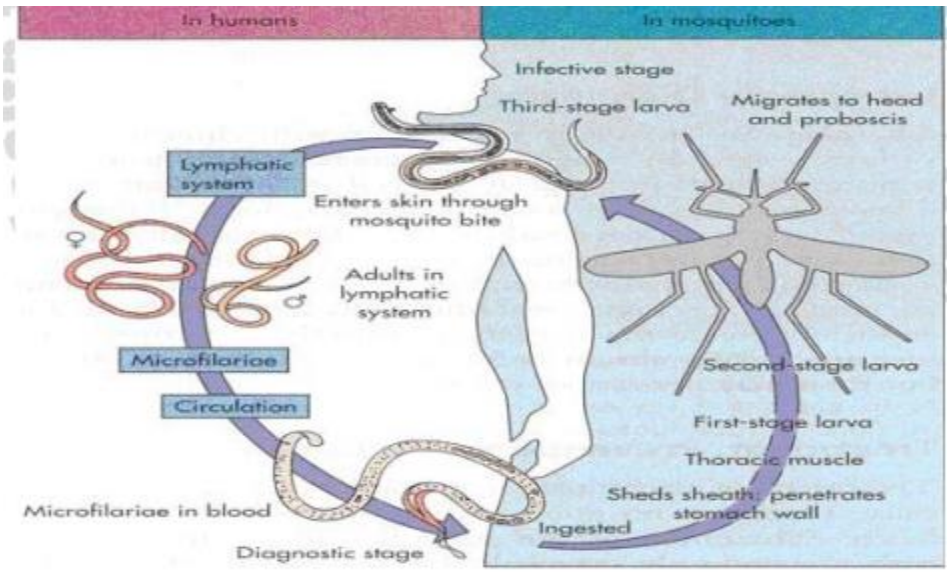


Figure1.13. Life cycle of *W. bancrofti*

Mode of transmission and pathogenesis

The filariform larvae are introduced through the skin by the bite of the arthropod intermediate host. The larvae invade the lymphatics, usually the lower limb, where they develop into adult worms. The microfilariae are librated into the blood stream. They remain in the pulmonary circulation during day, emerging into the peripheral circulation only during night, to coincide with the biting habit of the vector. Presence of the adult worms causes lymphatic blockage and gross lymphedema, which sometimes lead to elephantiasis.

Pathogenecity and clinical features:

♦ The adult worm obstructs the flow of lymph in the lymph nodes and the lymphatic vessels draining the lower limbs and the external genitalia.

♦ The lower limbs and external genitalia become swollen. The skin becomes thick and fissured. The disease is called bancroftian elephantiasis.

♦ The major symptoms and findings include: lymphangitis, lymphedema, fever, headache, myalgia, hydrocele and chyluria.

Diagnosis

♦ Blood film examination after staining by Giemsa or Leishman stain to detect microfilaria. The film should be taken by night.

Figure 1.14. Microfilaria of W. *bancrofti* in blood smear

Treatment - Diethyl carbamazine (DEC): 2 mg/kg 3x daily for 2 weeks.

Onchocerca volvulus

Infection by this filarial worm is common in Africa.

Morphology:

Male: Similar to that of *Wuchereria bancrofti*.

Female: The female measures 30-50 cm in length. It is present inside of a fibrous nodule (onchocercomata or onchocerca tumor).

Intermediate Host and vector

Female Simulium, (*Simulium damnosum*), Black fly, found around plantations following rivers or river basins.

Microfilaria

Measures 300 microns in length. It is non-sheathed microfilaria. It is present in the subcutaneous tissue fluids and not in blood.

