MEDICAL HELMINTHOLOGY

Ass. Prof. Dr. Nada. A. Saif

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Department of Microbiology Faculty of Medicine and Health Science
TAIZ University.

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MEDICAL HELMINTHOLOGY

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INTRODUCTION TO THE BOOK
Parasitic infections are most important infection spread in Yemen.

This book will be give general information in simple way to the undergraduate students about the helminthes that cause disease.

Also will describe the pathogenesis, clinical and controll of these helminthes.

In this book the author try to join the basic informations with the clinical field to be useful to all.

Some important helminths spread in Yemen(Taiz): Dog tape worm. H. nana, Schistosoma, T. saginata, Filaria. and according to our Problem based system, these parasites will be taken again in clinical field, surgery, tropical medicine and dermatology.

Any comments and suggestions for improvement of the book will be most welcome.

Thanks For

Head of Microbiology Department: Dr. Hafez
My Daughter
Manar Abdul Raheem
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CHAPTER-1
Introduction to Helminths and Trematoda

Introduction To Helminths
*Helminths or parasite worms are.
*Multicellular.
*Bilateral symmetrical metazoan.

The term helminth (Greek helmins - worm).
* Two phyla of medical importance.
1) Phylum Platyhelminthes
2) Phylum Nemathelminthes

Phylum Platyhelminthes (Flat worms)
*Flattened dorsoventrally
*Bilateral symmetrical
*Leaf like or tape- like.
*The alimentary canal is incomplete or lack.
*The various systems of the worms are embedded in parenchymatous tissue cells (No body cavity).
*Hermaphrodites (Monoecious).
(The worm has both male and female reproductive system).
*Self-fertilization or cross fertilization.
* Helminths which occur as parasites in humans belong to two phyla.

1) Phylum Nemathelminthes (Class Nematoda).
* Unsegment.
* Separate sexes.
* They have body cavity.
* No circulatory system.
* Body wall consisting of an outer layer of longitudinal muscles.
*Class Nematoda is divided into two subclasses.
Subclass a) Adenophorea (Aphasmidia).
b) Secernentea: (Phasmidia).
### Table 1: Difference between Adenophorea and Secernentea.

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<th>(A) Adenophorea</th>
<th>(B) Secernentea</th>
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<tr>
<td><strong>Eggs</strong></td>
<td>With plug at either end</td>
<td>Without plug</td>
</tr>
<tr>
<td><strong>caudal papillae</strong></td>
<td>No caudal papillae in male</td>
<td>Numerous</td>
</tr>
<tr>
<td><strong>stage of larva (infective stage)</strong></td>
<td>2nd stage larva</td>
<td>Third stage larva</td>
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<td><strong>super family</strong></td>
<td>Trichuroidea, has anterior part narrower than posterior, Male has one spicule</td>
<td>Has alteration of free-living and parasitic generation. Strongyloides</td>
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</table>

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2) Phylum Platyhelminths include two classes.

Class Trematoda (Flukes).

Class Cestoidea or subclass cestoda (Tapeworms).

**Phylum Platyhelminths.**

**Class Trematoda (Flukes).**

**Snail -transmitted-Parasite).**

**General characteristics**

* Leaf like structure.
* Unsegmented.
* Size from 1mm-Few cms.
* Organs of fixation in the form of 2 suckers (Distomata)

a) Oral sucker at anterior end surrounding the mouth, through which the digestive tract opens.

b) Ventral sucker (acetabulum) for attachment.

(Sometime genital sucker present)

* Hermaphrodites except blood flukes.
* The body is covered with integument which often bears spines, papillae or tubercles.

No body cavity, no respiratory organs, no circulatory system.

Alimentary tract is present but in complete, it consists of.

* A mouth
* Muscular pharynx
* Esophagus: which bifurcates in front of the ventral sucker into a pair of blind intestinal ceca, alimentary tract appears like an inverted Y.
*The anus is absent.

* Excretory system is bilateral symmetrical, it consists of flame cells which derive waste products and collecting tubules, which lead to a median bladder opening at the posterior end of the body.

*The nervous system is rudimentary, it consists of a group of paired ganglion cells and nervous trunk.

*Reproductive system is highly developed.

*Trematoda are oviparous and lay eggs which are: operculated except blood flukes.

![Reproductive system of trematoda.](image_url)

**Classification of Trematoda**

1) Zoological classification.

2) According to habitat (Liver trematoda-Intestinal trematoda-Lung trematoda and blood trematoda).

*Liver, intestinal and lung trematoda are hermaphrodites, But blood trematoda is unisexual.
Life cycle: Trematoda pass their life cycle in two different hosts.

Man: Final host, who harbors the adult.

