Medical Parasitology

Second class

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Introduction *:*

Medical Parasitology is the study of parasites and as such that does not include bacterial, fungal or viral parasites. Human parasites are separated into intestinal and blood borne parasites. For a parasite to be defined as intestinal it must have an intestinal life cycle stage, though it may have lifecycle stages in the heart, blood vessels, and lungs in the humans, other animals or the environment.

The association between two organisms may be one of the following:

Mutualism: mutual benefit is derived from the association.

Symbiosis: mutual benefit, but the two organisms cannot live independently.

Commensalism: one partner benefits (commensal) while the other (host) is unaffected. It may be called a non-pathogenic parasite.

When an animal lives on another organism from which it receives food and shelter without any compensation to it, and then this association is called **parasitism**. The animal, which enjoys advantages, is the **parasite.** All animals have parasites; hence there are more parasites than free-living animals. The habitat occupied by a parasite is very different from the environment of its free-living ancestors, hence it has either to adapt itself to this new habitat or perish.

Parasitism: one organism (parasite) lives at the expense of the other (host). The latter usually suffers from the association with pathogenic parasite).

Parasitism is the form of mutual relations between organisms of various kinds, from which one (parasite) uses another (host) as environment for living, and from which it obtains food causing him damage (disease).

Classification of Parasites

Each parasite belongs to a phylum, class, order, family, genus and species; the scientific designation of a parasite is binomial, a generic name (genus) and a specific name (species).

The parasites of humans in the phylum **protozoa** are now classified under three subphyla: **Sarcomastigophora** (containing the amoebae and flagellates); **Apicomplexa** (containing the sporozoan); and **Ciliophora** (containing the ciliates). The important human parasites are found within these great groups.

1. **Subphylum (Sarcodina)** is typically amoeboid and in represented in humans by class of ***Entamoeba, Endolimax, lodamoeba, Naegleria,* and *Acanthamoeba***.
2. **Subphylum Zoomastigophora,** the flagellates, have one or more whiplike flagella and, in some cases, an undulating membrane (e.g., trypanosomes). These include intestinal and genitourinary flagellates ***(Giardia, Trichomonas, Dientamoeba, Chilomastix)*** and blood tissue flagellates ***(Trypanosoma, Leishmania)***.

3. **Subphylum Sporozoa** undergoes a complex life cycle with alternating sexual and asexual reproductive phases, usually involving two different hosts (e.g., arthropod and vertebrate, as in the blood forms). The subclass **Coccidia** contains the human parasites ***Isospora, Toxoplasma,*** and others. One of these, ***Cryptosporidium,*** has been implicated as a cause of intractable diarrhea among the immunosuppressed. Among the **Haemosporina** (blood sporozoan) are the malaria parasite ***(Plasmodium* species)** and the subclass ***Piroplasmia,*** which includes ***Babesia*** species. ***Pneumocystis*** has recently been shown to be a member of the Fungi rather than the Protozoa. It is another opportunistic parasite of immunosuppressed individuals.
4. **Subphylum Ciliata** is a complex protozoan bearing cilia distributed in rows or patches, with two kinds of nuclei in each individual. Balantidium coli, a giant intestinal ciliate of humans and pigs, is the only human parasite representative of this group.

The Parasitic Worms, or helminths, of a human being, belong to two Subphyla:

1. **Subphylum Platyhelminths** (flatworms) lack a true body cavity (celom) and are characteristically flat in dorsoventral section. Medically important species belong to the classes **Cestoda** (tapeworms) and **Trematoda** (flukes). The tapeworms of humans are band-like and segmented; the flukes are typically leaf-shaped, and the schistosomes are narrow and elongate. The important tissue and intestinal cestodes of humans belong to the genera ***Diphyllobothrium, Spirometra, Taenia, Echinococcus, Hymenolepis,*** and ***Dipylidium***. Medically important trematode genera

include ***Schistosoma, Paragonimus, Clonorchis, Opistorchis, Heterophyes, Metagonimus, Fusciolopsis,*** and ***Fasciola.***

2. **Subphylum Nemathelminths** (worm-like, separate-sexed, insegmented roundworms) include many parasitic species that infect humans.

Phylum Protozoa

General Features

The single protozoal cell performs all functions. Most of the protozoa are completely nonpathogenic but few may cause major diseases such as malaria, leishmaniasis, and sleeping sickness. Protozoa like *Cryptosporidium parvum* and *Toxoplasma gondii* are being recognized as opportunistic pathogens in patients affected with human immunodeficiency virus (HIV) and in those undergoing immunosuppressive therapy. Protozoa exhibit a wide range of a size (1- 150 μm), shape, and structure; yet all possess essential common features.

Single-celled microorganisms belonging to the animal kingdom are classified as Protozoa (Greek Protos—first; zoon—animal). Within its single cell, the protozoon contains all structures required for performing its various functions. Some free-living protozoa resemble plants containing green plastids that enable them to perform photosynthesis. It is believed that these

represent the earliest forms of animal life. Numerous varieties of protozoa have evolved to suit all manner of environmental conditions.

Free-living protozoa are found in all habitats—in the deep ocean or in shallow freshwaters, in hot springs or in ice, under the soil, or in the snow on mountain tops. Parasitic protozoa have however adapted to different host species, with more restricted physicochemical requirements.

Protozoa exhibit a wide range of size, shape, and structure, yet all possess certain essential common features. The typical protozoan cell is bounded by a trilaminar unit membrane, supported by a sheet of contractile fibrils that enable the cell to change its shape and to move. The cytoplasm can often be differentiated into an outer rim of relatively homogeneous ectoplasm and a more granular inner endoplasm.

The ectoplasm serves as the organ of locomotion and for engulfment of food materials by putting forth pseudopodial processes. It also functions in respiration, discharging waste materials, and also as a protective covering for the cell. Within the endoplasm is the nucleus within a tough nuclear membrane. The nucleus is usually single but maybe double or multiple, some species having as many as a hundred nuclei in one cell. The nucleus contains one or more nucleoli or a central endosome or karyosome. The chromatin may be distributed along the inner surface of the nuclear membrane (peripheral chromatin) or as condensed masses around the karyosome. The endoplasm shows a number of structures-the endoplasmic reticulum, mitochondria, and Golgi bodies. Contractile vacuoles may be present which serve to regulate the osmotic pressure. Several food vacuoles also may be seen.

The active feeding and growing stage of the protozoa are called the trophozoite (G.trophos-nourishment). The cell may obtain nourishment from

the environment by diffusion or by active transport across the plasma membrane. Larger food particles are taken in by phagocytosis through pseudopodia. Some species ingest food through

Protozoa: General Features of special mouth-like structures or cytostomes. Minute droplets of food may also enter by pinocytosis. Several species possess a resting or resistant cystic stage which enables prolonged survival under unfavorable conditions. The cystic stage may also involve reproduction by the nucleus dividing once or more to give rise to daughter trophozoites on excystation. The cyst is usually the infective stage for the vertebrate host.

Reproduction is usually asexual. The most common method is binary fission by the mitotic division of the nucleus, followed by the division of the cytoplasm. In amoebae, division occurs along any plane, but in flagellates, the division is along the longitudinal axis and in ciliates in the transverse plane. Some protozoa, as for instance the malaria parasites exhibit schizogony in which the nucleus undergoes several successive divisions within the schizont to produce a large number of merozoites. Sexual stages are seen in ciliates and Sporozoa. In ciliates, the sexual process is conjugation in which two organisms join together and reciprocally exchange nuclear material. In Sporozoa, male and female gametocytes are produced, which after fertilization form the zygote giving rise to numerous sporozoites by sporogony.

SubPhylum Sarcomastigophora

Simple protozoa that have no fixed shape. They are classified under the Phylum-. The cytoplasm is bounded by a unit membrane and can be differentiated into an outer ectoplasm and an inner endoplasm. Pseudopodia

are formed by the ectoplasm thrusting out, being followed by the endoplasm flowing in, to produce blunt projections. Pseudopodial processes appear and disappear, producing quick changes in the shape of the cell. These are employed for locomotion and engulfment of food by phagocytosis. Amoebae may be free-living or parasitic. A few of the free-living amoebae can, on occasion act as human pathogens, producing meningoencephalitis and other infections. Some of them can act as carriers of pathogenic bacteria. The parasitic amoebae inhabit the alimentary canal.

Parasitic Amoebae

Parasitic amoebae belong to the following genera: Genus Species:

1. Entamoeba:

a) *E.histolytica.*

b) *Entamoeba dispar.*

c) *E.coli.*

d) *E.polecki.*

e) *E.hartmanni.*

f) *Entamoeba gingivalis.*

2. Endolimax.

a) E.nana.

3. Iodamoeba *I.butschlii.*

4. Dientamoeba.

a) *D.fragilis*.

Entamoeba histolytica

Is an important human pathogen, causing amoebic dysentery as well as hepatic amoebiasis and other extraintestinal lesions. *E.hartmanni* is

nonpathogenic, though it resembles *E. histolytica* very closely except for its smaller size and was therefore known as the 'small race' of *E. histolytica*. *E.polecki* a natural parasite of pigs and monkeys may sometimes infect humans causing diarrhoea. *E. coli* is a common commensal in the colon and its importance is that it may be mistaken for *E.histolytica*. *E.gingivalis* is present in the mouth, being found in large numbers when the oral hygiene is poor. It has no cystic stage and so the trophozoites depend for transmission on direct oral contact as in kissing, air-borne spread through salivary droplets and fomites such as shared drinking and eating utensils.

It is generally nonpathogenic, though it has been claimed that it contributes to periodontal disease. All the genera of intestinal amoebae other than Entamoeba are nonpathogenic commensals, except D.fragilis, which may occasionally cause chronic, but mild intestinal symptoms. Intestinal amoebae can be differentiated based on their morphological features.

History

History *Entamoeba histolytica* was discovered in 1875 by Losch in the dysenteric feces of a patient in St Petersburg, Russia. He also observed it in colonic ulcers at autopsy and produced dysentery in a dog by inoculation through the rectum. In 1890, William OsIer reported the case of a young man with dysentery who later died of liver abscess. Councilman and Lafleur in 1891 established the pathogenesis of intestinal and hepatic amoebiasis and introduced the terms 'amoebic dysentery' and 'amoebic liver abscess.

Geographical Distribution *E. histolytica* is world-wide in prevalence. It is much more common in the tropics than elsewhere, but it has been found wherever sanitation is poor, in all climatic zones, from Alaska (61° N) to the

Straits of Magellan (52°S). It has been reported that about 10 per cent of the world's population and 50 per cent of the inhabitants of some developing countries may be infected with the parasite. The infection is not uncommon even in affluent countries, about 1 per cent of Americans being reported to be infected. While the large majority of the infected are asymptomatic, invasive amoebiasis causes disabling illness in an estimated 50 million persons and death in 50,000 annually, mostly in the tropical belt of Asia. Africa and Latin America. It is the third leading parasitic cause of mortality, after malaria and schistosomiasis. *E. histolytica* is found in the human colon.

Natural infection also occurs in monkeys, dogs and probably in pigs also but these animals do not appear to be relevant as sources of human infection. Infection is mostly asymptomatic. It commonly occurs in the lumen of the colon as a commensal, but sometimes invades the intestinal tissues to become a pathogen.

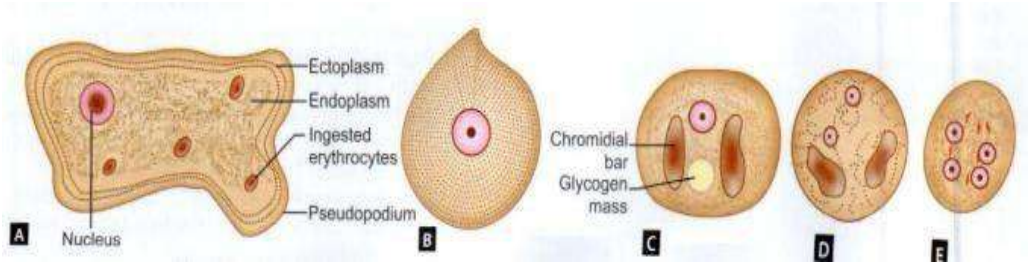
Morphology

***E. histolytica* occurs in three forms:**

a. Trophozoite.

b. Precystic.

c. Cystic stages.



(Figs. 1 A to E) *Entamoeba histolytica*: (A) Trophozoite; (B) Precystic stage; (C) Uninucleate. (D) Binucleate cyst; and (E) Mature quadrinucleate cyst.

Trophozoite

Trophozoite is the vegetative or growing stage of the parasite. It is the only form present in tissues. It is irregular in shape and varies in size from 12-60 μm; average being 20 μrn. It is large and actively motile in freshlypassed dysenteric stool, while smaller in convalescents and carriers.The parasite, as it occurs free in the lumen as a commensal is generally smaller in size, about 15-20 μm and has been called the ***minuta form*.**

Cytoplasm: Outer ectoplasm is clear, transparent and refractile. Inner endoplasm is finely granular, having a ground glass appearance. The endoplasm contains nucleus, food vacuoles, erythrocytes, occasionally leukocytes and tissue debris.

Pseudopodia are finger-like projections formed by sudden jerky movements of ectoplasm in one direction, followed by the streaming in of the whole endoplasm.

Typical ameboid motility is a **crawling** or **gliding movement** and not a free swimming one. The direction of movement may be changed suddenly, with another pseudopodium being formed at a different site, when the whole cytoplasm flows in the direction of the new pseudopodium. The cell has to be attached to some surface or particle for it to move. In culture tubes, the trophozoites may be seen crawling up the side of the glass tube.

Pseudopod formation and motility are inhibited at low temperatures.

Nucleus is spherical 4- 6 μm in size and contains **central karyosome**, surrounded by clear halo and anchored to the nuclear membrane by fine radiating fibrils called the Linin network, giving a **cartwheel appearance**. The nucleus is not clearly seen in the living trophozoites, but can be clearly

demonstrated in preparations stained with iron hematoxylin. Nuclear membrane is lined by a rim of chromatin distributed evenly as small granules. The trophozoites from acute dysenteric stools often contain phagocytosed erythrocytes. This feature is diagnostic as phagocytosed red cells are not found in any other commensaJ intestinal amebae. The trophozoites divide by **binary fission** in every 8 hours.Trophozoiles survive up to 5 hours at 37°C and are killed by drying, heat and chemical sterilization. Therefore, the infection is not transmitted by trophozoites. Even if live trophozoites from freshly-passed stools are ingested, they are rapidly destroyed in stomach and cannot initiate infection.

Precystic Stage

Trophozoites undergo encystment in the intestinal lumen. Encystment does not occur in the tissues nor in feces outside the body. Before encystment, the trophozoite extrudes its food vacuoles and becomes round or oval, about 10- 20 μmin size. This is the precystic stage of the parasite. It contains a **large glycogen vacuole** and two **chromatid bars**. It then secretes a highly retractile cyst wall around it and becomes cyst.

Cystic Stage

The cyst is spherical in shape about 10-20 μmin size. The early cyst contains a single nucleus and two other structures a mass of glycogen, and 1-4 chromatoid bodies or chromatoid bars, which are cigar-shaped. The chromatoid bodies are so-called because they stain with hematoxylin, like chromatin. As the cyst matures, the glycogen mass and chromidial bars disappear and the nucleus undergoes two successive mitotic divisions to form

two and then four nuclei. 1he mature cyst is, thus **quadrinucleate**. The cyst wall is a highly refractile membrane, which makes it highly resistant to gastric juice and unfavorable environmental conditions.The nuclei and chromidial bodies can be made out in unstained films, but they appear more prominently in stained preparations. With iron hematoxylin stain, nuclear chromatin and chromaroid bodies appear **deep blue or black**, while the glycogen mass appears unstained. When stained with iodine, the glycogen mass appears **golden brown**, the nuclear chromatin and karyosome bright yellow, and the chromatoid bodies appear as clear space, being unstained.

Life Cycle

Entamoeba histolytica passes its life cycle only in one host (man).

Cysts and trophozoites are typically found in diarrheal stool. Infection by *Entamoeba histolytica* occurs by ingestion of mature cysts in fecally contaminated food, and water. Excitation occurs in the small intestine and trophozoites are released, which migrate to the large intestine. The trophozoites multiply by binary fission and produce cysts, and both stages are passed in the feces. Because of the protection conferred by their walls, the cysts can survive days to weeks in the external environment and are responsible for transmission. Trophozoites passed in the stool are rapidly destroyed once outside the body, and if ingested would not survive exposure to the gastric environment. The cysts passing in stool of infected individuals. In some patients, the trophozoites invade the intestinal mucosa or, through the bloodstream, extraintestinal sites such as the liver, brain, and lungs, with resultant pathologic manifestations.

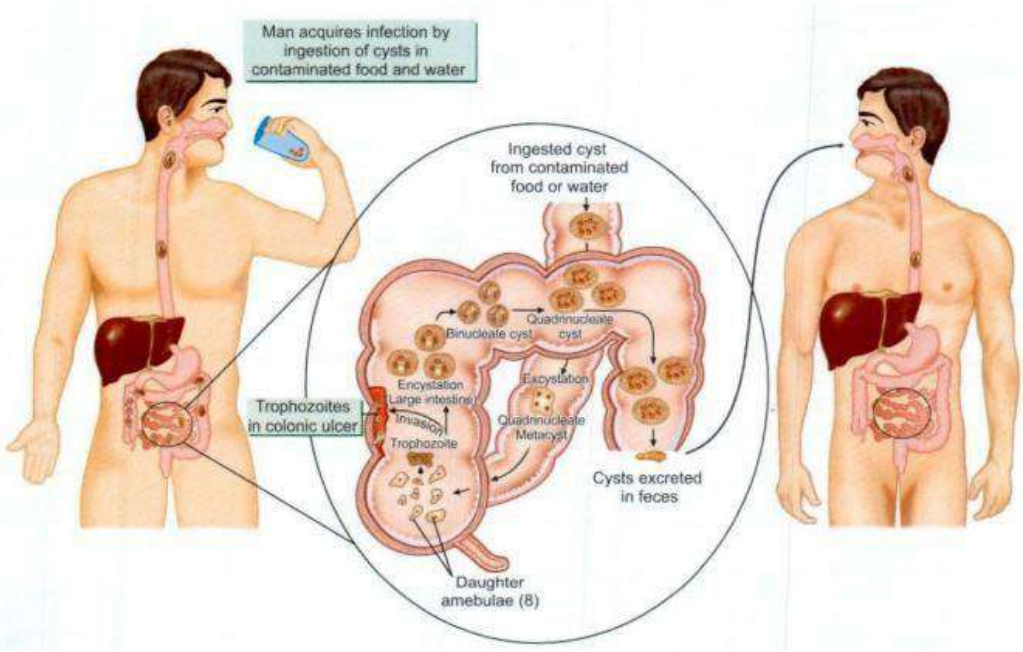


Figure (2): Life cycle of *E. histolytica*.

Infective Form

Mature quadrinucleate cyst passed in feces of convalescents and carriers. The cysts can remain viable under moist conditions for about I0 days.

Mode of Transmission

Man acquires infection by swallowing food and water contaminated with cysts.

Excystation: Is hapened when the cyst reaches cecum or lower part of the ileum, due to the alkaline medium, the cyst wall is damaged by trypsin. **Metacyst** mean the cytoplasm gets detached from the cyst wall and ameboid movements appear causing a tear in the cyst wall, through which quadrinucleate ameba is liberated.

Metacystic Trophozoites mean the nuclei in the metacyst immediately undergo division to form eight nuclei, each of which gets surrounded by its own cytoplasm to become eight small amebulae or metacystic trophozoites.

Pathogenesis and Clinical Features

E. histolytica causes intestinal and extraintestinal amebiasis. Incubation period is highly variable. On an average, it ranges from 4 days to 4 months. Amebiasis can present in different forms and degree of everity, depending on the organ affected and the extent of damage caused.

Intestinal Amebiasis

The lumen-dwelling amebae do not cause any illness. They cause disease only when they invade the intestinal tissues. This happens only in about 10% of cases of infection, the remaining 90% being

Asymptomatic.

Not all strains of *E. hislolylica* are pathogenic or invasive. Differentiation between pathogenic and nonpathogenic strains can be made by susceptibility to complementmediated lysis and phagocytic activity or by the use of genetic markers or monoclonal antibodies and zymodeme analysis.

Amebic ulcer

Is the typical lesion seen in intestinal amebiasis.The ulcers are multiple and are confined to the colon, being most numerous in the cecum and next in the sigmoidorectal region. The intervening mucous membrane between the ulcers remains healthy. Ulcers appear initially on the mucosa as raised nodules with pouting edges measuring pinhead to l inch. They later break down discharging brownish necrotic material containing large numbers of trophozoites.

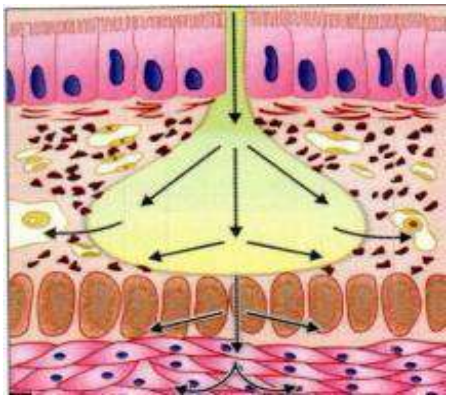


Figure (3): Flask-shaped amebic ulcer

The typical amebic ulcer is **flask-shaped** in cross section, with mouth and neck being narrow and base large and rounded. Multiple ulcers may coalesce to form large necrotic lesions with **ragged and undermined edges** and are covered with brownish slough.

Ameboma

Occasionally, a granulomatous pseudotumoral growth may develop on the intestinal wall by rapid invasion from a chronic ulcer. This amebic granuloma or ameboma may be mistaken for are malignant tumor.

Amebomas are most frequent at cecum and rectosigmoid junction.

Laboratory diagnosis of *Entamoeba histolytica*

Stool examination: Bloody and\or mucoid stool sample to see trophozoite and cyst stages by normal saline and Lugol's iodine direct smear respectively.