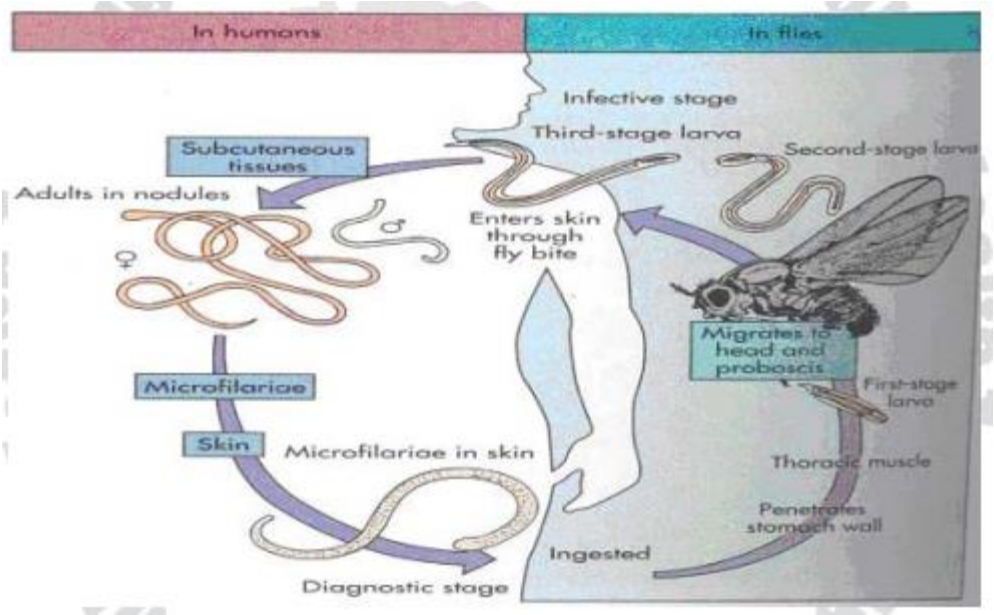


Figure 1.15. Life cycle of *O. volvulus*

Infective stage and mode of infection is similar to that of *Wuchereria bancrofti*.

Pathogenecity and clinical manifestations:

The disease, onchocerciasis or river blindness includes:

• Skin fibrous nodules (onchocercomata) enclosing female worms. The nodules are common in neck, iliac crest and the coccyx.

• Skin hypo- or hyper- pigmentation. Dermatitis is present. In advanced cases, the skin becomes thickened and wrinkled, showing lizard or leopard skin appearance.

• Elephantiasis of the external genitalia and corneal opacity and optic atrophy may finally cause blindness.

Diagnosis

Superficial biopsy (skin snip) is taken from the skin using sharp razor blade. The specimen is allowed to stand for 30 minutes in saline before it is examined microscopically for microfilariae.

Treatment

Ivermectin: 50 mg/kg bodyweight, given every 6 or 12 months. Because it kills microfilariae but not adult worms, retreatment is necessary over a period of years.

Prevention

• Vector control

• Mass treatment

• Establishment of villages away from Simulium breeding places.

• Use of repellents

• Protective clothing

Loa loa

The eye worm, *Loa loa,* causes Loiasis. The insect vectors include mango flies of Chrysops - *Chrysops silacea, Chrysops dimidiata*. Loiasis is endemic in Central and West Equatorial Africa. The abundant rubber plantations provide a favorable environment for the vector to transmit the disease.

Morphology

Adult male worms: 30-34 mm in length Adult female worms: 40-70 mm in length

Pathogenesis

The microfilaria have a sheath. Their diurnal periodicity corresponds to the feeding pattern of the insect vector, which bites humans from 10:00 AM to 4:00 PM.

Clinical Features

Incubation period is about one year. It causes calabar swelling beneath the skin due to parasites. There is fever, pain, pruritus, urticaria, allergic reactions, retinopathy, glomerulonephritis, meningo-encephalitis etc.

Laboratory diagnosis

• Detection of microfilaria in peripheral blood, urine, sputum, CSF - stained with Giemsa or unstained

• Eosinophilia

Treatment

DEC, 6 to 10 mg per kilogram per day for 2 to 3 weeks: but has side effects - allergic reactions

DRACUNCULUS MEDINENSIS (Guinea worm or Medina worm)

Dracunculus medinensis causes dracunculiasis. The infection is endemic to Asia and Africa: India, Nile Valley, central, western and equatorial Africa, lowlands of Ethiopia and Eritrea.

Morphology

Gravid female worms measure 70-120 cm in length. Their body cavity is almost fully occupied by a uterus greatly distended with rhabditiform larvae (250-750 μm in length). A digestive tube and cuticular annulations distinguish the larvae from microfilariae.