Freshwater snail: Intermediate host, that harbors the larva stage.

A second intermediate host (Carb, fish) is required in some trematoda.
*The eggs hatch in water to form the first stage larva (The motile –Ciliated miracidium).
*The miracidium infects the intermediate host snail in which further development(Metamorphosis) take place.
*The miracidium becomes the sac-like sporocyst.
*Within the sporocyst, certain cells proliferate to form the germ balls, which are responsible for asexual reproduction.
*In blood trematoda, the sporocyst develops into second generation sporocyst in which the infective larvae cercariae are formed.
*But in hermaphroditic trematoda, the sporocyst matures into a more complex larval stage (Redia), which produce cercariae.
*In blood trematoda, furcocercus cercaria infects the final host by direct penetration.
*In the hermaphroditic trematoda, the cercariae have an unsplit tail, and they encyst on vegetation or within a second intermediate host, which are the infective forms.

Cercaria
a- Consist of a body (The future worm)
b- Tail
It uses for swimming and the shape of tail is important in the naming of cercaria.
*The body contains oral and ventral suckers, mouth, esophagus, pharynx and primitive intestinal caeca, flame cells, excreatory vesicle, pore and genital primordium.
*It Contains 2 eye spots, The body may be covered it with spines.

Different types of glands may be found.
Escape gland
It presents in anterior part of the body which helps the cercariae penetrating their way through snail tissue.
Cystogenous gland
It helps in the formation of the cyst wall of encysted metacercariae through their secretions, e.g. Cercaria of *Fasciola*.

Penetration glands
*It Secretes certain enzymes that help in lyses of tissues of the final host (Skin or mucous membrane) thus facilitate entrance of the cercaria, e.g. Cercaria of *Schistosoma*.
*Some Cercariae may have both penetration and Cystogenous glands e.g.: *Heterophyes* to penetrate the skin of fish "Second intermediate host" and encysted in the fish muscles.
*Some cercariae have a "Sty let" in the oral sucker which is a sharp sternum- like rod used for the penetration of the hard cuticle of arthropod intermediate host. E.g. Cercaria of *Paragonimus*.

Types of Cercariae
a- Leptocercus cercaria
It has a simple tail e.g. Cercaria of *Fasciola*.
b- Parapleurolophocercus cercaria
It has a simple tail covered incompletely by a membrane e.g., cercaria of *Heterophyes*.
c- Furcocercus cercaria
It has a forked tail, e.g., cercaria of *Schistosoma*
d- Microcercous cercaria.

It has a very short stumpy tail e sty let, e.g., cercaria of *Paragonimus*.
*Cercariae leave the snail and are capable of: swimming in water only for short time and they die within 24hr-48hrs if unable to continue the life cycle.*
*Cercariae usually encysted by*
The formation of cyst around the body, leaving their tails.
*The encysted metacercaria is formed on.*
Aquatic vegetation, rare in water, in fishes, and become the infective stage In blood flukes, cercaria is the infective stage.
*In all trematoda except blood trematoda.
Infective stage is encysted metacercaria. Route of infection is oral.

![Image of Types of Cercaria](image_url)

**Fig. 2: Types of Cercaria.**

**Blood flukes: Schistosomes**

*Schistosomes are the causative agents of the disease schistosomiasis (Bilharzia).*

*It is the most important and prevalent of water-borne-parasitic disease.*

*The name schistosome (Greek Schisto-split and soma-body).*

*Schistosomes live in venous plexuses in the body of the final host, the location varying with the species.*

*The parasite was first observed in Cairo in 1851 by Theodor Billharz in the blood of mesenteric veins of a young Egyptian man on autopsy.*

**General characteristics**

*Schistosomes are unisexual (Diecious).*

*Males are shorter and broader than females.*

*Males possess a gynaecophoric canal, which is formed by the in folding of the lateral margins ventrally, for holding the female during copulation.*

*Suckers are armed with delicate spine.*

*Muscular pharynx is absent.*

*Intestinal ceca reunited behind the ventral sucker to form a single canal.*

*In the male the number of testes varies from 4-8.*

*Eggs are non-Operculated (with spine) and are fully embryonated (mature embryo) when laid.*

*Cercariae have bifid tail and act as infective stage. They can penetrate the unbroken skin of the final host.*

*Encysted metacercaria stage is absent.*
There are four schistosoma species

1) Schistosoma haematobium
2) Schistosoma mansoni
3) Schistosoma japonicum
4) Schistosoma intercalatum

Schistosoma haematobium.
(Endemic hematuria).

Geographical distribution
Nile valley-Part of Africa and Asia.

