CESTODES (TAPEWORMS)

INTRODUCTION

The tapeworms are hermaphroditic and require an intermediate host. The adulttapeworms found in humans have flat body, white or grayish in color. Theyconsist of an anterior attachment organ or scolex and a chain of segments (proglottids) also called strobilla.

The strobilla is the entire body except thescolex. The scolex has suckers or grooves.

It has rosetellum, which has 1 or 2rows of hooks situated on the center of the scolex.

Adult tapeworms inhabit the small intestine, where they live attached to themucosa.

Tapeworms do not have a digestive system. Their food is absorbed

from the host's intestine.

H*ymenolepis nana* (DWARF TAPEWORM)

Morphology

Adult worm measures 1-3 cm in length. It is made up of head (scolex), neck andsegmented body.

The head carries four suckers and a rostellum armed with one

row of hooks. The segments of the body are divided into mature and gravidsegments. In the mature segment, there are three testes in the middle.

Infective stage and mode of infection: The egg, which is immediately infective when passed by the patient, is rounded,about 40 microns in diameter.

It contains a six- hooked oncosphere within a rigidmembrane (the embryosphere).

This embryosphere has two polar thickening orknobs from which project 4-8 long, thin filaments called polar filaments

Infection takes place by:

1. Ingestion of egg with contaminated raw vegetables.

2. Direct infection from a patient

3. Auto infection: the eggs of H. nana are infective as soon as they are passed with feces by the patient. If the hands of the patient are contaminated by these eggs, she/he infects herself/himself again and again.

Pathogenecity

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

ECHINOCOCCUS

There are two different species. These are: *Echinococcusgranulosus* and*Echinococcusmultilocularis*.

***Echinococcusgranulosus* (dog tape worm)**

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

Morphology

The adult worm measures 3-6 mm in length (up to 1 cm). It has scolex, neck andstrobilla.

Adult worms live in small intestine of definitive host (dog). Man is an

intermediate host - carrying the hydatid cyst (larva). Man contracts infection byswallowing eggs in excreta of definitive host.

Life cycle and Pathogenecity

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

♦ Penetrate wall

♦ Enter portal veins

♦ Migrate via portal blood supply to organs: eg: lungs, liver, brain etc.,thus, causing extra intestinal infections. In these organs, larvae develop intohydatid cysts.

The cysts may be large, filled with clear fluid and containcharacteristic protoscolices (immature forms of the head of the parasite).

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

Mode of human infection

Ingestion of eggs by the following ways:

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

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

Clinical features

Asymptomatic infection is common, but in symptomatic patients

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

♦ Hepatomegaly - with abdominal pain and discomfort

♦ Pressure -from expanding cyst

♦ Rupture of cyst - severe allergic reaction - anaphylaxis.

Diagnosis:

♦ X-ray or other body scans

♦ Demonstration of protoscolices in cyst after operation

♦ Serology

Echinococcusmultilocularis

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

Taeniasaginata (beef tapeworm)

In adult stage, *T. saginata* inhabits the upper jejunum where it may survive for aslong as 25 years.

It causes intestinal infection, Taeniasis. It has worldwidedistribution.

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

1. Scolex: the hold fast organ

2. Neck: posterior to the scolex

3. Stobilla: the main bulk, made up of proglottids.

Morphology:

Adult worm measures 5-10 meters in length. The pyriformscolex has 4 suckersbut no rostellum.

The mature segments have irregularly alternate lateral genital

pores. Each of the terminal segments contains only a uterus made up of amedian stem with 15-30 lateral branches.

Life cycle

The adult worm lives in the small intestine of man. Gravid segments pass out inthe stool and become disintegrated and eggs come out to the soil.

The gravidproglottid uterus contains about 100,000 eggs. The egg of T. saginata is round,about 40 microns in diameter.

The 6-hooked embryo is enclosed in a radiallystriated embryophore.

Eggs are ingested by an intermediate host, cattle. The 6-hooked embryo escapes from its shell, penetrates through the intestinal wall intothe bloostage, cysticercusbovis (made up of aninvaginated/inverted

head andspherical body)d vessels and is carried to the muscles where it develops into a larval.

Infection to man takes place by the ingestion of raw orinsufficiently cooked beef.

In the small intestine of man, the head of thecysticercus gets invaginated and the body becomes segmented.

Pathogenecity

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

Diagnosis

Recovery of the gravid segments or the eggs from the stool

Prevention:

♦ Thorough cooking of meat (above 570C).

♦ Proper disposal of human excret

Taeniasolium (pork tapeworm)

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

Morphology:

Adult worm measures about 3 meters in length. The globular scolex has

rostellum with 2 rows of hooklets. There are <1000 proglottids.

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

Life cycle

Embryonated eggs passed with stool are ingested by pig and the embryo isreleased.

It penetrates the intestinal wall and is carried by vascular channels to

all parts of the body. After a period of 2-3 months of development the

encysted larval stage called cysticerci or bladder worm occurs in the striated muscles ofthe tongue, neck, trunk brain, eye, and the nervous system.

The cysticercussurvives for 5 years. Humans become infected by eating pork containing larvae,cysticercuscellulosae.

When improperly cooked cysticercus infected meat iseaten by man, the scolex remains undigested and attaches itself to the intestinalwall and chain of proglottids begin to grow to adult worm.

Clinical manifestations

Resembles that of *T. saginata* infection

Diagnosis

Demonstration of eggs in stool specimen

Prevention:

♦ Treatment of infected persons.

♦ Thorough cooking of pork and proper processing

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

Diphylobotriumlatum(fish tapeworm or broad tapeworm)

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

Morphology

Diphylobotriumlatum is the broadest and longest tapeworm. The adult wormmeasures up to 30 feet with 3000-4000 proglottids, which are wider than they arelong.

The tapeworm has no rostellum hooks or suckers.

Life cycle

Unlike Taenia, the gravid segments are retained by the worm. Operculated eggspassed in feces hatch into small ciliated coracidium larvae which swim about freely.

These are eaten by crustaceans -Cyclops or Diaptomus - in which the

larvae develop into second stage larvae- the procercoid. When the crustaceansare swallowed by fresh water fish, the larvae migrate into the flesh of the musclefish and develop to pleurocercoid or sparganum larvae.

Humans are infected byingesting raw or improperly cooked fish. The tapeworm matures in the intestineand after 3 weeks, the adult worm discharges eggs. The life cycle requires two intermediate hosts.

Clinical manifestation

Most infections are asymptomatic. Rarely, it causes severe cramping, abdominalpain, vomiting, weakness and weight loss.

Pernicious anemia can also result,due to interference of vitamin B12 absorption in jejunum.

Diagnosis

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

DIFFERENT KINDS OF PARASITES

• Ectoparasite – a parasitic organism that lives on the outer surface of its host, e.g.

lice, ticks, mites etc.

• Endoparasites – parasites that live inside the body of their host, e.g. *Entamoeba histolytica*.

• Obligate Parasite - This parasite is completely dependent on the host during a segment or all of its life cycle, e.g. Plasmodium spp .

• Facultative parasite – an organism that exhibits both parasitic and non-parasitic modes of living and hence does not absolutely depend on the parasitic way of life, but is capable of adapting to it if placed on a host. E.g*. Naegleria fowleri*

• Accidental parasite – when a parasite attacks an unnatural host and survives. E.g.

Hymenolepis diminuta (rat tapeworm).

• Erratic parasite - is one that wanders in to an organ in which it is not usually found.

E.g*. Entamoeba histolytica* in the liver or lung of humans.

DIFFERENT KINDS OF HOSTS

• Definitive host – a host that harbors a parasite in the adult stage or where the parasite undergoes a sexual method of reproduction .

• Intermediate host - harbors the larval stages of the parasite or an asexual cycle of development takes place. In some cases, larval development is completed in two different intermediate hosts, referred to as first and second intermediate hosts.

• Paratenic host – a host that serves as a temporary refuge and vehicle for reaching an obligatory host, usually the definitive host, i.e. it is not necessary for the completion of

the parasites life cycle.

• Reservoir host – a host that makes the parasite available for the transmission to another host and is usually not affected by the infection.

• Natural host – a host that is naturally infected with certain species of parasite.

• Accidental host – a host that is under normal circumstances not infected with the host

. CLASSIFICATION OF THE PATHOGENIC PROTOZOA:

PROTOZOA ORGAN OF IMPORTANT HUMAN

LOCOMOTION PATHOGENS

1. Rhizopoda Pseudopodia *Entamoeba histolytica*

(Amoeba)

2. Mastigophora Flagella Trypanosomes

(Flagellates) Leishmania

Trichomonas

Giardia

3. Sporozoa None, exhibit a slight Plasmodium.Spp

Amoeboid movement

4. Ciliates Cilia *Balantidium coli*

Entamoeba Histolytica - Amoebiasi

Entamoeba histolytica is a protozoan parasite responsible for a disease called amoebiasis. It occurs usually in the large intestine and causes internal inflammation as its name suggests (histo = tissue, lytic = destroying). 50 million people are infected worldwide, mostly in tropical countries in areas of poor sanitation. In industrialized countries most of the infected patients are immigrants, institutionalized people and those who have recently visited developing countries.

Inside humans *Entamoeba histolytica* lives and multiplies as a trophozoite. Trophozoites are oblong and about 15–20 µm in length. In order to infect other humans they encyst and exit the body. The life cycle of *Entamoeba histolytica* does not require any intermediate host. Mature cysts (spherical, 12–15 µm in diameter) are passed in the feces of an infected human. Another human can get infected by ingesting them in fecally contaminated water, food or hands. If the cysts survive the acidic stomach, they transform back into trophozoites in the small

intestine. Trophozoites migrate to the large intestine where they live and multiply by binary fission. Both cysts and trophozoites are sometimes present in the feces. Cysts are usually found in firm stool, whereas trophozoites are found in loose stool. Only cysts can survive longer periods (up to many weeks outside the host) and infect other humans. If trophozoites are ingested, they are killed by the gastric acid of the stomach. Occasionally trophozoites might be transmitted during sexual intercourse.