Pathogenecity and life cycle

Infection is acquired by drinking unfiltered or not boiled water that contains Cyclops species. The larvae are released in the stomach, penetrate the intestinal wall and find their way to the subcutaneous tissue. Mating takes place in the axillary or inguinal regions 3 months after infection. The male worms then die in the tissue and the female worms move down to the limbs within 10 months. In about 1 year, female worms in the subcutaneous tissue provoke the formation of a burning blister in the skin of the legs. When in water, the blister bursts, and about 5 cm of the worm is extruded from the resulting ulcer - thus releasing many thousands of first stage larvae. The larvae swim in water and are ingested by the intermediate host - Cyclops species- within about 4 days. Inside the Cyclops, the larvae molt twice and become infective in 2 weeks

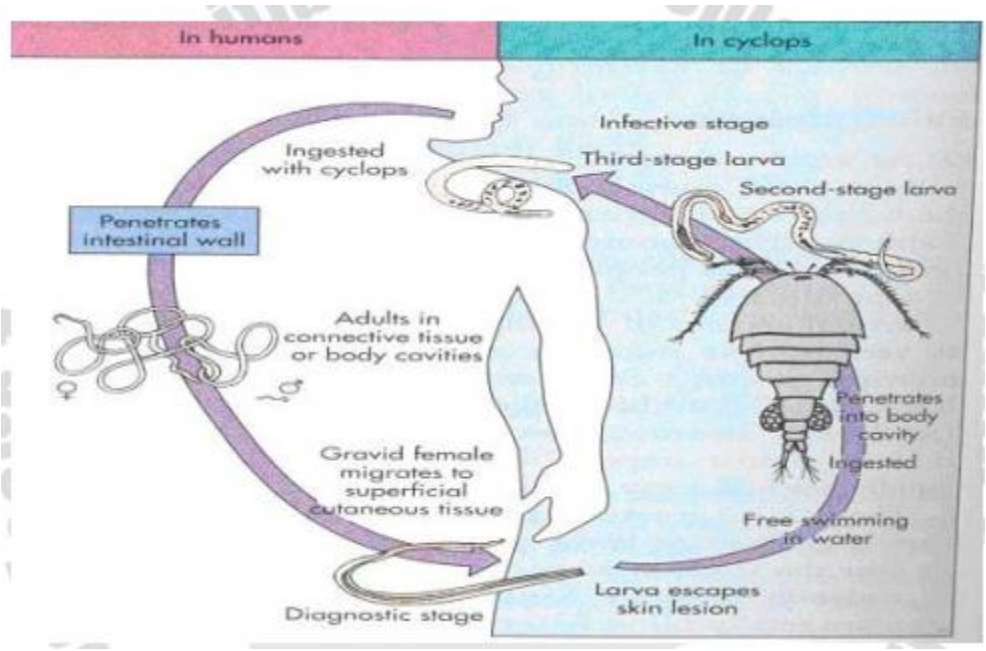


Figure 1.17. Life cycle of *Drancunculus medinesis*

Clinical feature

The female parasites in the subcutaneous tissue release toxic byproducts of histamine-like nature, which cause systemic allergic reactions, like erythema, urticaria, pruritus, fainting, asthma, dyspnea, etc. This is followed by the appearance of a blister on the legs, which ruptures on contact with water releasing larvae into the water by the female worm. The wound may ulcerate. The worms migrate into other tissues and may cause arthritis, pericarditis, abscesses etc. It occasionally penetrates the eyeball and causes loss of the eye.

Diagnosis

1. Clinical: Observation of blister, worm or larvae
2. Histologic features of subcutaneous sinus tract
3. Eosinophilia and radiographic evidence

Treatment

Surgical excision when the worm is in the leg Niridazole (Ambilhar) or DEC

Prevention

Health education on:

♦ Boiling or filtering of drinking water

♦ Treating of patients and educating them not to enter water bodies

♦ Using insect larvicides to kill Cyclops in water.

TRICHINOSIS

Etiologic agent - *Trichinella spiralis*

This is the only important species in this group. It causes trichinosis - a cosmopolitan infection. More than 100 different animal species can be infected with Trichinella species, but the major reservoir host for human infections is swine.

Morphology

Adult female worm measures 3-4 mm in length and the adult male worm measures 1.4-2.6 mm in length. The encysted larvae measure 800-1300 μm in length.