Morphology
* The male: 10-15mm long by 1mm thick, and covered by a finely tuberculate cuticle.
* The oral sucker small, the ventral sucker large and prominent.
* The adult female: Long and slender, 20mm by 0.25mm with the cuticular tubercles confined to the two end.
* The egg: Oval, 100-170umx40-70um., with a brownish yellow transparent shell carrying a terminal spine at one pole, contains mature embryo.

Life Cycle
* The adult worms live in the vesicle and pelvic plexuses.
* The gravid worm contains 20-30 eggs in its uterus, and may pass up to 300 eggs a day.
* The terminal spine eggs are laid in the small venules of the vesicle and pelvic plexuses, sometimes they are laid in the mesenteric portal system, pulmonary arterioles and other ectopic sites.
* The eggs are laid one behind the other with the spine pointing posteriorly.

From the venules, the eggs make their way through the vesicle wall by.

a) Piercing action of spine.
b) Lytic enzyme released by eggs.
c) Moulting pressure within the venules.

* The eggs pass into the lumen of urinary bladder together with some extravasated blood.
* The eggs are discharged in the urine toward the end of micturition.
*The eggs are passed in urine more during midday than at any other time of day or night.

*The eggs laid in ectopic sites generally die and evoke local tissues reaction.

*The eggs may be found in rectal biopsies, semen, rare feces.

*The eggs that are passed in water hatch and release ciliated miracidium.

*The miracidium enters the *Bulinus* snail, there miracidium loses its cilia, after 4-8 weeks the 1st and 2nd sporocyst generation develop.

*Large number of cercariae are produced by asexual reproduction within the second generation sporocyst.

*Furcocercus cercariae go out of snail to water, they penetrate unbroken human skin by (lytic substance secreted from penetration gland present in cercariae).

*On entering the skin cercariae shed their tails and become Schistosomulae.

*Schistosomulae enter the peripheral venules and start long migration through vena cava to the right heart then pulmonary circulation then left heart then systemic circulation.

*From systemic circulation reach to liver, in intra hepatic veins Schistosomulae grow, pairing of worms take place on sexual reproduction.

*They then start migration against the blood stream in to the inferior mesenteric veins, until reach the vesicle and pelvic plexuses, where they mature, mate and begin laying eggs.

*Eggs start appear in urine after 10-12 weeks from skin penetration.

*The adult worms may live for 20-30 years.

*Human are the only natural final host.

*No animal reservoir is known.
Introduction to Helminths and Trematoda

Fig. 3: Eggs of Schistosoma.

Fig. 4: Life cycle of Schistosoma.
Introduction to Helminths and Trematoda

Fig. 5: Snails of liver and blood trematoda.

Fig. 6: Anatomical routes for *Shistosoma* to reach their places.
Pathology, clinical and complications

1) Penetration of the skin by cercariae
* Skin penetration may not be apparent.
* Human and some non-human cercariae *Schistosoma* species cause cercarial dermatitis (Swimmer's itch).
* Swimmer itch manifests with papules, macules, vesicles and intense itching.

2) Migration and maturation of immature worms
There are general toxic and allergic symptoms including.
Urticaria with eosinophilia, abdominal pain and tender hepatosplenomegaly.
This is known as Katayama or Snail fever.

3) Damage by eggs in tissue
* An inflammatory granuloma forms with epithelial, giant, plasma and eosinophil cells and fibroblasts (Hoeppli reaction).
* There is subsequent fibrosis and calcification.
* Such damage may be local and/or ectopic.

4) Urinary Schistosomiasis
* Caused by *S. haematobium*.
* The bladder and ureter are typically involved with: Hyperaemia, terminal hematuria (Endemic hematuria), dysuria and Frequency Of micturition, papules, papillomata, and ulceration.
* There is generalized hyperplasia and fibrosis of Vesicle mucosa with a granular appearance (Sandy patches).
* There may be cystitis and calculus formation, with calcification and squamous cell carcinoma.
* Fistulae may develop.
* There may be hydro ureter and hydronephrosis.
* Schistosomal Corpulmonale.

5) Intestinal schistosomiasis: Caused by *S. mansoni*.

Geographical distribution: Nile Delta, Africa, South America.
*S. mansoni* is similar to *S. haematobium* in morphology and life cycle.
Adult worms are smaller and their integument studded with prominent coarse tubercles.

