Topic 1:INTRODUCTION TO PARASITOLOGY

**Historical perspective of parasitology**

**Antony van Leunhoek** of Holland first described microscopic organisms in the faeces of man and animals and named them **animalcules.**

He described free living and parasitic protozoa, filamentous fungi of globular bodies (yeasts) in 1683.

The term protozoa was first used in 1817 by Goldfus of Germany.

Tape warms were first isolated in 1782.

**DEFINITION OF TERMS IN MEDICAL PARASTOLOGY.**

1. **Parasitology** is the area of biology concerned with the phenomenon of dependence of one living organism on another.
2. **Medical parasitology** is the science that deals with organisms living in the human body (the host) and the medical significance of this host-parasite relationship. It deals with the parasites which infect man, the diseases they produce, the response generated by him against them and various methods of diagnosis and prevention.
3. **Parasite:**is a living organism, which takes its nourishment and other needs from a host. The term parasite is usually applied to ***Protozoans ( Unicellular micro-organism)***and ***Helminths (Multicellular organism).***
4. **Host:** is an organism which supports the parasite.The parasites included in medical parasitology are**protozoa**, **helminthes**, and some **arthropods**.
5. **Ecto-parasite(Ectozoa) –**a parasitic organism that lives on the outer surface of its host, e.g.lice, ticks, mites etc.
6. **Endoparasites**– parasites that live inside the body of their host in the blood, tissues, body cavities, digestive tract and other organs.e.g. *Entamoeba* *histolytica.*
7. **Obligate Parasite**- This parasite is completely dependent on the host during a
8. segment or all of its life cycle, e.g. Plasmodium spp. It cannot exist without a parasitic life.
9. **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*(Lives a parasitic life when opportunity arises).
10. **Accidental parasite –**when a parasite attacks an unnatural host and survives. E.g. *Hymenolepis diminuta*(rat tapeworm). It’s also known as occasional parasite
11. **Temporary parasite-**Visits its host for a short period.
12. **Permanent parasite**-leads a parasitic life throughout the whole period of its life.
13. **Wandering or Aberrant parasite-**Happens to reach a place where it cannot live.
14. **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.

**CLASSES OF HOSTS-**

1. **Definitive host –**a host that harbors a parasite in the adult stage or where the parasite undergoes the sexual method of reproduction. In majority of human parasitic infections, man is the definitive host
2. **Intermediate host**- harbors the larval stages of the parasite or the 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. In Malaria and Hydatid disease man is the intermediate host.
3. **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.
4. **Reservoir host –**a host that makes the parasite available for the transmission to another host and is usually not affected by the infection.
5. **Natural host –**a host that is naturally infected with certain species of parasite.
6. **Accidental host –**a host that is under normal circumstances is not infected with the parasite.

**VECTORS**

1. **Vector**-A vector is an agent, usually an insect, that transmits an infection from one human host to another.
2. **Mechanical vector-**This is a vector which assists in the transfer of parasitic forms between hosts but is not essential in the life cycle of the parasite.e.g. a housefly that transfers amoebic cysts from infected feaces to food that is eaten by humans.
3. **Biological vector-** In this group the parasite undergo a biological development or multiplication in the body of the vector. e.g Mosquito in the transmission of plasmodium.

**LIFE CYCLE OF PARASITES**

These are developmental stages that the organisms pass before a stage is attained and the organism reproduces and new cycle of development begins. The life cycle of parasite differ in different species. It maybe sexual or asexual.

1. **Direct life cycle (Simple**): When a parasite requires only one species of host to complete its development, it is referred to as direct life cycle. e.g.E. histolytica .
2. **Indirect life cycle (Complex**): When a parasite requires two or more species of hosts to complete its development, then the life cycle is referred to as indirect life cycle. E.g. Filariasis, Plasmodium.

**HOST PARASITE RELATIONSHIP**

1. **Symbiosis**-It is an association in which both organisms are so dependent upon each other that one cannot live without the help of the other. None of the partner suffers any harm from the association.

* N/B: Any organism that spends a portion or all of its life cycle intimately associated with another organism of a different species is considered as **Symbiont (symbiote**) and this relationship is called **symbiosis (symbiotic relationships).**

1. **Commensalism**-An association in which the parasite only is deriving benefit without causing injury to its host. A commensal is capable of leading an independent life.

* N/B: Most of the normal floras of the human body can be considered as commensals.

1. **Mutualism -**an association in which both partners are metabolically dependent upon each other and one cannot live without the help of the other; however, none of the partners suffers any harm from the association.
2. **Parasitism**-An association in which the parasite derives benefit and the host gets nothing in return but always suffers some injury. A parasite has lost its power of independent life. E.g. Worms like *Ascaris lumbricoides*reside in the gastrointestinal tract of man, and feed on important items of intestinal food causing various illnesses.
3. **Zoonosis:**Parasitic infection which are primarily confined to vertebrate animals but can cause disease in man if they become infected e.g. Leishmaniasis, echinococcosis trichinosis.
4. **Anthroponosis:**Parasitic infections that are confined to man only e.g. Malaria & filarial.
5. **Zooanthroponosis:**Infections in which human being is not an accidental host, but serves as an essential link in the life cycle of the parasite e.g. beef and pork tapeworm.