Pathogenecity and life cycle

After ingesting infected meat, the capsule of the encysted larvae is digested by gastric juice, and the larvae are released in the duodenum or jejunum where they molt four times to become adult worm. After mating, the male worm dies and the female worm begins to deliver the embryos 4-7 days after the infection. The larvae penetrate the intestinal wall and migrate through the lymphatic vessels to the blood stream, which carries them to various organs. Skeletal muscles and diaphragm are most frequently parasitized. Others include the tongue, masseter and ocular muscles.

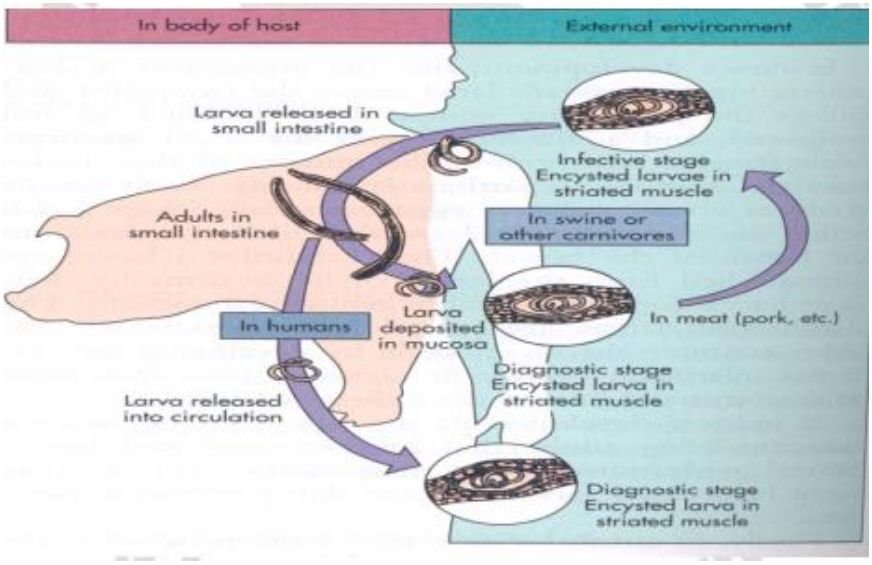


Figure 1.18 Life cycle of *Trichinella spiralis*

Clinical features

There are two clinical phases.

1. The intestinal phase: lasting 1-7 days - asymptomatic; sometimes cause nausea, vomiting, diarrhea, constipation, pain, etc, and

2. The muscle phase: which causes myalgia, palpabral edema, eosinophilia, fever, myocarditis, meningitis, bronchopneumonia etc.

Diagnosis:

♦ Muscle Biopsy

♦ Detection of larvae in blood or CSF

♦ Detection of larvae and adult worms in stool (rare).

♦ ELISA

Treatment - Thiabendazol

Prevention

♦ Cooking of all meat before consumption

♦ Inspection of pigs

♦ Pork must be stored at -150C for 20 days.

CESTODES (TAPEWORMS)

INTRODUCTION

The tapeworms are hermaphroditic and require an intermediate host. The adult tapeworms found in humans have flat body, white or grayish in color. They consist of an anterior attachment organ or scolex and a chain of segments (proglottids) also called strobilla. The strobilla is the entire body except the scolex. The scolex has suckers or grooves. It has rosetellum, which has 1 or 2 rows of hooks situated on the center of the scolex.

Adult tapeworms inhabit the small intestine, where they live attached to the mucosa. Tapeworms do not have a digestive system. Their food is absorbed from the host's intestine.

HYMENOLEPIS NANA (DWARF TAPEWORM)

Morphology

Adult worm measures 1-3 cm in length. It is made up of head (scolex), neck and segmented body. The head carries four suckers and a rostellum armed with one row of hooks. The segments of the body are divided into mature and gravid segments. In the mature segment, there are three testes in the middle.

Infective stage and mode of infection

The egg, which is immediately infective when passed by the patient, is rounded, about 40 microns in diameter. It contains a six- hooked oncosphere within a rigid membrane (the embryosphere). This embryosphere has two polar thickening or knobs from which project 4-8 long, thin filaments called polar filaments.

Infection takes place by:

1. Ingestion of egg with contaminated raw vegetables.
2. Direct infection from a patient
3. Auto infection: the eggs of H. nana are infective as soon as they are passed with feces by the patient. If the hands of the patient are contaminated by these eggs, she/he infects herself/himself again and again.