Most *Entamoeba histolytica* infections are asymptomatic and trophozoites remain in the intestinal lumen feeding on surrounding nutrients. About 10–20 % of the infections develop into amoebiasis which causes 70 000 deaths each year. Minor infections (luminal amoebiasis) can cause symptoms that include:

gas (flatulence)

intermittent constipation

loose stools

stomach ache

stomach cramping.

Severe infections inflame the mucosa of the large intestine causing amoebic dysentery. The parasites can also penetrate the intestinal wall and travel to organs such as the liver via bloodstream causing extraintestinal amoebiasis. Symptoms of these more severe infections include:

anemia

appendicitis (inflammation of the appendix)

bloody diarrhea

fatigue

fever

gas (flatulence)

genital and skin lesions

intermittent constipation

liver abscesses (can lead to death, if not treated)

malnutrition

painful defecation (passage of the stool)

peritonitis (inflammation of the peritoneum which is the thin membrane that lines the abdominal wall)

pleuropulmonary abscesses

stomach ache

stomach cramping

toxic megacolon (dilated colon)

weight loss.

Prevention:

To prevent spreading the infection to others, one should take care of personal hygiene. Always wash your hands with soap and water after using the toilet and before eating or preparing food. Amoebiasis is

common in developing countries. Some good practices, when visiting areas of poor sanitation:

Wash your hands often.

Avoid eating raw food.

Avoid eating raw vegetables or fruit that you did not wash and peel yourself.

Avoid consuming milk or other dairy products that have not been pasteurized.

Drink only bottled or boiled water or carbonated (bubbly) drinks in cans or bottles.

Natural water can be made safe by filtering it through an "absolute 1 micron or less" filter and dissolving iodine tablets in the filtered water. "Absolute 1 micron" filters are found in outdoor/camping supply stores. Micron = micrometer = 0.001 mm.

Amoebiasis is diagnosed by your health care provider under a microscope by finding cysts and (rarely trophozoites) from a stool sample. The results are usually said to be negative, if Entamoeba histolytica is not found in three different stool samples. But it still does not necessarily mean that you are not infected because the microscopic parasite is hard to find and it might not be present the particular samples. A blood test might also be available but is only recommended, if your health care provider believes that the infection could have spread to other parts of the body. Trophozoites can be identified under

a microscope from biopsy samples taken during colonoscopy or surgery.

Entamoeba histolytica should be differentiated from the non-pathogenic Entamoeba dispar. The two are morphologically identical and differentiation must be based on immunologic or isoenzymatic analysis or molecular methods. They can be distinguished under a microscope, if *Entamoeba histolytica* has ingested red blood cells. Entamoeba dispar is about 10 times more common. If either one is found, then you are usually treated.

Most of these amoebae are commensal organisms that can parasitize the human

OTHER AMEBAE INHABITING THE ALIMENTARY CANAL

gastrointestinal tract.

Entamoeba hartmanni in all of its life–cycle stage, *E.hartmanni* resembles *E.histolytica*

except in size, yet there is a slight overlap in the size range. The trophozoites do not

ingest red blood cells, and their motility is generally less vigorous than that of E.histolytica. As in other amebae, infection is acquired by ingestion of food or water contaminated with cyst-bearing faeces. Identification is based on examination of small amebae in unstained or iodine-stained preparations. Usually no treatment is indicated,

measures generally effective against faecal-borne infections will control this amoebic

Entamoeba coli the life cycle stages include; trophozoite, precyst, cyst, metacyst, and metacystic trophozoite. Typically the movements of trophozoites are sluggish, with broad short pseudopodia and little locomotion, but at a focus the living specimen cannot

be distinguished from the active trophotozoite of *E.histolytica*. However, the cysts are remarkably variable in size. Entamoeba coli is transmitted in its viable cystic stage through faecal contamination. E.coli as a lumen parasite is non-pathogenic and produces no symptoms. The mature cyst (with more than four nuclei) is the infective stage

treatment to differentiate E.coli from the pathogenic E.histolytica. Specific is not indicated since this amoeba is non-pathogenic. The presence of *E.coli* in stool specimen is evidence for faecal contamination

. Prevention depends on better personal hygiene and sanitary disposal of human excreta.

Entamoeba polecki- arelatively cosmopolitan parasite of hog and monkey. It can cause human disease but is rarely isolated. The disease is manifested as mild, transient diarrhoea. The diagnosis of *E.polecki* infection is confirmed by the microscopic diarrhoea. The diagnosis of E.polecki infection is confirmed by the microscopic

detection of cysts in stool specimens. Treatment is the same as for

E.histolytic

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Entamoeba gingivalis - only the trophozoite stage presents, and encystation probably does not occur. E.gingivalis is a commensal, living primarily on exudate from the margins of the gums, and thrives best on unhealthy gums. No specific treatment isindicated
. However the presence of *E.giingivalis* suggests a need for better oral hygiene

. The infection can be prevented by proper care of the teeth and gums.

Blastocystis hominis- is an inhabitant of the human intestinal tract previously regarded as non-pathogenic yeast

. Its pathogenecity remains controversial. The organism is

found in stool specimen from asymptomatic people as well as from people with persistent diarrhoea. B.hominis is capable of pseudopodia extension and retraction,

reproduces by binary fission or sporulation. The classic form that is usually seen in the human stool specimen varies tremendously in size, from 6-40µm. There are thin – human stool specimen varies tremendously in size, from 6-40µm. There are thin –

walled cysts involved in autoinfection, and thick–walled cysts responsible for external

transmission via the faecal-oral route. The presence of large numbers of these parasites

(five or more per oil immersion microscopic field) in the absence of other intestinal

pathogens indicates disease. The organism may be detected in wet mounts or trichome

–stained smears of faecal specimens. Treatment with iodoquinol or metronidazole has

infection. Prevention is achieved by good personal hygiene

Endolimax nana is a lumen dweller in the large intestine, primarily at the cecal level,

where it feeds on bacteria. The life cycle is similar to *E.histolytica*. Motility is typically

sluggish (slug-like) with blunt hyaline pseudopodia, Projects shortly. Human infection

results from ingestion of viable cysts in polluted water or contaminated food. Typical

ovoid cysts of *E.nana* are confirmative. Rounded cysts and living trophozoites are often

confused with *E.hartmanni* and *E.histolytica*. No treatment is indicated for this nonpathogenic

infection. Prevention can be achieved through personal cleanliness and

community sanitation.

Iodamoeba buetschlii: - the natural habitat is the lumen of the large intestine, the

principal site probably being the caecum. The trophozoite feeds on enteric bacteria; it is

a natural parasite of man and lower primates. It is generally regarded as a nonpathogenic

lumen parasite. No treatment is ordinarily indicated. Prevention is based on

good personal hygiene and sanitation in the community.

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(five or more per oil immersion microscopic field) in the absence of other intestinapathogens indicates disease. The organism may be detected in wet mounts or trichome

–stained smears of faecal specimens. Treatment with iodoquinol or metronidazole ha

been successful in eradicating the organism from intestine and alleviating symptoms.

However, the definitive role of *B.hominis* in disease remains to be demonstrated. The

incidence and apparent worldwide distribution of the infection indicates preventive

measures to be taken, which involve improving personal hygiene and sanitary condition

PATHOGENIC FLAGELLATES

INTRODUCTION

Flagellates are unicellular microorganisms. Their locomotion is by lashing a tail-like

appendage called a flagellum or flagella and reproduction is by simple binary fission.

There are three groups of flagellates:

• Luminal flagellates

Giardia lamblia

Dientmoeab fragilis

• Hemoflagellates

Trypanosoma species.

Leishmania species.

• Genital flagellates

Trichomonas vaginalis

Luminal flagellates

Giardia lamblia

Important features – the life cycle consists of two stages,

the trophozoite and cyst. The trophozoite is 9-12 µm long

and 5-15µm wide anteriorly. It is bilaterally symmetrical,

pear-shaped with two nuclei (large central karyosome),

four pairs of flagella, two axonemes, and a suction disc

with which it attaches to the intestinal wall. The oval cyst

is 8-12µm long and7-10µm wide, thick-walled with

four nucleus and several internal fibera? Each cyst gives

rise to two trophozoites during excystation in the intestinal

tract.

Transmission is by ingestion of the infective cyst

Pathogenesis

Infection with G.lamblia is initiated by ingestion of cysts. Gastric acid stimulates

excystation, with the release of trophozoites in duodenum and jejunum. The

trophozoites can attach to the intestinal villi by the ventral sucking discs without

penetration of the mucosa lining, but they only feed on the mucous secretions. In

symptomatic patients, however, mucosa-lining irritation may cause increased mucous

secretion and dehydration. Metastatic spread of disease beyond the GIT is very rare

Epidemiology

Giardia lamblia has a worldwide distribution, particularly common in the tropics and

subtropics. It is acquired through the consumption of inadequately treated contaminated

water, ingestion of contaminated uncooked vegetables or fruits, or person-to-person

spread by the faecal-oral route. The cyst stage is resistant to chlorine in concentrations

used in most water treatment facilities. Infection exists in 50% of symptomatic carriage,

and reserves the infection in endemic form.

Clinical features

Clinical disease: Giardiasis

Symptomatic giardiasis ranges from mild diarrhea to severe malabsorption syndrome.

Usually, the onset of the disease is sudden and consists of foul smelling, watery

diarrhea, abdominal cramps, flatulence, and streatorrhoea. Blood & pus are rarely

present in stool specimens, a feature consistent with the absence of tissue destruction.

Immunity

The humoral immune response and the cellular immune mechanism are involved in

giardiasis. Giardia – specific IgA is particularly important in both defense against and clearance of parasite.

Laboratory diagnosis

Examination of diarrhoeal stool- trophozoite or cyst, or both may be recovered in wet

preparation. In examinations of formed stool (e.g. in asymptomatic carriers) only cysts

are seen. Giardia species may occur in “showers”, i.e. many organisms may be present

in the stool on a given day and few or none may be detected the next day. Therefore

one stool specimen per day for 3 days is important If microscopic examination of the stool is negative in a patient in whom giardiasis is

highly suspected duodenal aspiration, string test (entero-test), or biopsy of the upper

small intestine can be examined.