**CLASSIFICATION OF PARASITES**

a)      Parasites are divided into two**phyla**:

1. **Protozoa**: These are unicellular parasites
2. **Metazoa :**These are multi cellular parasites.

d)      Parasites of medical importance come under the kingdom called **protista** and **animalia**.

e)      **Protista** includes the **microscopic** **single-celled eukaroytes** known as **protozoa**.

f)        In contrast, helminthes are **macroscopic, multicellular** worms possessing well differentiated tissues and complex organs belonging to the kingdom **animalia**.

1. **Medical Protozoology -**Deals with the study of medically important protozoa.
2. **Medical Helminthology -**Deals with the study of helminthes (worms) that affect man.
3. **Medical Entomology -**Deals with the study of arthropods which cause or transmit disease to man.

Top of Form

Bottom of Form

**.   Phylum protozoa:**

These parasites are unicellular eukaryotic organisms and the single cell carries out all the functions of the parasite like reproduction, digestion, respiration and excretion.

They usually measure from 1-150 μm.

Examples of protozoa are:

**Entamoeba histolytica, Giardia lamblia, Plasmodia,**

**Leishmania and Trypanosoma.**

**According to the site of infection the protozoan paraites are classified as under:**

**Blood & Tissue Flagellates**:              Leishmania, Trypanosoma

**Intestinal Flagellates**:                       Giardis lamblia, Trichomonas hominis  Enteromonas hominis,Retromonas intestinalis

**Oral flagellates**:                                 Trichomonas tenax

**Genital flagellates**:                            Trichomonas vaginalis.

**B.    Phylum Metazoa:**

These parasites are multicellular. They consist of helminths. Examples of metazoan are Cestodes, Nematodes, and Trematodes.

 Helminths are further classified into platyhelminths and nemathelminths. The platyhelminths are further classified into cestodes and trematodes.

**Helminths:**The following are common morphological features of helminths:

·         No organs of locomotion

·         Have tough cuticle

·         Gastro intestinal tract (GIT) absent or rudimentary or developed

·         Nervous system primitive

·         Very well developed reproductive system

·         Hermaphrodites or separate sexes

·         Enormous number of eggs produced

·         Do not multiply in humans (generally).

**CLASSIFICATION OF MEDICALLY IMPORTANT PARASITES**

**PROTOZOA                                                     METAZOA (HELIMINTHS)**

**Sarcodina (Amoebae):                                   Platyhelminthes:**

(a) Genus, Entameba:                                     **Trematodea:**

E.g. *Entameba histolytica*(a) Genus Schistosoma

(b) Genus Endolimax                                            E.g. *S. mansoni*

E.g. *Endolimax nana*(b) Genus Fasciola

(c) Genus Iodameba                                          E.g. *F. hepatica*

E.g. *Iodameba butchlii*

**Cestoda:**

(d) Genus Dientmeba                                  (a) Genus Diphylobotrium

E.g. *Dientameba fragilis*E.g. *D. latum*

**Mastigophora (Flagellates):**(b) Genus Taenia

(a) Genus Giardia                                                       E.g. *T. saginata*

E.g. *G. lamblia*(c) Genus Echinococcus

(b) Genus Trichomonas                                                            E.g. *E. granulosus*

E.g. *T. vaginalis*(d) Genus Hymenolepsis

(c) Genus Trypanosoma                                                          E.g. *H. nana*

E.g. *T. brucci***Nemathelminthes:**

(d) Genus Leishmania                                                              (a) Intestinal Nematodes

E.g*. L. donovani*E.g. *A. lumbricoides*

**Sporozoa**(b) Somatic Nematodes

(1) Genus Plasmodium                                                                       E.g. *W. bancrofti*

E.g. *P. falciparum*

(2) Genus Toxoplasma

E.g. *T. gondi*

(3) Genus Cryptosporidum

E.g. *C. parvum*

(4) Genus Isospora E.g. *I. beli*

**Ciliates**

E.g. *Balantidium coli.*

**OURCES OF PARASITIC INFECTIONS:**

1. Contaminated soil and water
2. Freshwater fishes-Diphyllobothrium latum and Clonorchis sinensis.
3. Crab and crayfishes-Paragonimus wertermani.
4. Raw or undercooked pork-Trichinella spiralis, T.solium.
5. Raw or undercooked beef-T.saginata, Toxoplasma gondii.
6. Watercress-Fasciola hepatica
7. Blood sucking insects
8. Housefly-Mechanical carrier-E.histolytica
9. Dog-Echinococcus granulosus and Toxocara canis.
10. Cat-T.gondii.
11. Man-E.histolytica, Enterobius vermicularis and H.nana.
12. Autoinfection-May occur with E.vermicularis and S.stercoralis leading to hyperinfection.