The sample (stool) should be collected into a wide mouth container and examined without delay, and should be inspected via: Macroscopy and Microscopy.

Macroscopy: The stool is characterized by:

Foul-smelling, copious, semiliquid, brownish -black in color, intermingled with blood and mucus, and it does not adhere to the container.

Microscopy

Saline preparation:

- The cellular exudate is scanty and consists of only the nuclear masses (pyknotic bodies) of a few pus cells, epithelial cells and macrophages.
- The RBCs are in clumps and yellow or brown -red in color.
- Charcot-Leyden crystals are often present. These are diamond-shaped, clear and refractile crystals.
- Actively motile trophozoites throwing pseudopodia can be demonstrated in freshly-passed stool. Presence of ingested RBCs clinches the identity of *E. hislolytica*. Nucleus is not visible but a faint outline may be detected.
- Cyst has a smooth and thin cell wall and contains round refractile chromatoid bars. Glycogen mass is not visible.

Iodine preparation: For the demonstration of cysts or dead trophozoites, stained preparations may be required for the study of the nuclear character. Iodine-stained preparation is commonly employed for this purpose. The trophozoite of *E. histolytica* stains yellow to light brown. The nucleus is clearly visible witl1 a central karyosome. The cytoplasm of the cystic stage shows a smooth and hyaline appearance. Nuclear chromatin and karyosome appear bright yellow. Glycogen masses stain golden brown and chromatoid bars are not stained.

The trichrome stain is useful to demonstrate intracellular features of both trophozoites and cysts. Since the excretion of cysts in the stool is often imminent, at least three consecutive samples should be examined.

Mucosal scrapings: Scraping obtained by sigmoidoscopy is often contributory. The examination method includes a direct wet mount and iron

hematoxylin and immunofluorescent staining with anti-*E.hislolytica* antibodies.

Stool culture: Stool culture is a more sensitive method in diagnosing chronic and asymptomatic intestinal amebiasis. The culture of stools gives higher positivity for *E. histolytica* as compared to direct examination.

Media used for stool culture include to diagnosis the *E. histolytica*: Boeck and Drbohlav's biphasic medium. NIH polygenic medium, Craig's medium, Nelson's medium, Robinson's medium, and Balamuth's medium.

Serodiagnosis: Serological tests become positive only in invasive amebiasis.

Antibody detection: Amebic antibodies appear in serum only in the late stages of intestinal amebiasis. Tests for antibodies in scrum help in the diagnosis of mainly extraintestinal infections. Serological tests include indirect hemagglutination assay (IHA), indirect fluorescent antibody (IFA), enzyme-linked immunosorbent assay (ELISA), counter-current immunoelectrophoresis (CIEP), and latex agglutination tests.

Serum with an antibody titer of 1:256 or more by IHA and 1:200 by IFA are considered to be significant.

Amebic antigen detection: Amebic antigen in serum are detected only in patients with active infections and disappears after clinical cure. Antigen like Lipophosphoglycan (LPG) amebic lectin, serine-rich *E. histolytica* protein (SREHP) are detected using monoclonal antibodies by ELISA. **Molecular diagnosis**: Recently, deoxyribonucleic acid (DNA) probes and radioimmunoassay have been used to detect E. histolytica in the stool. It is a rapid and specific method.

Treatment

Both luminal and tissue amebicides: Metronidazole and related compounds like tinidazole and om imidazole act on both sites and are the drug of choice for treating amebic colitis and amebic liver abscess.

Nonpathogenic Intestinal Ameba

Entamoeba dispar

Entamoeba dispar is morphologically indistinguishable (both cyst and trophozoite) from *E. histolytica*, so it may be considered as a subspecies of *E. histolytica*. It can be distinguished from *E. histolytica* by: Zymodeme study (hexokinase isoenzyme pattern), molecular methods, PCR amplifying, detection of lectin antigen in stool, and RBC inside trophozoites—present only in *E. histolytica*.

E. dispar parasite is nonpathogenic, usually colonizes in the large intestine (10 times more than *E. histolytica*) but doesn't invade intestinal mucosa. It grows well in polyxenic media, however, poorly grows on axenic media. *E. dispar* doesn't induce antibody production.

Life Cycle and Morphology

The life cycle is essentially identical to that of *Entamoeba coli* or any of the other nonpathogenic intestinal protozoa, and the cyst form is the infective form for humans.

Morphology of Trophozoites

Living trophozoites vary in diameter from about 12 to 60 μm. Motility has been described as rapid and unidirectional, with pseudopods forming quickly in response to the conditions around the organism; it may appear to be sporadic. Although this characteristic is often described, it is rare to diagnose these organisms on the basis of motility seen in a direct wet mount. The cytoplasm is differentiated into a clear outer ectoplasm and a more granular inner endoplasm. Based on the recent ability to culture these organisms in axenic culture systems and on light and electron microscopy studies, there may be some morphologic differences between *E. histolytica* and *E. dispar*. However, these differences would not be recognized by routine diagnostic methods such as the permanent stained smear. If organisms were seen that were consistent with *E. histolytica* /*E. dispa*r on the permanent stained smear, the laboratory report would indicate that fact and would be written as "*Entamoeba histolytica* /*E. dispar*".

When the organism is examined on a permanent stained smear (trichrome or iron hematoxylin), the morphological characteristics are easily seen. The nucleus generally has evenly arranged chromatin on the nuclear membrane and has a small, compact, centrally located karyosome. The cytoplasm is described as finely granular with few ingested bacteria or debris in vacuoles. Ingested RBCs are never seen in the trophozoites; if ingested

RBCs are seen, this finding identifies the organism as *E. histolytica*, not *E. dispar*

Entamoeba moshkovskii

Entamoeba moshkovskii is also morphologically indistinguishable from *E. histolytica* and *E. dispar* (may be the third subspecies of *E. histolytica*).

This species was first described from Moscow sewage by Tshalaia in 1941 and was thereafter reported to occur in many different countries including India. It can be distinguished from *E. histolytica* by isoenzyme analysis, molecular methods, and detection of lectin antigen.Though it is a non-pathogen harboring in the intestine recent studies from Bangladesh and India have reported *E. moshkovskii* as a sole potential pathogen in patients presenting with gastrointestinal symptoms and/or dysentery, highlighting the need for further study to investigate the pathogenic potential of this organism.

Entamoeba moshkovskii is found worldwide and is generally considered to be a free-living ameba. Based on microscopic morphology, this organism is indistinguishable from *E. histolytica* and *E. dispar*, except in cases of invasive disease when *E. histolytica* contains ingested RBCs. Although first isolated from sewage, *E. moshkovskii* can also be found in clean riverine sediments to brackish coastal pools. Apparently, there are some differences that separate this organism from *E. histolytic* and *E. dispar*. However, these differences pertain to physiology rather than morphology; *E. moshkovskii* is osmotolerant, can be cultured at room temperature, and is resistant to emetine.

Life Cycle and Morphology

The life cycle is essentially identical to that of *E. dispar*, and morphological differences are minimal to none. In wet preparations, trophozoites usually range in size from 15 to 20 μm and cysts normally range in size from 12 to 15 μm. It is important to remember that on the permanent stained smear there is a certain amount of artificial shrinkage due to dehydration; therefore, all of the organisms, including pathogenic *E. histolytica*, may be somewhat smaller (from 1 to 1.5 μm) than the sizes quoted for the wet-preparation measurements.

Morphology of Trophozoites

Trophozoites do not ingest RBCs, and the motility is similar to that of both *E. histolytica* and *E. dispar*. Nuclear and cytoplasmic characteristics are very similar to those seen in *E. histolytica*; however, trophozoites of *E. moshkovskii* do not contain ingested RBCs.

Morphology of Cysts: Nuclear characteristics and chromatoidal bars are similar to those in *E. histolytica* and *E. dispar*.

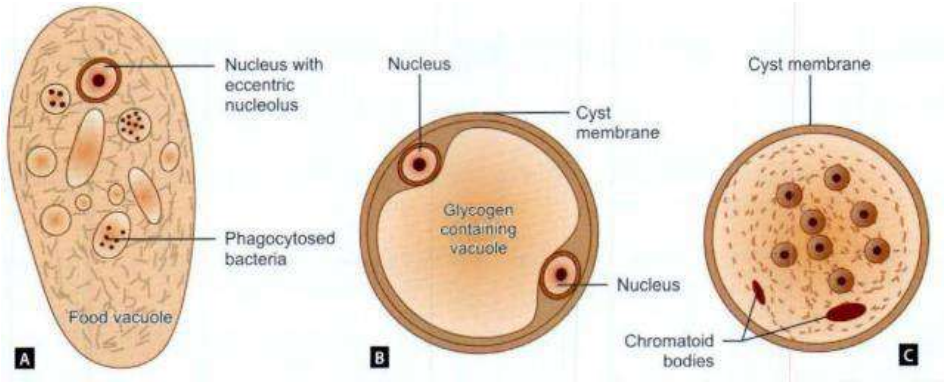
Entamoeba Coli

Entamoeba coli was first described by Lewis (1870) and Cunningham (1871) in Kolkata and its presence in healthy persons was reported by Grassi (1878). It is worldwide in distribution and a nonpathogenic commensal intestinal ameba. It is a larger than *E. histolytica* about 20-50 μm with sluggish motility and contains ingested bacteria but no red cells. The nucleus is clearly visible in unstained films and has a large eccentric karyosome and thick nuclear membrane lined with coarse granules of chromatin. Cysts are large,

10- 30 µm in size, with a prominent glycogen mass in the early stage. The chromatoid bodies are splinter-Like and irregular. The mature cyst has eight nuclei.

Life cycle

The life cycle is the same as in *E. histolytica* except that it remains a luminal commensal without tissue invasion and is nonpathogenic.



Figuer (4)A to C: Schematic diagram of the morphological forms of ***Entamoeba coli***. (A) Vegetative form; (B) Binucleate cyst; and (C) Eight-nucleate cyst

Entamoeba hartmanni

Entamoeba hartmanni occurs wherever *E. histolytica* is found. It is now considered to be a separate species of nonparhogenic commensal intestinal ameba. It is much smaller than *E. histolytica*, the trophozoirc measuring 4- 12 µm and cyst 5-10 µmin size. Trophozoites do nor ingest red cells and their motility is less vigorous. The cyst resembles that of Endolimax nana.

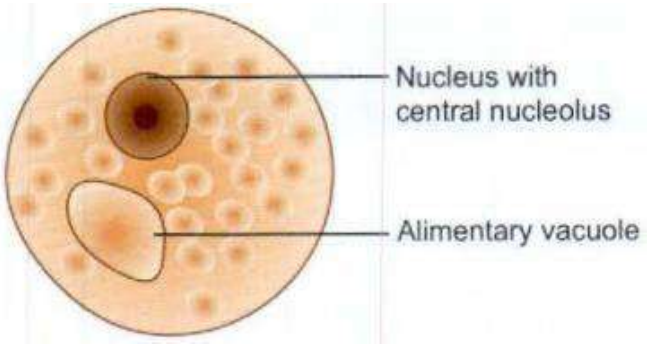


Figure (5): Trophozoite of ***Entamoeba hartmanni.***

Entamoeba Gingivalis

Entamoeb gingivalis was the first ameba of humans, discovered by Gros in 1849. It is global in distribution. Only the trophozoite is found; the cystic stage being apparently absent. The trophozoite is about 10-20 µm, actively motile with multiple pseudopodia. The cytoplasm contains food vacuoles with ingested bacteria, leukocytes, and epithelial cells. Nucleus is round with central karyosome lined by coarse chromatin granules. The ameba lives in gingival tissues and is abundant in unhygienic mouths. It is a commensal and is not considered to cause any disease. It is transmitted by direct oral contact. *E. gingivalis* have been found in bronchial washings and vaginal and cervical smears, where it can be mistaken for *E.histolytica*.

Endolimax Nana

This common commensal ameba is widely distributed. It lives in the human intestine.The trophozoite is small (nana: small), less than 10 µm in size with sluggish motility.The nucleus has a conspicuous karyosome

connected to the nuclear membrane by one or none coarse strands. The cyst is small, oval, and quadrinucleate with glycogen mass and chromidial bars, which are inconspicuous or absent. It is nonpathogenic.

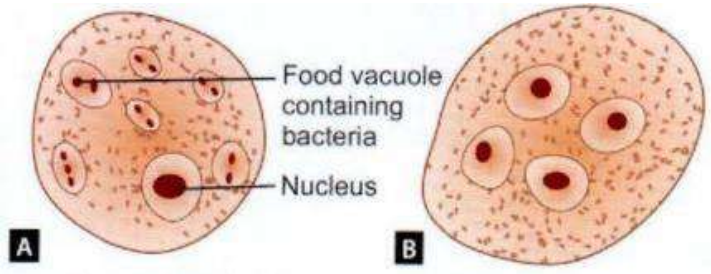


Figure (6): ***Endolimax nana***. (A) Vegetative form: and (B) Quadrinucleate cyst.

Iodamoeba Butschlii

This is widely distributed, though less common than *E. coli* and *E. nana*. Trophozoite is small, 6- 12 µm, with a conspicuous nucleus. The prominent karyosome is half the size of the nucleus, having a bull's eye appearance. The cyst is oval, uninucleate and has a prominent iodine staining glycogen mass (iodophilic body). Hence, the name lodamoeba. It is nonpathogenic. The comparative morphology of amebae infecting humans is illustrated in.

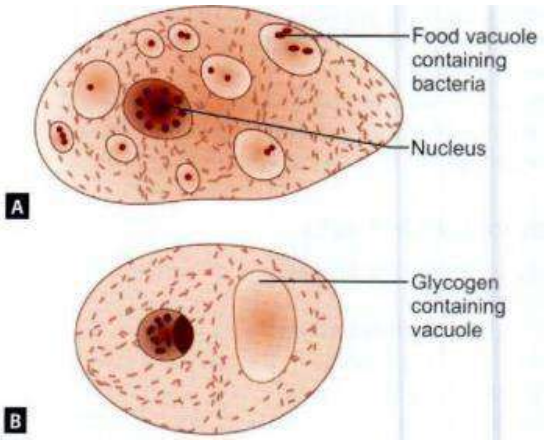


Figure (7): ***Iodamoeba butschlii***. (A) Vegetative form: and (B) Cyst.

Subphylum Mastigophora

Parasitic protozoa which possess whip-like flagella as their organs of locomotion are classified under the Phylum-Sarcomastigophora, Subphylum-Mastigophora, class Zoomastigophorea (from mastix-whip, photos-bearing). Depending on their habitat, they can be considered under two headings:

1. **Lumen-dwelling flagellates (Intestinal, Oral and Genital Flagellates):** Flagellates found in the alimentary and urogenital tracts.
2. **Haemoflagellates:** Flagellates found in blood and tissues.

Flagellates classified as:

Subphylum: Mastigophora

Class: Zoomastigophora (mastix: whip)

Depending on their habitat, they can be considered under:

Lumen-dwellingflagellates: Flagellates found in the alimentary tract and urogenital tract.

Hemoflagellates: Flagellates found in blood and tissues. Most luminal flagellates are nonpathogenic commensals.

Two of them cause clinical diseases: **(1) *Giardia lamblia***, which can cause diarrhea, and **(2) *Trichomonas vaginalis***, which can produce vaginitis and urethritis.

Intestinal Flagellates

Most luminal flagellates are nonpathogenic commensals. Two of them cause clinical disease, *Giardia lamblia* which can cause diarrhea, and *Trichomonas vaginalis* which can produce vaginitis and urethritis. Intestinal

flagellates found in humans are listed below, with the sites affected by them shown in parenthesis.

1. *Giardia lamblia* (duodenum, jejunum).
2. *Trichomonas vaginalis* (vagina, urethra).
3. *Trichomonas. tenax* (mouth).
4. *Trichomonas*. *hominis* (caecum).
5. *Chilomastix mesnili* (caecum).
6. *Dientamoeba fragilis* (See Chapter-2).

Giardia Lamblia

History and Distribution

This flagellate was observed by Leeuwenhoek (1681) in his own stools and was thus one of the earliest of protozoan parasites to have been recorded. It is named Giardia after Professor Giard of Paris and lamblia after Professor Lambl of Prague who gave a detailed description of the parasite. Worldwide in distribution, it is the most common intestinal protozoan pathogen. Infection may be asymptomatic or cause diarrhoea.

Giardiasis (gee-ar-die-a-sis with a soft "G") is an infection of the small intestine that is caused by the parasite, *Giardia duodenalis*, also known as *Giardia lamblia* and *Giardia intestinalis*.

It is the most common cause of parasitic gastrointestinal disease; it is estimated that 20,000 cases of giardiasis occur each year in the U.S., and there is a 20% to 40% prevalence in the world's population.

Giardia lamblia exists in two forms, an active form called a trophozoite, and an inactive form called a cyst. The active trophozoite attaches to the lining of the small intestine with a "sucker" and is responsible for causing the signs and symptoms of giardiasis.

The trophozoite cannot live long outside of the body, therefore it cannot spread the infection to others. The inactive cyst, on the other hand, can exist for prolonged periods outside the bod.

When it is ingested, stomach acid activates the cyst, and the cyst develops into the disease-causing trophozoite. It takes ingestion of only ten cysts to cause infection. Trophozoites are important not only because they cause the symptoms of giardiasis, but also because they produce the cysts that exit the body in the feces and spread the infection to others.

Cysts of *Giardia* are present in the feces of infected persons. Thus, the infection is spread from person to person by contamination of food with feces, or by direct fecal-oral contamination. Cysts also survive in water, for example in fresh water lakes and streams. As a result, giardiasis is the mostcommon cause of water-borne, parasitic illness in the U.S.

Domestic mammals (for example, dogs, cats, calves) and wild mammals (for example, beavers) can become infected with Giardia; however, it is not clear how often domestic or wild mammals transmit giardiasis to humans.

Morphology

It occurs in two forms: trophozoite and cyst.

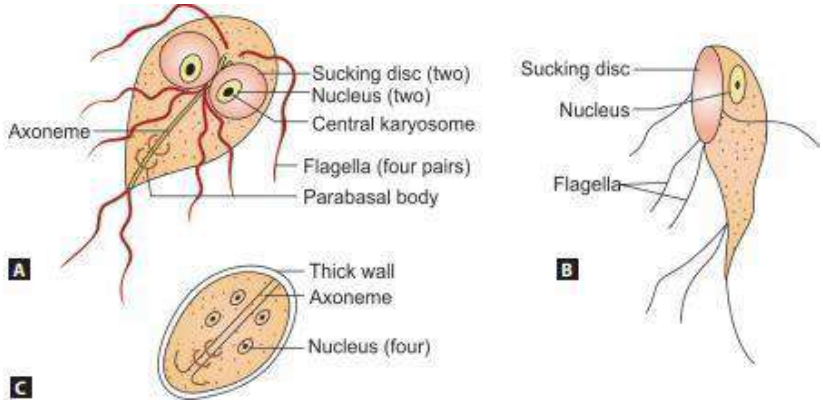


Figure (8): *Giardia lamblia* (schematic diagram): A- trophozoite front view; B- trophozoite lateral view; C-cyst.

Trophozoite

The trophozoite has a falling leaf-like motility, usually measures 10–20 µm in length and 5–15 µm in width.

Shape: In front view, it is pear shaped (or tear drop or tennis racket shaped) with rounded anterior end and pointed posterior end. Laterally, it appears as a curved portion of a spoon (sickle shaped).

It is convex dorsally while the ventral surface has a concavity bearing a bilobed adhesive disc. Hence, it appears as sickle-shaped in the lateral view. Trophozoite is bilaterally symmetrical; on each side from the midline it bears.

One pair of nuclei, Pair of median bodies. Four pairs of basal bodies or blepharoplast (from which the axoneme arises).

Four pairs of flagella—two lateral, one ventral and one caudal pair of flagella, pair of parabasal bodies (connected to basal bodies through which the axoneme passes) and pair of axoneme or axostyle (the intracellular portion of the flagella).

Cyst

Giardia cyst is oval shaped, measures 11–14 µm in length and 7–10 µm in width, it contains four nuclei and remnants of axonemes, basal bodies and parabasal bodies and it is the infective form as well as the diagnostic form of the parasite.

Life cycle

Giardia lamblia possesses a simple life cycle which is composed of 2 stages: (1) the trophozoite and (2) the cyst.

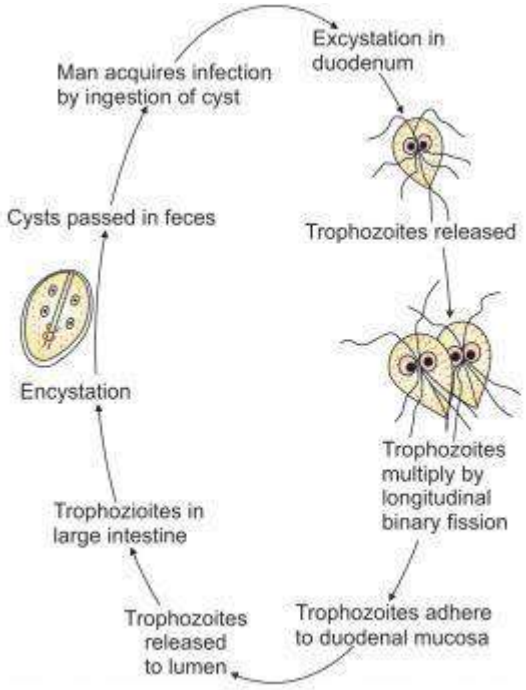


Figure (9): Life cycle of *Giardia lamblia*

Host: *Giardia lamblia* life cycle in one host.

Infective form: Mature cyst.

Mode of transmission: Man acquires infection by ingestion of food and water contaminated with mature cysts or rarely by sexual route (mainly in homosexuals).

Development of Life cycle in Man:

Excystation: Two trophozoites are released from each cyst in the duodenum within 30 minutes of entry.

- **Multiplication:** Trophozoites multiply by longitudinal binary fission in the duodenum.
- **Adhesion:** Trophozoites adhere to the duodenal mucosa by the bilobed adhesive ventral disc.