**Habitat:** Portal veins and veins of Inferior mesenteric plexus.

**Intermediate host:** Fresh water snail of genus *Biomphalaria*.

**Final host:** Man, monkeys and rodents (Reservoir host).

**Eggs:** Oval with lateral spine. 140-180umx45-70um.

**Life cycle:** *S. mansoni* resembles *S. haematobium* in morphology and life cycle.
*In human the Schistosomulae mature in the liver and the adult worm moves against the blood stream to the venules of inferior mesenteric group in sigmoidorectal area, the eggs penetrate the gut wall reach colonic lumen and shed in feces.
*There are marked initial toxic and allergic symptoms.
*Large intestine and rectum are typically involved with.
*Polyposis, papules, abscess, ulcers, papillomata and fistulae..
*The bladder is sometimes involved.
*There can be ectopic lesions; the liver is frequently involved, receiving eggs via the: Portal vein with inflammatory reaction and fibrosis leading to periportal(Pipe-stem) fibrosis with portal hypertension, esophageal varicose, splenomegaly and ascites.
*There can also be lesions in the brain, spinal cord, and lung.

6) **Oriental schistosomiasis**
*Caused by *S. japonicum*. The dangerous species.
**Geographical distribution:** Far east, Philippine, Japan, China, and Taiwan.
*The adult male is slender (0.5mm thick) and doesn't have cuticular tuberculation. The eggs are with rudimentary lateral protuberance. 70-100umx50-80um.

**Habitat:** Superior mesenteric veins.

**Intermediate host:** Snail of genus *Onchomelania*.

**Final host:** Man, domestic animals and rodents.
*Intestinal lesions are similar to *S. mansoni*.
*The brain may also involve.
*They are more severe specially at early stages where the patient may get: Fever, urticaria, edema and lung disturbances, this known as katayama syndrome.

**Katayama Syndrome**
It is an immune complex disease caused by antibodies to Schistosomulae, adult worm and eggs.

**Diagnosis of S.haematobium**
*History and clinical.
*Eosinophilia may be present.
*Egg with terminal spine found by Nuclepore filtration or after centrifugation.
*Eggs may also found in semen.
*Eggs may also found in stool.
*Biopsy material obtained through the proctoscope (S.mansoni and S.japonicum) and cystoscope for S.haematobium and examined microscopically after compression or sectioning.
*X-rays.
In chronic cases: Immunodiagnosis is useful as: circumoval precipitation test, ELISA and indirect haemagglutination.
*Intradermal(Fairley's test).
*Ultrasonography.

**Update methods for diagnosis.**
PCR
A diagnostic tests for Ag detection.
Proxy markers for morbidity assessments.

**Diagnosis of S.manson**
*The same as that of S.haematobium.
*Stool examination.

**Control and prevention**
1) Prevention of pollution of water with human excreta.
2) Eradication of molluscan hosts in endemic area by molluscicidal campaigns: Physical methods, Biological methods and Chemical methods.
3) Avoidance of swimming, bathing or washing in infected water.
4) Effective treatment for infected man by using Praziquantil drug. According to Road map of WHO for control schistosomiasis still the Prazquantil the less expensive and more specific methods for control.

*WHO (World Health Organization).

**Cercarial Dermatitis**

swimmer or Bathers or Paddy field (Farmers dermatitis).

*Invasion of human skin by non-human cercariae of birds or mammals example *Schistosoma Spindale*(Final host of it are cattle, goat, sheep).

*Non-human cercariae penetrate the human skin but cannot proceed further beyond the skin.

**This Produces**

*severe itching-edema-skin eruption and secondary bacterial infection.

*This condition may last 3-5 days.

Treatment: Antibiotics, Anti-allergy, Niclosamide.

*Notes: Reservoir host act as source of infection to human, the disease transmitted from animals to human called zoonotic diseases.

**Hermaphroditic Flukes**

**Liver flukes**

*Flukes inhibiting the human biliary tract are.

1) *Colonorchis sinensis*

2) *Fasciola hepatica* and *F. gigantica*

3) *Opisthorichis species*

4) *Dicrocoelium dendriticum*

**Colonorchis sinensis**

(Chinese liver fluke)

*C.sinensis* was describe in 1875 by McConnell in biliary tract of Chinese in Calcutta.
Geographical distribution: Japan, Korea, Taiwan, China.
Morphology
* Adult has a flat, transparent, spatulate body, pointed anteriorly and round posteriorly, 10-25mm long and 3-5mm broad.
* Oral sucker is slightly larger than ventral sucker.
* Eggs are broadly ovoid, 30um x 15um with yellowish brown shell, it has an operculum at one pole and a small hook-like spine at the other.

Habitat: Biliary tract.

Final host: Man, dog and other fishing-eating canines.

Intermediate host
Snail and Fish.

Mode of transmission
* Eat frozen, dried or pickled fish.
* Infection occur through.
Contaminated fingers or cooking utensils.

Infective stage.
Encyst metacercaria

Diagnostic Stage: Eggs.