**MODE OF INFECTION OF PARASITIC INFECTIONS:**

***a)      Ingestion***

It is called faecal-oral route by contaminated food, water soiled finger or formites

a)      Ingest parasites in raw or undercooked meat e.g. Tinea saginata, Tinea Solium, Diplyllobothrium latun or paragoninus westermani in crab or cray fish.

b)      By ingesting food or drink contaminated by infective form of parasite e.g. cysts of E.hitolytica, ova of Ascaries lumbricoides Tricluri,Trichura & Enterobius vermicularies.

c)      By ingesting water containing infected Cyclops e.g. Drucuculus medinensis.

d)      By ingesting aquatic plants containing metacercarial forms of parasite that have come out of its intermediate host & encyst in acquatic plants.

***b)      By skin transmission;***

a)      The ***Filariform larvae*** of Ancylostoma duodenale & strongyloides stercoralis in contaminated soil penetrate intact skin.

b)      By contact with water containing cercarial forms of S.haematobium, S.Manson; S.Japonicam and penetrate the skin.

***c)      By the agency of insect host***

Some anthropods (blood-sucking insects) may introduce the parasite while sucking blood e.g. Plasmodium by anoplyline mosquito, Trypanosoma by Tsetsefly, Leishmania by sand fly, Wucheria bancrofti by culex mosquito.

In this group the parasite undergo a biological development or multiplication in the body of the vector. These vectors are called ***Biological Vectors.***

***d)      Direct transmission***

a)      By sexual contact e.g. Trichomonas vaginalis.

b)      By kissing in the case of gingival amoebae.

***e) Inhalation-***

***f). Iatrogenic infection-***Malaria parasites may be transmitted by transfusion of blood from the     donor with malaria containing asexual forms of erythrocytic schizogony.This is known as trophozoite induced malaria or transfusion malaria. Malaria parasites may also be transmitted by the use of contaminated syringes and needles.This may occur in drug addicts.

**PATHOGENIC EFFECT OF PARASITIC INFECTIONS**

Parasite infections may remain inapparent or may cause disease of clinical significance depending on:-

a)      **Parasite** – strain, number, size, metabolic process

b)      **Host** – age, natural immunity, immunity to infection, co-existing disease, nutritional status, lifestyle.

**Protozoal infections**

The lesions are greatly influenced by proliferation, multiplication and metastatic spread to distant organs.

**Helminthic infections**

In most helminthic infections, man serves as the definitive host and adult parasites are found within the body.

No multiplication takes place except in cases of strongyloides sterecoralis & Hymenolepis nana.

The infection depend on the number of invading organisms

Clinically they present as:-

***1.      Physical obstruction***

Round worms (Ascaris Lubricoides) causing intestinal obstruction. A single warm may also block the appendix or bile duct.

***2.      Pressure effect***

Hydatid cyst may cause pressure effect on surrounding tissues.

Pressure effects if parasites are present on vulnerable sites e.g. brain, eyes.

***3.      Vascular or Lymphatic obstruction***

P.falciparum infection causing blockage of capillaries of cerebral cortex leading to fatal cerebral malaria.

***4.      Trauma***

Clinical disease may result from trauma e.g. Hookworm infection.

***5.      Allergic Manifestations.***

Secretions and excretions of growing larvae and products released from dead parasites may lead to allergic manifestations.

***6.      Predisposition to Malignancy***

Parasitic infection may contribute to development of neoplastic growths. E.g. liver flukes; schistosoma haemalobium.

**Immunity in parasitic infections.**

Parasitic infection generate antibody response and effector T cell (cell mediated) response but the parasite elimination is much less efficient than that against bacterial & viral infectious.

***Nb:***

-          Parasites have evolved to be closely adapted to the host

-          The infections are chronic and show host specificity

**Acquired immunity**

***1.      Antibody response***

Immune response to parasites results in production of immunoglobulins type IgG. &IgM. IgE is produced in Helminthic infectious while IgA is produced in intestinal protozoal (Entamoeba, Gardia).

**How do they work?**

***Neutralization (How anti-body response work).***

Antibody combines with various surface molecules of parasites & neutralize them and thereby block or interfere with their proper functioning.

***2.      Agglutination***

Agglutination of blood parasites by IgM prevents spread of the parasite.

***3.      Physical clearance***

Antibodies that block orifices of certain worms e.g. oral or genital opening – interfere with their physiological functions & can cause starvation or curtail reproduction.

***4.      Opsonisation***

Antibody can increase the clearance of the parasite by phygocytic cells.