In addition to conventional microscopy, several immunologic tests can be implemented

for the detection of parasitic antigens.

Treatment

For asymptomatic carriers and diseased patients the drug of choice is quinacrine

hydrochloride or metronidazole.

Prevention

- Asymptomatic reservoirs of infection should be identified & treated.

- Avoidance of contaminated food and water.

- Drinking water from lakes and streams should be boiled, filtered and/or iodine treated.

- Proper waste disposal and use of latrine.

Trichomonas vaginalis

Important features- it is a pear-shaped organism with a central nucleus and four

anterior flagella; and undulating membrane extends about two-thirds of its length. It

exists only as a trophozoite form, and measured 7-23µm long & 5-15µm wide.

Transmission is by sexual intercourse.

Figure 6; Life cycle of *Trichomonas vaginalis*

Pathogenesis

The trophozoite is found in the urethra & vagina of women and the urethra & prostate

gland of men. After introduction by sexual intercourse, proliferation begins which results

in inflammation & large numbers of trophozoites in the tissues and the secretions. The

onset of symptoms such as vaginal or vulval pruritus and discharge is often sudden and

occurs during or after menstruation as a result of the increased vaginal acidity. The

vaginal secretions are liquors, greenish or yellowish, sometimes frothy, and foul

smelling. Infection in the male may be latent, with no symptoms, or may be present as

self limited, persistent, or recurring urethritis.

Epidemiology

This parasite has worldwide distribution, and sexual intercourse is the primary mode of

transmission. Occasionally, infections can be transmitted by fomites (toilet articles,

clothing), although this transmission is limited by liability of the trophozoite. Rarely

Infants may be infected by passage through the mother's infected birth canal. The

prevalence of this flagellate in developing countries is reported to be 5% –20% in

women and 2% –10% in men.

Clinical features

Clinical disease - trichomoniasis.

Most infected women at the acute stage are asymptomatic or have a scanty, watery

vaginal discharge. In symptomatic cases vaginitis occurs with more extensive

inflammation, along with erosion of epithelial lining, and painful urination, and results in

symptomatic vaginal discharge, vulvitis and dysuria.

Immunity

The infection may induce humoral, secretory, and cellular immune reactions, but they

are of little diagnostic help and do not appear to produce clinically significant immunity.

Laboratory diagnosis

• In females*, T.vaginalis* may be found in urine sediment, wet preparations of vaginal

secretions or vaginal scrapings.

• In males it may be found in urine, wet preparations of prostatic secretions or

following massage of the prostate gland.

• Contamination of the specimen with faeces may confuse *T.vaginalis* with

T.hominis.

Prevention

- Both male & female sex partners must be treated to avoid reinfection

- Good personal hygiene, avoidance of shared toilet articles & clothing.

- Safe sexual practice

Dientamoeba fragilis

Dientamoeba fragilis was initially classified as an amoeba; however, the internal

structures of the trophoziote are typical of a flagellate. No cyst stage has been

described. The life cycle and mode of transmission of *D. fragilis* are not known. It has

worldwide distribution. The transmission is postulated, via helminthes egg such as those

of Ascaris and Enterobius species. Transmission by faecal- oral routes does occur.

Most infection with *D. fragilis* is asymptomatic, with colonization of the cecum and upper

colon. However, some patients may develop symptomatic disease, consisting of

abdominal discomfort, flatulence, intermittent diarrhea, anorexia, and weight loss. The

therapeutic agent of choice for this infection is iodoquinol, with tetracycline and

parmomycine as acceptable alternatives. The reservoir for this flagellate and lifecycle

are unknown. Thus, specific recommendation for prevention is difficult. However,

infection can be avoided by maintenance of adequate sanitary conditions.

Other flagellates inhabiting the alimentary canal

Trichomonas hominis – The trophozoites live in the caecal area of the large intestine

and feed on bacteria. It is considered to be non-pathogenic, although it is often

recovered from diarrheic stools. Since there is no known cyst stage, transmission

probably occurs in the trophic form. There is no indication of treatment.

Trichomanas tenax – was first recovered from the mouth, specifically in tartar from the teeth. There is no known cyst stage. The trophozoite has a pyriform shape and is

smaller and more slender than that of *T.hominis*. Diagnosis is based on the recovery of

the organism from the teeth, gums, or tonsillar crypts, and no therapy is indicated.

Chilomastix mesnli – has both a trophozoite and cyst stage. It normally lives in the

cecal region of the large intestine, where the organism feeds on bacteria and debris. It

is considered to be a non-pathogenic, and no treatment is recommended

Parasitology

Lecture 12

Lumaalhili

INTESTINAL FLUKES

♦Fasciolopsisbuski: These giant intestinal flukes (2-7.5 cm in length) arefound in some Asian countries.

♦Heterophyids: Minute flukes acquired by ingestion of raw fresh water fish.

They are found in Asian countries.

LIVER FLUKES

♦Clonorchissinensis: Chinese liver fluke - adult worms live in bile ducts.

♦Faciola hepatica: Sheep liver fluke - is a common parasite, cosmopolitan indistribution.

. It is large (3 cm in length). Adult worms reside in the large biliary

passages and gall bladder.

♦ Other: Faciolagigantica: lives in the liver of cattle. Human infections arevery rare.

LUNG FLUKES

At least eight different species of lung flukes, all belonging to the genus

Paragonimus, are known to infect man. Paragonimuswestermani, best knownspecies, affects man causing paragonimiasis (lung disease).

It is found in Asia(China, India, Indonesia, Malaya etc) and some African countries.

NEMATODES (ROUND WORMS)

All the important human parasites of the Phylum Nemathelminthes

(Aschelminthes) belong to the Class Nematoda.

GENERAL CHARACTERISTICS OF NEMATODES :

They are un-segmented, elongated and cylindrical. They have separate sexeswith separate appearances.

They have a tough protective covering or cuticle.

They have a complete digestive tract with both oral and anal openings.

The nematodes are free living (Majority) or parasites of humans, plants or animals.

The parasitic nematodes:

The nematodes are generally light cream-white colored. Their life cycle includes:

egg, larvae and adult.

The parasitic nematodes are divided into:

1. Intestinal nematodes

1.1. Intestinal nematodes with tissue stage

A. Ascarislumbricoides

B. Hookworms

C. Strongyloidesstercoralis

2.1 Intestinal nematodes without tissue stage

A. *Enterobiusvermicularis*

B. Trichuristrichuira.

2. Tissue and blood dwelling nematodes

2.1. Filarial worms

2.2. *Dracunculusmedinensis*

2.3. Trichinella

2.4. Larva migrans.

2.1. INTESTINAL NEMATODES WITH TISSUE STAGE

2.1.1. ***ASCARIS LUMBRICOIDES***

These are common roundworms infecting more than 700 million peopleworldwide.

Morphology:

Male adult worm measures 15-20 cm in length. The posterior end is curvedventrally.

The female worm measures 20-40 cm in length. Its posterior end is

straight.

Infective stage and modes of infection:

The egg containing larva when ingested with contaminated raw vegetablescauses ascariasis.

Life cycle:

Ingested eggs hatch in the duodenum. The larvae penetrate the intestinal wall

and circulate in the blood. From the heart they migrate to the lungs, ascend to

the trachea, descend to the esophagus and finally reach the small intestine to

become adult. The female pass immature eggs which pass to the soil and maturein 2 weeks.

Pathogenicity and clinical features:

Adult worms in the intestine cause abdominal pain and may cause intestinalobstruction especially in children.

Larvae in the lungs may cause inflammation of

the lungs (Loeffler's syndrome) – pneumonia-like symptoms.

Diagnosis

1. Examination of stool for eggs by direct saline smear method. The egg isovoidal, 75x60 microns, covered by albuminousmamillatins.

2. Demonstration of adult worm.

2.1.2. HOOK WORMS

There are two species of hookworm:

1*. Ancylostomaduodenale*

2. Necatoramericanus

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

2.1.2.1. Ancylostomaduodenale:

Grayish-white in color. The body is slightly ventrally curved. The anterior endfollows the body curvature.

The buccal cavity is provided ventrally with pairs ofteeth and dorsally with a notched dental plate.

Distribution: This species is found in the northern part of the world including

China, Japan, Europe, North Africa and Ethiopia.

Morphology:

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

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

Necatoramericanus

This species, so called American hookworm, is found in predominantly thetropics.

The anterior end is hooked against the body curvature. The mouth is

provided ventrally and dorsally with cutting plate.

Morphology:

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

Female: The female measures 10 cm in length. The posterior end is straight

Infective stage and methods of infection:

The filariform larva infects by skin penetration.

Life cycle:

Adult male and female worms live in the small intestine. The female lays eggs

(oval, 60x40 microns), which contain immature embryo in the 4 cell stage.When the eggs pass in the stool to the soil and under favorable conditions of

temperature, moisture and oxygen, they hatch into larvae, which molt twice andbecome infective.

When the filariform larvae penetrate the skin, they circulate in

the blood, reach the lungs, ascend to the trachea, descend to esophagus toreach the small intestine and become adults.

Pathogenicity:

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

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

LARVA MIGRANS

There are three types of larva migrans:

a. Cutaneous larva migrans (Creeping eruption)

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

1. Ancylostomabraziliens: infects both dogs and cats.

2. Ancylostomacaninum: infects only dogs.

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

The dermatitis leads to scratching and then bacterial superinfection.

Treatment

Thiabendazole: Applied topically.

b. Visceral larva migrans

A syndrome caused by the migration of parasitic larvae in the viscera of a

host for months or years. It may be caused by transient larval migration in the

life cycles of several parasites such as hookworm, Ascarislumbricoides, T.

spiralis, S. strecoralis and other filarial worms.

Toxocariasis

This is a kind of visceral larva migrans caused by

♦Toxocaracanis (Dog ascarid) and

♦Toxocaracatis (Cat ascarid).

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

called toxocariasis.

Morphology

♦ The larvae of Toxocaracanis and Toxocaracatis measure about 400 µm in

length.

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

lumbricoides in humans.

Epidemiology

Visceral larva migrans is cosmopolitan in distribution.