This is achieved by the microtubules of median bodies, contractile proteins and lectins present on the surface of adhesive disc that binds to the intestinal receptors.

- In active stage of the disease, sometimes the trophozoites are excreted in diarrhea stool.
- **Encystation:** Gradually when the trophozoites pass down to the large intestine, encystation begins.

Pathogenicity and Clinical Features

Giardia lamblia is typically seen within the crypts of duodenal and jejunal mucosa. It does not invade the tissue but remains tightly adhered to the intestinal epithelium by means of the Sucking-disk.

They may cause abnormalities of villous architecture by cell apoptosis and increased lymphatic infiltration of lamina propria. Loss of brush border epithelium of the intestine leads to a deficiency of enzymes including disaccharides.

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Often they are asymptomatic, but in some cases, Giardia may lead to mucus diarrhea, fat malabsorption (steatorrhea), dull epigastric pain, belching, and flatulence. The stool contains excess mucus and fat but no blood.

Children may develop chronic diarrhea, malabsorption of fat, vitamin A, vitamin B12, folic acid, protein, sugars like xylose disaccharides, weight loss and sprue-like syndrome.

Signs and symptoms usually appear one to three weeks after exposure and may include: Watery, sometimes foul-smelling diarrhea that may alternate with soft, greasy stools, fatigue or malaise, abdominal cramps and bloating, gas or flatulence, nausea and weight loss.

Laboratory diagnosis of Giardia lamblia

- Stool examination-detects cysts and trophozoites.
- Entero-test.

Antigen detection in stool (copro-antigen) by ELISA and rapid immunochromatography test (ICT).

Antibody detection in serum by ELISA and IFA, culture, molecular method—PCR, and radiological findings—barium meal, X-ray.

Stool examination

Multiple stool samples (at least 3) should be tested before a negative result is reported.

Wet Mount In bright-field microscopy, cysts appear ovoid to ellipsoid in shape and usually measure 11 to 14 μm (range: 8 to 19 μm). Immature and mature cysts have 2 and 4 nuclei, respectively. Intracytoplasmic fibrils are visible in cysts.

Giardia cysts can be demonstrated by iodine and saline wet mount preparations but they cannot differentiate active disease from carriers.

Fig .(10) Wet mount for *Giardia lamblia* cyst stage with saline and iodine

Demonstration of the trophozoites with falling leaf like motility by saline mount indicates active stage of the disease. Giardia adheres firmly to the duodenal mucosa by adhesive disc leading to intermittent shedding. Hence, repeated stool examination (at least three consecutive samples) should be done.

Sensitivity varies from 60% to 80% with one stool and more than 90% after three stools examination.

Concentration techniques like zinc sulfate floatation or formalin ether sedimentation methods are employed to increase the chance of detection.

Duodenal sampling: If stool examination is negative, then direct duodenal samples like aspirates (obtained by entero-test) or biopsy (done by endoscopy) should be processed.

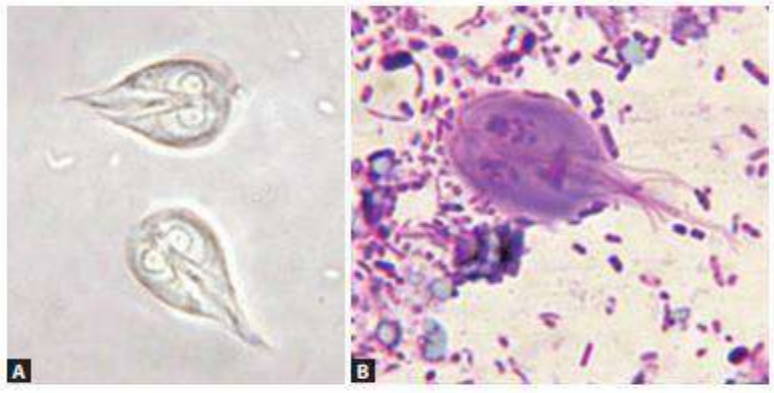


Fig .(11) Trophozoite of *Giardia lamblia* (A) saline mount front view; (B) Giemsa stain

Entero-test

It uses a gelatin capsule attached to a thread.

Antigen Detection in Stool (Copro-antigen)

The enzyme-linked immunosorbent assay (ELISA) and direct fluorescent antibody tests (DFA) are available using labeled monoclonal antibodies against cyst wall protein antigens.

Molecular methods

Detection of *Giardia* nucleic acid by polymerase chain reaction (PCR) or by gene probes is highly sensitive and specific

Treatment

Several drugs can be used to treat Giardia infection. Effective treatments include metronidazole, tinidazole, and nitazoxanide. Other medications include paromomycin, quinacrine, and furazolidone.

Trichomonas vaginalis

Geographical Distribution: It is encountered in all climates and all social groups.

Habitat: In female it is found mainly in vagina and in male it is in urethra.

Morphology: It is found only in trophozoite form which bears following characters, pear shaped measuring 10 to 30 μ × 5 to 10 μ, it has short

undulating membrane which comes up to the middle of the body, possesses 4 anterior flagellae, a prominent axostyle which bifurcates the body into two and projects posteriorly, there is a costa, parabasal body, rounded nucleus (anteriorly), chromatin granules are present all over, more densely near costa and axostyle, flagellae give characteristic webbing or rotatory motility, and it multiplies by binary fission in longitudinal axis.

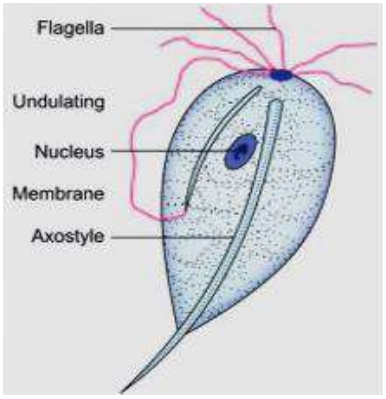


Fig.(12)Trichomonas vaginalis

Mode of Transmission

It is primarily a venereal disease in which transmission can also be from person-to-person contact. However, newborns may get infected during birth. Fomites (such as clothes, utensils, and furniture) also form another way of transmission of infection.

Incubation Time: It varies from 4 to 30 days.

Pathogenesis and Pathology

Within a few days following the introduction of viable *Trichomonas vaginalis* into the vagina, the proliferating colonies of this flagellate cause degeneration and desquamation of the vaginal epithelium. It is followed by leukocytic inflammation of the tissue layer. Very large numbers of

trichomonads and leukocytes are now present in the vaginal secretion, which is liquid, greenish, or yellow, and covers the mucosa down to the urethral orifice, vestibular glands, and clitoris. As the acute condition changes to the chronic stage, the secretion loses its purulent appearance due to a decrease in the number of trichomonads and leukocytes, *Trichomonas vaginalis* in male genitalia may be symptomless or may be responsible for an irritating, persistent, or recurring urethritis.

Clinical Picture

The vaginal secretion is extremely irritating, almost unbearable and is constantly flowing. The symptoms may continue from a few days to many months. After each menstruation there is a tendency for acute stage to recur. The chronic condition transforms into latent one and secretions become normal with no manifestation although trichomonas are still present. Difference in the intensity of symptoms may be due to differences in virulence of strains of this organism. In male patients it may be symptomless or may cause urethritis and prostatitis.

Laboratory Diagnosis

In female patient, *Trichomonas vaginalis* maybe demonstrated in sedimented urine, vaginal secretion, or from vaginal scraping.

In male patient *Trichomonas vaginalis* maybe found in the centrifuged urine and prostatic secretions following massage of the prostatic gland.
However, care should be taken to prevent contamination of the specimen with feces, since Trichomonas hominis maybe has seen and thus misdiagnosed as

Trichomonas vaginalis. The smear is stained using Giemsa, PAS, Papanicolaou, Leishman, Diff Quick and acridine orange.

Treatment

Trichomoniasis is usually treated quickly and easily with antibiotics. Most people are prescribed an antibiotic called metronidazole which is very effective if taken correctly. You'll usually have to take metronidazole twice a day, for 5 to 7 days. Sometimes this antibiotic can be prescribed in a single, larger dose.

Trichomonas tenax

Harmless commensal of the oral cavity, periodontal area, carious cavities of the tooth, tonsillary crypts, etc. Measures 5 to 10 μ, i.e. smaller in size as compared to *Trichomonas vaginalis*. Transmission is through fomites, salivary droplets, and kissing.

Hemoflagellate

Including genus: Leishmania and Trypanosoma (blood tissue species): There are four morphological forms of clinical significance associated with the hemoflagellates: Amastigote, promastigote, epimastigote and trypomastigote.

General characteristics

1- They live in the blood and tissues of man and other vertebrate hosts and in the gut of the insect vector.
2- Members of this family have a single nucleus, a kinetoplast and a single flagellum.
3- Nucleus is round or oval and is situated in the central part of the body.
4- Kineloplast consists of a deeply staining parabasal body and adjacent dot-Like blepharoplast.
5- The parabasal body and blepharoplast are connected by one or more thin fibr.
6- Flagellum is a thin, hair-like structure, which originate from the blepharoplast.

7- The portion of the flagellum ,which is inside the body of the parasite and extends from the blepharoplasl to surface of the body is known as axoneme.

8- A free flagellum at the anterior end traverses on the surface of the parasite as a narrow undulating membrane.

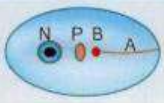
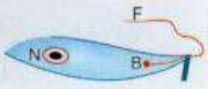
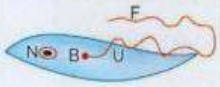
9- Hemoflagellates exist in two or more of four morphological stages.

The transmission of hemoflagellates

Is accomplished by the bite of an arthropod vector. Flagellate protozoa found in blood or tissues of human and there are two genera of medical importance (Leishmania and Trypanosoma). The major difference between these two genera is that primary diagnostic form found in Leishmania is the amastigote, whereas that of Trypanosoma is the trypomastigote

1. Amastigotes: It is Roundish to oval in shape, Consist of a nucleus and kinetoplast. The large single nucleus is typically located off-center .The dotlike blepharoplast is attached to a small axoneme, this axoneme extends to the edge of the organism.The single parabasal body is located adjacent to the blepharoplast.
2. Promastigotes: It is Long and slender in appearance. The large single nucleus is located in or near the center .The kinetoplast is located in the anterior end of the organism .A single free flagellum extends anteriorly from the axoneme.
3. Epimastigotes: It is long and slightly wider than promastigote form.The large single nucleus is located in posterior end .The kinetoplast located anterior to the nucleus .Undulating membrane extending half of the body length .A single free flagellum extends anteriorly from the axoneme.

4. Trypomastigotes: It is C or U shape in stained blood films .Long and slender in appearance .One nucleus located anterior to the kinetoplast .The kinetoplast is located in the posterior end of the organism .Undulating membrane extending entire body length .A single free flagellum extends anteriorly from the axoneme when present

	Amastigote	*Promastigote*	*Epimastigote*	*Trypomastigote*
Morphological characteristics	Rounded or ovoid, without any external flagellum. The nucleus, kinetoplast and axial filaments can be seen. The axoneme extends up to the anterior end of the cell	Lanceolate in shape. Kinetoplast is anterior to the nucleus (antinuclear kinetoplast) near the anterior end of the cell, from which flagellum emerges. There is no undulating membrane	Elongated, with the kinetoplast placed more posteriorly, though close to and in front of the nucleus (juxtanuclear kinetoplast). The flagellum runs alongside the body as a short undulating membrane, before emerging from the anterior end	This stage is elongated, spindle-shaped with a central nucleus. The kinetoplast is posterior to the nucleus (postnuclear kinetoplast) and situated at the posterior end of the body. The flagellum runs alongside the entire length of the cell to form a long undulating membrane before emerging as a free flagellum from the anterior end
Seen in	*Trypanosoma cruzi* and *Leishmania* as intracellular form in vertebrate host	It is the infective stage of *Leishmania*, found in the insect vector as well as in cultures *in vitro*	It is the form in which *Trypanosoma brucei* occur in salivary gland of the vector tsetse fly and *Trypanosoma cruzi* in the midgut of the vector reduviid bug. *Note:* This stage is lacking in *Leishmania*	This is the infective stage of trypanosomes found in arthropod vector and in the blood of infected vertebrate. *Note:* This stage is lacking in *Leishmania*
Schematic illustration				

Table(1): Morphological form of hemofagellate

Genus *Leishmania*:

Leishmaniasis There are many different species of Leishmania and the disease that they cause. directly linked to the species of Leishmania with which a person Several species of Leishmania are pathogenic for man: L. donovani causes visceral leishmaniasis (Kala-azar, black disease, dumdum fever); L. tropica cause cutaneous leishmaniasis (oriental sore, Delhi ulcer, Aleppo, Delhi or Baghdad boil); and L. braziliensis (also, L. mexicana and L. peruviana) are etiologic agents of mucocutaneous leishmaniasis .

Life Cycle

Leishmania are transmitted by arthropod. In this case it is a small biting fly known as a **sand fly**. Leishmaniae spend part of their life cycle in the gut of the sandfly, but their life cycle is completed in a vertebrate host. Within the sandfly gut, the protozoa are carried as extracellular promastigotes, these parasites multiply in the gut and migrate toward the pharynx. Sandflies transferred promastigotes to the vertrebrate host when the sandfly takes a meal blood by expelling leishmaniae into the bite wound of the mammalian host. From where they pass into the blood and tissues of the human host.

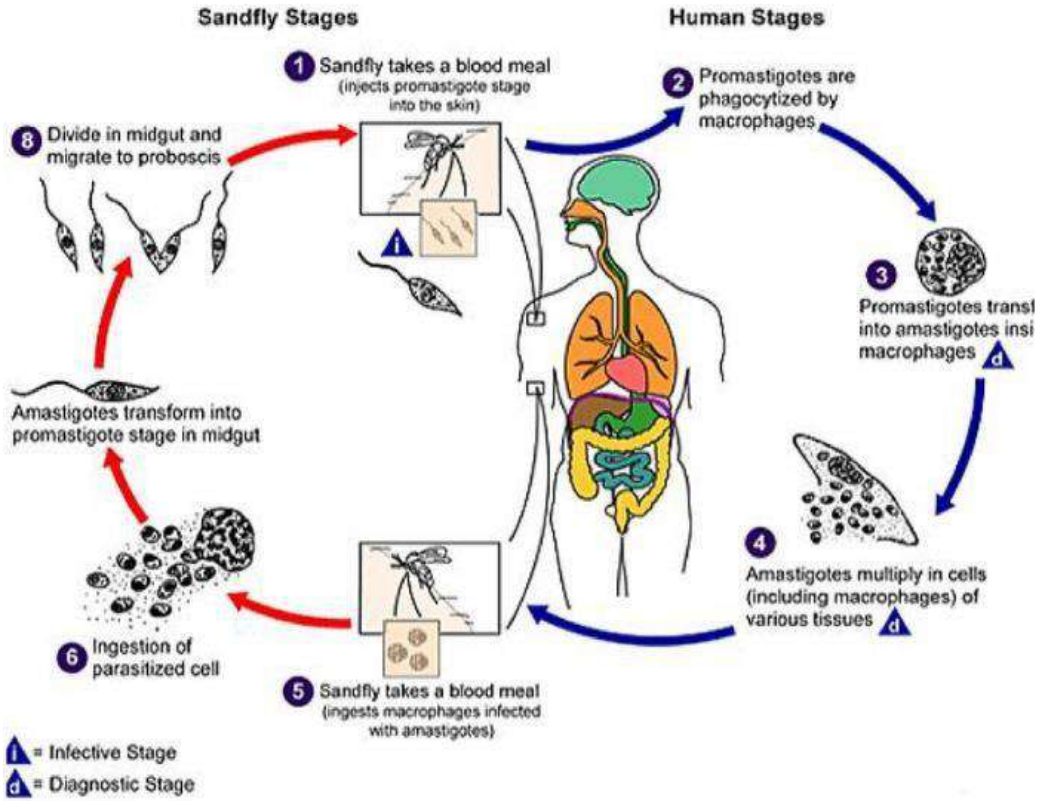


Figure (12): Life Cycle of leishmamia spp.

Pathogensis and clinical finding

Leishmaniasis is a parasitic disease caused by several species of genus Different species of leishmania cause different disease.

A. *L. donovani* causes visceral leishmaniasis also called Kala-azar and Dum Dum fever. Spleenomegaly & hepatomegaly the infection is generalized and the parasite is distributed in the internal organs. The parasite may also cause a variety of skin lesions (dermal leishmaniasis) without any visceral manifestations.

Laboratory Diagnosis 1. Giemsa-stained slides of blood , bone marrow , lymph node aspirates and biopsies of the infected areas for the diagnosis of amastigote forms.

2. Culture of blood, bone marrow and other tissues these samples show the promastigote forms.

3. Serological tests.

B. *L. tropica*: causes tropic sore or Baghdad boil, oriental sore and cutenaeous Leishmianiasis. The infection is limited to a local lesion of the skin and subcutaneous tissues.

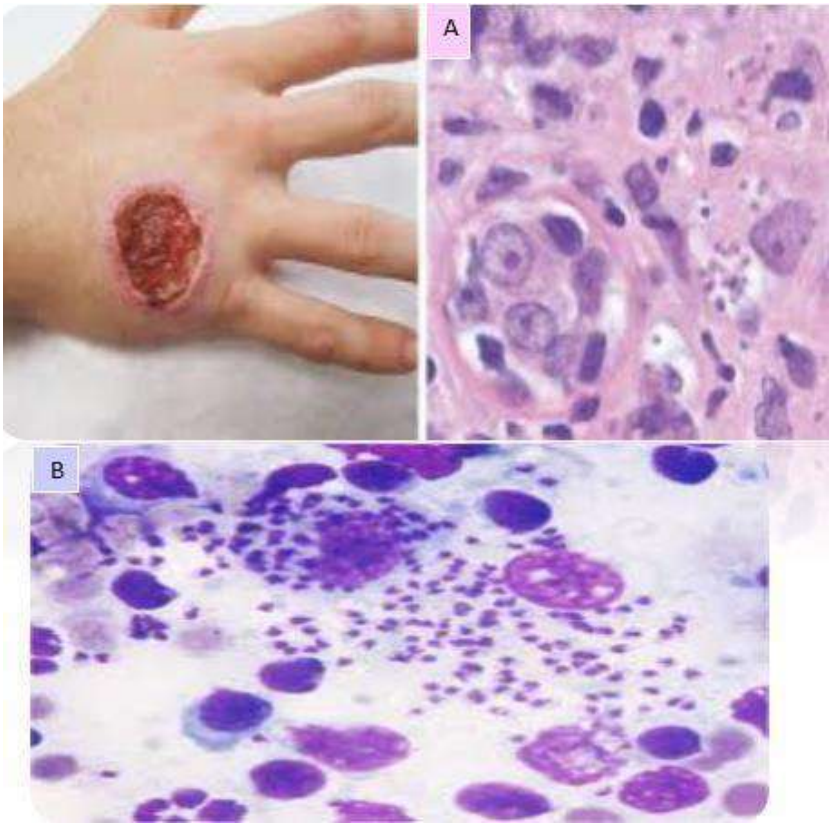


Figure (13): *L. tropica* and *L.major* amastigote a-intracellular b-intercellular.

Laboratory Diagnosis

1. The specimen of choice for identify the amastigotes of Leishmania braziliensis is a biopsy of the infected ulcer.
2. Microscopic examination of the Giemsa-stained preparations should reveal the typical amastigotes. Promastigotes may be present when the sample is collected immediately after introduction into the patient.
3. Culturing the infected material, which often demonstrated the promastigote stage.
4. Serological tests.

Treatment

Pharmacologic therapies include the following:

- Pentavalent antimony (sodium stibogluconate or meglumine antimonate): Used in cutaneous leishmaniasis
- Liposomal amphotericin B (AmBisome): Effective against pentavalent antimony–resistant mucocutaneous disease and visceral leishmaniasis

SubPhylum Apicomplexa

Coccidia

Coccidia are small protozoans (one-celled organisms), which are members of the class Sporozoa, an exclusively parasitic group that typically requires alternation of sexual and asexual reproduction in the life cycle.

The coccidia are characterized by a thick walled oocyst stage that is typically excreted with the feces. Some coccidia (*Cryptosporidium, Cyclospora, Isospora*) carry out their entire life cycle within the intestinal epithelial cells of the host and are transmitted by the fecal-oral route. Other coccidia (*Sarcocystis, Toxoplasma*) have a more complicated life cycle involving tissue cysts and multiple hosts (ie, heteroxenous).

1- Locomotive organelles absent, the flagella present only in male game.
2- Life cycles are complex, with well-developed a sexual(which produce merozoites , some of these merozoites differentiate into macro and microgametes) and sexual stages (which produce oocysts.
3- Sporozoa produce special spore like cells called sporozoites.
4- It is intracellular parasite with complex cycle alternating between humans and mosquitoes as in malaria, while in T.gondii which causes an acute infection in human is acquired from cats and other animals.
5- Have similar independent gametocytes ,the male or microgametocyte and female or macrogametocyte. The female produce a single

macrogamete and the male produce multiple gametes , followling an oocyst is formed after fertilization.