Life cycle
* Adult fluke in biliary tract of human or animals.
* Eggs passed in stools reach water, and are ingested by first intermediate host (Snail).
* Miracidium emerges from egg and penetrates into tissues of snail.
* Redia metamorphose to cercaria.
* Cercariae leave the snail and swim about water to infect the second intermediate host (Fish).
* Encysted metacercaria develop in the muscles of fish.
(This is the infective form to man or animals).
Pathogenesis

*The migration of the larva up the bile duct induce: Desquamation, hyperplasia and adenomatous change.

*The adult worm may causes obstruction and blockage of common bile duct lead to cholangitis.

Chronic infection may result in.

*Cholangiocarcinoma.

*Calculus formation.

Few cases go on to

Biliary cirrhosis.

Portal hypertension.

Fig. 7: Life cycle of C.sinensis.
Clinical Picture
* Fever, epigastric pain, diarrhea, tender hepatomegaly, biliary colic, obstructive jaundice.
* Many infection are asymptomatic.

Diagnosis
* Eggs may be demonstrated in feces or aspirated bile (They don't float in concentrated saline).
* Serological tests.
* Intradermal allergic tests.

Control and prevention
* Proper cooking of fish.
* Health education.
* Snail control.

**Opisthorichis Felineus**
(Cat liver fluke)
* *O.felineus* which resemble *C.sinensis* can cause human infection.
* Infection is usually a symptomatic.
* Geographical distribution: Europe, Soviet Union, Thailand.

![Fig. 8: Adult of *O.felineus.*](image)

Genus Fasciola

* **Fasciola hepatica** (Sheep liver fluke)
  * It is the largest and most common liver fluke found in human(Greater liver fluke).
*It causes the economically important disease in sheep (Liver Rot).

Geographical distribution: Cattle raising countries, Egypt.
*Final host: Herbivorous animals and accidently man.
*Intermediate host: Snail (Lymnaea cailliaudi) for F. gigantica and (L. truncatula) for F. hepatica.
*Infective stage: Encyst metacercaria.
(Encyst on aquatic vegetation and grass).
Diagnostic stage: Operculated immature egg.*
Disease: Fascioliasis (Liver Rot).

**Morphology**

**Adult**
*Leaf like with anterior cone, its length 30mm long and 15mm broad.
*Alimentary canal, intestinal ceca have compound lateral branches and medial branches T or Y shape.
*Testes: Two, branched in shape located at the middle of the body of adult.
*Ovary: One, branched, anterolateral to testes.

**Eggs:** 140umx80um, oval, Operculated. They have thin shell and immature embryo.

**Life cycle**
*Adult in biliary tract of sheep and human.
*Egg passes in stool reaches water.
*Miracidium escapes and penetrates the tissues of snail in which it develops into.
Sporocyst.
Redia, first and second generation.
*Cercaria releases into water plants.
*Cercaria losses its tail and encyst in vegetation (Infective to final host).

**Pathogenesis, clinical and complications**
Asymptomatic.
Acute or invasive phase.
Chronic.
Ectopic.
*Signs and symptoms appear 2 weeks after the infection.
Acute stage is due to migration of immature worm in liver tissue which lead to.
* Mechanical injury.
* Parasite toxin.
* Allergy to parasite ex-product.
* Anemia and dyspepsia.
* Traid signs: Hepatomegaly, Fever and eosinophilia.
This stage occurs in heavy infection in animals and rare in (human).

**Chronic stage:** It is due to presence of maturing worm in the bile duct which is lead to.
* Cholecystitis
* Cholelithiasis
* Jaundice
* Liver fibrosis

**Ectopic fascioliasis**
* In lung
* Subcutaneous tissue
* Rare central nervous system

**Halzoun (Suffocation)**
* It is form of acute laryngopharyngitis.
* It results from ingesting nymphs of liver of herbivores, *(Lingatulla Serrata).*

**Symptoms**
* Hoarseness of voice
* Dysphagia
* Dyspnea
* Suffocation

**Diagnosis of fascioliasis**
* Eggs in feces or aspirated bile.
* Eosinophilia.
* Serology.
Control and prevention
* Snail control.
* Elimination of water vegetation.
* Mass treatment of infected animals.
* Safe water supply.
* Proper cooking of animal liver and water plants.

![Diagram of Reproductive System of F. hepatica](image)

Fig. 9: Reproductive system of *F. hepatica*.

*Fasciola gigantica*
* It is the largest of human liver flukes.
* It measures up to 75mm in length and 12mm in width.
* It has long rounded posterior end, shorter cephalic cone, a larger ventral sucker and more anterior position of testes.
* The eggs of *F. gigantica* are larger (180um x 80um).
* The life cycle and habitat are similar to *F. hepatica*.
* Pathology and clinical pictures are similar to those of *F. hepatica*.
**Dicrocoelium dendriticum**

*(Lancet fluke)*

It is the Lesser liver fluke.