Transmission:

Ingestion of eggs of Toxocara species in contaminated food or soil or direct

contact with infected patients. Children are more at risk.

Clinical features:

♦ Majority are asymptomatic.

♦ Eosinophilia

♦ Cerebral, myocardial and pulmonary involvement may cause death.

Diagnosis - Identification of larvae in tissue.

C. Intestinal larva migrans

This is an extremely rare kind of larva migrans

2.1.4. STRONGYLOIDES STERCORALIS

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

Morphology:

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

two spicules

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

Infection: follows skin penetration by filariform larvae.

Life cycle

Adult male and female worms live in the small intestine. After fertilization, the

female penetrates the mucosa of the small intestine and lay eggs in the

submucosa. The eggs hatch and the larvae penetrate the mucosa back to the

lumen. If the environmental conditions are favorable, the larvae will come out

with the stool to the soil. They transform into adults, which lay eggs, and hatching

larvae get transformed to adults and so on. If the environmental conditions are

not favorable, the larvae in the stool will moult and transform into infective

filariform larvae, which pierce the intestine (auto-infection). Larvae penetrating

the skin from the soil or by autoinfection are carried by the blood to the lungs,

ascend to the trachea, descend to the esophagus and mature in the small

intestine.

Clinical presentation

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

pneumonia.

Disseminated strongyloidiasis:

Multiplicity of symptoms are present due to the injury of other organs by the

migrating larvae. Organs such as liver, heart adrenals, pancreas, kidneys, and

CNS, etc. may be affected. This is usually seen in immunocompromized

individuals.

Diagnosis - Detection of rhabditiform larvae of strongyloides in stool.

INTESTINAL NEMATODES WITHOUT TISSUE STAGE

ENTEROBIUS VERMICULARIS (PIN WORM OR THREAD WORM)

Enterobiusvermicularis is a small white worm with thread-like appearance. Thed eggs

MEDICAL HELMINTHOLOGY

INTRODUCTION

Medical helminthology is concerned with the study of helminthes or parasiticworms.

Helminthes are trophoblastic metazoa (multi-cellular organisms).

Helminthes are among the common parasitic causes of human suffering. Theyare the cause of high morbidity and mortality of people worldwide

They cause different diseases in humans, but few helminthic infections cause life- threateningdiseases.

They cause anemia and malnutrition. In children they cause a

reduction in academic performance. Helminthes also cause economic loss as aresult of infections of domestic animals

There is age dependent distribution ofinfections from geo-helminthes and schistosomes.

. As a result of predisposingbehavioral and immunological status, children disproportionately carry the burdenof schistosomes and geo-helminthes.

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

1. Contaminated soil (Geo-helminthes), water (cercariae of blood flukes) andfood (Taenia in raw meat).

2. Blood sucking insects or arthropods (as in filarial worms).

3. Domestic or wild animals harboring the parasite (as in echinococcus in dogs).

4. Person to person (as in *Enterobiusvermicularis, Hymenolopis nana*).

5. Oneself (auto-infection) as in *Enterobiusvermicularis*.

They enter the body through different routes including: mouth, skin and thefood (Taenia in raw meat).

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

1. Trematodes (Flukes)

2. Nematodes (Round worms)

3. Cestodes (Tape worms)

The Trematodes and Cestodes are groups of flat worms.

MEDICALLY IMPORTANT TREMATODES (FLUKES)

INTRODUCTION

Trematodes belong to the phylum platyhelminthes. They are found in a widerange of habitats.

The great majority inhabit the alimentary canal, liver, bile duct,

ureter and bladder of vertebrate animals.

According to the sites they inhabit, there are four groups of flukes. These are:

Blood flukes, Intestinal flukes, Liver flukes, and Lung flukes

BLOOD FLUKES

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

SCHISTOSOMIASIS (BILHARZIASIS)

It is estimated that about 600 million people in 79 countries suffer from

schistosomiasis (Bilharziasis). The schistosomes cause intestinal, hepatosplenic,pulmonary, urogenital, cerebral and other forms of schistosomiasis.

Schistosome is the only fluke with separate sexes. The female worm lies gynecophoral canal of the male. This condition is important for transportation.

There are five medically important species:

1. *Schistosomamansoni*: causes intestinal schistosomiasis.

2. *Schistosomahaematobium*: causes vesical (urinary) schistosomiasis.

3. *Schistosomajaponicum*: causes intestinal schistosomiasis.

4. *Schistosomaintercalatum*: causes intestinal schistosomiasis.

5. *Schistosomamekongi*: causes intestinal schistosomiasis. This seems to cause milder disease in man. It causes disease in other vertebrate hosts.

Schistosomamansoni

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

Geographical distribution: It is found in Africa, South America, Middle East(some Arab countries) etc. Stream and lake-based transmission is common.

The snail hosts that harbor *S. mansoni* are the genera (B. *: Biomphalaria glabrata*) and Trobicorbis. These have oval shells.

Morphology

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

Female:The female is 1.5-2.0 cm in length. The ovary is present in the anteriorthird and Vitelline glands occupy the posterior two-thirds. It lays about 100-300eggs daily.

The uterus is short containing few ova.

URINARY SCISTOSOMIASIS

Schistosomahaematobium

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

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

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

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

Schistosomajaponicum

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

Schistosomaintercalatum

This is the rarest and least pathogenic schistosome that matures in man. It isfound in Western and Central Africa.

The daily egg output is about 300. The eggshave a terminal spine.

LIFE CYCLE OF SCHISTOSOMES

Adult worms reside in pairs: the female lying in the gynecophoral canal of themale.

After fertilization, eggs are passed into the venules. A larval form – the

miracidium - develops within the egg. Its lytic enzymes and the contraction of thetissues of the intestine *(S. mansoni*) or urinary bladder (*S. haematobium*).

The eggs pass into the lumens and organs and are evacuated in the feces (*S.mansoni*) or the urine (*S. haematobium*).

On contact with fresh water themiracidia hatch from the eggs and swim about until they find the appropriatesnail, which they penetrate.

After twogenerations of sporocyst development andmultiplication within the snail, the fork-tailed cercariae emerge.

Infection to mantakes place during bathing or swimming.

The cercariae penetrate the skin, arecarried into the systemic circulation and pass through to the portal vessels.

Within the intrahepatic portion of the portal system, the worms feed and grow to maturity.

Symptoms and complications

Patients infected with *S. haematobium* suffer from terminal haematuria andpainful micturition.

There is inflammation of the urinary bladder (cystitis), and

enlargement of spleen and liver.

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

S. haematobium causes squamous cell carcinoma in the bladder.

Laboratory Diagnosis

S. mansoni

♦ Microscopic examination of the stool for eggs after concentration by

sedimentation method. The egg has characteristic lateral spine.

♦ Rectal snip

S. haematobium:

♦ Examination of the urine after allowing it to sediment in a conical urinalysisglass.

A drop from the sediment is taken and examined for eggs. Egg has

terminal spine.

♦ Biopsy from bladder

Prevention:

1. Health education:

A. On use of clean latrines and safe water supply

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

water

2. Snail control:

A. Physical methods:

i. Periodic clearance of canals from vegetations.

ii. Manual removal of snails and their destruction.

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

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

2.2. INTESTINAL NEMATODES WITHOUT TISSUE STAGE

***Enterobiusvermicularis*(pin worm or thread worm)**

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

Morphology

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

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

Infective stage

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

Mode of infection

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

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

• Aerosol inhalation from contaminated sheets and dust.

Life cycle

Adult worm lives in the large intestine. After fertilization, the male dies and the female moves out through the anus to glue its eggs on the peri-anal skin. This takes place by night. The egg is 50x25 microns, plano-convex and contains

larva. When the eggs are swallowed, they hatch in the small intestine and the larvae migrate to the large intestine to become adult.

Clinical presentation

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

Diagnosis

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

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

Trichuristrichiura (whip worm)

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

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

Female: The female measures 4-5 cm in length. Its posterior end is straight

Infective stage and mode of infection

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

Life cycle:

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

Symptoms

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

Diagnosis

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

2.3. TISSUE NEMATODES

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

FILARIAL WORMS

The filarial worms have complex life cycles involving a developmental stage in an insect vector. They require an arthropod vector for their transmission. The worms inhabit either the lymphatic system or the subcutaneous tissues of man. The female worm gives rise to a young worm called microfilaria. The microfilariae,

when taken by the arthropod intermediate host during biting, develop into filariform larvae, which are the infective stages. Humans get infected when bitten by the infected arthropod intermediate host.

Wuchereriabancrofti

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

Mode of transmission and pathogenesis

The filariform larvae are introduced through the skin by the bite of the arthropod intermediate host. The larvae invade the lymphatics, usually the lower limb, where they develop into adult worms. The microfilariae are librated into the bloodstream.

They remain in the pulmonary circulation during day, emerging into the peripheral circulation only during night, to coincide with the biting habit of the vector. Presence of the adult worms causes lymphatic blockage and gross lymphedema, which sometimes lead to elephantiasis.

Pathogenecity and clinical features:

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

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

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

Diagnosis

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

Loa loa

The eye worm, Loa loa, causes Loiasis. The insect vectors include mango flies of Chrysops .Loiasis is endemic in Centraland West Equatorial Africa.

The abundant rubber plantations provide a favorableenvironment for the vector to transmit the disease.

Morphology

Adult male worms: 30-34 mm in length

Adult female worms: 40-70 mm in length

Pathogenesis

The microfilaria have a sheath. Their diurnal periodicity corresponds to the feeding pattern of the insect vector, which bites humans from 10:00 AM to 4:0

Clinical Features

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

Laboratory diagnosis

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

• Eosinophilia.

TRICHINOSIS

Etiologic agent- *Trichinellaspiralis*

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

Morphology

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

Pathogenecity and life cycle

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

diaphragm are most frequently parasitized. Others include the tongue, masseter and ocular muscles.

Clinical features

There are two clinical phases.