Plasmodium spp

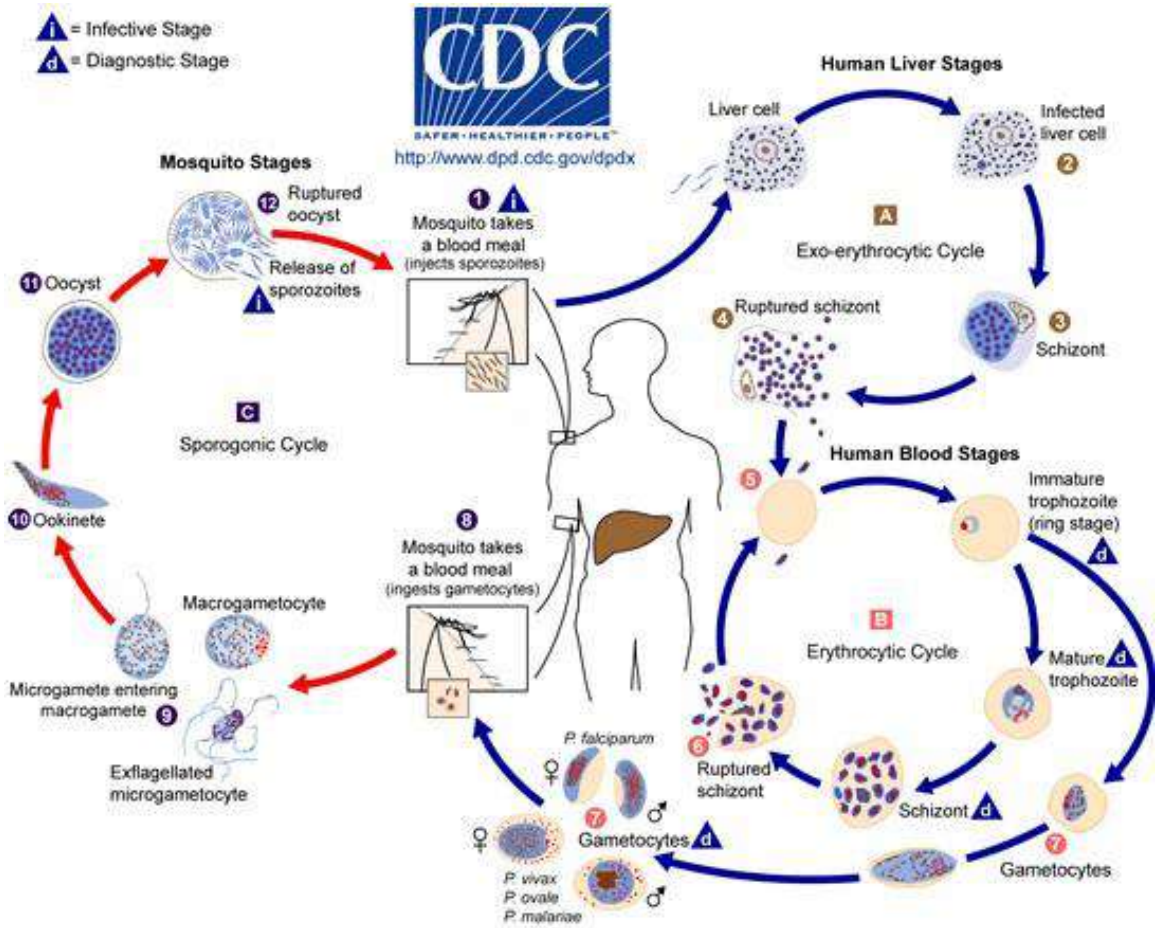


Figure (14): Life cycle of *plasmodium spp*.

Plasmodium falciparum

Plasmodium falciparum is the most important malaria parasite, found in the tropics and sub-tropics, being responsible for approximately 50% of all malaria cases. The incubation period of *P. falciaprum* malaria is the shortest, between 8 and 11 days and has a periodicity of 36 – 48 hours. It can be differentiated from the other species by the morphology of different stages found in the peripheral blood. In infections with *Plasmodium falciparum* usually only young **trophozoites** and **gametocytes** are seen in peripheral blood smears, the **schizonts** are usually found in capillaries sinuses of internal

organs and in the bone marrow. The disease runs an acute course and often has lethal outcome. It is a significant cause of abortions and stillborns and even death of non-immune pregnant women.

Life cycle

The aspects of the life cycle, which are specific to P. falciparum, are as follows:

a) It attacks all ages of erythrocytes so that a high density of parasites can be reached quickly. In extreme cases up to 48% of the red blood cells can be parasitised.
b) Multiple infections resulting in several ring forms in a corpuscle are not uncommon.
c) The latter stages in the asexual cycle do not occur in the peripheral blood as in other forms of malaria, so that only rings and crescents are found in blood films. After 24 hours the ring forms and older trophozoites show a tendency to clump together and adhere to the visceral capillary walls. They become caught up in the vessels of the heart, intestine, brain or bone marrow in which the later sexual stages are completed.

Morphology of Trophozoites

Red blood cells in *Plasmodium falciparum* infections are not enlarged and they may have a spiky outline, common in cells, which have dried slowly. The typical arrangement of cytoplasm in young trophozoites is the well-known ring formation, which thickens and invariably contains several vacuoles as the trophozoite develops. Chromatin is characteristically found as a single bead, but double beads and small curved rod forms frequently occur.

Maurer's dots are slow to appear and are first seen as minute purplish dots, 6 or less in number. The points become spots, still few in number and are now characteristic enough to be recognised. Maurer describes them as fine ringlets, loops or streaks. They are seldom absent from the red blood cells containing large rings but the staining of the spots is very sensitive to pH and is seldom seen if the pH falls below 6.8.

Trophozoites of *P. falciparum* can be found on the edge of the red blood cells. These are known as **acole**

Gametocytes

Gametocytes are the sexual stage of the malaria parasite. *Plasmodium falciparum* gametocytes appear in the peripheral circulation after 8 – 11 days of patent parasitaemia and they have assumed their typical crescentic shapes. They soon reach their peak density, and then decline in numbers, disappearing in about 3 months as a rule.

The female form, or **macrogametocyte**, is usually slenderer and somewhat longer than the male, and the cytoplasm takes up a deeper blue colour with Giemsa stain. The nucleus is small and compact, staining dark red, while the pigment granules are closely aggregated around it. The male form, or **microgametocyte**, is broader than the female and is more inclined to be sausage shaped. The cytoplasm is either pale blue or tinted with pink and the nucleus, which stains dark pink, is large and less compact than in the female, while the pigment granules are scattered in the cytoplasm around it. In humans, **gametocytes** neither multiply, nor cause symptoms but they are the forms, which are infective to the mosquito. When a female Anopheline mosquito takes a blood meal, the male and female **gametocytes** continue their sexual development.

Schizonts

Schizonts are rarely seen in the peripheral blood and their presence may indicate a potentially serious parasitaemia. Schizonts have 8 – 36 merozoites and a large mass of golden brown pigment (haemozoin) seen in the preschizont and schizont stage.

Clinical Signs of Disease

Symptoms include headache, photophobia, muscle aches and pains, anorexia, nausea and vomiting. Complications include severe anaemia cerebral malaria, renal disease, black water fever, dysentery, pulmonary oedema and tropical splenomegaly syndrome.

Plasmodium vivax

Introduction

Plasmodium vivax is found almost in all places, where malaria is endemic and is the most predominant of malaria parasites. Causing 43% of all cases of malaria in the world, it also has the widest geographical distribution. Although the disease itself is not usually life threatening, it can cause severe acute illness.

Plasmodium vivax does not infect West Africans due to the fact that West Africans do not possess the Duffy Antigen on the red blood cells, which the parasite requires to enter the red blood cell. It has an incubation period of between 10 and 17 days, which is sometimes prolonged to months or years due to the formation of hypnozoites. It has a periodicity of 48 hours. *Plasmodium vivax* infections are usually characterised by the presence of more than one developmental stage in the peripheral blood film. The parasites parasitise young enlarged erythrocytes and Schüffner's dots develop on the erythrocyte membrane.

Life cycle

The aspects of the life cycle, which are specific to P. vivax, are as follows:

a) The degree of infectivity is low, only the young immature corpuscles are infected; about 2% of erythrocytes are parasitised.
b) The periodicity of the asexual cycle is closely synchronised.
c) Hypnozoites develop in the liver, so that relapses may occur.

Morphology of Trophozoites

Most trophozoites of *P. vivax* are already several hours old when they appear in peripheral blood and by that time the Schüffner's dots are already visible. The trophozoites are actively amoeboid and contain single or sometimes double chromatin dots that are either circular or ovoid. As the trophozoites mature, the Schüffner's dots increase in number and size and the parasite changes from large irregular rings to rounded or ovoid forms in mature trophozoites.

Gametocytes

Mature female gametocytes are large rounded parasites, which fill or nearly fill the host cell. The cytoplasm is blue and fairly homogenous. The nuclear chromatin is a single, well-defined purplish mass, varied in form and usually peripheral in distribution. Mature male gametocytes can be distinguished from females by the large, loose and ill-defined mass of chromatin and by their paler colour and smaller mass.

Schizonts

The parasitised red cells are much enlarged containing Schüffner's dots. The parasites are large, filling the enlarged red cell. There are between 12-24

merozoites in the schizonts (usually16). The pigment is a golden brown central loose mass.

Clinical Signs of Disease

Symptoms include headache, photophobia, muscle aches and pains, anorexia, nausea and vomiting. Complications due to *P. vivax* are relatively rare and arise due do a previous debility or pre-existing disease.

Plasmodium ovale

Introduction

Plasmodium ovale is widely distributed in tropical Africa especially the west coast, despite that it is a species that is rarely encountered. It has also been reported in South America and Asia. It has an incubation period of 10 – 17 days, which is sometimes prolonged to months or years due to the formation of hypnozoites. It has a periodicity of 48 hours; the fever it produces is milder than the benign tertian *P. falciparum.*

Life cycle

The features of the life cycle, which are specific to *P. ovale,* are as follows:

a) It morphologically resembles *P. malariae* in most of its stages;
b) The changes produced in the erythrocytes in general are similar to those produced by *P. vivax,* but Schüffner's dots appear considerably earlier (in the ring stage) and are coarser and more numerous;
c) In the oocyst the pigment granules are (usually) characteristically arranged in two rows crossing each other at right angles;

d) Hypnozoites develop in the liver so that relapses may occur.

Morphology

Parasites of *P. ovale* are usually found in enlarged and stippled red blood cells (James's dots), similar to those found in *P. vivax* infections. Host cells show an oval shape, particularly those containing younger stages of the parasites and the host cell may also show "spiking" or fimbriation.

Trophozoites

Young trophozoites are found as compact rings in cells containing Schüffner's dots. The trophozoite rings remain compact as they develop and show little of the amoeboid features common in *P. vivax*. Small, scattered pigment granules can be seen in developing trophozoites, which disperse as the trophozoite matures. Late trophozoites are round and consolidated with an increase in cytoplasm, they are very similar to *P. vivax* at this stage.

Gametocytes

The mature gametocytes are round, filling two thirds of the red cell. The red blood cell is slightly enlarged and stippled and contains pigment, which has a distinct arrangement of concentric rods, mostly at the periphery.

Schizonts

The parasite is smaller than red blood cells and contains 6-12 merozoites, usually 8 in a single ring. The pigment is a brown / greenish central clump. The red cell is slightly enlarged, stippled, frequently oval and fimbriated.

Clinical Signs of Disease

Symptoms, like those of *P. vivax,* include headache, photophobia, muscle aches and pains, anorexia, nausea and vomiting. Complications due to *P. ovale* are relatively rare and arise due do a previous debility or preexisting disease.

Plasmodium malariae

Introduction

Plasmodium malariae occurs mainly in the subtropical and mild areas where *P. falciparum and P. vivax* occur. However, it is less frequently seen, responsible for approximately 7% of all malaria in the world. It has an incubation period of 18 – 40 days and a periodicity of 72 hours.

Life cycle

The features of the life cycle, which are specific to P. malariae, are as follows:

a) Infected erythrocytes are not larger than uninfected ones and sometimes even smaller;
b) Mature erythrocytes are attacked and rarely reticulocytes, so that the density of parasites is very low; about 0.2% of erythrocytes are parasitised;
c) It is often difficult to distinguish between a large trophozoite and an immature gametocyte.

Morphology

Parasites of *P. malariae* are typically compact heavily pigmented parasites, which are usually smaller and more deeply stained than normal.

They tend to parasitise small, old red blood cells, they do not contain any inclusion dots and the parasitaemia is usually low.

Trophozoites

Trophozoites are found as fairy large fleshy rings with a single chromatin dot. These can be much distorted and can often take the form of bands across the cell. All trophozoites have a single chromatin dot and contain pigment.

Gametocytes

Gametocytes contain large amounts of black pigment, with chromatin present as a compact mass in females and diffuse in males. They occupy less than two thirds of the red blood cell.

Schizonts

Schizonts are usually few in numbers with 6 – 12 large merozoites in a single ring. Pigment is usually present as a central black mass. The parasites present are generally only found at one stage of schizogony development.

Clinical Signs of Disease

Symptoms include headache, photophobia, muscle aches and pains, anorexia, nausea and vomiting. *Plasmodium malariae*, like *P. vivax* and *P. ovale* are relatively benign. However, chronic infections in children may lead to nephrotic syndrome due to immune complexes depositing on the glomerular wall.

Diagnosis of malaria parasites

The definitive diagnosis of malaria infection is still based on finding malaria parasites in blood films. In thin films the red blood cells are fixed so the morphology of the parasitised cells can be seen. Species identification can be made, based upon the size and shape of the various stages of the parasite and the presence of stippling (i.e. bright red dots) and fimbriation (i.e. ragged ends). However, malaria parasites may be missed on a thin blood film in case of low parasitaemia. Therefore, examination of a thick blood film is recommended. With a thick blood film, the red cells are approximately 6 – 20 layers thick which results in a larger volume of blood being examined.

Thick Blood Films

In examining stained thick blood films, the red blood cells are lysed, so diagnosis is based on the appearance of the parasite. In thick films, organisms tend to be more compact and denser than in thin films.

Thin Blood Films

When examining thin blood films for malaria you must look at the infected red blood cells and the parasites inside the cells

Serological diagnosis

Various test kits are available to detect antigens derived from malaria parasites. Such immunologic ("immunochromatographic") tests most often use a dipstick or cassette format, and provide results in 2-15 minutes. These "Rapid Diagnostic Tests" (RDTs) offer a useful alternative to microscopy in situations where reliable microscopic diagnosis is not available. Malaria

RDTs are currently used in some clinical settings and programs. The World Health Organization is conducting comparative performance evaluations of many of the RDTs which are commercially available worldwide based on a panel of parasites derived from a global network of collection sites. Results of this testing is available at:

Molecular Diagnosis

Parasite nucleic acids are detected using polymerase chain reaction (PCR). Although this technique may be slightly more sensitive than smear microscopy, it is of limited utility for the diagnosis of acutely ill patients in the standard healthcare setting. PCR results are often not available quickly enough to be of value in establishing the diagnosis of malaria infection. PCR is most useful for confirming the species of malarial parasite after the diagnosis has been established by either smear microscopy or RDT.

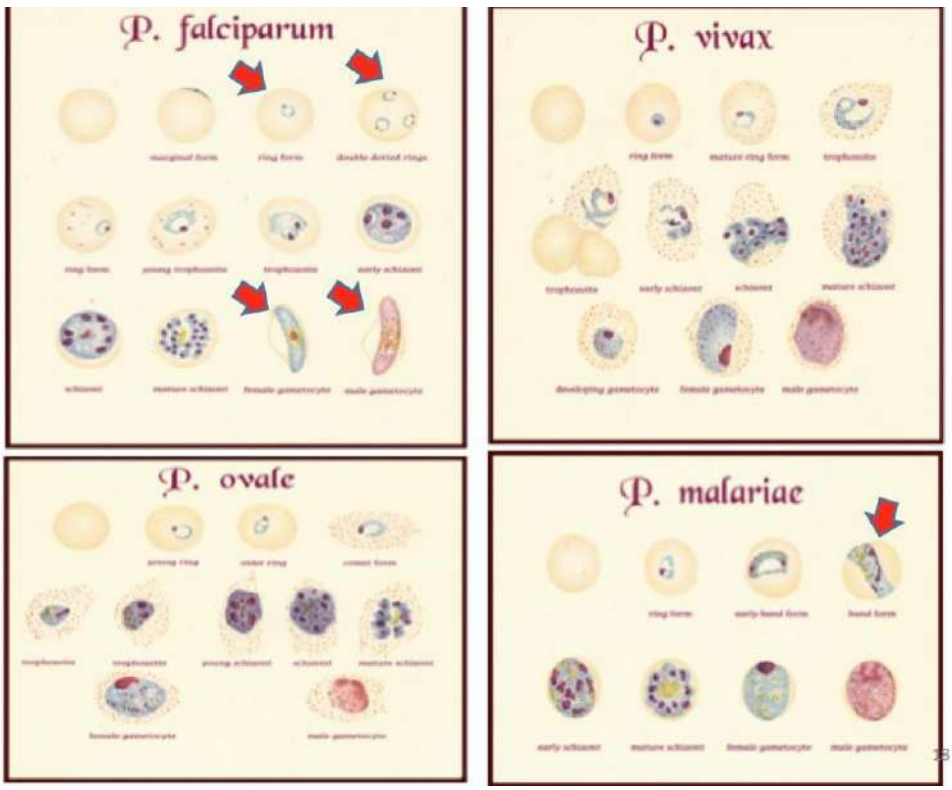


Figure (15): Differences between stages of *Plasmodium spp*

Laboratory Diagnosis

1. Microscopic examination of giemsa-stained slides of aspiration of fluid underneath the ulcer bed for typical amastigotes.
2. Culture of the ulcer tissue may also reveal the promastigote forms.
3. Serological tests such as Indirect Fluorescent Antibody (IFA) also vailable.

C. *L. brasiliensis* causes Espundia Mucocutaneous leishmaniasis. The infection is limited to a local lesion of the skin but may metastasise to other areas of skin and oro-nasal mucosa.The primary lesion often disappears spontaneously, followed by mucocutaneous lesions that destroy the mucosal surface of the nose, pharynx, and larynx. If the condition is untreated, potentially fatal secondary bacterial infections and disfigurement may occur.

Genus: *Toxoplasma gondii*

Toxoplasma gondii: The parasite probably is the only protozoan, whose all the stages (tachyzoite, tissue cyst and oocyst) are infected for man. *Toxoplasma gondii* was first described by Nicolle and Manceaux in 1908 in gundi (*Ctenodactylus gundi*), a small rodent of North Africa. It was named as *Toxplasma,* due to crescent shape of its tachyzoite .The parasite was subsequently demonstrated in man by Darling. It was found in congenitally infected child in 1937.

The life cycle of *Toxoplasma gondii* was fully described only in 1970, when it was known that cats are the definitive hosts, man and other

warmblooded animals are the intermediate hosts. *T. gondii* is an obligate intracellular parasite, which is found inside the reticuloendothelial cells

Morphology

There are five forms in *T. gondii* life cycle: trophozoite (tachyzoite), tissue cyst (bradyzoite,,)schizont gametocyte) and oocyst .Tachyzoites, tissue cysts and oocysts are important stages seen during the life cycle of the parasite, all these stages are infectious to man.

Trophozoite (tachyzoite)

It is oval to crescent-shaped with a pointed anterior end and arounded posterior end. It measures 4-7μm in lengths and 2-4μm in breadth. An ovoid nucleus is present in the posterior end of the parasite. Tachyzoite is the active, multiplying form seen during the acute stage of the infection. It can invade any type of cell in a host and once inside a cell, it multiplies within a vacuole by a process known as endodyogeny, or by binary fission or schizogony. Tachyzoites divide until they fill the host cell, which then liberates them, and they reinvade (oringested by) other macrophages, repeating the process.

Tissue cyst

It is spherical and may vary in size from 5 to 100μm in diameter. This is the resting form and is found during chronic stage of the infection. The tissue cysts can be found in any organ of the body but are commonly found in the brain and the skeletal and heart muscels.

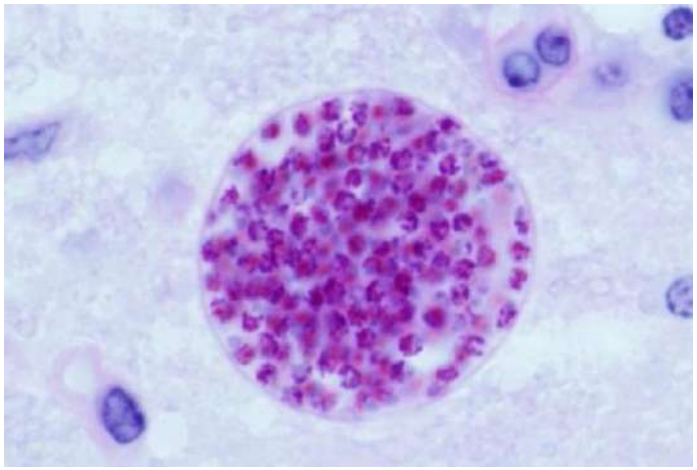


Figure (16): Cyst stage of *Toxoplasma gondii*.

Oocyst

This stage is only present in cat and other felines but not in humans. It is oval and measures10-12μm in diameter. Each cyst is surrounded by a thick resistant wall which encloses a spheroplast .The oocyst is liberated from the intestinal epithelial cell while still immature; it complete its development while passing down the gut and after expulsion in the faeces.

Life cycle

Toxplasma gondii needs two hosts to complete its life cycle. The definitive hosts are domestic cat and other members of the family Felidae (domestic cats and their relatives) such as bob cats, ocelots, Bengal tigers, mountain lion, etc. The sexual multiplication or gametogony (the intestinal cycle) take place in the epithelial cells of the small intestine. The oocysts are passed in the unsporulated form in the faeces. The intermediate hosts are human and mice and other non-feline hosts (e.g., goat, sheep, pig, cattle, etc.). The asexual multiplication or sporogony (the extra-intestinal cycle) occurs in the extraintestinal tissue.