***5.      Antibody reacting with surface antigens***

Antibodies binding to the cells of parasites resulting in direct damage or lysis.

***6.      Antibody dependent cell-mediated cytotoxicity (ADCC).***

It is usually seen in some parasitic infections e.g. Trypansoma cruzi, S.Mansoni, Trichinella spirallis & Filarial worms.

**Cellular Response.**

***1.      T.Lympghocytes***

CD4 + T cells acts as helper cells in antibody production & CD8 + T cells are cytotoxic in several instances.

***2.      Macrophages***

Play a dominant role in the process of elimination of Protozoa and worms.

***3.      Granulocytes***

Neutrophils & eosinophils play important roles in elimination of protozoa & helminthes.

Unlike bacterial infection, complete elimination of infecting parasites followed by immunity to reinfection is seldom observed.

**Evasion Mechanisms**

Like bacteria some parasites have evolved effective mechanism to avoid elimination by the host defense systems. The intracellular location of many protozoa helps them to evade immunological attack.

**Escape mechanisms of parasites.**

1.      Intracellular habitat. E.g. Malaria parasites, trypanosome, leishmania

2.      Encystment – Toxoplama godii, trypanosome cruzi.

3.      Resistance to microbicidal products of phagocytes. Leishmania Donovani

4.      Variation of antigen – Malaria parasite, trypanosome

5.      Masking of antigen – Schistosomes

6.      Interference by antigens – trypanosomes

7.      Polyclonal activation – Trypanosomes

8.      Suppression of immune response – Most parasite (Malaria parasites, T.Spirallis, S.mansoni).

9.      Sharing of antigens – Schistosomes between parasite & host

10.  Continuous turnover and schistosomes release of surface  antigens

Topic 1.1: CESTODES (Taeniasis/Cysticercosis)

**1. TAENIASIS AND CYTICERCOSIS**

* Taeniasis and cysticercosis are diseases resulting from infection with parasitic tapeworms belonging to Taenia species. Approximately 45 species of Taenia have been identifi ed; however, the two most commonly responsible for human infection are;

1. The pork tapeworm Taenia solium and
2. The beef tapeworm Taenia saginata.

* Infection with adult tapeworms of either T. solium or T. saginata cause taeniasis in humans. Th e metacestode, or larval stage, of Taenia solium causes the tissue infection, cysticercosis.
* **Life-Cycle of Taeniasis;**
* The complete life-cycle of **Taenia solium** involves two hosts:

1. The pig and
2. The human,

whereas that of**Taenia saginata** involves;

1. The cow and
2. The human

* Humans act as the definitive host and harbor the adult tapeworm in the small intestine.
* Infection is acquired either through the accidental ingestion of embryonated eggs passed in the feces of an individual infected with the adult tapeworm, or through the consumption of raw or poorly cooked meat containing cysticerci.
* The cysticerca develops into an adult worm in the gut; these worms can survive up to 25 years.
* Depending on the species of Taenia, an adult worm can reach lengths between 2-25 meters and may produce as many as 300,000 eggs per day.
* The morphology of the adult worm consists of a scolex and a strombila. The scolex acts as the organ of attachment and consists of four suckers equipped with hooklets. The strombila consists of several segments (proglottids) with the gravid or egg-carrying proglottids located  toward the posterior end of the worm.
* Individual proglottids may contain as many as 40,000 eggs in T. solium or as many as 100,000 eggs in T. saginata. Both the proglottids and the eggs are released with the feces of infected individuals and serve as a source of infection for pigs and cattle, which act as intermediate hosts for these parasites.
* Following the ingestion of eggs, mature larvae (onchospheres) are released in the gut. These onchospheres enter the blood
* stream by penetrating the small intestine and migrate to skeletal and cardiac muscles where they develop into cysticerci. Cysticerci may survive in the host tissues for several years causing cysticercosis
* The consumption of raw or undercooked meat containing cysticerci facilitates the spread of infection from pigs to humans. In humans, cysticerci transform into adult tapeworms which persist in the small intestines for years causing taeniasis. The time between initial infection and the development of the adult worm occurs over a period of  approximately 2 months. In some instances, an infected individual harboring the adult worm can become auto-infected through the accidental  ingestion of eggs released in the feces.
* **Signs and symptoms;**
* **1. Taeniasis**
  1. Minor gastrointestinal irritation.
  2. Nausea,
  3. Diarrhea,
  4. Constipation,
  5. Hunger pains,
  6. Passage of proglottids in the feces.
* **2. Cysticercosis;**
* The clinical manifestations associated with cysticercosis are a direct result of the
* inflammatory response induced to control parasite growth and may occur months to years after initial infection.
* Manifestations of disease are dependent upon a variety of factors including the site of infection as well as the number of
* cysticerci present within the tissues, which most often localize to sites within the eyes, skeletal muscles and brain.
* Cysticercosis is the most common intra-orbital parasitic infection and is observed in 13-46% of infected individuals. Infection may involve the sub-retinal space (intra-ocular) or the extraocular muscles, eyelid and/or lachrymal glands (extra-ocular) surrounding the eye(s).
* Patients infected with cysticerci in the skeletal muscles and/or subcutaneous tissues are usually asymptomatic. In most cases, multiple cysts are present within the tissues, although solitary cysts may also be detected. Cysts range from 10-15
* mm in length and arrange themselves in the same orientation as the muscle fibers.
* Leakage of fluid into the tissues, or death of the parasite, can trigger a strong inflammatory response, resulting in sterile abscess formation accompanied by localized pain and swelling.
* **Diagnosis of Taeniasis and Cysticercosis;**