Pathogenecity

Light infections produce no symptoms. In fairly heavy infections, children may show lack of appetite, abdominal pain and diarrhea.

Treatment - Niclosamide: 4 tablets chewed in a single dose daily for 5 days.

HYMENOLEPIS DIMINUTA (RAT TAPEWORM)

Hymenolepis diminuta differs from *Hymenolepis nana* in that:

♦ The adult worm measures about 10-60 cm

♦ The rosetellum on the head has no hooks

♦ In the mature segment, there are two testes at one side and another testis on the other side.

Life cycle

The adult worms are present in the small intestine of man and rats. Eggs passed in stool are similar to the eggs of *H. nana* but are brown in color with no polar filaments arising from the polar thickening. The eggs are ingested by the rat flea where they develop to cysticercoid stage. Infection to man takes place accidentally by food or contaminated hands by cysticercoid stage.

Pathogenecity

Most infections are asymptomatic, but occasionally, patients may present with nausea, anorexia and diarrhea.

Treatment

same as *Hymenolepis nana*.

ECHINOCOCCUS

There are two different species. These are: *Echinococcus granulosus* and *Echinococcus multilocularis*

Echinococcus granulosus (dog tape worm)

Responsible for most cases of echinococcosis. Echinococcosis is caused by larval tapeworms. The disease is common in East Africa (the highest prevalence is seen in Kenya: 10-15%).

Morphology

The adult worm measures 3-6 mm in length (up to 1 cm). It has scolex, neck and strobilla. Adult worms live in small intestine of definitive host (dog). Man is an intermediate host - carrying the hydatid cyst (larva). Man contracts infection by swallowing eggs in excreta of definitive host.

Life cycle and Pathogenecity

Oncosphere hatch in duodenum or small intestine into embryos (oncosphere) which:

♦ Penetrate wall

♦ Enter portal veins

♦ Migrate via portal blood supply to organs: eg: lungs, liver, brain etc., thus, causing extra intestinal infections. In these organs, larvae develop into hydatid cysts. The cysts may be large, filled with clear fluid and contain characteristic protoscolices (immature forms of the head of the parasite).

These mature into developed scolices, which are infective for dogs.

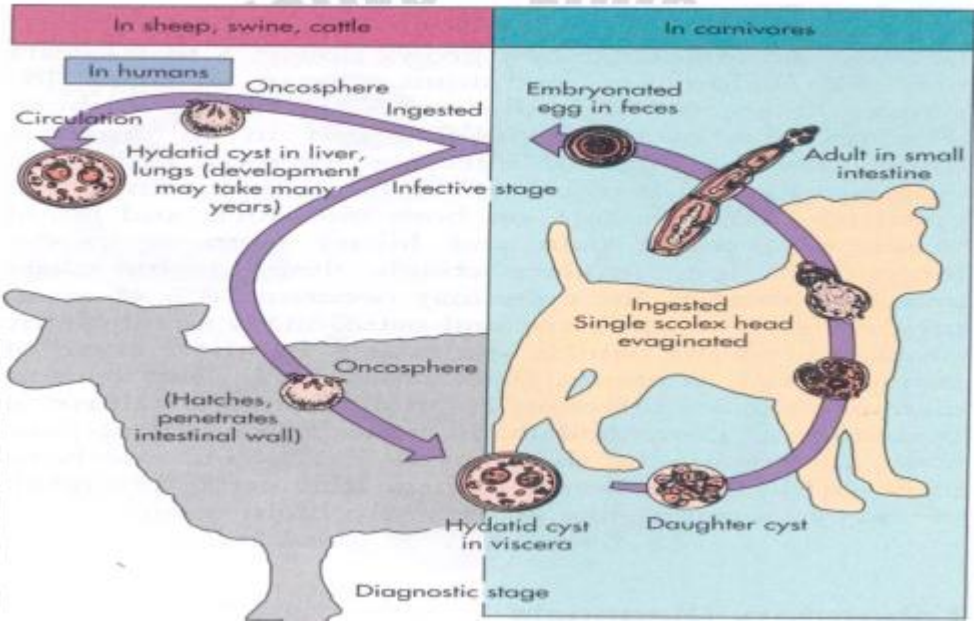


Figure 1.19. Life cycle of *Echinococcus granulosus*

Mode of human infection

Ingestion of eggs by the following ways:

i) Ingestion of water or vegetables polluted by infected dog feces.

ii) Handling or caressing infected dogs where the hairs are usually contaminated with eggs.