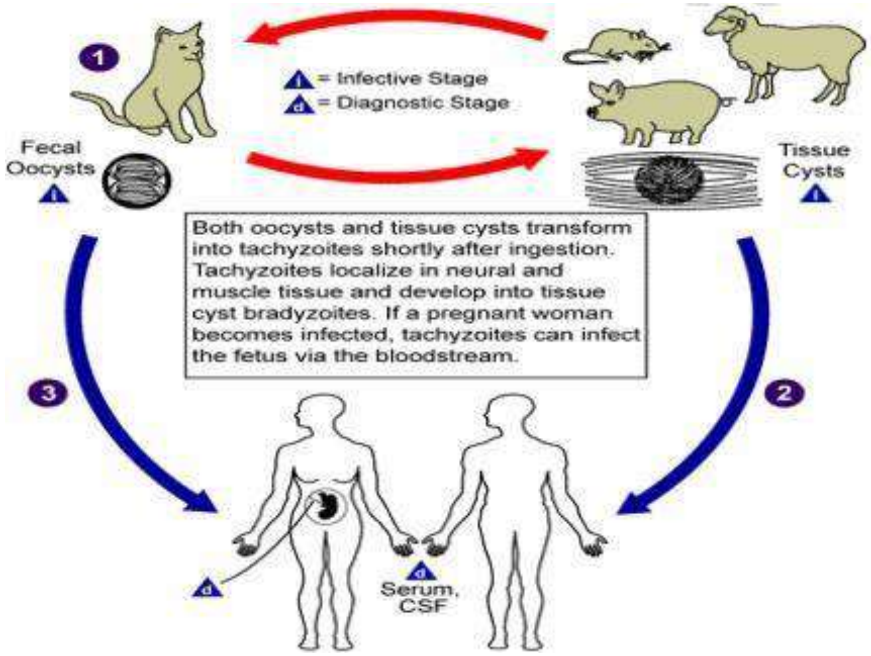
Develop in intermediate hosts

Human infection may be acquired in several ways: a) ingestion of undercooked infected meat containing Toxoplasma cysts; b) ingestion of the oocyst from fecally contaminated hands or food and water; c) organ transplantation or blood transfusion; d) transplacental transmission; e) accidental inoculation of tachyzoites.

Develop in definitive hosts

Members of the cat family (Felidae) are the only known definitive hosts for the sexual stages of *T. gondii* and thus are the main reservoirs of infection. Cats become infected with *T. gondii* by their predatory habit of feeding on the muscles, brain and other tissues of infected mice, which harbour the tissue cysts. They also get infection by being fed raw meat of domesticated animals containing these cysts. After the cat ingests tissue cysts or pseudocysts or oocysts, viable organisms are released and invade epithelial cells of the small intestine where they undergo an asexual followed by a sexual cycle and then form oocysts, which are then excreted. The sexual cycle consists of a limited number of merogonies, producing merozoites which reinvade other mucosal cells, until the final generation of merozoites enter mucosal cells and commence the sexual cycle of gametogony, gametogony fertilization and sporogony within the developing oocyst.The oocysts are then released into the lumen of the intestine by rupture of the host cell. These oocysts, which are non-infectious, are shed in non-sporulated form up to 21days in cat's faeces. Millions of oocysts are excreted in the faeces daily, up to 3 weeks. The oocysts sporulate outside the host with the formation of two sporocysts, each

containing four sporozoites, within few days. These sporulating oocysts can survive in the environment for several months Man acquires infection by ingesting these sporulating oocysts and the cycle is repeated. Surprisingly the extra-intestinal cycle, which is seen in man and other non-feline hosts, also occurs in cat itself, possible by direct invasion of lymphatics or lymph nodes by tachyzoites produced in its own intestine.



. **Figure (17):** life cycle of *Toxoplasma gondii***.**

Pathogenesis

Toxoplasmosis in immunocompetent: Toxoplasmosis in adults and children past the neonatal stage is usually benign and asymptomatic. Acquisition of the infection via either oocysts or tissue cysts results in an acute infection in which tachyzoites are disseminated throughout the body via the lymphatic and hematogenously. This acute stage will persist for several weeks as immunity develops.

Congenital Toxoplasmosis

Congenital (i. e, transplacental) infections are more likely to be symptomatic than postnatal infections and can be particularly severe. transmission only possible during acute stage ,transmission is more frequent later in pregnancy infection can result in: spontaneous abortion, premature birth, or full-term with or without progressive disease typical disease manifestations include: retinochoroiditis, intracerebral calcification, hydrocephaly, microcephaly, psychomotor disturbances, mental retardation, blindness and other visual defects.

Toxoplasmic Encecphalitis

Noted as an opportunistic infection in regards to reactivation of latent infections due to immunosuppression associated with organ transplants and certain cancer treatments and AIDS patients.

Ocular Toxoplasmosis:

Originally the ocular manifestations were more often associated with congenital infections (develop weeks to years after birth) or a late manifestation due to the reactivation of a congenital infection.

Diagnosis:

Parasites can be detected in biopsied specimens, buffy coat cells, or cerebral spinal fluid.These materials can also be used to inoculated mice or tissue culture cells. Diagnosis relies heavily on serological procedures (detection of T. gondii Ag or anti Toxoplasma IgM &IgG Abs. High IgM titer present in acute infection while high IgG titer represent past or chronic infection. Imaging techniques, such as computed tomography (CT) scanning and magnetic resonance imaging (MRI), are useful in the diagnosis of

toxoplasmic encephalitis. Sabin-Feldman dye test, is an important test in diagnosis of toxoplasmosis.

Treatment

Recommended: anti-folates (pyrimethamine + sulfadiazine). Spiramycin for infection during pregnancy.

Cryptosporidium

Since its initial identification in 1907 several *Cryptosporidium* species have been identified in a wide variety of animals ranging from fish to humans. The first human cases of cryptosporidiosis were reported in 1976 and were characterized as a diarrheal disease associated with immune suppression. Initially it was believed to be a rare and exotic disease. During the 1980's *Cryptosporidium* was recognized as a major cause of diarrhea in AIDS patients and often resulted in death. However, it is now recognized that *Cryptosporidium* is a common cause of diarrhea in immunocompetent persons and has probably been a human pathogen since the beginning of humanity. Two species infecting humans have been identified: *C.*
parvum and *C. homini*

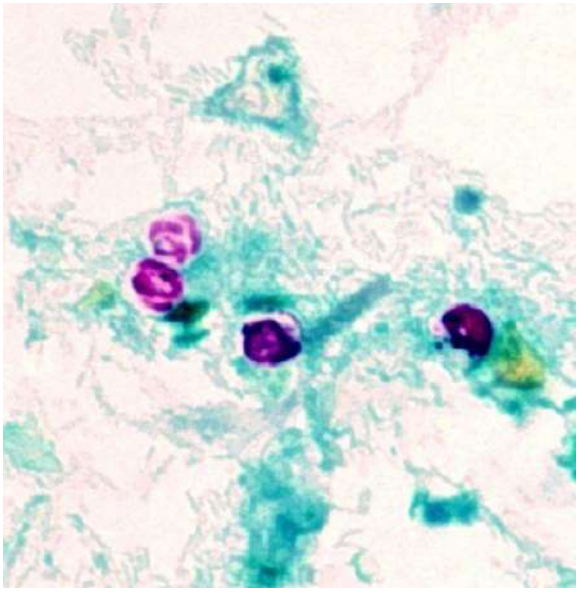


Figure (18): photomotograph of *Cryptosporidium* oocyst.

Life cycle

Cryptosporidium is often classified as a coccidian and exhibits a life cycle similar to other intestinal coccidia. However, *Cryptosporidium* is more closely related to the gregarines and this is reflected in some aspects of its life cycle. The infection is acquired through the ingestion of sporulated oocysts . The pH changes associated with passage through the gut and bile and pancreatic fluids in the small intestine trigger excystation. Sporozoites emerge from the oocyst and attach to intestinal epithelial cells. In contrast to other coccidia, *Cryptosporidium* sporozoites do not invade the enterocytes. Instead they induce the fusion and expansion of the microvilli resulting in the parasite becoming surrounded by a double membrane of host origin. A junction, called the 'feeder organelle' or the 'adhesion zone', forms between the parasite and the host enterocyte. The parasite, now called a trophozoite , likely derives nutrients from the host cell via this junction.

Trophozoites undergo an asexual replication (ie, merogony) and produce 4-8 merozoites (Mz) which are released into the intestinal lumen. The merozoites infect new intestinal epithelial cells and undergo additional rounds of merogony. The increased severity of the disease in immunocompromised patients is due in part to their inability to limit these additional rounds of merogony.

As an alternative to merogony, the merozoites can develop into either macro- or microgametocytes following the infection of an enterocyte. Microgametogenesis involves several rounds of replication followed by the release of numerous microgametes into the intestinal lumen. The microgametes fertilize macrogametes still attached to the intestinal epithelial cells. The resulting zygote (Zg) undergoes sporogony and the sporulated oocysts (Oo) are excreted with the feces. An autoinfection is also possible and this too may contribute to the increased disease severity in immunocompromised patients.

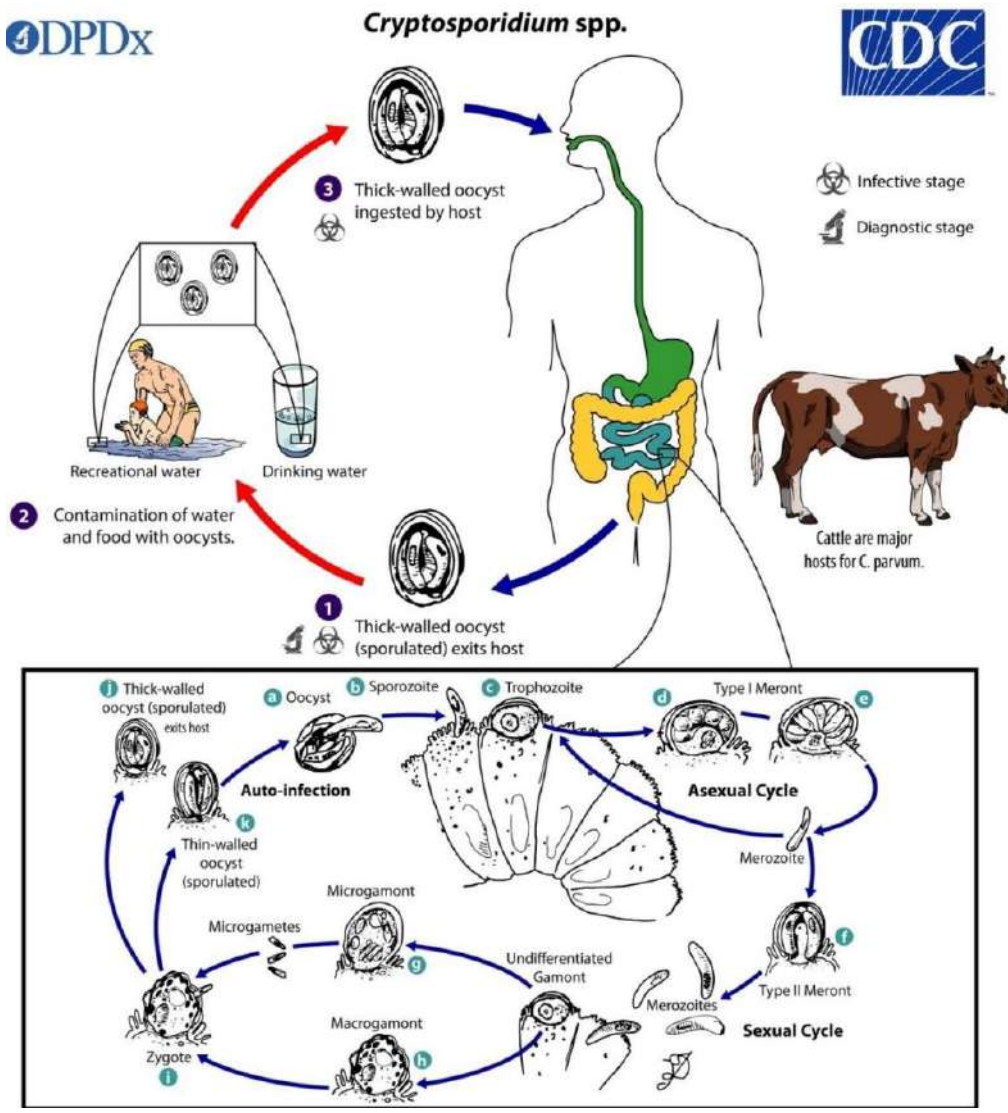


Figure (19): life cycle of *Cryptosporidium* spp.

Transmission

The risk factors of transmission for *Cryptosporidium* are similar to other fecal-oral diseases. However, waterborne cryptosporidiosis outbreaks have been especially notable. The most infamous is an outbreak in Milwaukee during the spring of 1993 in which an estimated 400,000 people developed symptomatic cryptosporidiosis (Factors that contribute to the increased risks of *Cryptosporidium* waterborne outbreaks are:

- small size of oocysts

- wide range of host specificity and monoxenous development
- close associations between human and animal hosts
- large number of oocysts excreted (up to 100 billion per calf)
- low infective dose
- robust oocysts which are resistant to chlorine
- infectious sporulated oocysts excreted

Despite the impressiveness of some waterborne outbreaks, human-tohuman transmission appears to predominate. For example, asymptomatic infected children are common, secondary cases in households are high, and outbreaks tend to occur in hospitals, institutions and day care centers-situations typical for fecal-oral transmission.

Pathogenesis

The most common clinical manifestation of cryptosporidiosis is a mild to profuse watery diarrhea. This diarrhea is generally self-limiting and persists from several days up to one month. Recrudescences are common. Abdominal cramps, anorexia, nausea, weight loss and vomiting are additional manifestations which may occur during the acute stage. The disease can be much more severe for persons with AIDS which manifests as a chronic diarrhea lasting for months or even years. Some AIDS patients exhibit a fulminant cholera-like illness which requires intravenous rehydration therapy. The fatality rate can be quite high in these fulminant cases.

Diagnosis

Microscopic demonstration of the large, typically shaped oocysts, is the basis for diagnosis. Because the oocysts may be passed in small amounts and intermittently, repeated stool examinations and concentration procedures are recommended as Zinc sulfate or sugar flotation which is the most sensitive stool concentration technique. Fluorescent stains, modified acid-fast, hematoxylin/eosin, and Giemsa staining are helpful in identifying the translucent oocysts

Diarrhea can have osmotic, inflammatory, or secretory components The watery nature of the diarrhea associated with *Cryptosporidium* infections has suggested the presence of an enterotoxin.

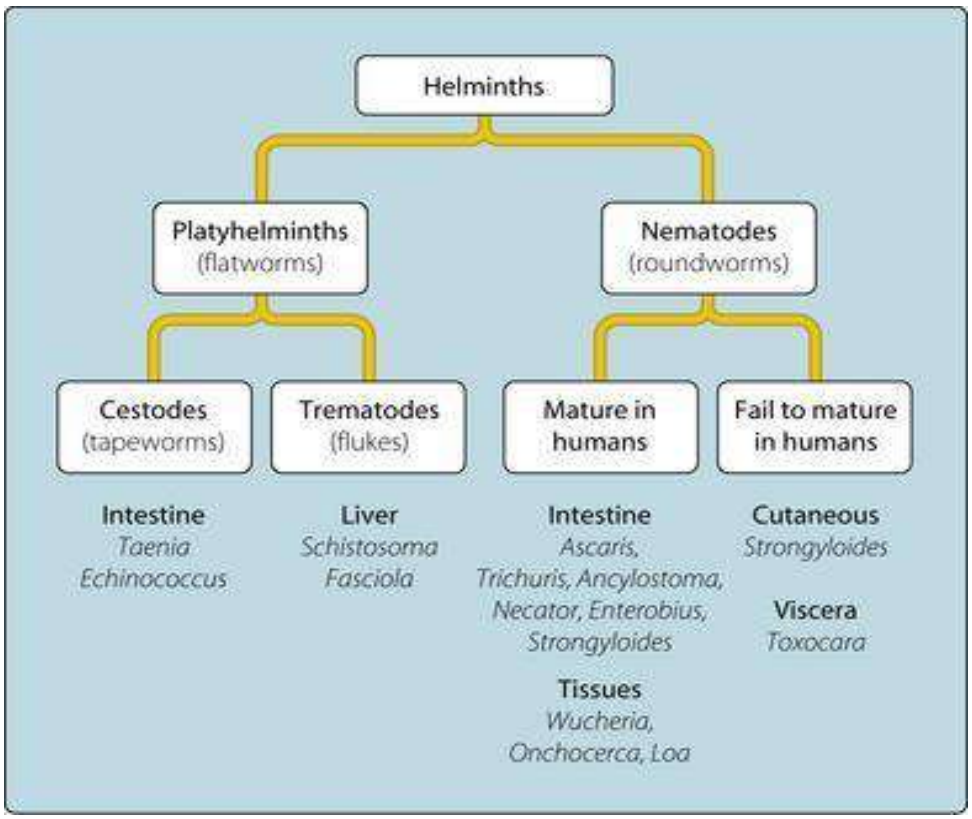
Treatment

Like most gastrointestinal infection in humans involves fluid rehydration, electrolyte replacement, and management of any pain recently, nitazoxanide is the only drug approved for the treatment of cryptosporidiosis in immunocompetent hosts.Paromomycin may alleviate some of the diarrhoeal symptoms continuing antiretroviral drugs for HIV infection to boost the immune system may also control infection

Phylum Metazoa (Helminthes)

Helminths are multicellular (eukaryotic) organisms and thus belong to kingdom Animalia. As such, they also belong to a group of animals known as metazoa.

While there is still confusion on how to group helminths in terms of taxonomy, they are divided into the following(see table below



Class Trematoda

Schistosoma spp.

Schistosomiasis (Bilharziasis) is caused by some species of blood trematodes (flukes) in the genus *Schistosoma*. The three main species infecting humans are S*chistosoma haematobium*, *S. japonicum*, and *S. mansoni*. Three other species, more localized geographically, are *S. mekongi*,

S. intercalatum, and *S. guineensis* (previously considered synonymous with *S. intercalatum*). There have also been a few reports of hybrid schistosomes of cattle origin (*S. haematobium*, *S. bovis*, *S. curassoni,* and *S. mattheei*) infecting humans. Unlike other trematodes, which are hermaphroditic, *Schistosoma* spp. are dioecous (individuals of separate sexes).

In addition, other species of schistosomes, which parasitize birds and mammals, can cause cercarial dermatitis in humans but this is clinically distinct from schistosomiasis.

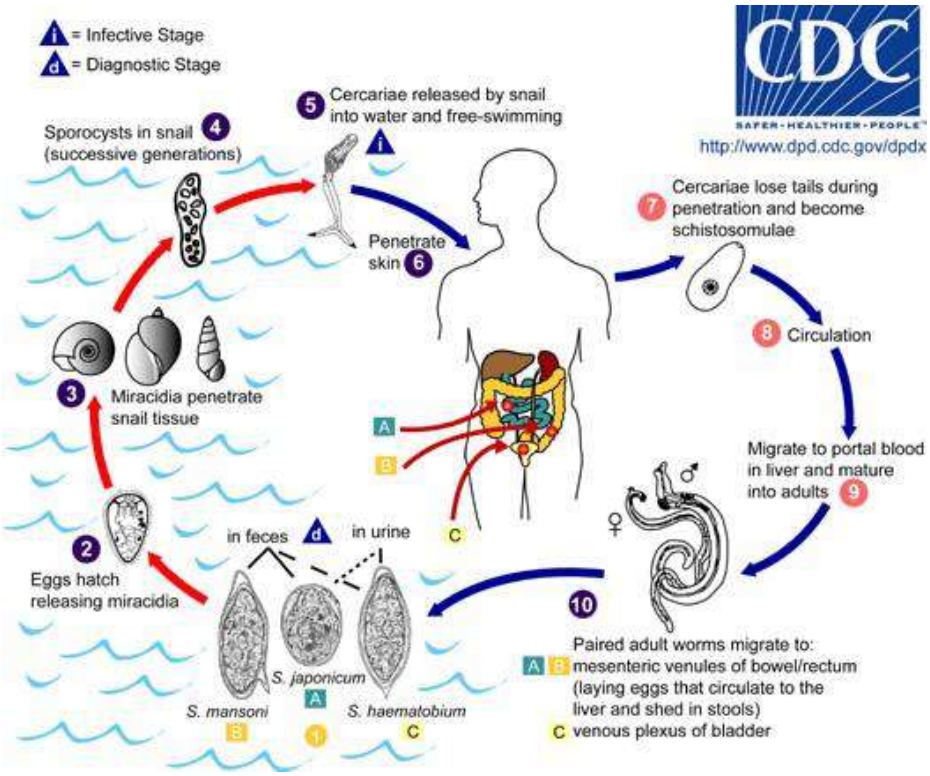


Figure (20): Life Cycle of *schistosoma spp*.

Life Cycle

Schistosoma eggs are eliminated with feces or urine, depending on species . Under appropriate conditions the eggs hatch and release miracidia , which swim and penetrate specific snail intermediate hosts . The stages in the snail include two generations of sporocysts and the production of cercariae .

Upon release from the snail, the infective cercariae swim, penetrate the skin of the human host , and shed their forked tails, becoming schistosomulae . The schistosomulae migrate via venous circulation to lungs, then to the heart, and then develop in the liver, exiting the liver via the portal vein system when mature, . Male and female adult worms copulate and reside in the mesenteric venules, the location of which varies by species (with some exceptions) For instance, *S. japonicum* is more frequently found in the superior mesenteric veins draining the small intestine and *S. mansoni* occurs more often in the inferior mesenteric veins draining the large intestine However, both species can occupy either location and are capable of moving between sites. *S. intercalatum* and *S. guineensis* also inhabit the inferior mesenteric plexus but lower in the bowel than *S. mansoni*. *S. haematobium* most often inhabitsin the vesicular and pelvic venous plexus of the bladder , but it can also be found in the rectal venules. The females (size ranges from 7–28 mm, depending on species) deposit eggs in the small venules of the portal and perivesical systems. The eggs are moved progressively toward the lumen of the intestine (*S. mansoni*,*S. japonicum, S. mekongi, S. intercalatum/guineensis*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively.

Hosts

Various animals such as cattle, dogs, cats, rodents, pigs, horses, and goats, serve as reservoirs for *S. japonicum*, and dogs for *S. mekongi*. *S. mansoni* is also frequently recovered from wild primates in endemic areas but is considered primarily a human parasite and not a zoonosis.

Intermediate hosts are snails of the genera *Biomphalaria,* (*S. mansoni*), *Oncomelania (S. japonicum*), *Bulinus* (*S. haematobium, S. intercalatum, S. guineensis*). The only known intermediate host for *S. mekongi* is *Neotricula aperta*.