1. Stool for ova and cysts.- Proglottids and eggs are identified by microscopy.
2. Histology for cysticercosis.
3. Serology detection tests with Enzyme Linked Immunosorbent Assay (ELISA).

**Treatment;**

1. Albendazole.

### 2. Praziquantel for cysticercosis Topic 1.2: CESTODES (Hydatidosis)

Hydatid disease, also called hydatidosis or echinococcosis, is a cyst-forming disease resulting from an infection with the metacestode, or larval form, of **parasitic dog tapeworms from the genus Echinococcus.**

* To date, five species of Echinococcus have been characterized. The vast majority of human diseases are from;

1. Echinococcus granulosus and
2. Echinococcus multioccularis which cause cystic echinococcosis and alveolar echinococcosis, respectively.

* **Life Cycle**
* Hydatid disease is caused by infection with the larval form of E. granulosus (and/ or E. multiocularis) and results in the formation of cysts within various host tissues.
* The complete life cycle of Echinococcus granulosus requires two hosts.Domestic dogs act as the primary definitive host of the mature adult worms and a single infected dog may harbor millions of adult worms within its intestines.
* Other canines such as wild dogs, wolves, coyotes, foxes and jackals may also act as a definitive host harboring the adult tapeworms. Intermediate hosts become infected with the larval form of the parasite and include a wide range of herbivorous animals, primarily sheep, cattle, pigs, goats and horses.
* The life cycle is completed by the ingestion of one or more cysts and its contents by the canine host through the consumption of infected viscera of sheep and and/or other livestock. Protoscoleces released in the small intestine attach to the intestinal wall through the action of four suckers and a row of hooks and within two months mature into adult worms capable of producing infective eggs.
* Humans may become infected though the ingestion of food and/or water contaminated with infective eggs released in the feces of dogs harboring the adult
* tapeworm(s). Once ingested, the eggs release oncospheres capable of actively
* penetrating the intestinal mucosa.
* These oncospheres gain access to the blood stream via the hepatic portal vein and migrate to various internal organs where they develop into cysts. Hydatid cysts most oft en localize within the liver and the lungs; however, cysts may also form in the bones, brain, skeletal muscles, kidney and spleen.
* The clinical manifestations of hydatid disease vary depending on a variety of factors including the location, size and number of cysts present within the infected tissues.
* Life cycle of Echinococcus.
* Similar to E. granulosus, the complete life cycle of E. multiocularis also requires two hosts. Th e primary defi nitive host for E. multiocularis is the fox, although the parasite may also infect wild and domesticated dogs and occasionally cats. Rodents such as field mice, voles and ground squirrels act as natural intermediate hosts and acquire infection by ingesting infective eggs released into the environment.
* **Signs and Symptoms;**
* Echinococcus granulosus and Echinococcus multiocularis are the two species most oft en identified in human hydatid disease. Cystic echinococcosis, caused by E. granulosus, is the most common and accounts for approximately 95% of all
* global cases.
* Cystic echinococcosis may affect people of all ages, but hydatid cysts are most oft en present in patients between 15-35 years of age. Infection with E. granulosus results in the rapid growth of large, uniocular cysts fi lled with fluid . Most cysts develop within the tissues of the liver and lung, with 55-75% of cysts found in the liver and 10-30% of cysts found in the lungs.
* Cysts may survive in the liver for several years and oft en do not cause any symptoms in the infected host. Symptoms arise when the cysts become large enough to be palpable and/or cause visual abdominal swelling and pressure. Patients frequently experience abdominal pain in the right upper quadrant, oft en accompanied by nausea and vomiting.
* The rupture or leakage of cysts within the tissue can result in anaphylactic shock and facilitate the spread of secondary cysts through the release and dissemination of germinal elements. Biliary tract disease and portal hypertension may complicate liver involvement and post obstructive infection due to erosion of cysts into the biliary tract may further complicate echinococcal infection. Pulmonary cystic echinococcosis is acquired early during childhood, but the clinical manifestations associated with the disease do not typically appear until the third or fourth decade of life.
* Cysts residing within the lung tissue oft en remain silent producing little to no symptoms. Problems arise when cysts grow large enough to obstruct or erode a bronchus, oft en causing the rupture of cysts and the dissemination of cystic fluids. Patients infected with pulmonary cysts frequently experience;