Geographical distribution: Europe, North Africa, Northern Asia, part of the Far East.

*Host:* Herbivorous animals as sheep and cattle, rarely man.

**Habitat:** Biliary tract.

**Life cycle**

Egg contains fully mature embryo with thick shell, dark brown in color, slightly flattened on one side, Operculated, 45x30u, pass in feces.

*They ingested by land snail(The first intermediate host, snail(*Helicella* and *Zebrina*).*

*They hatched inside the snail.

*Cercariae appear in slime balls secreted by snails, and eaten by second intermediate host, brown ant(*Genus Formica, *F. fusca*).*

*Inside the ant metacercariae develop.

*Herbivores get infected when they accidentally eat the ants while grazing.

*The eggs can be passed in feces for several days by persons eating infected sheep liver.

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*Fig. 10: Life cycle of *D.dentriticum*.**
**Pathogenesis**
It is less severe than Fasciola because the parasite reach the bile duct through cystic duct without migration into liver.

**Diagnosis:** Eggs in stool.

**Intestinal flukes**
Flukes parasitize the human small intestine are.
- *Heterophyes heterophyes*
- *Metagonimus yokogawai*
- *Fasciolopsis buski*
- *Echinostoma*

*Only one flukes* *Gastrodiscoid hominis* *parasitize* the human large intestine.

**Fasciolepsis buski**
*(Giant intestinal flukes)*
*It is the largest trematoda infected human.*
*It is a common parasite of man and pig.*

**Geographic distribution**
China, South and Far East Asian countries, In India (Assam and Bengal).

**Habitat:** Duodenum and jejunum.

**Intermediate host:** Snail (Genus *Segmentina*).

**Infective stage:** Encyst metacercaria

**Diagnostic stage:** Egg with immature embryo and operculum.

**Disease:** Fasciolopsiasis.

**Morphology and life cycle**

**Adult**
*No cephalic cone. Large, 20-75mm long and 8-20mm broad.*
*Oral sucker sub terminal.*
*Ventral sucker large.*
*Two branches testes one above the other in the posterior half of the body.*
*Branched ovary in the middle of the body.*
*Life span of about 6 months.*
Eggs
They are identical to those of *F. hepatica*. Each worm lays 25000 eggs/day.

Life cycle
*Eggs are laid the lumen of small intestine.
*Eggs passed in feces hatch in water in about 6 weeks, releasing the miracidium which swim about.
*They penetrate the snail tissues to undergo development as sporocyst, first and second redia generation and cercariae.
*The cercariae escape from snail and encyst on the roots of lotus, bulb of water chestnut and on other aquatic vegetation.
*When they are eaten, the metacercariae excyst in the duodenum, become attached to mucosa and develop to adults in about 3 months.

Mode of transmission: Orally.

Fig. 11: Life cycle of *F. buski*. 
Clinical and complications
1-Traumatic: At the site of attachment lead to: inflammation and ulceration
2-Mechanical: Partial obstruction of the bowel.
3-Toxic: Intoxication and sensitization that result from absorption of worms metabolites lead to defect in absorption of vitamin B12.

*Others
Hemorrhagic diarrhea, anemia, asthenia, edema, ascites.

Diagnosis
*Stool examination(eggs and adult can be seen).
*Eosinophilia and leukocytosis.

Control and prevention
*Washing of water vegetables by hot water.
*Preventing contamination of ponds and other waters with pigs or human excreta.
*Anti snail measures.

*Heterophyes heterophyes*
Habitat: Between the villi of small intestine.
Geographic distribution: Nile Delta, Turkey, Far East.
Final host: Man and fish-eating bird and animals.
Intermediate host: first one is snail (*Pirenella conica*)-Second one is fish(Mugil, Tilapia).
Inf ective stage: Encyst metacercariae in fish.
Diagnostic stage: Operculated eggs with thick shell.
Disease: Heterophyasis.

Morphology and life cycle
Adult
*It is the smallest trematoda(1.5mm in length and 0.3mm in breadth).
*Life span: 2 months.
*Shape: Pear shape, genital sucker is posterolateral to ventral sucker.
*Testes: Two ovoid in posterior part of the body.
*Ovary: Globular in front of the testes.
**Eggs:** Oval, Operculated, thick shell and knob at posterior end (30um by 15um).