Geographic Distribution

Schistosoma mansoni is found primarily across sub-Saharan Africa and some South American countries (Brazil, Venezuela, Suriname) and the Caribbean, with sporadic reports in the Arabian Peninsula.

S. haematobium is found in Africa and pockets of the Middle East.

S. japonicum is found in China, the Philippines, and Sulawesi. Despite its name, it has long been eliminated from Japan.

The other, less common human-infecting species have relatively restricted geographic ranges. *S. mekongi* occurs focally in parts of Cambodia and Laos. *S. intercalatum* has only been found in the Democratic Republic of the Congo; *S. guineensis* is found in West Africa. Instances of infections with hybrid/introgressed *Schistosoma* (*S. haematobium, S. bovis, S. curassoni, S. mattheei*) have occurred in Corsica, France, and some West African countries.

Clinical Presentation

Symptoms of schistosomiasis are not caused by the worms themselves but by the body's reaction to the eggs. Many infections are asymptomatic. A local cutaneous hypersensitivity reaction following skin penetration by cercariae may occur and appears as small, itchy maculopapular lesions. Acute schistosomiasis (Katayama fever) is a systemic hypersensitivity reaction that may occur weeks after the initial infection, especially by *S. mansoni* and *S.*

japonicum. Manifestations include systemic symptoms/signs including fever, cough, abdominal pain, diarrhea, hepatosplenomegaly, and eosinophilia.

Occasionally, *Schistosoma* infections may lead to central nervous system lesions. Cerebral granulomatous disease may be caused by ectopic *S. japonicum* eggs in the brain, and granulomatous lesions around ectopic eggs in the spinal cord may occur in *S. mansoni* and *S. haematobium* infections. Continuing infection may cause granulomatous reactions and fibrosis in the affected organs (e.g., liver and spleen) with associated signs/symptoms.

Pathology associated with *S. mansoni* and *S. japonicum* schistosomiasis includes various hepatic complications from inflammation and granulomatous reactions, and occasional embolic egg granulomas in brain or spinal cord. Pathology of *S. haematobium* schistosomiasis includes hematuria, scarring, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord.

Diagnosis

Examination of stool and/or urine for ova is the primary method of diagnosis for suspected schistosome infections. The choice of sample to diagnose schistosomiasis depends on the species of parasite likely causing the infection. Adult stages of *S. mansoni*, *S. japonicum*, *S. mekongi*, and *S. intercalatum* reside in the mesenteric venous plexus of infected hosts and eggs are shed in feces; *S. haematobium* adult worms are found in the venous plexus of the lower urinary tract and eggs are shed in urine.

Serologic testing for antischistosomal antibody is indicated for diagnosis of travelers or immigrants from endemic areas who have not been treated

appropriately for schistosomiasis in the past. Commonly used serologic tests detect antibody to the adult worm. For new infections, the serum sample tested should be collected at least 6 to 8 weeks after likely infection, to allow for full development of the parasite and antibody to the adult stage. Serologic testing may not be appropriate for determination of active infection in patients who have been repeatedly infected and treated in the past because specific antibody can persist despite cure. In these patients, serologic testing cannot distinguish resolved infection from active infection. An antigen test has been developed that can detect active infection based on the presence of schistosomal antigen, but this test is not commercially available in the United States and at this time is undergoing field evaluations for accurate diagnosis of low-intensity infections.

Prevention

The best way to prevent schistosomiasis is to take the following steps if you are visiting or live in an area where schistosomiasis is transmitted: Avoid swimming or wading in fresh water when you are in countries in which schistosomiasis occurs. Swimming in the ocean and in chlorinated swimming pools is safe.

Treatment

Safe and effective medication is available for the treatment of both urinary and intestinal schistosomiasis. Praziquantel, a prescription medication, is taken for 1-2 days to treat infections caused by all schistosome species.

Class: Cestoda *Taenia*

***spp*.**

Is a tapeworms consist of two main parts: a rounded head called a **scolex** and a flat body of multiple segments called **proglottids.** The scolex has specialized means of attaching to the intestinal wall, namely, suckers, hooks, The worm grows by adding new proglottids from its germinal center next to the scolex. The oldest proglottids at the distal end are gravid and produce many eggs, which are excreted in the feces and transmitted to various intermediate hosts such as cattle, pigs, Humans usually acquire the infection when undercooked flesh containing the larvae is ingested. However, in two important human diseases, cysticercosis it is the eggs that are ingested and the resulting larvae cause the disease.

There are four medically important cestodes: *Taenia solium, Taenia saginata,* There are two important human pathogens in the genus *Taenia: T. solium* (the pork tapeworm) and *T. saginata* (the beef tapeworm).

Taenia solium

Disease

The adult form of *T. solium* causes taeniasis. *T. solium* larvae cause cysticercosis.

Important Properties

T. solium can be identified by its scolex, which has **four suckers and circle of hooks,** and by its gravid proglottids, which have 5–10 primary uterine branches. The eggs appear the same microscopically as those of *T. saginata* and Echinococcus species.

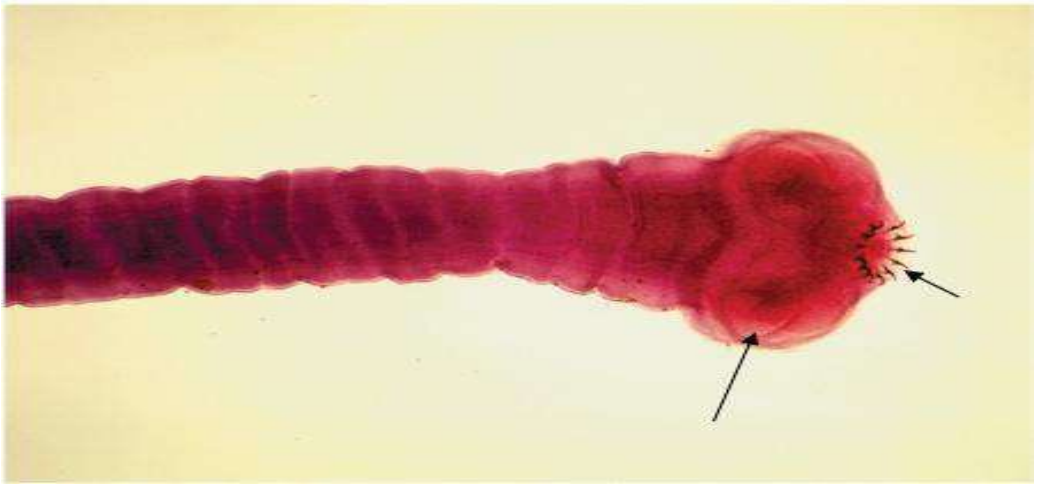


Figure (21): *Taenia solium* - Scolex and several proglottids. Long arrow points to one of the four suckers on the scolex of *Taenia solium*. Short arrow points to the circle of hooklets. Proglottids can be seen extending from the scolex toward the left side of the image.

Life cycle

In taeniasis, the adult tapeworm is located in the human intestine. This occurs when humans are infected by eating raw or undercooked **pork** containing the larvae, called **cysticerci.** (A cysticercus consists of a peasized fluid-filled bladder with an invaginated scolex.) In the small intestine, the larvae attach to the gut wall and take about 3 months to grow into adult worms measuring up to 5 m. The gravid terminal proglottids containing many eggs detach daily, are passed in the feces, and are accidentally eaten by pigs. Note that pigs are infected by the worm eggs; therefore, it is the larvae (cysticerci) that are found in the pig. A six-hooked embryo (oncosphere) emerges from each egg in the pig's intestine. The embryos burrow into a blood vessel and are carried to skeletal muscle. They develop into cysticerci in the muscle, where they remain until eaten by a human.

Humans are the definitive hosts, and pigs are the intermediate hosts.

In cysticercosis, a more dangerous sequence occurs when a person **ingests the worm eggs** in food or water that has been contaminated with human feces. Note that in cysticercosis, humans are infected by eggs excreted in human feces, not by ingesting undercooked pork. Also pigs do not have the adult worm in their intestine, so they are not the source of the eggs that cause human cysticercosis. The eggs hatch in the small intestine, and the oncospheres burrow through the wall into a blood vessel. They can disseminate to many organs, especially the eyes and brain, where they encyst to form cysticerci Image on cysticercosis

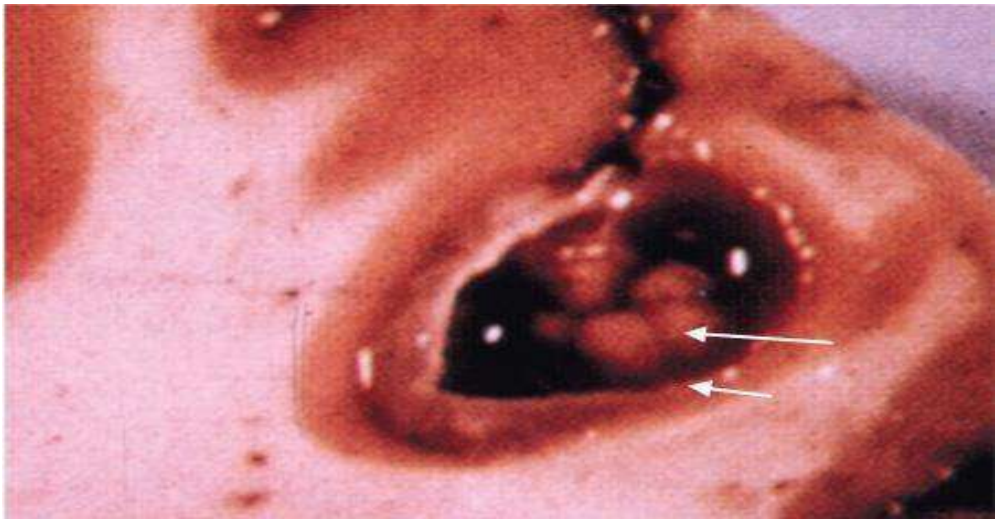


Figure (22): Cysticercus of *Taenia solium* in brain—Long arrow points to a larva of *Taenia solium*. Short arrow points to the wall of the cysticercus (sac) that surrounds the larva

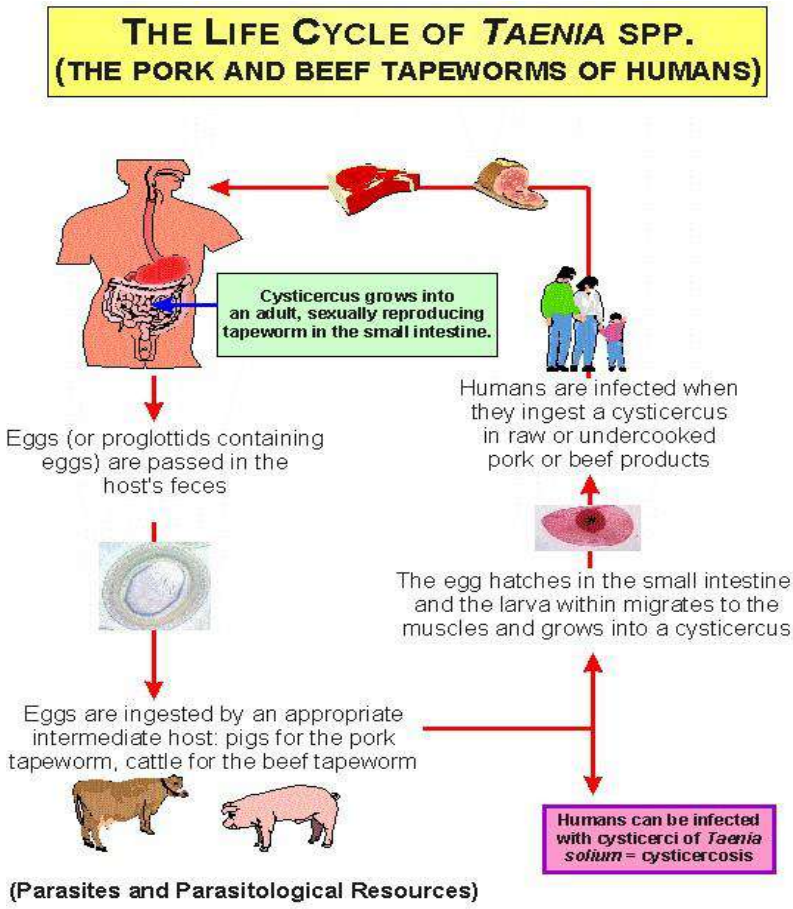


Figure (23): life cycle of *Taenia spp*.

Pathogenesis & Epidemiology

The adult tapeworm attached to the intestinal wall causes little damage. The **cysticerci,** on the other hand, can become very large, especially in the **brain,** where they manifest as a **space-occupying lesion.** Living cysticerci do not cause inflammation, but when they die they can release substances that provoke an inflammatory response. Eventually, the cysticerci calcify. The epidemiology of taeniasis and cysticercosis is related to the access of pigs to human feces and to consumption of raw or undercooked pork. The disease occurs worldwide but is endemic in areas of Asia, South America, and eastern Europe.

Clinical Findings

Most patients with adult tapeworms are asymptomatic, but anorexia and diarrhea can occur. Some may notice proglottids in the stools. Cysticercosis in the brain causes headache, vomiting, and seizures. Cysticercosis in the eyes can appear as uveitis or retinitis, or the larvae can be visualized floating in the vitreous. Subcutaneous nodules containing cysticerci commonly occur.

Laboratory Diagnosis

Identification of *T. solium* consists of finding gravid proglottids with 5–10 primary uterine branches in the stools. In contrast, *T. saginata* proglottids have 15–20 primary uterine branches. Eggs are found in the stools less often than are proglottids. Diagnosis of cysticercosis depends on demonstrating the presence of the cyst in tissue, usually by surgical removal or computed tomography (CT) scan. Serologic tests, e.g., ELISA, that detect antibodies to *T. solium* antigens are available, but they may be negative in neurocysticercosis.

Prevention

One way to prevent taeniasis is to cook meat to safe temperatures. A food thermometer should be used to measure the internal temperature of cooked meat. Do not sample meat until it is cooked.

Treatment

The treatment of choice for the intestinal worms is praziquantel. The treatment for cysticercosis is either praziquantel or albendazole, but surgical excision may be necessary.

Taenia saginata

Disease

Taenia saginata causes taeniasis. *T. saginata* larvae do not cause cysticercosis.

Important Properties & life cycle

Taenia saginata has a scolex with four suckers but, in contrast to *T. solium,***no hooklets.** Its gravid proglottids have 15–25 primary uterine branches, in contrast to *T. solium* proglottids, which have 5–10. The eggs are morphologically indistinguishable from those of *T. solium.*

Humans are infected by eating raw or undercooked **beef** containing larvae (cysticerci). In the small intestine, the larvae attach to the gut wall and take about 3 months to grow into adult worms measuring up to 10 m. The gravid proglottids detach, are passed in the feces, and are eaten by cattle. The embryos (**oncospheres**) emerge from the eggs in the cow's intestine and burrow into a blood vessel, where they are carried to skeletal muscle. In the muscle, they develop into cysticerci. The cycle is completed when the cysticerci are ingested. Humans are the definitive hosts and cattle the intermediate hosts. Unlike *T. solium, T. saginata* **does not cause cysticercosis** in humans.

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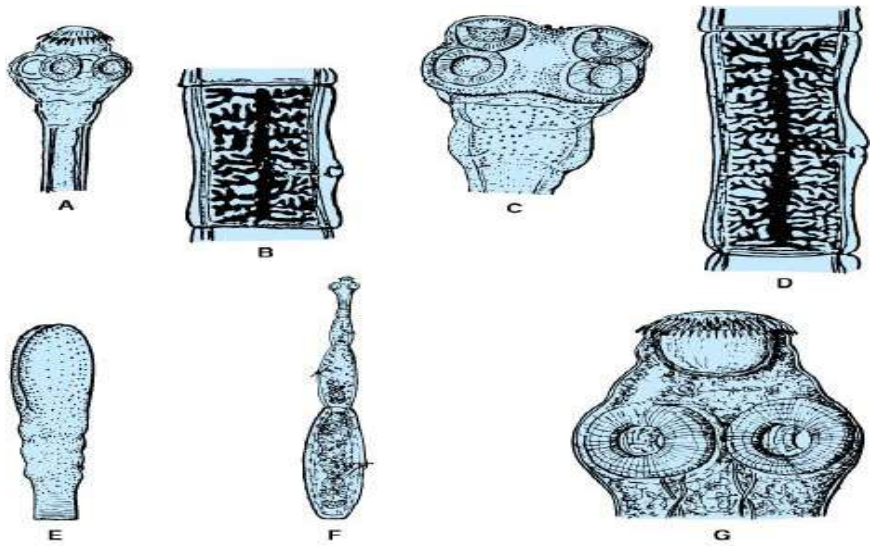

Figure (24): Image on cestoda segment & comparism between (*T.solium & T.saginata*. A: *Taenia solium* scolex with suckers and hooks B:*Taenia solium* gravid proglottid. This has fewer uterine branches than does the proglottid of *Taenia saginata* (see panel D) C:*Taenia saginata* scolex with suckers D:*Taenia saginata* gravid proglottid E:*Diphyllobothrium latum* scolex with sucking grooves F: Entire adult worm of *Echinococcus granulosus* G:*Echinococcus granulosus* adult scolex.

Pathogenesis & Epidemiology

Little damage results from the presence of the adult worm in the small intestine. The epidemiology of taeniasis caused by *T. saginata* is related to the access of cattle to human feces and to the consumption of raw or undercooked beef. The disease occurs worldwide but is endemic in Africa, South America, and eastern Europe. In the United States, most cases are imported.

Clinical Findings

Most patients with adult tapeworms are asymptomatic, but malaise and mild cramps can occur. In some, proglottids appear in the stools and may even protrude from the anus.

Laboratory Diagnosis

Identification of *T. saginata* consists of finding gravid proglottids with 15–20 uterine branches in the stools. Eggs are found in the stools less often than are the proglottids.

Treatment

Praziquantel with a single oral dose of 5 or 10 mg/kg.

Alternatively, niclosamide a single 2-g dose (outside the US).

***Hymenolepis nana* (Dwarf tape worm)**

Hymenolepis nana occurs worldwide in humans and is also a common murine parasite, occasionally developing its cysticercoids stage in beetles. It is the MOST COMMON tapeworm infection in North America. Main properties

The dwarf tapeworm, is the smallest cestode with an adult length of 15-44mm. Scolex of *H. nana* has four suckers, and an armed rostellum that is clearly visible. Eggs are oval or subspehrical and smaller, ranging 40-60 um x 30-50 um. On the inner membrane are two poles, from which 4 to 8 polar filaments spread out between the two membranes. The oncosphere has six hooks.

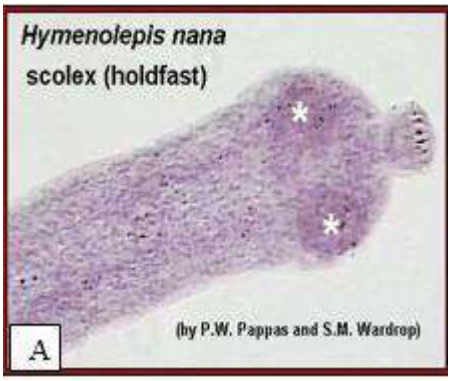

Figure (25): A- scolex B- Egg

Life cycle

The **life cycle** of *Hymenolepis nana* starts, when microscopic **eggs** are passed with the stool of an infected human. They then get ingested either by rodents, humans (definite hosts) or insects (intermediate hosts).

If a person ingests eggs (from contaminated fingers, water, food or soil), oncospheres (hexacanth larvae) hatch in the small intestine. **A larva** penetrates an intestinal villus and develops into a **cysticercoid**.

A cysticercoid develops to look more like an adult having a scolex (head) and a neck. It bursts out of the villus, attaches to the intestinal mucosa and matures into an adult in the last part of the small intestine, ileum. Its long neck starts producing segments, proglottids, which make up the body.

A gravid proglottid releases thousands of eggs through its genital atrium or when its membrane disintegrates. Eggs are immediately infective when passed with the stool and cannot survive more than 10 days in the external environment.

If the eggs are ingested by an insect (a beetle or a flea), the larvae develop inside the insect. **If** the insect is ingested by a rodent or accidentally by a human, the cysticercoid larvae get inside the intestine and develop into adults.

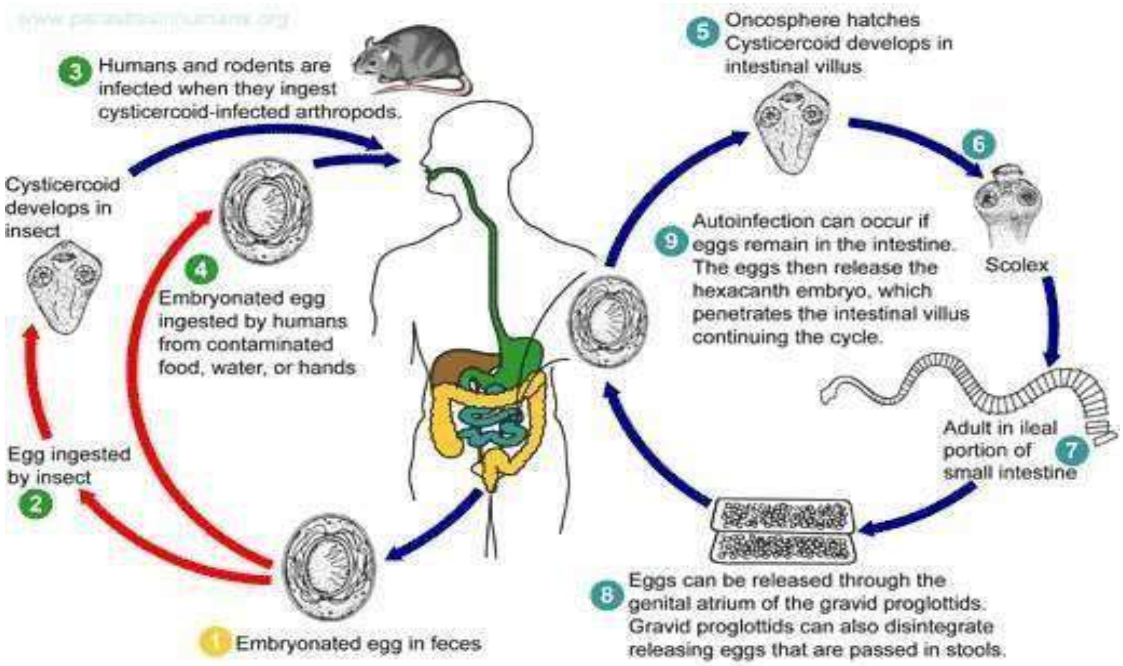

Figure (26): life cycle of *Hymenolepis nana*.