1. Chronic dry cough,
2. Chest pain and hemoptysis often  accompanied by
3. Headache,
4. Sweating,
5. Fever and malaise.

* Hydatid disease can affect a wide range of organs including the bones, central nervous system, heart, spleen, kidneys, muscles and eyes. Patients diagnosed with the disease should be screened for the presence of multiple cysts in various tissues.
* **Diagnosis of hydatidosis;**
* Individuals affected normaly remain asymptomatic for many years;

1. Ultrasound of abdominal organs.
2. CT scan.
3. MRI.
4. Serological assay.
5. DNA PCR.

**Treatment of hydatidosis;**

1. Surgery.
2. Albendazole 400mg BD X 1-6/12.

opic 1.3:CESTODES (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**

1. Most infections are asymptomatic.
2. Rarely, it causes
   1. Severe cramping,
   2. Abdominal pain,
   3. Vomiting,
   4. Weakness and
   5. Weight loss.
   6. Pernicious anemia can also result, due to interference of vitamin B12 absorption in jejunum.

**Diagnosis**

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

**Treatment**

Niclosamide: 2 gm PO stat after light breakfast.

Prevention:

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

Personal protection: cooking of all fresh water fish.

opic 1.4: CESTODES (HYMENOLEPSIS NANA/DWARF TAPEWORM)

**Morphology**

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

**Infective stage and mode of infection**

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

Infection takes place by:

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

**Pathogenecity**

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

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

opic 1.5: TREMATODES (SCHISTOSOMIASIS/BILHARZIOSIS)

It is estimated that about 600 million people in 79 countries suffer from schistosomiasis (Bilharziasis). The schistosomes cause intestinal, hepatosplenic, pulmonary, urogenital, cerebral and other forms of schistosomiasis.

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

**There are five medically important species:**

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

The first two schistosomes (S. mansoni and S. haematobium) are the most prevalent.

**SCHISTOSOMA MANSONI**

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

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

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

Morphology

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

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

**URINARY SCHISTOSOMIASIS**

Etiology - Schistosoma haematobium

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

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

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

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

Distribution: In Ethiopia, S. haematobium is found in the Lower Awash Valley in the east and in Benshangul-Gumuz (Assossa) regional state in the west in low altitudes below 1000 meters above sea level.

**SCHISTOSOMA JAPONICUM**

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

**SCHISTOSOMA INTERCALATUM**

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

**LIFE CYCLE OF SCHISTOSOMES**

Adult worms reside in pairs: the female lying in the gynecophoral canal of the male. After fertilization, eggs are passed into the venules. A larval form – the miracidium - develops within the egg. Its lytic enzymes and the contraction of the venule rupture the wall of the venule liberating the egg into the perivascular tissues of the intestine (S. mansoni) or urinary bladder (S. haematobium).

The eggs pass into the lumens and organs and are evacuated in the feces (S.mansoni) or the urine (S. haematobium). On contact with fresh water the miracidia hatch from the eggs and swim about until they find the appropriate snail, which they penetrate. After two generations of sporocyst development and multiplication within the snail, the fork-tailed cercariae emerge. Infection to man takes place during bathing or swimming.

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

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

Symptoms and complications

1. Patients infected with S. haematobium suffer from terminal haematuria and painful micturition. There is inflammation of the urinary bladder (cystitis), and enlargement of spleen and liver.
2. Patients infected with S. mansoni suffer from cercarial dermatitis (swimmers itch) and dysentery (mucus and blood in stool with tenesmus) as well as enlargements of the spleen and liver.
3. S. haematobium causes squamous cell carcinoma in the bladder.

**Laboratory Diagnosis**

**1. S. mansoni**

1. Microscopic examination of the stool for eggs after concentration by sedimentation method. The egg has characteristic lateral spine.
2. Rectal snip

**2. S. haematobium:**

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

**Treatment:**

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

**Prevention:**

1. Health education:

1. On use of clean latrines and safe water supply
2. Avoid urination and defecation in canals, avoid contact with canalwater

2. Snail control:

A. Physical methods:

1. Periodic clearance of canals from vegetations.
2. 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 the

snails. e.g. Endod

Topic 1.6: TREMATODES (FLUKES)

**INTESTINAL FLUKES**

1. Fasciolopsis buski: These giant intestinal flukes (2-7.5 cm in length) are found in some Asian countries.
2. Heterophyids: Minute flukes acquired by ingestion of raw fresh water fish. They are found in Asian countries.

**1. LIVER FLUKES**

1. Clonorchis sinensis: Chinese liver fluke - adult worms live in bile ducts.
2. 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.
3. Faciola gigantica: lives in the liver of cattle. Human infections are very rare.