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

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

Diagnosis:

♦ Muscle Biopsy

♦ Detection of larvae in blood or CSF

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

♦ ELISA

Prevention

♦ Cooking of all meat before consumption

♦ Inspection of pigs

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

Parasitology

Lacuter 4 Luma al hili

Haemoflagelates

Leishmania Species

Clinical disease - Veseralleishmaniasis

-Cutaneous leishmaniasis

- Mucocutaneousleishmaniasis

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

Visceral leishmaniasis

Leishmaniadonovani

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

The amastigote stage appears as an ovoidal or rounded body, measuring about

2-3µm in length; and the promastigotes are 15-25µm lengths by 1.5-3.5µm

breadths.

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver,

spleen and bone marrow) are the most severely affected organs. Reduced bone

marrow activity, coupled with cellular distraction in the spleen, results in anaemia,

leukopenia and thrombocytopenia. This leads to secondary infections and a

tendency to bleed. The spleen and liver become markedly enlarged, and

hypersplenism contributes to the development of anaemia and lymphadenopathy

also occurs. Increased production of globulin results in hyperglobulinemia, and

reversal of the albumin-to-globulin ratio.

Epidemiology

L. donovanidonovani, infection of the classic kala-azar (“black sickness”) or

dumdum fever type occurs in many parts of Asia, Africa and Southeast Asia.

Kala-azar occurs in three distinct epidemiologic patterns. In Mediterranean basin

(European, Near Eastern, and Africa) and parts of China and Russia, the

reservoir hosts are primarily dogs & foxes; in sub-Saharan Africa, rats & small

carnivores are believed to be the main reservoirs. In India and neighboring

countries (and Kenya), kala-azar is anthroponosis, i.e. there is no other

mammalian reservoir host other than human. The vector is the Phlebotomus

sand fly. Other variants of *L. donovani* are also recognized: *L. donovani*infantum

with similar geographical distribution, reservoir host and vector; with *L. donovani*

donovani*. L. donovani*chagasi is found in South America, Central America,

especially Mexico, and the West Indies. Reservoir hosts are dogs, foxes, and

cats, and the vector is the Lutzomiya sand fly.

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and

sweating that may resemble malaria symptoms are also common early in Clinical featurestheinfection. As organisms proliferate & invade cells of the liver and spleen, markedenlargement of the organs, weight loss, anemia, and emaciation occurs. With

persistence of the disease, deeply pigmented, granulomatous lesion of skin,

referred to as post-kala-azar dermal leishmaniasis, occurs.

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

Immunity

Host cellular and humoraldefence mechanisms are stimulated.

Laboratory diagnosis

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

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

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

• Serologic testing is also available.

Prevention

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

• Protection from sand flies by screening and insect repellents.

Old World Cutaneous Leishmaniasis (Oriental sore)

Clinical disease

L.tropica minor - dry or urban cutaneous leishmaniasis

L.tropica major - wet or rural cutaneous leishmaniasis

L.aethiopica - cutaneous leishmaniasis.

Important features

These are parasites of the skin found in endothelial cells of the capillaries of the

infected site, nearby lymph nodes, within large mononuclear cells, in neutrophilic

leukocytes, and free in the serum exuding from the ulcerative site. Metastasis to

other site or invasion of the viscera is rare.

Pathogenesis

In neutrophilic leukocytes, phagocytosis is usually successful, but in

macrophages the introduced parasites round up to form amastigote and multiply.

In the early stage, the lesion is characterized by the proliferation of macrophages

that contain numerous amastigotes. There is a variable infiltration of lymphocytes

and plasma cell. The overlying epithelium shows acanthosis and hyperkeratosis,

which is usually followed by necrosis and ulceration.

Epidemiology

Cutaneous leishmaniasis produced by L.tropica complex is present in many parts

of Asia, Africa, Mediterranean Europe and the southern region of the former

Soviet Union. The urban Cutaneous leishmaniasis is thought to be an

anthroponosis while the rural cutaneous leishmaniasis is zoonosis with human

infections occurring only sporadically. The reservoir hosts in L. major are rodents.

L.aethopica is endemic in Ethiopia and Kenya. The disease is a zoonosis with

rock & tree hyraxes serving as reservoir hosts. The vector for the old world

cutaneous leishmaniasis is the Phlebotomus sand fly.

Clinical features

The first sign, a red papule, appears at the site of the fly's bite. This lesion

becomes irritated, with intense itching, and begins to enlarge & ulcerate.

Gradually the ulcer becomes hard and crusted and exudes a thin, serous

material. At this stage, secondary bacterial infection may complicate the disease.

In the case of the Ethiopian cutaneous leishmaniasis, there are similar

developments of lesions, but they may also give rise to diffuse cutaneous

leishmaniasis (DCL) in patients who produce little or no cell mediated immunity

against the parasite. This leads to the formation of disfiguring nodules over the

surface of the body.

Immunity

Both humoral and cell mediated immunity (CMI) are involved

Prevention

- Prompt treatment & eradication of ulcers

- Control of sand flies & reservoir hosts.

New World Cutaneous and MucocutaneousLeishmaniasis

(American cutaneous leishmaniasis)

Clinical disease:

Leishmaniamexicana complex- Cutaneous leishmaniasis.

Leishmaniabraziliensis complex- mucocutaneous or cutaneous leishmaniasis

Important features:

The American cutaneous leishmeniasis is the same as oriental sore. But some of

the strains tend to invade the mucous membranes of the mouth, nose, pharynx,

and larynx either initially by direct extension or by metastasis. The metastasis is

usually via lymphatic channels but occasionally may be the bloodstream.

Pathogenesis

The lesions are confined to the skin in cutaneous leishmaiasis and to the mucous

membranes, cartilage, and skin in mucocutaneousleishmaniasis. A granulomatous

response occurs, and a necrotic ulcer forms at the bite site. The lesions tend to

become superinfected with bacteria. Secondary lesions occur on the skin as well

as in mucous membranes. Nasal, oral, and pharyngeal lesions may be polypoid

initially, and then erode to form ulcers that expand to destroy the soft tissue and

cartilage about the face and larynx. Regional lymphadenopathy is common.

Epidemiology

Most of the cutaneous &mucocutaneousleishmaniasis of the new world exist in

enzootic cycles of infection involving wild animals, especially forest rodents.

Leishmaniamexicana occurs in south & Central America, especially in the Amazonbasin, with sloths, rodents, monkeys, and raccoons as reservoir hosts. The

mucocutaneousleishmaniasis is seen from the Yucatan peninsula into Central &

South America, especially in rain forests where workers are exposed to sand fly

bites while invading the habitat of the forest rodents. There are many jungle

reservoir hosts, and domesticated dogs serve as reservoirs as well. The vector is

the Lutzomyia sand fly.

Clinical features

The types of lesions are more varied than those of oriental sore and include

Chiclero ulcer, Uta, Espundia, and Disseminated Cutaneous Leishmaniasis.

Laboratory diagnosis

• Demonstration of the amastigotes in properly stained smears from touch

preparations of ulcer biopsy specimen.

• Serological tests based on fluorescent antibody tests.

• Leishman skin test in some species.

Immunity

The humoral and cellular immune systems are involved

Treatment

The drug of choice is sodium stibogluconate.

Prevention

• Avoiding endemic areas especially during times when local vectors are most

active.

• Prompt treatment of infected individuals.

Trypanosomiasis

Etiologic agents

Trypanosoma brucei complex – African trypanosomiasis (sleeping sickness)

Trypanosoma cruzi – American trypanosomiasis (Chagas' disease)

Important features

These species may have amastigote, promastigote, epimastigote, and

trypomastigote stages in their life cycle. In human trypanosomes of the African

form, however, the **amastigote** and **promastigote** stages of development are absent.

Typical trypanosome structure is an elongated spindle-shaped body that more or less tapers at both ends, a centrally situated nucleus, a kinetoplast posterior to nucleus, an undulating membrane arising from the kinetoplast and proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end.

African trypanosomiasis

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

Pathogenesis

The trypomastigotes spread from the skin through the blood to the lymph node and the brain.

. The typical somnolence **(sleeping sickness**) usually progresses to coma as a result of demyelinating encephalitis. In acute form, cyclical fever spike (approximately every 2 weeks) occurs that is related to antigenic variation. As antibody mediated agglutination and lysis of the trypomastigotes occurs, the fever subsides.

With a few remains of antigenic variants new fever spike occurs and the cycle repeats itself over a long period.

Epidemiology

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

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

This transmission and vector cycle makes the organism more difficult to control than *T.b. gambiens*.

Clinical features

Although both species cause sleeping sickness, the progress of the disease is different.

T.gambiense induced disease runs a low-grade chronic course over a few years. One of the earliest signs of disease is an occasional ulcer at the site of the fly bite.

As reproduction of organisms continues, the lymph nodes are invaded, and fever, myalgia, arthralgia, and lymph node enlargement results. Swelling of the posterior cervical lymph nodes is characteristic of Gambian sleeping sickness and is called winterbottom's sign.

Chronic disease progresses to CNS involvement with lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration. In the final stages, convulsions, hemiplegia, and incontinence occur. The patient becomes difficult to arouse or obtain a response from, eventually progressing to a comatose state.

Death is the result of CNS damage and other infections, such as pneumonia.

In *T.rhodesiense*, the disease caused is a more acute, rapidly progressive disease that is usually fatal

. This more virulent organism also develops in greater numbers in the blood. Lymphadenopathy is uncommon, and early in the infection, CNS invasion occurs, resulting in lethargy, anorexia, and mental disturbance

The chronic stages described for *T.gambiense* are not often seen, because in addition to rapid CNS disease, the organism produces kidney damage & myocarditis, leading to death.

Immunity

Both the humoral and cellular immunity involve in these infections. The

immune responses of the host to the presence of these parasites, however, is faced with antigenic variation, in which organisms that have changed their antigenic identity

can escape the host immune response and initiate another disease process with increased level of parasitemia

Laboratory examination

Examination of thin and thick films, in concentrated anticoagulated blood preparations, and in aspiration from lymph nodes and concentrated spinal fluid.

Methods for concentrating parasites in blood may be helpful approaches including

centrifugation of heparinized samples and an ion–exchange chromatography.

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

Prevention

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

• Treatment of human cases to reduce transmission to flies.

• Avoiding insect bite by wearing protective clothing & use of screen,

bed netting and insect repellants.