Mode of transmission

Ingestion of contaminated grain and flour with beetles. Autoinfection very common in children in day-care centers. Children are especially at risk of infection.

Clinical manifestations

In cases with low intestinal parasite burden, there are no symptoms. In heavy infections, especially if autoinfection and hyperinfection occur, patients can experience diarrhea, abdominal pain, headache, anorexia, and other vague complications.

Pathology

Life cycle is simple and does not require an intermediate. Embryonated eggs are ingested and develop in the intestinal villi into a larval cysticercoid stage. This larva attaches its four muscular suckers and crown of hooklets to the small intestine and the adult worm produces a strobila of egg-laden proglottids. Eggs passed in the feces are then immediately and directly infective, initiating another cycle. Infection may also be acquired by ingesting infected insect intermediate hosts. AUTOINFECTION (reinfection of the small intestine when eggs are released internally) and HYPERINFECTION also occur, causing subsequent increases in worm burden. Eggs hatch in the intestine, develop into a cysticercoid larvae, and then grow into adult worms without leaving the host.

Diagnosis

The diagnosis is usually suggested by the presence of characteristic *H. nana* eggs upon stool examination (8-10 polar filaments lying between the membrane of the 6-hooked embryo and the shell).

Prevention

Good hygiene, public health and sanitation programs, and the elimination of infected rats help to prevent the spread of hymenolepiasis. Preventing fecal contamination of food and water in institutions and crowded areas is of primary

importance. General sanitation and rodent and insect control (especially control of fleas and grain insects) are also essential for prevention of H. nana infection.

Treatment

Praziquantel is the drug of choice-, an alternative is niclosamide.

Genus: *Echinococcus Granulosus*

Disease

The larva of *Echinococcus granulosus* (dog tapeworm) causes unilocular hydatid cyst disease. Multilocular hydatid disease is caused by *E.multilocularis,* which is a minor pathogen.

Important Properties

Echinococcus granulosus is composed of a scolex and only three proglottids, making it **one of the smallest tapeworms** .The scolex has a circle of hooks and four suckers similar to *T. solium.* **Dogs** are the most important definitive hosts. The intermediate hosts are usually **sheep.** Humans are almost always dead-end intermediate hosts.

In the typical life cycle, worms in the dog's intestine liberate thousands of eggs, which are ingested by sheep (or humans). The oncosphere embryos emerge in the small intestine and migrate primarily to the liver but also to the lungs, bones, and brain. The embryos develop into large fluid-filled **hydatid cysts,** the inner germinal layer of which generates many protoscoleces within "brood capsules." The life cycle is completed when the entrails (e.g., liver containing hydatid cysts) of slaughtered sheep are eaten by dogs.

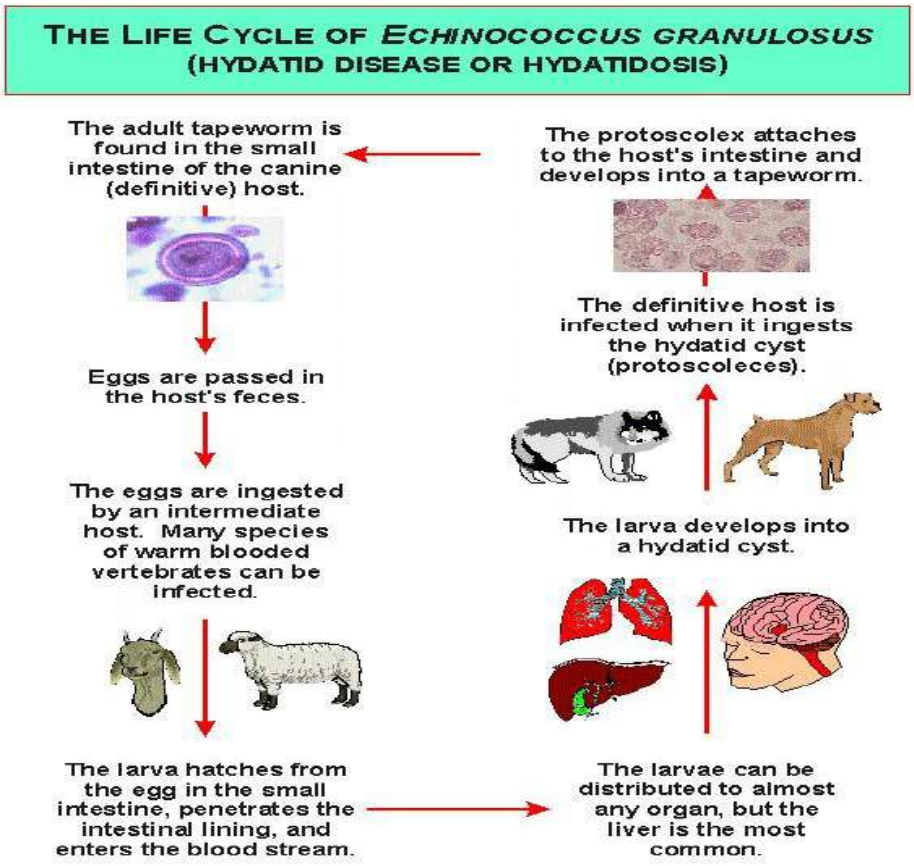

Figure (27): life cycle of *E. granulosus*.

Pathogenesis & Epidemiology

Echinococcus granulosus usually forms one large fluid-filled cyst (unilocular) that contains thousands of individual scoleces as well as many daughter cysts within the large cyst. Individual scoleces lying at the bottom of the large cyst are called "hydatid sand." The cyst acts as a space-occupying lesion, putting pressure on adjacent tissue. The outer layer of the cyst is thick, fibrous tissue produced by the host. The cyst fluid contains parasite antigens, which can sensitize the host. Later, if the cyst ruptures spontaneously or during trauma or surgical removal, lifethreatening **anaphylaxis** can occur. Rupture of a cyst can also spread protoscoleces widely.

Clinical Findings

Many individuals with hydatid cysts are asymptomatic, but **liver cysts** may cause hepatic dysfunction. Cysts in the lungs can erode into a bronchus, causing bloody sputum, and cerebral cysts can cause headache and focal neurologic signs.

Rupture of the cyst can cause fatal anaphylactic shock.

Laboratory Diagnosis

Diagnosis is based either on microscopic examination demonstrating the presence of brood capsules containing multiple protoscoleces or on serologic tests, e.g., the indirect hemagglutination test.

Treatment

Treatment involves albendazole with or without surgical removal of the cyst. Extreme care must be exercised to prevent release of the protoscoleces during surgery. A protoscolicidal agent, e.g., hypertonic saline, should be injected into the cyst to kill the organisms and prevent accidental dissemination.

Diphyllobothrium latum

Disease

Diphyllobothrium latum, the fish tapeworm, causes diphyllobothriasis.

Important Properties

In contrast to the other cestodes, which have suckers, the scolex of *D. latum* has two elongated **sucking grooves** by which the worm attaches to the intestinal wall .The scolex has no hooks, unlike *T. solium* and Echinococcus. The proglottids are wider than they are long, and the gravid uterus is in the form of a rosette. Unlike other tapeworm eggs, which are round, *D. latum* eggs are oval and have a lid like opening (**operculum**) at one end .*D. latum* is the longest of the tapeworms, measuring up to 13 m. Humans are infected by ingesting raw or undercooked **fish** containing larvae (called plerocercoid or sparganum larvae). In the small intestine, the larvae attach to the gut wall and develop into adult worms. Gravid proglottids release fertilized eggs through a genital pore, and the eggs are then passed in the stools. The immature eggs must be deposited in fresh water for the life cycle to continue. The embryos emerge from the eggs and are eaten by tiny copepod crustacea (first intermediate hosts). There, the embryos differentiate and form procercoid larvae in the body cavity. When the

copepod is eaten by freshwater fish, e.g., pike, trout, and perch, the larvae differentiate into plerocercoids in the muscle of the fish (second intermediate host). The cycle is completed when raw or undercooked fish is eaten by humans (definitive hosts).

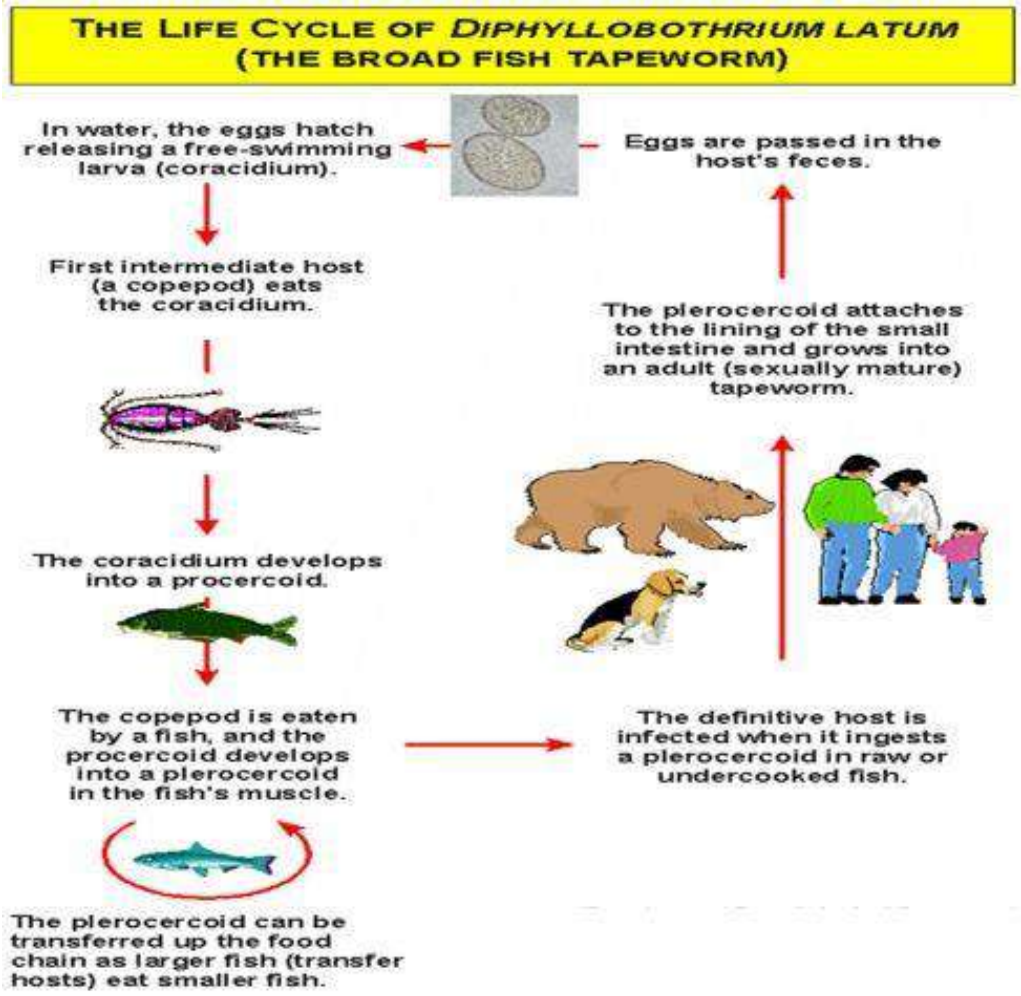

Figure (28): life cycle of *Diphyllobothrium latum*

Pathogenesis

Infection by *D. latum* causes little damage in the small intestine. In some individuals, megaloblastic anemia occurs as a result of vitamin B12 deficiency caused by preferential uptake of the vitamin by the worm.

Clinical Findings

Most patients are asymptomatic, but abdominal discomfort and diarrhea can occur.

Laboratory Diagnosis

Diagnosis depends on finding the typical eggs, i.e., oval, yellow-brown eggs with an operculum at one end, in the stools. There is no serologic test.

Treatment

The standard treatment for diphyllobothriasis (as well as many other tapeworm infections) is a single dose of praziquantel, 5–10 mg/kg orally once for both adults and children.

Nematoda (Roundworm) The general characteristics

Nematodes (Gr., nema thread+ eidos, form) are commonly referred to as nonsegmented roundworm, threadworm, or pinworm, as distinct from flatworm and higher segmented annelids.

1- Generally a light cream-white color, but the females may appear darker when filled with dark-colored eggs.
2- The body is symmetrical, non-segmented, cylindrical, tapered at both ends and, covered in a complex cuticle.
3- The genders are separated (male and female), and generally, males are such female worms but the female slightly longer than the male.
4- Body cavity is pseudocoel filled with parenchyma substances in most cases.
5- They have a complete digestive tract with both oral and anus.
6- Has a nervous system with pharyngeal nerve ring, but no circulatory system.
7- The life cycle of nematodes is direct and required several molts to become an adult worm.
8- The many nematodes are free-living, while others are parasitic.
9- The females are Oviparous, viviparous, or ovoviviparous.

Enterobius vermicularis

Its common name is a pinworm, and its original name was *Oxyuris vermicularis*. *E. vermicularis* is thought to cause the world's most common human parasitic infection. The infection is more prevalent in the cool and temperate zones, where people tend to bathe less often and change their underclothes less frequently.
It is worldwide in distribution and commonly affects children.

Habitat

Adult worm (female resides in cecum and appendix of man).

Morphology

Female 8 to 13 mm by 0.3 to 0.5 mm and has a pointed tail (hence the name "pinworm") (Figure.1).Male is much smaller, 2 to 5 mm by 0.1 to 0.2 mm, and has a curved tail.

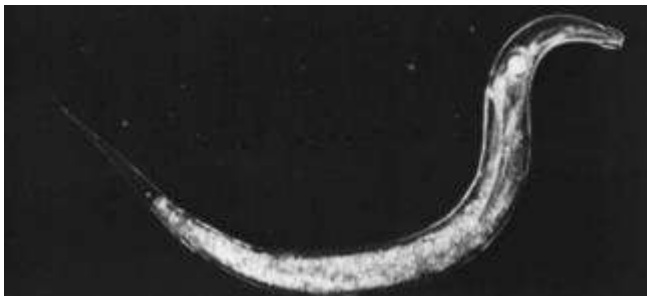

Figure (29): *Enterobius vermicularis*, adult female pinworm.

Life Cycle

Eggs are laid by the gravid female worm in perianal region. Larvae inside the eggs mature within 4–6 h. Embryonated eggs are ingested by humans (fingers to anus to mouth or via inhalation). Eggs hatch in the intestine. Adults develop in the lumen of the large intestine (caecum). The male worm is dies after mating. Gravid female migrates to perianal region at night to lay eggs.

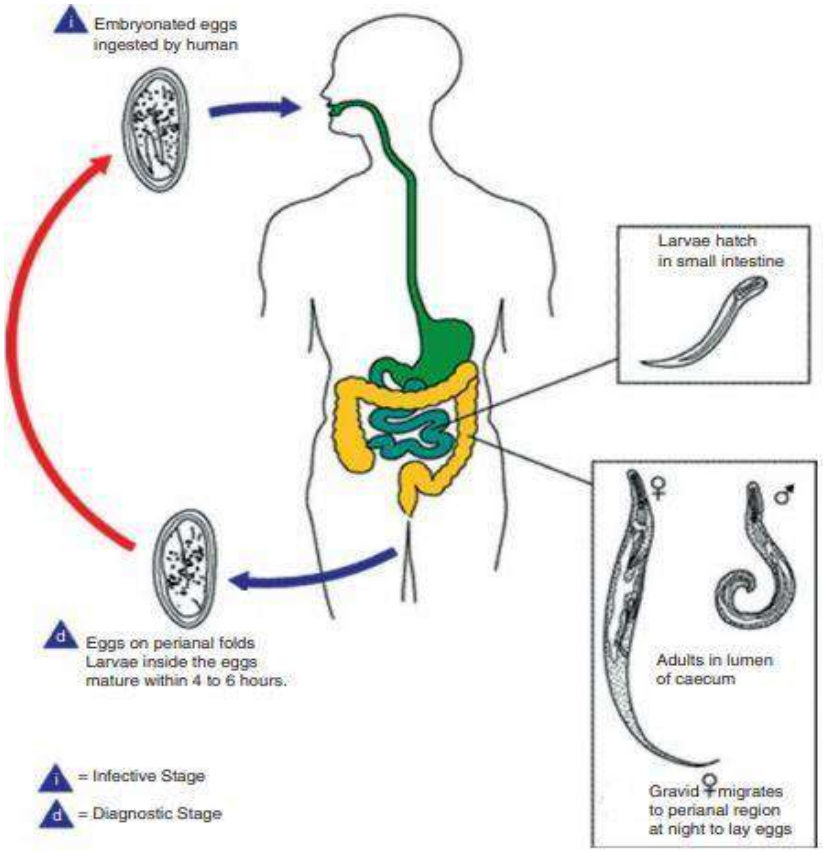

Figure (30): Life cycle of *Enterobius vermicularis*.

Pathogenesis and Clinical Features

Enterobiasis mostly in children, enterobiasis may be asymptomatic, irritation and pruritus of the perianal area especially at night, causes acute appendicitis in children may be and asymptomatic chronic appendicitis.

In women

Vagina irritation, may migrate the adult worms up to the uterus and fallopian tubes, then cause cervicitis and chronic salpingitis.

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.

Treatment: Mebendazole; Piperazine.

Prevention

Washing your hands with soap and warm water after using the toilet, changing diapers, and before handling food is the most successful way to prevent pinworm infection.

Ascaris lumbricoides

Geographical Distribution It is cosmopolitan.

Habitat: Adult worm lives in the lumen of the small intestine (jejunum) of man.

Morphology Adult Worm

They are the largest of the common nematode parasites of humans; females measure 20 to 35 cm long, and males are 15 to 31 cm long, with a curved posterior end (Figure 10.1). Also, the three well developed lips are characteristic of this group.

Elongated cylindrical measuring 15 to 50 cm × 2 to 6 mm. Oral cavity has 2 lateral lips and one median lip, body cavity contains a toxin (ascaron) in which digestive and reproductive organs float, posterior end of male is curved ventrally and male worm is smaller than female worm.

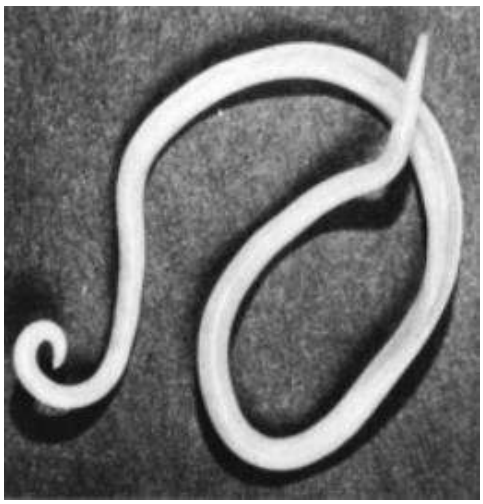

Figure (31): Adult *Ascaris lumbricoides* (male).

Egg a. Fertilized

45 o 75 µ × 35 to 50 µ, the shell has an innermost very thin vitelline membrane, a thick glycogenic middle layer, and a coarsely laminate outermost layer, embryo is unsegmented and made up of coarse lecithin granules and floats in a saturated salt solution.

b. Unfertilized

Larger in size 88 to 94 µ × 45 µ, the shell is relatively thin, the innermost layer vitelline is absent, embryo contains a disorganized mass containing refractile granules, it does not develop into larvae and it does not float in a saturated salt solution.

Life cycle

Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces. Unfertilized eggs may be ingested but are not infective. Larvae develop to infectivity within fertile eggs after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed, the larvae hatch, invade the intestinal mucosa, and are carried via the portal, then systemic

circulation to the lungs. The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed.

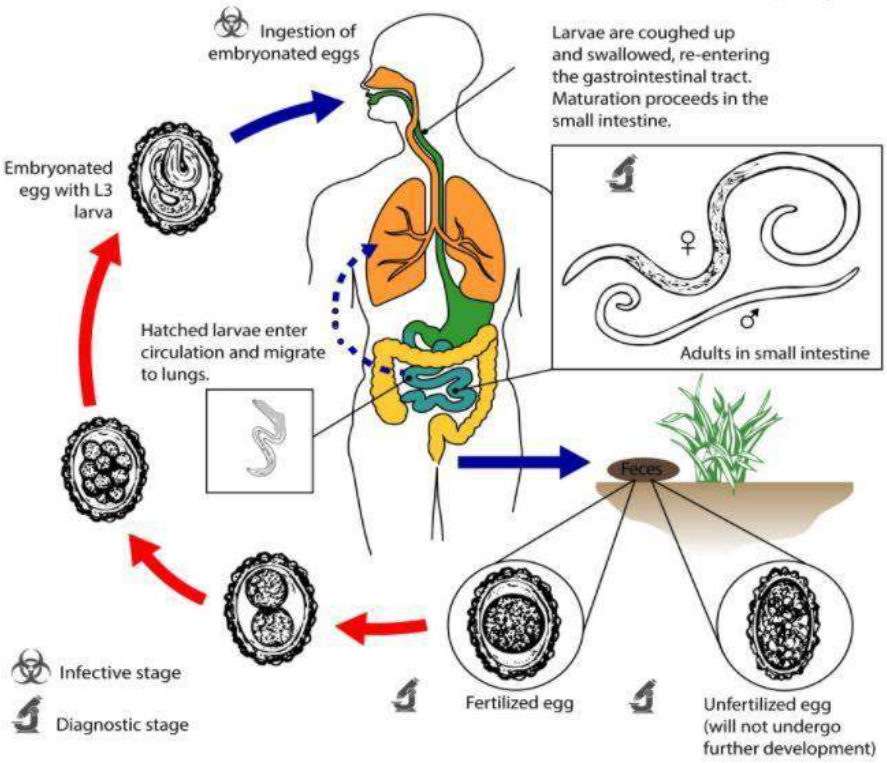

Figure (32): Life cycle of *Ascaris lumbricoides*.