**2. 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.

Topic 1.7: NEMATODES (INTRODUCTION)

* All the important human parasites of the Phylum Nemathelminthes (Aschelminthes) belong to the Class Nematoda.
* **GENERAL CHARACTERISTICS OF NEMATODES**

1. They are un-segmented, elongated and cylindrical.
2. They have separate sexes with separate appearances.
3. They have a tough protective covering or cuticle.
4. They have a complete digestive tract with both oral and anal openings.
5. 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) Intestinal nematodes with tissue stage**

1. Ascaris lumbricoides
2. Hookworms
3. Strongyloides stercoralis

* **2) Intestinal nematodes without tissue stage**

1. Enterobius vermicularis
2. Trichuris trichuira.

* **2. Tissue and blood dwelling nematodes**

1. Filarial worms
2. Dracunculus medinensis
3. Trichinella
4. Larva migrans.

opic 1.8:NEMATODES (ASCARIOSIS)

**ASCARIS LUMBRICOIDES**

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

**Morphology:**

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

Infective stage and modes of infection: The egg containing larva when ingested with contaminated raw vegetables causes ascariasis.

**Life cycle:**

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

**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

**Treatment**

1. Mebendazole,
2. Albendazole and
3. Piperazine

Topic 1.9: NEMATODES (HOOK WORM)

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 Kenya, 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. Thelarv ae may cause inflammation of the lungs.

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

**Treatment**

Mebendazole: 1 tab 2x daily for 3 days.

opic 1.10; NEMATODES (LARVAE MIGRANS)

**There are three types of larva migrans:**

* **1. Cutaneous larva migrans (Creeping eruption)**
* Various animals harbor hookworms. Two species of dogs and cats are important.
  1. Ancylostoma braziliens: infects both dogs and cats.
  2. Ancylostoma caninum: infects only dogs.

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

They may evoke a fairly severe reaction - pruritus and dermatitis. The dermatitis leads to scratching and then bacterial superinfection.

**Treatment**

1. Thiabendazole: Applied topically.

**2. 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.

**3. Toxocariasis**

This is a kind of visceral larva migrans caused by

1. Toxocara canis (Dog ascarid) and
2. Toxocara catis (Cat ascarid).

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

**Morphology**

1. The larvae of Toxocara canis and Toxocara catis measure about 400 μm in length.
2. 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:**

1. Majority are asymptomatic.
2. Eosinophilia
3. Cerebral, myocardial and pulmonary involvement may cause death.

**Diagnosis** - Identification of larvae in tissue.

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

**4. C. Intestinal larva migrans**

This is an extremely rare kind of larva migrans

Topic 1.11:NEMATODES (STRONGYLOIDES STERCORALIS)

**Morphology;**

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.

**Treatment:**

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

### opic 1.12: NEMATODES (ENTEROBIUS VERMICULARIS/PIN WORM/THREAD WORM)

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

##### MorphologyMale:**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.**

##### **Treatment Mebendazole; Piperazine.**

opic 1.13:NEMATODES (TRICHURIS TRICHURIA/WHIP WORM)

**Morphology;**

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.

**Treatment**

1. Mebendazole: 1 tablet twice daily for 2 days.

### opic 1.14: NEMATODES (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.

#### 1. Wuchereria bancrofti

This is a parasite of lymph nodes and lymphatic vessels- causing lymphatic filariasis. This filarial worm is transmitted by the bite of various species of mosquitoes. It is believed that over 100 million people are infected. The microfilariae are nocturnal – seen in greatest numbers in peripheral blood in the night between 10 PM -2 AM.

The physiological basis of this nocturnal periodicity is not understood.

**Mode of transmission and pathogenesis**

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

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

**Epidemiology**: W. bancrofti infection is not reported in higher , but limited to lowlands of Gambella. The epidemic area covers a long distance along the Baro River.

**Pathogenecity and clinical features:**

1. The adult worm obstructs the flow of lymph in the lymph nodes and the lymphatic vessels draining the lower limbs and the external genitalia.
2. The lower limbs and external genitalia become swollen. The skin becomes thick and fissured. The disease is called bancroftian elephantiasis.
3. The major symptoms and findings include:
   1. Lymphangitis,
   2. Lymphedema,
   3. Fever,
   4. Headache,
   5. Myalgia,
   6. Hydrocele and
   7. Chyluria.

**Diagnosis**

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

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

#### 2. Endemic non-filarial elephantiasis (Podoconiosis)

Non-filarial elephantiasis of the lower limbs is common in mining areas. Silicon, aluminium and iron particles in the red clay soil are absorbed through skin abrasions in bare footed persons. The mineral particles cause obstruction of the lymphatics.