**Life cycle**
*The Operculated eggs are passed in feces and hatch after ingestion by intermediate host, snail.*
*Miracidium transform to sporocyst and two generations of rediae, the cercariae escape and encyst on suitable fishes.*
*When the infected fish are eaten raw or inadequately cooked, the final host become infected.*
*Encyst metacercaria excyst in the small intestine of final host, and embed themselves between the villi and develop to mature worm.*

![Fig. 12: Life cycle of H.heterophyes.](image)

**Clinical and complications**
a) Intestinal picture: Colicky abdominal pain, mucous diarrhea.
b) Extra intestinal picture: The eggs are carried in the lymphatic and portal circulation to ectopic sites such as: Brain, spinal cord and myocardium., where they induce granulomas.
Rarely the worms themselves may be carried to these sites as emboli.
**Prevention and control**

*The infection can be avoided by ensuring that fish intended as food are well cooked or frozen prior to ingestion.

**Metagonimus yokogawai**

*Resemble *H.heterophyes.*

*Only in Far East.*

*No genital sucker in adult.*

*Adult slightly larger than *H.heterophyes.*

*The ventral sucker is situated to right of the midline.

**Eggs**

*Resemble those of *H.heterophyes.*

*They measure 26-28umx15-17um in size.*

*Snail of genus *Semisulcospira.*

**Life cycle**

*Similar to *H.heterophyse.*

**Clinical picture**

*The parasite causes diarrhea.*

*Ectopic sites: Myocardium, brain, spinal cord.*

![Life cycle of *M.yokogawi.*](Fig. 13a)
Fig. 13b: Adult of \textit{M.yokogawi}.

\textbf{Diagnosis}

* Present of eggs in stools.

Prevention is similar to \textit{H.heterophes}.

\textbf{Echinostoma}

* Medium in size, the adult worm is less than 20mm long and 2mm wide.
* It is common in Japan, Philippines, Far East.
* Characteristic feature is a crown of spine on the disc surrounding the oral sucker.
* \textit{Echinostome} means spiny mouth.
* Eggs resemble the \textit{F.buski}.
* Clinically: Asymptomatic.

\textbf{Flukes of large intestine}

\textit{Gastrodiscoid hominis}

* It is the only fluke inhibiting large intestine(Caecum and ascending colon).
* It is common in: Assam, Bengal, Vietnam, Philippines.
* Pigs, monkeys, rats are the reservoir hosts.
* Adult is pyriform with a conical anterior and hemispherical posterior portion.
* It is about 5-14mm long and 4-6mm broad.
* The eggs are Operculated with immature embryo, 150um x 70um.
Introduction to Helminths and Trematoda

* Cercariae encyst on water plant.
* Infected person develop mucoid diarrhea.

Lung trematoda
(Endemic hemoptysis)
Paragonimus westermani
Oriental lung trematoda.
* Habitat: Lung.
* Parasite is endemic in the Far East, Japan, Korea, Taiwan, China, South East Asia, Sri lanka, India.
* Ectopic sites: Spleen, liver, other organs.

Final host: Human, cat, dog, fox, pig.

Intermediate host: First: Snail belong to the genera Semisulcospira, Second: Fresh water Cray fish or Crabs(Crustacean).

Infective stage: Encyst metacercariae in crab or Cray fish.

Diagnostic stage: Eggs in sputum or feces.
Mode of transmission: Orally.

Morphology and life cycle
* Adult worm is egg shape 10mm long, 5mm broad and 4mm thick.
* Adult usually live in pair in cystic space communicate with bronchi.
* Life Span: 20 years in human.

Eggs: Operculated, 100umx50um, golden brown in color, thick shell, contain immature embryo.

Disease: Paragonimiasis.
Life cycle
*Eggs escape into bronchi and coughed up and voided in sputum, or swallowed and passed in feces.
*The eggs mature in about 2 weeks and hatch to release free-swimming miracidiae.
*These infect the first intermediate host (Snail).
*Inside the snail, the miracidium passes through the stages of sporocyst, first generation rediae, second generation rediae, finally giving birth to cercariae.
*The mature cercariae escape from snail into water and penetrate gills and muscles of its second intermediate host, crabs or Cray fish.
*Crabs and Cray fish can also become infected by eating the infected snail.
*Inside the crabs, the cercariae transform into: metacercariae in the viscera, muscles and gills.
*Humans become infected after ingestion of raw or poorly cooked or salted crabs.
*Ingested metacercariae excyst in the duodenum, releasing larvae that attach to the duodenal mucosa.
*These larvae penetrate the intestine and enter into abdominal cavity.
*Then they migrate upwards, piercing through or around the diaphragm to the pleural cavity and lung.
*Finally arriving the bronchioles, where they develop into adult worms usually in pair, encapsulated by hosts inflammatory response to produce eggs.
*Eggs are expelled in the sputum.