Pathogenicity and Clinical Picture

The larvae in lung alveoli cause migrating pneumonitis (allergic reaction) and this allergic reaction may be due to ascaron. The adult worm may cause malnutrition, vague abdominal pain, colic pain, poor digestion, diarrhea, etc.

Perforation of bowel, appendicitis and diverticulitis may occur.

Laboratory Diagnosis

Detection of adult worms in stool. Microscopic detection of eggs in feces or bile obtained by duodenal intubation. Eosinophilia. Dermal reaction (allergic), scratch test with powdered ascaris antigen.

Treatment

Piperazine, decaris, albendazole, mebendazole are effective.

HOOK WORMS

There are two species of hookworm:

1. *Ancylostoma duodenale* (Old World hookworm).
2. *Necator americanus* (New World hookworm).

Ancylostoma duodenale (Old World hookworm)

Characteristics

Intestinal nematode, Adult hookworms are small cylindrical and creamy-white; males measure 8-11 mm in length and 0.45 mm width, females are 10-13 mm long, 0.60 mm wide, Eggs are 50-60 µm long and 35-40 µm wide and head continues in the same direction as curvature of the body.

Geographic Range: It is widely found in tropical and subtropical countries. **Habitat:** The adult worm resides in the small intestine of man particularly in the jejunum.

Morphology: Adult female hookworms are about 11 mm x 50 micrometers. Males are smaller. The anterior end of A. duodenale is equipped with one or more pairs of teeth. Hookworm eggs are 60 micrometers x 35 micrometers.

Life Cycle

Eggs are in the stool. The larvae hatch in the soil after 1 to 2 days. Rhabditiform larvae become filariform larvae after 5 to 10 days which are the infective stage. The larvae penetrate the human skin. The larvae reach the heart and lungs, then reach into the pharynx. The larvae swallowed to reach the small intestine, where they mature into adults. Most adult worms are eliminated in 1 to 2 years, may reach several years.

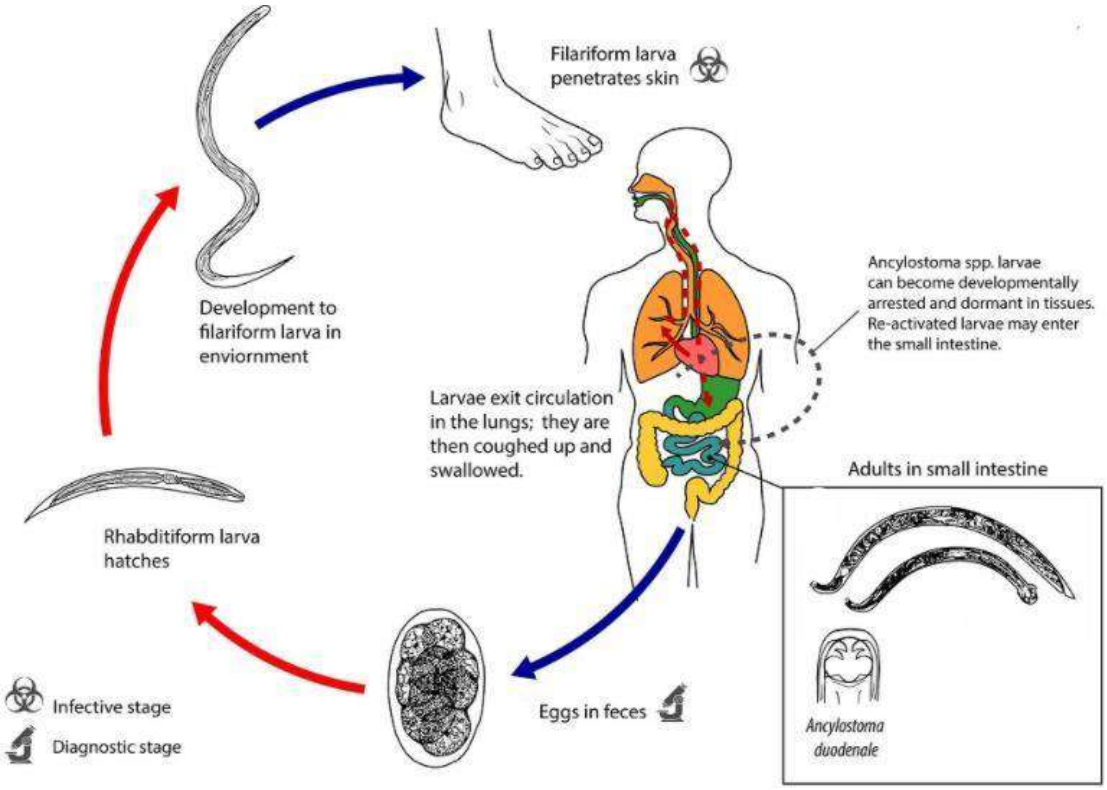

Figure (33): Life cycle of *Ancylostoma duodenale* (hookworms).

Clinical Features

In acute infection.

From minimal to severe pruritus. Secondary infection if the lesions are opened by scratching. Pneumonitis and increased peripheral eosinophilia. Necrosis of the intestinal tissue. Blood loss. Fatigue, nausea, vomiting, abdominal pain. Diarrhea with black to red stools. Weakness, laziness, and pallor.

In chronic infection.

Anemia and edema of the face and feet. Hemoglobin levels of 5 g/dl or less. There may be cardiomegaly. Mental and physical retardation. Death especially in children.

Laboratory Diagnosis

Stool examination for adult worm (naked eye). Microscopic examination of stool for ova. Study of duodenal contents for adult worm and ova. Blood examination for

anemia and eosinophilia. Stool examination for occult blood and Charcot-Leydon crystals.

Prevention

Do not walk barefoot in areas where hookworm is common and where there may be fecal contamination of the soil. Avoid other skin-to-soil contact and avoid ingesting such soil. Fecal contamination occurs when people defecate outdoors or use human feces as fertilizer.

Treatment

Anthelminthic medications (drugs that rid the body of parasitic worms), such as albendazole and mebendazole, are the drugs of choice for treatment of hookworm infections.

***Necator americanus* (New World hookworm)**

Necator americanus is a species of hookworm (a type of helminth) commonly known as the New World hookworm. Like other hookworms. It is lives in the small intestine of human.

Geographic Range: *Necator americanus* is found in Africa, Asia, and Europe but is predominately found in the Americas and in Australia. In the United States, the largest concentration is found in the southern and southwestern United States. In the rest of the world, *N. americanus* is found in tropical climates, it infects an estimated 576-740 million people.

Habitat: It is lives in the small intestine of human.

Morphology: This parasite has two dorsal and two ventral cutting plates around the anterior margin of the buccal capsule. It also has a pair of subdorsal and a pair of subventral teeth located close to the rear. Males are usually 7–9 mm long, whereas females are about 9–11 mm long. The typical lifespan of these parasites is 3–5 years. They can produce between 5,000 and 10,000 eggs per day.

Life Cycle

The unembryonated egg in the soil. After 24–48 hours under favorable conditions, the eggs become embryonated and hatch what is known as 'rhabditiform'. The **rhabditiform** larvae grow and molt in the soil, transforming into a juvenile stage 2. The juvenile stage 2 molts once more until reaching the juvenile 3 stage, which is also called **'filariform'**; this is also the infective form. The transformation from rhabditiform to the filariform usually takes 5-10 days. This larval form is able to penetrate human skin, travel through the blood vessels and heart, and reach the lungs. Once there, it burrows through the pulmonary alveoli and travels up the trachea, where it is swallowed and carried to the small intestine. There, it attaches to the intestinal wall, and matures into an adult and begins reproduction. Adults live in the lumen of the intestinal wall, where they cause blood loss to the host. The eggs produced by the adults end up on the soil after leaving the body through the feces; female hookworms produce up to 30,000 eggs per day. On average, most adult worms are eliminated in 1–2 years. The *N. americanus* lifecycle only differs slightly from that of *A. duodenale*. *N. americanus* has no development arrest in immune hosts and it must migrate through the lungs.

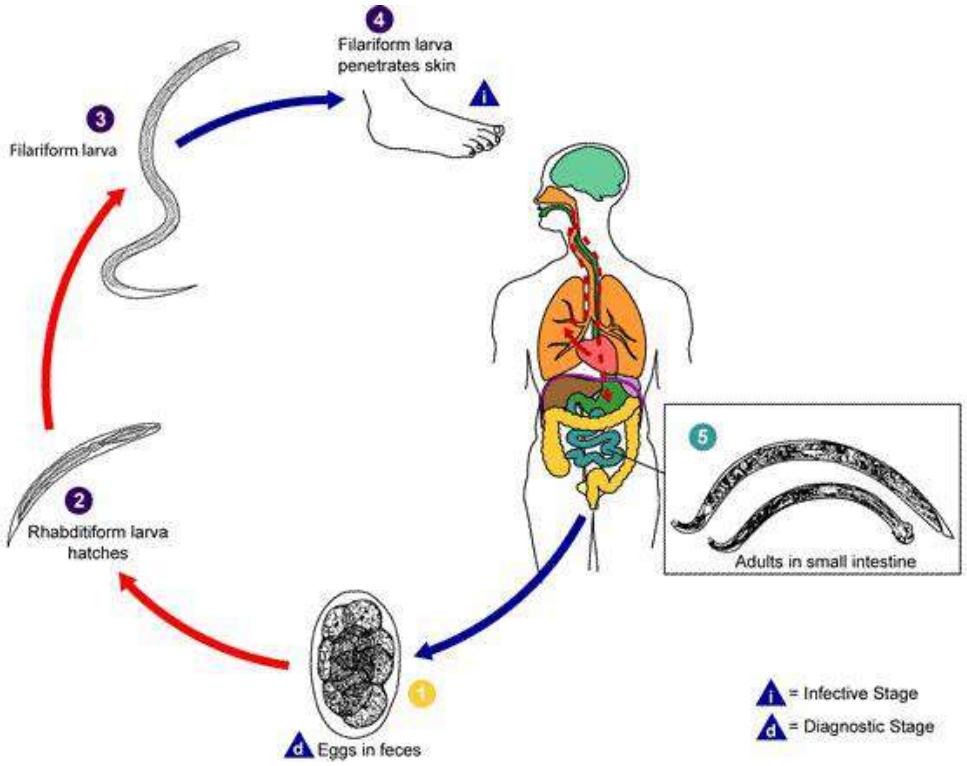

Figure (34): Life cycle of *N. americanus* (hookworms).

Pathogenesis and symptoms

The larvae penetrate the skin and travel into the respiratory tract and lymph nodes so entering the blood, lungs, and intestine. Some larvae cannot readily enter the dermis and remain trapped in the skin, causing skin irritation and cutaneous larva migrans. Other symptoms include excessive coughing and dyspnea (short of breath).

In the intestine, larvae reside and mature into adults, and sucks blood causes anemia. The incubation process arise for up to 40 days. The anemia causing mental retardation and growth insufficiency in children also this worm cause abdominal pain with diarrhea, bloating, and nausea.

Laboratory Diagnosis

Diagnose hookworm by taking a stool sample and using a microscope to look for the presence of hookworm eggs.

Prevention hookworm infection

Do not walk barefoot in areas where hookworm is common and where there may be fecal contamination of the soil. Avoid other skin-to-soil contact and avoid ingesting such soil. Fecal contamination occurs when people defecate outdoors or use human

feces as fertilizer. The infection of others can be prevented by not defecating outdoors or using human feces as fertilizer, and by effective sewage disposal systems.

Treatment hookworm infection

Once you have been diagnosed with hookworm disease, your healthcare provider may prescribe medicine mebendazole or albendazole. You might also be given an iron supplement with this treatment.

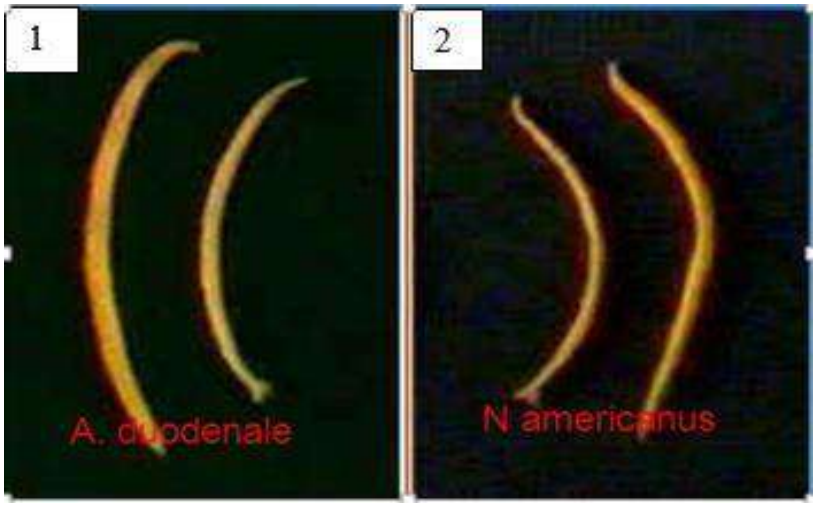

Figure (35): Difference in morphology:

1- ***Ancylostoma Duodenales*. 2-** ***Necator americanus*.**

Strongyloides strocoralis

Geographical Distribution

Strongyloides stercoralis is broadly distributed in tropical and subtropical areas across the globe.

Habitat

The adult parasitic stage lives in tunnels in the mucosa of the human's small intestine

Morphology

Morphology. Strongyloides stercoralis is one of the smallest parasites known to infect humans. Female filariform larvae (males are thought to be non-parasitic) are slender and fast-moving, being approximately 50 μm in diameter and between 350600 μm in length. Rhabditiform larvae are shorter and slower, 60 μm in diameter and between 250-300 μm in length.

Life cycle

The life cycle of *Strogyloides stercoralis* alternates between free-living and parasitic cycles, and includes adult worms, two different larval stages, and eggs. These cycles form the basis for autoinfection and multiplication within the host, features relatively unique among the helminths to Strongyloides. Soil-living adult worms produce eggs, which give rise to non-infective rhabditiform larvae. These either continue the free-living cycle by maturing into adults or become infective filariform larvae. Filariform larvae can penetrate intact human skin, after which they migrate to the lungs. From there, they are expectorated, swallowed, and reach the small intestine; this journey takes about 3-4 weeks. In the intestine, S. stercoralis matures into adult worms, which are semi-translucent and about 2mm long. These produce eggs, which hatch and become rhabditiform larvae.

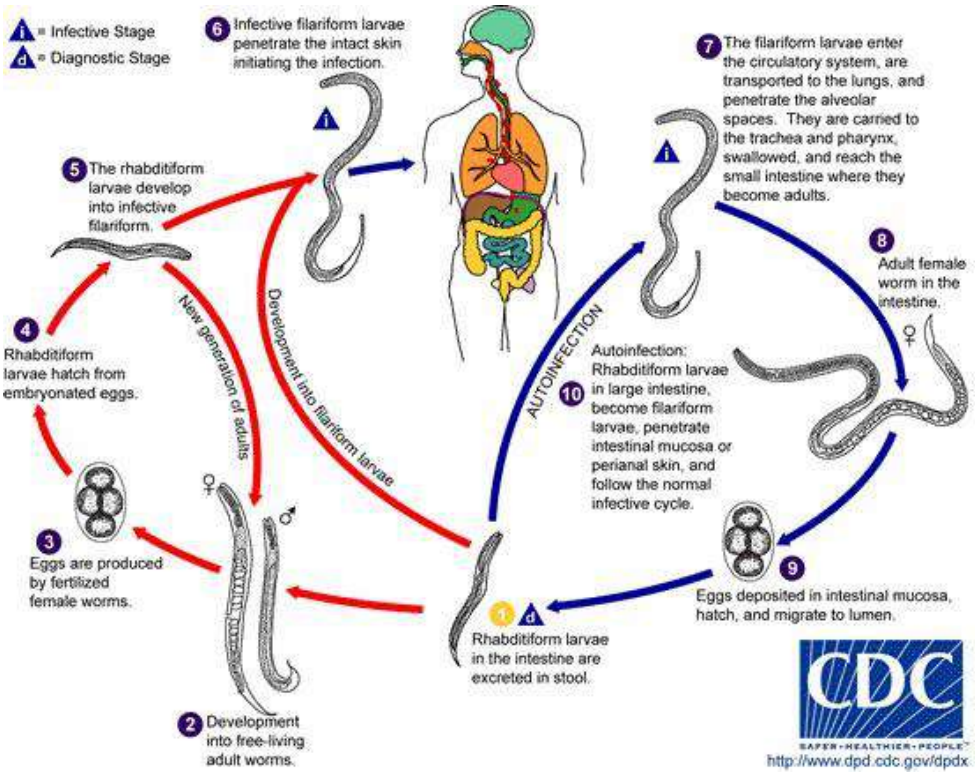

Figure (36): life cycle of *Strongyloides strocoralis*.

Pathogenesis and symptoms

Many people infected are asymptomatic at first. Symptoms include dermatitis: swelling, itching, larva currens, and mild hemorrhage at the site where the skin has been penetrated. Spontaneous scratch-like lesions may be seen on the face or elsewhere. If the parasite reaches the lungs, the chest may feel as if it is burning, and wheezing and coughing may result, along with pneumonia-like symptoms (Löffler's syndrome). The intestines could eventually be invaded, leading to burning pain, tissue damage, sepsis, and ulcers. Stools may have yellow mucus with a recognizable smell. Chronic diarrhea can be a symptom. In severe cases, edema may result in obstruction of the intestinal tract, as well as loss of peristaltic contractions.

Laboratory Diagnosis

If found, either rhabditiform or filariform, in recent stool samples will confirm the presence of this parasite. Other techniques used include direct fecal smears, culturing fecal samples on agar plates, serodiagnosis through ELISA, and duodenal fumigation. Still, diagnosis can be difficult because of the day-to-day variation in juvenile parasite load, and larvae can be found in the stool about 3 to 4 weeks later.

Prevention & Control

The best way to prevent Strongyloides infection is to wear shoes when you are walking on soil, avoid contact with fecal matter or sewage, and clean up after dogs. Proper sewage disposal and fecal management are keys to prevention.

Treatment

The drug of choice for strongyloidiasis is ivermectin, which kills the worms in the intestine at 200 μg/kg (7). Two doses are given 1–14 days apart, which has a cure rate of 94–100%.

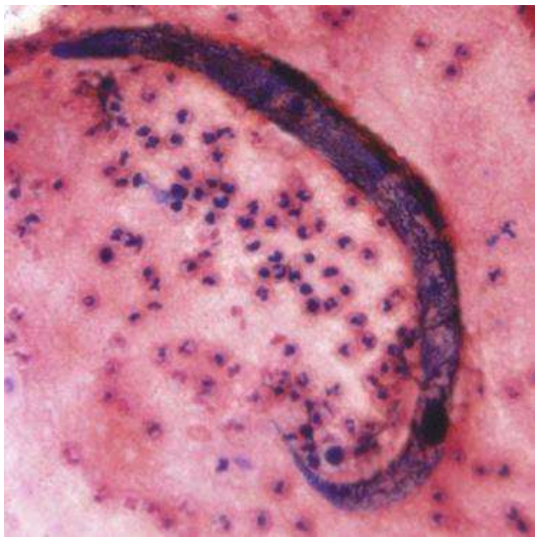

Figure (37): Strongyloides larva in sputum gram stain.

Diagnosis

Although most of these larvae exit the gastrointestinal tract via the stool and subsequently develop into adult worms in the soil, a small number directly become infective (filariform) larvae within the gut and penetrate the intestinal mucosa or perianal skin, completing the life cycle without leaving the host This is termed autoinfection, and distinguishes S. stercoralis from nearly all other helminths in several ways, including indefinite persistence in a host (in the absence of treatment), multiplication in the absence of exogenous re-infection, and potential person-toperson transmission.

Pretest: الاختبار القبلي:

Q :What is medical parasitology ?

Q :write the difference in morphology between. Amastigotes and promastigotes of *Leishmania spp*?

Q- Enumerate the advantages of sedimentation techniques?

Q- Which protozoans is transmitted primarily by the motile trophozoite form ?

Q:Discuss how Cysticercus cellulosae invasion the human body?

Q: Explain how the invasion of schistosomes into the human body

1- The life cycle of *Ascaris lumbricoides* 2- *Schistosom spp*
3-Scolex and egg of *Hymenoleps nana* 4- The life cycle of *Taenia spp*

Q -Give the habitat, infective stage, and modes of infection, for <u>two</u> of the following parasites :
1- Echinococcus granulosus . 2. Taenia solium.*3- *Schistosom mansoni

Q Fill in the blanks

1-The larval stage of dog tapeworm is ----------.
2-Man could be final and intermediate host in some cestoda like ----------
3-Hydatid cyst is mainly covered by two layers: ----------&----------
4-Adult *Schistosoma haematobium* inhabit ----------.

Q: Enumerate the methods of transmission of pathogens through arthropods with an example of each type of transmission?

Q: Explain in detail the differentiation between Subfamily Anophelinae and Subfamily Culicinae with an example for each species?Trichinella spiralis

Posttest الاختبار البعدي:

Q Draw with labeling (choose only three):

:

Posttest الاختبار البعدي:

References: المصادر:

References :

1-Paniker’s Textbook of Medical ParasitologyJAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD New Delhi • London • Philadelphia • Panama (2013).

2- -AI-kaysi, M.A;AI-SaIihi M.A; Mohammed,A.A(2022). Practical Diagnostic ParasitoIogy & EntomoIogy ,Noor OmniScriptum Publishing group,Russo,

3-AI-kaysi, M.A; Mohammed,A.A(2022). Medical parasitology , lambert publisher