Clinical features

Asymptomatic infection is common, but in symptomatic patients

♦ It may cause cough - with hemoptysis in lung hydatid disease.

♦ Hepatomegaly - with abdominal pain and discomfort

♦ Pressure -from expanding cyst

♦ Rupture of cyst - severe allergic reaction - anaphylaxis.

Diagnosis:

♦ X-ray or other body scans

♦ Demonstration of protoscolices in cyst after operation

♦ Serology

Treatment

♦ Surgery

♦ Albendazole 400 mg twice a day for one to eight periods of 28 days each, separated by drug-free rest intervals of 14 to 28 days.

Echinococcus multilocularis

Foxes are the definitive hosts, while various rodents such as mice serve as intermediate hosts.

TAENIA SAGINATA (BEEF TAPEWORM)

In adult stage, *T. saginata* inhabits the upper jejunum where it may survive for as long as 25 years. It causes intestinal infection, Taeniasis. It has worldwide distribution.

These are one of the true and segmented tapeworms. Their body is divided into three regions;

1. Scolex: the hold fast organ
2. Neck: posterior to the scolex
3. Stobilla: the main bulk, made up of proglottids.

Morphology:

Adult worm measures 5-10 meters in length. The pyriform scolex has 4 suckers but no rostellum. The mature segments have irregularly alternate lateral genital pores. Each of the terminal segments contains only a uterus made up of a median stem with 15-30 lateral branches.

Life cycle

The adult worm lives in the small intestine of man. Gravid segments pass out in the stool and become disintegrated and eggs come out to the soil. The gravid proglottid uterus contains about 100,000 eggs. The egg of *T. saginata* is round, about 40 microns in diameter. The 6-hooked embryo is enclosed in a radially striated embryophore. Eggs are ingested by an intermediate host, cattle. The 6- hooked embryo escapes from its shell, penetrates through the intestinal wall into the blood vessels and is carried to the muscles where it develops into a larval stage, **cysticercus bovis** (made up of an invaginated /inverted head and spherical body). Infection to man takes

place by the ingestion of raw or insufficiently cooked beef. In the small intestine of man, the head of the cysticercus gets invaginated and the body becomes segmented.

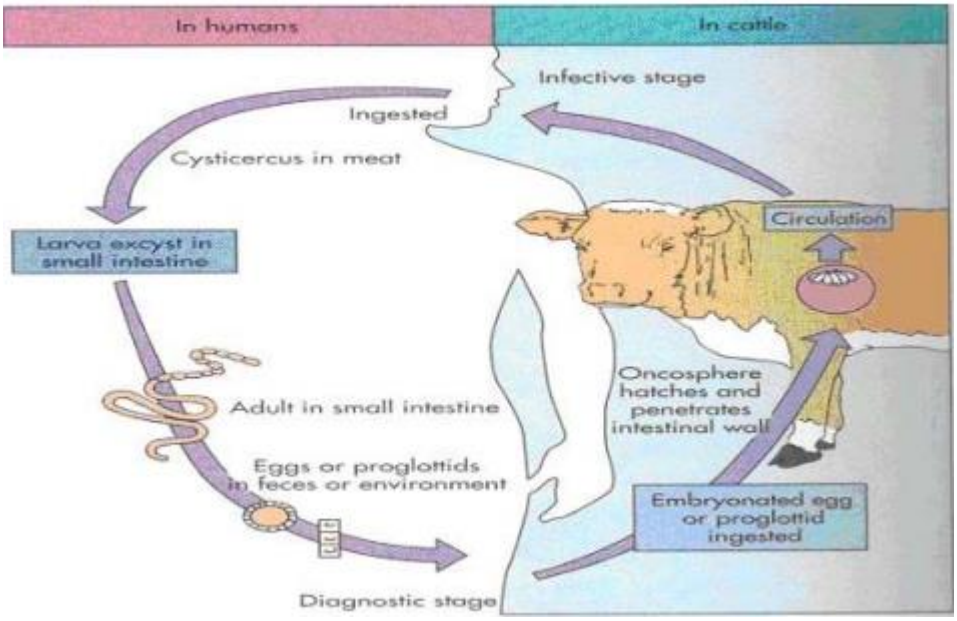


Fig 1.20. Life cycle of *Taenia saginata*

Pathogenecity

Infected persons may complain of epigastric pain, abdominal discomfort, diarrhea, weight loss, hunger sensation, vomiting, etc.