American trypanosomiasis

Trypanosoma cruzi is a pleomorphic trypanosome that includes an additional form of amastigote in its life cycle. The vector for transmission are **reduviid bugs**.

Pathogenesis

During the acute phase, the organism occurs in blood as a typical trypomastigote and in the reticuloendothelial cells as a typical amastigote. The amastigotes can kill cells and cause inflammation, consisting mainly of mononuclear cells.

Cardiac muscle is the most frequently and severely affected tissue. In addition, neuronal damage leads to cardiac arrhythmias and loss of tone in the colon (megacolon) and esophagus (megaesophagus)

In the chronic phase, the organism persists in the amastigote form.

Epidemiology

T.cruzi occurs widely in both reduviid bugs and a broad spectrum of reservoir animals in North, Central, and South America.

Human disease is found most often among children in South and Central America, where there is direct correlation between infected

wild animal reservoir hosts and the presence of infected bugs

whose nests are found in human dwellings.

Clinical features

Chagas' disease may be asymptomatic acute or chronic disease. One of

the earliest signs is development at the site of the bug bite of an erythematous and indurated area called a chagoma.

This is often followed by a rash and edema around the eyes and face;

in young children frequently an acute process with CNS involvement may occur. Acute infection is also characterized by fever, chills,

malaise, myalgia, and fatigue. The chronic Chagas' disease is hepatosplenomegaly, myocarditis, and enlargement of the esophagus and colon hepatosplenomegaly, myocarditis, and enlargement of the esophagus and colon as a result of the destruction of nerve cells (E.g. Auerbach's plexus) and other tissues that control the growth of these organs. Involvement of the CNS may produce granulomas in the brain

with cyst formation and a meningoencephalitis

Death from chronic Chagas' disease results from tissue destruction in the many areas invaded by the organisms, and sudden death results from complete heart block and brain damage

Laboratory diagnosis

Examine thin or thick stained preparations for trypomastigotes. Wet preparations should also be examined to look for motile organisms that leave the blood stream and become difficult to find.

Biopsy of lymph nodes, liver, spleen, or bone marrow may demonstratei

Immunity

Unlike African trypanosomiasis, the antigenic variation is less common in T.cruzi

infection. Therefore, the humoral and cellular immune responses function in the.

Treatment

The drug of choice is nifurtimox. Alternative agents include allopurinol & benzimidazole

Prevention

• Bug control, eradication of nests

• Treating infected person & exclusion of donors by screening blood.

• Development of vaccine.

MEDICALLY IMPORTANT CILIATES

Balantidiasis

The intestinal protozoan *Balantidium coli* is the only member of the

ciliate group that is pathogenic for humans. Disease produced by B. coli is similar to amebiasis, because the organisms elaborate proteolytic and cytotoxicsubstances that mediate tissue invasion and intestinal ulceration

Life cycle

The life cycle of *B. coli* is simple, involving ingestion of infectious cysts,

excystation, and invasion of trophozoites into the mucosal lining of the

large intestine, caecum, and terminal ileum. The trophozoite is covered

with rows of hair like cilia that aid in motility. Morphologically more complex than amebae, *B. coli* has a funnel-like primitive mouth called a

cytostome, a large (macro) nucleus and a small (micro) nucleus involved in reproduction

Epidemiology

B. coli are distributed worldwide. Swine and (less commonly) monkeys

are the most important reservoirs. Infections are transmitted by the faecal-oral route; outbreaks are associated with contamination of water supplies with pig faeces.

Person-to-person spread, spread, including through food handlers, has been implicated in outbreaks. Risk factors associated with human disease include contact contact with swine and substandard hygienic conditions.

Clinical features

As with other protozoan parasites, asymptomatic carriage of B. coli can exist.

Symptomatic disease is characterized by abdominal pain, tenderness, tenesmus, nausea, anorexia, and watery stools with blood and pus.

Ulceration of the intestinal mucosa, as with amebiasis, can be seen; a

secondary complication caused by bacterial invasion into the eroded intestinal mucosa can occur.

Extra intestinal invasion of organs is extremely rare in balantidiasis.

Laboratory Diagnosis

Microscopic examination of faeces for trophozoite and cysts is performed.

The trophozoite is very large, varying in length from 50 to 200µm and in width from 40 to 70µm.

The surface is covered with cilia

L8

COCCIDIA (SPOROZOA)

INTRODUCTION

Coccidia are members of the class sporozoa, Phylum Apicomplexa.

The life cycle is characterized by an alternation of generations, i.e. sexual (gametogony) and asexual (schizogony) reproduction and most members of the group also share alternative hosts.

The locomotion of a mature organism is by body flexion, gliding.

The genus Plasmodium that are the causes of malaria is the prototype of this class.

Malaria

There are four species normally infecting humans, namely, *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, and Plasmodium malariae.*

Life cycle

The life cycle of malaria is passed in two hosts (alternation of hosts) and has sexual and asexual stage (alternation of generations).

Vertebrate host - man (intermediate host), where the asexual cycle takes place.

The parasite multiplies by schizogony and there is formation of male

and female gametocytes (gametogony).

Invertebrate host - mosquito (definitive host) where the sexual cycle takes place.

Union of male and female gametes ends in the formation ofsporozoites (sporogony).

The life cycle passes in four stages:

<u>Three</u> in man:- Pre - erythrocyticschizogony

- Erythrocyticschizogony

- Exo- erythrocyticschizogony

<u>One</u>-in mosquito–SporogonyIntroduction into humans when infective female Anopheles mosquito bitesman, it inoculates saliva containing sporozoites (infective stage).

Pre- Erythrocyticschizogonysporozoites reach the blood stream and within30 minutes enter the parenchymal cells of the liver, initiating a

cycle of **scyhizogon.** Multiplication occurs in tissue schizonts, to form thousands of tinymerozoites. Merozoites are then liberated on rupture of schizonts about 7th – 9thday of the bites and enter into the blood stream. These merozoites either invadethe RBC's or other parenchymal liver cells.

In case of *P. falciparum* and possibly *P. malariae*, all merozoites invade RBC's without re-invading liver cells.

However,for*P. vivax* and *P. ovale*, some merozoites invade RBC's and

some re-invade liver cells initiating further Exo-erythrocyticschizogony, which is responsible forrelapses.

Some of the merozoites remain dormant (hypnozoites) becoming active later on.

Erythrocyticschizogony(blood phase) is completed in 48 hrs in P. vivax, *P.ovale*, and *P. falciparum*, and 72 hrs in *P. malariae*. The merozoites reinvadefresh RBC's repeating the schizogonic cycles

Erythrocyticmerozoites do not reinvade the liver cells. So malaria blood transfusion reproduces only erythrocytic cycle

<u>Gametogony</u>

Some merozoites that invade RBC's develop into sexual stages (male and female gametocytes). These undergo no further development until taken by themosquito.

Sporogony (extrinsic cycle in mosquito)

When a female Anopheles mosquito vector bites an infected person, it Sucks blood containing the different stages of malaria parasite. All stages other thangametocytes are digested in the stomach.

The microgametocyte undergoes ex-flagellation. The nucleus divides by reduction division into 6-8 pieces, which migrate to the periphery. At the same,time 6-8 thin filaments of cytoplasm are thrust out, in each passes a piece ofchromatin.

These filaments, the microgametes, are actively motile and separate

from the gametocyte.

The macrogametocyte by reduction division becomes a macrogamete.

Fertilization occurs by entry of a micro gamete into the macro gamete forming azygote.

The zygote changes into a worm like form, the ookinete, which wall of penetrates thethe stomach to develop into a spherical oocyst between the epitheliumand basement membrane.

The oocystes increase in size.

sporozoites develop inside the oocysts. Oocysts rupture and Thousands of sporozoites areliberated in the body cavity and migrate everywhere particularly to the salivaryglands.Now the mosquito is infective.

The sporogonous cycle in the mosquito takes 8-12 days depending on

temperature

Plasmodium falciparum

Plasmodium falciparum demonstrates no selectivity in host erythrocytes, i.e. itinvades young and old RBCs cells. The infected red blood cells also do notenlarge and become distorted.

• Multiple sporozoites can infect a single erythrocyte, and show multiple

infections of cells with small ring forms.

• The trophozoite is often seen in the host cells at the very edge or peripheryof cell membrane at accole position.

• Occasionally, reddish granules known as Maurer's dots are observed

• Mature (large) trophozoite stages and schizonts are rarely seen in

blood films, because their forms are sequestered in deep capillaries, liver andspleen.

• Peripheral blood smears characteristically contain only young ring

forms and occasionally crescent shaped gametocytes.

Epidemiology

P.falciparum occurs almost exclusively in tropical and subtropical regions. Weather(rainfall, temperature & humidity) is the most obvious cause of seasonality malaria transmission.

To date, abnormal weather conditions are also importantcauses of significant and widespread epidemics.

Moreover, drug-resistant infectionof *P.falciparum*is the commonest challenge in many parts of the world.

In Ethiopia even though all the four species of plasmodium infecting man have been recorded,*P.falciparum* is the one that most causes the epidemic disease andfollowed byvivax and malariae. *P.ovale* is rare.

Clinical features

Of all the four Plasmodia, *P. falciparum* has the shortest incubation period, whichranges from 7 to 10 days.

After the early flu-like symptoms, *P.falciparum* rapidlyproduces daily (quotidian) chills and fever as well as severe nausea, vomiting and diarrhea.

The periodicity of the attacks then becomes tertian (36 to 48 hours),

and fulminating disease develops. Involvement of the brain (cerebral malaria) is mostoften seen in *P.falciparum* infection.

Capillary plugging from an adhesion ofinfected red blood cells with each other and endothelial linings of capillaries causeshypoxic injury to the brain that can result in coma and death.

Kidney damage isalso associated with *P.falciparum* malaria, resulting in an illness called “blackwater” fever.

Intravascular hemolysis with rapid destruction of red blood cells

produces a marked hemoglobinuria and can result in acute renal failure, tubularnecrosis, nephrotic syndrome, and death.

Liver involvement is characterized byabdominal pain, vomiting of bile, hepatosplenomegally, severe diarrhea, and rapiddehydration.