#### 3. Onchocerca volvulus/river blindness

Morphology:

**Male**: Similar to that of Wuchereria bancrofti.

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

**Intermediate Host and vector**

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

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

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

**Pathogenecity and clinical manifestations:**

1. The disease, onchocerciasis or river blindness includes:
2. Skin fibrous nodules (onchocercomata) enclosing female worms. The nodules are common in neck, iliac crest and the coccyx.
3. Skin hypo- or hyper- pigmentation. Dermatitis is present. In advanced cases, the skin becomes thickened and wrinkled, showing lizard or leopard skin appearance.
4. Elephantiasis of the external genitalia and corneal opacity and optic atrophy may finally cause blindness.

**Diagnosis**

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

**Treatment**

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

**Prevention**

1. Vector control
2. Mass treatment
3. Establishment of villages away from Simulium breeding places.
4. Use of repellents
5. Protective clothing

#### 4. Loa loa/ Eye worm

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

**Morphology**

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

**Pathogenesis**

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

**Clinical Features**

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

**Laboratory diagnosis**

1. Detection of microfilaria in peripheral blood, urine, sputum, CSF - stained with Giemsa or unstained
2. Eosinophilia

**Treatment**

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

Topic 1.15: NEMATODES (DRACUNCULUS MEDINENSIS/GUINEA WORM/MEDINA WORM) AND TRICHINOSIS

**DRACUNCULUS MEDINENSIS;**

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

**Morphology**

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

**Pathogenecity and life cycle**

Infection is acquired by drinking unfiltered or not boiled water that contains Cyclops species. The larvae are released in the stomach, penetrate the intestinal wall and find their way to the subcutaneous tissue. Mating takes place in the axillary or inguinal regions 3 months after infection.

The male worms then die in the tissue and the female worms move down to the limbs within 10 months. In about 1 year, female worms in the subcutaneous tissue provoke the formation of a burning blister in the skin of the legs. When in water, the blister bursts, and about 5 cm of the worm is extruded from the resulting ulcer - thus releasing many thousands of first stage larvae.

The larvae swim in water and are ingested by the intermediate host - Cyclops species- within about 4 days. Inside the Cyclops, the larvae molt twice and become infective in 2 weeks

**Clinical features;**

1. The female parasites in the subcutaneous tissue release toxic byproducts of histamine-like nature, which cause systemic allergic reactions, like;
   1. Erythema,
   2. Urticaria,
   3. Pruritus,
   4. Fainting,
   5. Asthma,
   6. Dyspnea, etc.
2. This is followed by the appearance of a blister on the legs, which ruptures on contact with water releasing larvae into the water by the female worm. The wound may ulcerate.
3. The worms migrate into other tissues and may cause;
   1. Arthritis,
   2. Pericarditis,
   3. Abscesses etc. It occasionally penetrates the eyeball and causes loss of the eye.

**Diagnosis;**

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

**Treatment**

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

**Prevention;**

Health education on:

1. Boiling or filtering of drinking water
2. Treating of patients and educating them not to enter water bodies
3. Using insect larvicides to kill Cyclops in water.

**TRICHINOSIS**

**Etiologic agent**- Trichinella spiralis

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

**Morphology**

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

**Pathogenecity and life cycle**

After ingesting infected meat, the capsule of the encysted larvae is digested by gastric juice, and the larvae are released in the duodenum or jejunum where they molt four times to become adult worm. After mating, the male worm dies and the female worm begins to deliver the embryos 4-7 days after the infection.

The larvae penetrate the intestinal wall and migrate through the lymphatic vessels to the blood stream, which carries them to various organs. Skeletal muscles and diaphragm are most frequently parasitized. Others include the tongue, masseter and ocular muscles.

**Clinical features**

There are two clinical phases.

1. The intestinal phase: lasting 1-7 days - asymptomatic; sometimes cause ;
   1. Nausea,
   2. Vomiting,
   3. Diarrhea,
   4. Constipation,
   5. Pain, etc, and
2. The muscle phase: which causes;
   1. Myalgia,
   2. Palpabral edema,
   3. Eosinophilia,
   4. Fever,
   5. Myocarditis,
   6. Meningitis,
   7. Bronchopneumonia etc.

**Diagnosis:**

1. Muscle Biopsy
2. Detection of larvae in blood or CSF
3. Detection of larvae and adult worms in stool (rare).
4. ELISA

**Treatment**- Thiabendazol

Prevention

1. Cooking of all meat before consumption
2. Inspection of pigs
3. Pork must be stored at -150C for 20 days.

SUMMARY OF HELMINTHS

1. Helminths are parasites that can inhabit the intestinal tract, blood, tissue and other body organs.
2. Helminths are broadly classified into three;
   1. Nematodes- the roundworms.
   2. Cestodes- the tapeworms.
   3. Trematodes- the flukes.
3. Helminths have several attaching structures including;
   1. Rostellum-crown of thorns with hooks.
   2. Cutting teeth.
   3. Cutting plate.
4. Helminths cause disruption of the host nutrients absorption by utilizing all nutrients passing through the intestinal tract.

Top of Form