Diagnosis

Recovery of the gravid segments or the eggs from the stool

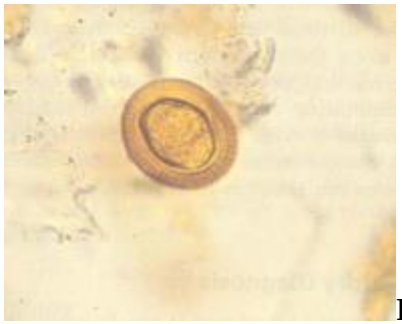


Figure1.21. Egg of *Taenia* Spp

Treatment:

Niclosamide: Four tablets chewed in a single dose. Mebendazole 100mg twice daily for three days

Prevention:

♦ Thorough cooking of meat (above 570C). ♦ Proper disposal of human excreta

TAENIA SOLIUM (PORK TAPEWORM)

The adult worms of *T. solium* reside or inhabit the upper jejunum. Infection has worldwide distribution.

Morphology:

Adult worm measures about 3 meters in length. The globular scolex has rostellum with 2 rows of hooklets. There are <1000 proglottids.

Gravid proglottid liberates about 30,000-50,000 eggs.

Life cycle

Embryonated eggs passed with stool are ingested by pig and the embryo is released. It penetrates the intestinal wall and is carried by vascular channels to all parts of the body. After a period of 2-3 months of development the encysted larval stage called cysticerci or bladder worm occurs in the striated muscles of the tongue, neck, trunk brain, eye, and the nervous system. The cysticercus survives for 5 years. Humans become infected by eating pork containing larvae, *cysticercus cellulosae*. When improperly cooked cysticercus infected meat is eaten by man, the scolex remains undigested and attaches itself to the intestinal wall and chain of proglottids begin to grow to adult worm.

Clinical manifestations

Resembles that of *T. saginata* infection

Diagnosis

Demonstration of eggs in stool specimen

Treatment

Niclosamide: 2 gm PO stat

Prevention:

♦ Treatment of infected persons.

♦ Thorough cooking of pork and proper processing

♦ Proper disposal of human excreta (good hygiene/sanitation).

Table 1: Comparison between *Taenia saginata* and *Taenia solium* species

Taenia saginata	*Taenia solium*	
Length (m)	5-10	2-3
Proglottid number	1000-2000	800-900
Hooklets	Absent	Present
Suckers	Pigmented	Non- Pigmented
Uterus branch	15-30	5-10

DIPHYLOBOTRIUM LATUM (FISH TAPEWORM OR BROAD TAPEWORM

The broad tapeworm infecting man has worldwide distribution, occurring in areas where improperly cooked or raw fresh water fish is prominent in diet.

Morphology

Diphylobotrium latum is the broadest and longest tapeworm. The adult worm measures up to 30 feet with 3000-4000 proglottids, which are wider than they are long. The tapeworm has no rostellum hooks or suckers.

Life cycle

Unlike Taenia, the gravid segments are retained by the worm. Operculated eggs passed in feces hatch into small ciliated coracidium larvae which swim about freely. These are eaten by crustaceans -Cyclops or Diaptomus - in which the larvae develop into second stage larvae- the procercoid. When the crustaceans are swallowed by fresh water fish, the larvae migrate into the flesh of the muscle fish and develop to pleurocercoid or sparganum larvae. Humans are infected by ingesting raw or improperly cooked fish. The tapeworm matures in the intestine and after 3 weeks, the adult worm discharges eggs. The life cycle requires two intermediate hosts.

Clinical manifestation

Most infections are asymptomatic. Rarely, it causes severe cramping, abdominal pain, vomiting, weakness and weight loss. Pernicious anemia can also result, due to interference of vitamin B12 absorption in jejunum.

Diagnosis

Eggs in stool: Single shell with operculum at one end and a knob on the other.

Treatment

Niclosamide: 2 gm PO stat after light breakfast.

Prevention:

Prohibiting the disposal of untreated sewage into fresh water /lakes. Personal protection: cooking of all fresh water fish.