Plasmodium vivax:

P.vivax is selective in that it invades only young immature erythrocytes.

Infections of *P. vivax* have the following characteristics:

• Infected red blood cells are usually enlarged and contain numerous

pink granules or schuffner’s dots.

• The trophozoite is ring-shaped but amoeboid in appearance.

• More mature trophozoites and erythrocyticschizonts containing up to24 merozoites are present.

• The gametocytes are round

Epidemiology

P. Vivax is the most prevalent of the human plasmodia with the widest geographicdistribution, including the tropics, subtropics, and temperate regions.

However, it isthe second most prevalent in Ethiopia following *P.falciparum.*

Clinical features:

After an incubation period (usually 10 to 17 days), the patient experiences vagueflu-like symptoms, such as headache, muscle pains, photophobia, anorexia,nausea and vomiting.

As the infection progresses, increased numbers of rupturing

erythrocytes liberate merozoites as well as toxic cellular debris and hemoglobin into circulation.

In combination, these substances produce the typical pattern chills,

fever and malarial rigors. These paroxysms usually reappear periodically(generally every 48 hours) as the cycle of infection, replication, and cell lysesprogresses.

The paroxysms may remain relatively mild or may progress to severe

attacks, with hours of sweating, chills, shaking persistently, high temperatures(1030F to 1060F) and exhaustion.

Since *P.vivax* infects only the reticulocytes, theparasitemia is usually limited to around 2 to 5% of the available RBCs.

Plasmodium malariae:

In contrast with *P.vivax* and *P.ovale, P.malariae* can infect only mature

erythrocytes with relatively rigid cell membranes. As a result, the parasite's growthmust conform to the size and shape of red blood cell.

This requirement produces no red cell enlargement or distortion, but it results indistinctive shapes of the parasite seen in the host cell, "band and bar forms" aswell as very compact dark staining forms.

The schizont of *P.malariae* is usuallycomposed of eight merozoites appearing in a rosette.

Epidemiology:

P. malariae infection occurs primarily in the same sub-tropical and temperateregions as infections with the other plasmodia but is less prevalent.

Clinical features:

The incubation period for *P. malariae* is the longest of the plasmodia, usually 18 to40 days, but possibly several months to years. The early symptoms are flu-like withfever patterns of 72 hours (quartan or malarial) in periodicity.

Plasmodium ovale:

P. ovale is similar to *P. vivax* in many respects, including its selectivity for young,pliable erythrocytes. As a consequence the classical characteristics.

include:

• The host cell becomes enlarged and distorted, usually in an oval

form.

• Schiffner’s dots appear as pale pink granules.

• The infected cell border is commonly fimbriated or ragged

• Mature schizonts contain about 10 merozoites.

Epidemiology:

P.ovale is distributed primarily in tropical Africa. It is also found in Asia and SouthAmerica.

Clinical features:

The incubation period for *P.ovale* is 16-18 days but can be longer. Clinically, ovaleAmerica.

malaria resembles vivax malaria with attacks recurring every 48-50 hours.

There are however, fewer relapses with P.ovale. Less than 2% of RBCs usually becomeinfected.

Laboratory diagnosis:

Microscopic examination of thick and thin films of blood is the method of choice forconfirming the clinical diagnosis of malaria and identifying the specific speciesresponsible for disease.

Malaria parasites in thick and thin blood films are best stained at pH 7.1 – 7.2using a Romanowsky stain (contains azure dyes and eosin).

The thick film is a concentration method that may be used to detect the presenceof organisms.

The thin film is most useful for establishing species identification.

Serologic procedures are available but they are used primarily for epidemiologicalsurveys or for screening blood

Prevention:

- Chemoprophylaxis and prompt diagnosis and treatment.
- Control of mosquito breeding
- Protection of insect bite by screening, netting and protective clothing
- Use of insect repellents.

L9

Other Coccidian parasites

Toxoplasma gondii – causes toxoplasmosis. The definitive host is the

domestic cat thend other felines. Humans and other mammals are intermediate hosts .*T.gondii* is usually acquired by ingestion and transplacental transmissionfrom an infected mother to the fetus can occur.

Human–to–human transmission,other than transplacental transmission, does not occur.

After infection of intestinal epithelium, the organisms spread to other organs, especially the brain,lungs, liver, and eyes.

Most primary infections in immunocompetent adults areasymptomatic.

Congenital infection can result in abortion, stillbirth, or neonatal

disease with encephalitis, chorioretinitis and hepatosplenomegaly.

Fever,jaundice, and intracranial calcifications are also seen. For the diagnosis of acuteand congenital infections, an immunofluorescence assay for detection of antibodyis used.Microscopic examination of Giemsa–stained preparations showscrescent–shaped trophozoite. Cysts may be seen in the tissue.

Isospora belli - is an intestinal protozoan that causes diarrhea, especially inimmunocompromized patients, e.g., those with AIDS.

Its life cycle parallels that ofother members of the Coccidia

The organism is acquired by fecal-oraltransmission of oocysts from either human or animal sources.

The oocystsexcysin the upper small intestine and invade the mucosa, causing destruction of thebrush border.

The disease in immunocompromized patients presents as a chronic, profuse,watery diarrhea. The pathogenesis of the diarrhea is unknown.

Diagnosis:

by finding the typical oocysts in fecal specimens. Serologic tests are not available.

The treatment of choice is trimethoprim-sulfamethoxazole.

Cryptosporidium parvum – causes cryptosporidiosis, the main **symptom**

of which is diarrhea. It is most severe in immunocompromized patients, e.g., thosewith AIDS.

The organism is acquired by faecal-oral transmission of Oocysts from

either human or animal sources. The oocystsexcyst in the small intestine, wherethe trophozoite (and other forms) attach to the gut wall. Invasion does not occur.

The jejunum is the site most heavily infested. The pathogenesis of the diarrhea isunknown; no toxin has been identified.

Cryptosporidium causes diarrhea worldwide, for large outbreaks of diarrheacaused by Cryptosporidium are attributed to inadequate purification of drinkingwater.

The disease in immunocompromized patients presents primarily as a

watery, non-bloody diarrhea causing large fluid loss. Symptoms persist

For long periods in immunocompromized patients, whereas self-limited

In immunocompetent individuals. Although immunocompromized patients usually donot die of cryptosporidiosis, the fluid loss and malnutrition are severely debilitating.

Diagnosis is made by finding oocysts in fecal smears when using a modifiedKinyoum acid–fast stain. Serological tests are not available.

Review Questions

1) What are the two distinctive characteristics that differentiate protozoa from

other Eukaryotic protists?

2) What are the ecological advantages of protozoa?

3) Explain the reproductive process, transmission route and pathogenesis of

protozoan parasites.

4) How are medically important protozoa classified?

5) Describe the pathogenesis of E.histolytica.

6) Explain the clinical features of Giardia lamblia.

7) What are the drugs of choice for treatment of Trichomoniasisvaginalis?

8) What is the hemoflagellate responsible for causing kala-azar?

9) What are the protozoal species responsible for old world cutaneous

leishmaniasis?

10) Explain the pertinent clinical syndrome of Leishmaniaaethiopica.

11) What are the causative protozoa for African tryponosomiasis?

12) Explain the immune systems involved and the immune phenomenon in the

MEDICAL HELMINTHOLOGY

INTESTINAL FLUKES

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

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

LIVER FLUKES

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

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

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

LUNG FLUKES

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

NEMATODES (ROUND WORMS)

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

GENERAL CHARACTERISTICS OF NEMATODES

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

The parasitic nematodes:

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

The parasitic nematodes are divided into:

1. Intestinal nematodes

1.1. Intestinal nematodes with tissue stage

A. *Ascaris lumbricoides*

B. Hookworms

C. *Strongyloides stercoralis*

1.2. Intestinal nematodes without tissue stage

A. *Enterobius vermicularis*

B. *Trichuris trichuira.*

2. Tissue and blood dwelling nematodes
1. Filarial worms

2. *Dracunculus medinensis*

3. Trichinella

4. Larva migrans.

2.1. INTESTINAL NEMATODES WITH TISSUE STAGE

Ascaris lumbricoides

These are common roundworms infecting more than 700 million people

measures 15-20 cm in length. The posterior end is curved

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

Infective stage and modes of infection:

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

Life cycle:

Ingested eggs hatch in the duodenum. The larvae penetrate the intestinal wall and circulate in the blood. From the heart they migrate to the lungs, ascend to the trachea, descend to the esophagus and finally reach the small intestine to become adult. The female pass immature eggs which pass to the soil and mature in 2 weeks.

Pathogenecity and clinical features

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

Diagnosis

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

2. Demonstration of adult worms

HOOK WORMS

There are two species of hookworm:

1. *Ancylostoma duodenale*

2. *Necator americanus*

The adults are found in the small intestines of man. Mixed infection is common. Both of the species are found in Ethiopia, but *N. americanus* is more common.

***Ancylostoma duodenale*:**

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

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

Morphology

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

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

Necator americanus

This species, so called American hookworm, is found in predominantly the

tropics. The anterior end is hooked against the body curvature. The mouth is provided ventrally and dorsally with cutting plate.

Morphology

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

Female: The female measures 10 cm in length. The posterior end is straight

Infective stage and methods of infection:

The filariform larva infects by skin penetration.

Life cycle

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

Pathogenecity

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

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

LARVA MIGRANS

There are three types of larva migrans:

a. Cutaneous larva migrans (Creeping eruption)

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

1. *Ancylostoma braziliens*: infects both dogs and cats.

2. *Ancylostoma caninum*: infects only dogs.

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

b. Visceral larva migrans

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

Toxocariasis

This is a kind of visceral larva migrans caused by

♦ *Toxocara canis* (Dog ascarid) and

♦ *Toxocara catis* (Cat ascarid).

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

Morphology

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

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

Epidemiology

Visceral larva migrans is cosmopolitan in distribution.

Transmission:

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

Clinical features:

♦ Majority are asymptomatic.

♦ Eosinophilia

♦ Cerebral, myocardial and pulmonary involvement may cause death.

Diagnosis - Identification of larvae in tissue.

C. Intestinal larva migrans

This is an extremely rare kind of larva migrans

Strongyloides stercoralis

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

Morphology:

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

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

Infection: follows skin penetration by filariform larvae.

Life cycle

Adult male and female worms live in the small intestine. After fertilization, the female penetrates the mucosa of the small intestine and lay eggs in the submucosa. The eggs hatch and the larvae penetrate the mucosa back to the lumen. If the environmental conditions are favorable, the larvae will come out with the stool to the soil. They transform into adults, which lay eggs, and hatching larvae get transformed to adults and so on. If the environmental conditions are not favorable, the larvae in the stool will moult and transform into infective filariform larvae, which pierce the intestine (auto-infection). Larvae penetrating the skin from the soil or by autoinfection are carried by the blood to the lungs, ascend to the trachea, descend to the esophagus and mature in the small intestine.

Clinical presentation

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

Disseminated strongyloidiasis:

Multiplicity of symptoms are present due to the injury of other organs by the

migrating larvae. Organs such as liver, heart adrenals, pancreas, kidneys, and CNS, etc. may be affected. This is usually seen in immunocompromized individuals.

Diagnosis - Detection of rhabditiform larvae of strongyloides in stool. **gy**

CESTODES (TAPEWORMS)

INTRODUCTION

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

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

Hymenolepis nana (dwarf tapeworm)

MorpholoAdult worm measures 1-3 cm in length. It is made up of head (scolex), neck and

segmented body. The head carries four suckers and a rostellum armed with one row of hooks. The segments of the body are divided into mature and gravid

segments. In the mature segment, there are three testes in the middle.

Infective stage and mode of infection

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

Infection takes place by:

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

Pathogenecity

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

Hymenolepis diminuta (RAT TAPEWORM)

Hymenolepis diminuta differs from *Hymenolepis nana* in that:

♦ The adult worm measures about 10-60 cm

♦ The rosetellum on the head has no hooks

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

on the other side.

Life cycle

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

Pathogenecity

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

ECHINOCOCCUS

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

Echinococcus granulosus (dog tape worm)

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

Morphology

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

Life cycle and Pathogenecity

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

♦ Penetrate wall

♦ Enter portal veins

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

Mode of human infection

Ingestion of eggs by the following ways:

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

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

Clinical features

Asymptomatic infection is common, but in symptomatic patients

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

♦ Hepatomegaly - with abdominal pain and discomfort

♦ Pressure -from expanding cyst

♦ Rupture of cyst - severe allergic reaction - anaphylaxis.

Diagnosis:

♦ X-ray or other body scans

♦ Demonstration of protoscolices in cyst after operation

♦ Serology

Echinococcus multilocularis

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

Taenia saginata (BEEF TAPEWORM)

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

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

1. Scolex: the hold fast organ

2. Neck: posterior to the scolex

3. Stobilla: the main bulk, made up of proglottids.

Morphology:

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

Life cycle

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

Pathogenecity

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

Diagnosis

Recovery of the gravid segments or the eggs from the stool

Prevention:

♦ Thorough cooking of meat (above 570C).

♦ Proper disposal of human excret

Taenia solium (pork tapeworm)

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

Morphology:

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

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

Life cycle

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

Clinical manifestations

Resembles that of *T. saginata* infection

Diagnosis

Demonstration of eggs in stool specimen

Prevention:

♦ Treatment of infected persons.

♦ Thorough cooking of pork and proper processing

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

Diphylobotrium latum (FISH TAPEWORM OR BROAD TAPEWORM)

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

Morphology

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

Life cycle

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

Clinical manifestation

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

Diagnosis

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

Prevention:

Prohibiting the disposal of untreated sewage into fresh water /lakes.

Personal protection: cooking of all fresh water fish.

INTRODUCTION

Medical entomology is a science, which deals with the study of arthropods. Members of the phylum arthropoda are the most numerous and widely distributed of all animal groups. Their medical importance lies in their ability to cause morbidity and mortality, and their extensive distribution over the face of the

earth. They may be found in every part of the world and in every type of environment. Many, particularly those within the class insecta and arachnida, live in close association with humans; others while primarily parasites of animals, will readily attack or feed upon humans and some may specifically adapt as human parasites.

ARTHROPODS

The arthropods include animals varying considerably in size and shape but have fundamental features in common.

Generally all arthropods have the following characteristics in common:

- They are bilaterally symmetrical
- Their bodies are divided into a number of rings or segments.
- They have jointed appendages, which may take the form of legs, antennae, or mouthparts.
- They have a hard chitinous exoskeleton (cuticle), which helps for the protection and insertion of muscles.
- The exoskeleton is partitioned by chitinous plates (scleritis) in order to

allow movement. The dorsal and ventral sections, the tergum, and sternum respectively are heavily chitinized. The lateral section, joining the tergum and sternum (pleuron) is less heavily chitinized and thus more flexible.

• They have a body cavity called haemocele, which contains haemolymph (blood and lymph) that bathes internal organs.

• Ecdysis or moulting is a phenomenon characteristic of all arthropods whereby the cuticle is shed at regular intervals in order to accommodate the growing tissues.

BIOLOGY OF ARTHOPODS

Arthropods use the following systems for survival and perpetuation.

(a) Digestive system

The alimentary canal comprises three distinct regions: the foregut or stomodium, the midgut or mesenteron, and the hindgut or proctodaeum:

Foregut - extends from the mouth to the proventriculus (muscular sac provided with stony cuticular plates or teeth serving for grinding and mixing of food.)

Mid gut - this is the stomach; physiologically the most active part of the alimentary canal, being concerned with digestive function.

Hind gut -consists of the ileum, colon, and rectum and finally opens to the anus.

(b) Circulatory system

The circulatory system of all arthropods is of the “open” type, i.e. the fluid that circulates is not restricted to a network of conducting vessels as for example in vertebrates, but flows freely among the body organs. A consequence of the open system is that insects have only one extra cellular fluid, hemolymph, in contrast to vertebrates which have two such fluids, blood and lymph. Through this system hemolymph is pumped from the heart to the aorta then to the whole body. The circulatory system has no respiratory function.

(c) Respiratory system

In the vast majority of insects, respiration is by means of internal air tubes known as trachea. These ramify through the organs of the body and its appendages, the finest branches being termed tracheolea. The air generally enters the trachea through paired, usually lateral openings termed spiracles, which are segmentally arranged along the thorax and abdomen. Respiratory spiracles also serve as exit of air conducting branches from the tracheal tube. Respiratory spiracles serve as exit of air conducting braches from the tracheal tube.

(d) Nervous system

The many diverse activities of the various systems of an insect are coordinated by the nervous system. This system is composed of elongated cells, or neurons, which carry information in the form of electrical impulses from internal and external sensory cells to appropriate effectors. These consist of Nerve ganglia in the head, ventral part of the body, which later

extends to body parts.

(e) Excretory System

The function of the excretory system is to maintain hemostasis. i.e. maintaining the uniformity of the hemolymph. It accomplishes this by the elimination of metabolic wastes and excesses, particularly nitrogenous ones, and the regulation of salt and water. The malpigian tubules are the major organs involved in filtration of the hemolymph. These tubules lie freely in the body cavity (haemocele) and open to the junction between the mid gut and the hindgut. After joining the digestive tract, waste fluids are excreted through the anus. The hindgut (specially the rectum) is involved in reabsorption of important ions and water.

(f) Reproductive System

Arthropods have separate sexes. Male contains testes, vas deference, seminal vesicle and ejaculatory duct, which open by aedeagus (penis). The female contains two ovaries, oviduct, and uterus that opens to the vagina.

DEVELOPMENT OF ARTHROPODS

The development of arthropods, which is called metamorphosis, is from egg to adult. This development could be:

Incomplete development from the egg to nymph, which looks like the adult

OR

Complete development, which extends from the egg to larva, pupa

that later differentiate to the adult arthropod.

IMPORTANCE OF ARTHROPODS IN PARASITOLOGY

Arthropods affect the health of man by being:

(a) Direct agents for disease /discomfort.

The following effects may be seen by the direct effect of arthropods.

・Annoyance – comes from disruptive activities of insects, such as flying around or landing on the head, and from feeding, possibly causing blood loss, though they don't remove sufficient blood to cause a medical problem in humans.

・Entomophobia – is an irrational fear of insects. One extreme form of entomophobia is delusory parasitosis, in which individuals become convinced that they are infested with insects when no actual infestation exists. This may cause undue alarm and anxiety, leading to unwarranted use of insecticides, and in severe cases, requiring professional treatment.

・Envenomization – is the introduction of a poison into the body of humans and animals. Arthropods may also inoculate poison to the host. E.g. Scorpion

・Allergic reactions – a hypersensitive response to insect proteins. All of the mechanisms associated with envenomization can also cause exposure to allergens. In fact, human deaths from bee and wasp stings usually are associated with a hypersensitive reaction

rather than direct effect of a toxin.

・Dermatosis and dermatitis – dermatosis is a disease of the skin and dermatitis is an inflammation of the skin. Both dermatosis and dermatitis can be caused by arthropod activities. Many mite species, such as scabies mites produce acute skin irritations.

(b) Agents for disease transmission

Arthropods can carry disease causative agents in the following two ways.

・Mechanical carrier

Here they lodge the disease causative agent without altering its development or multiplication

e.g. house fly

• Biological carrier

When arthropods become biological carriers for transmission of disease, it means that certain stages in the life cycle of parasite takes place in the body of the insect.

e.g. Anopheles mosquitoes.

Biological carrier is any of the following types:

 Propagative- where there is multiplication of the parasite with no developmental change

e.g. Yellow fever virus in Aedes mosquito.

 Cyclopropagative – in this type both multiplication and developmental change are going on.

e.g. Plasmodium species in Anopheles mosquito

 Cyclodevelopmental – here there is developmental change

of the parasite but no multiplication

E.g. Wucherera bancrofiti in Culex mosquito

 Transovarian- when the pararasite passes to progeny

arthropods through the ova

E.g. Ricketsia typhi in ticks

If we are clear about the importance of arthropods as a source of human infection, it is important to accurately identify and classify them for crucial treatment, prevention, and control of infection