Clinical and complications
a) Pulmonary picture
*In the lungs the worms lie in cystic spaces surrounded by a fibrous capsule formed by the host tissues.
*The cysts, about a centimeter in diameter are usually in communication with a bronchus.
*The adult worms in the lungs provoke granuloma, consisting mainly of eosinophil's and neutrophils, followed by development of broad layer of fibrous tissues outside, thus producing a thick cystic encapsulation of the parasite. The cyst enlarges as adult fluke grows, reaching up to 1.5-5cm in diameter.

Leakage of fluid into the bronchioles causes
*Paroxysmal coughing
*Hemoptysis.
*Up to 50 ml of gelatinous, rusty-brown sputum containing trace of blood and yellowish-brown parasite eggs may be expectorated daily during paroxysmal coughing.

* Chest pain.
* Clubbing fingers.
* Chronic case resemble tuberculosis.

b) Extra pulmonary picture
* Brain, lymph nodes, heart, subcutaneous tissues, abdomen, liver, groin. Flukes which lodge in these sites invoke inflammatory response, similar to that seen in lung, leading to ulceration and abscesses.

**Diagnosis**
1) Stool examination.
   Eggs in sputum and feces.
2) Serology test.
3) Skin test.
4) Biopsy.
5) Chest x-ray.

**Prevention and control**
1) Health education and sanitation improvement.
2) Destruction of snails. 3) Adequate cooking of crabs.
CHAPTER-2
CESTODA

Class Cestoidea
Subclass Cestoda

General characteristics of Cestodes.
*Cestodes are segmented.
*Dorsoventrally compressed.
*Tape – like or ribbon in shape.
*They vary from a few millimeters to several meters in length.
*They have no body cavity.
*Alimentary canal absent.
*Adult Cestodes live attached to the mucosa of small intestine and absorb food from the host intestine.

*Cestodes belong to the class Cestoidea.

Adult worm consists of three parts
-Scolex
-Neck
-Strobila

a) Scolex
*It is distended muscular organ.
*Scolex helps in the attachment of parasite into small intestine (In the order pseudophyllidae the scolex does not possess suckers, it possess a pair of longitudinal grooves).
*In order Cyclophyllidae the scolex possess four suckers.
**Cestoda**

**Rostellum**
- Apical protrusion in scolex.
- It is usually armed with hooks.

**b) Neck:** Actively part forming Strobila.

**c) Strobila:** It is composed of a chain of proglottides or segments.

**The proglottides near the neck are:**- Immature-Mature and Gravid segments.

![Classification of Cestodes](image)

**Pseudophyllidean tapeworms**

*Diphylobothrium latum*
(Broad tapeworm, Fish tapeworm)

**Host:** Man, Fish – eating animals.

**Habitat:** Small intestine, usually in the ileum.

**Geographical distribution:** Lake regions in Europe, Baltic, Some foci in America and Russia.

**Morphology and life cycle**

**Adult:** 3-10ms in size.

**Male system:** Comprises numerous testes distributed in the dorsal part of the segment.

**Scolex:** Elongated (2.5mm × 1mm) with 2 elongated dorsal and ventral groove (Bothria).

**Mature segments**
Broader than long containing the male and female genital system.
**Cestoda**

**Male system:** It Comprises numerous testes distributed in the dorsal part of the segment.

**Female system**
* It Comprises a bilobed ovary lying posteriorly in the segment, it lead to oviduct, open in ootype(surrounded by shell gland).
* From ootype arise the uterus and vagina.
* Uterus proceeds anteriorly as a convoluted tube (Rossete-shape), open posteriorly in common genital pore.
* Vittaline glands are distributed in the ventral part of the segment.

**Eggs**
* Oval, Operculated, Thick shelled.
* Content: Immature embryo.

**Coracidium:** Spherical 6 – hooked embryo enclosed in a ciliated embryophore.

**Procercoid:** Solid elongated (0.5 mm) with a spherical caudal end having 6 hooks.

**Plerocercoid:** Solid elongated (1- 2 Cm) with invaginated anterior end. The body is striated but not segmented.

**Life cycle**
* Adult may pass about a million eggs a day.
* Eggs are passed in feces in large numbers.
* Eggs doesn't float in saturated salt solution.
* They are not infecting to man.
* Eggs are resistant to chemical but are killed by drying.
* In water, egg matures in about 10-15 days and emerges through the operculum as ciliated; First stage Larva (Coracidium), it can survive in water about 12 hours.
* It should be ingested by fresh water Cyclops (1️⃣st intermediate host).
* In about 3 weeks Coracidium in cyclop transformed into elongated second stage larva about 0.5mm long, which called Procercoid larva.
* The infected Cyclops are swallowed by second intermediate host which is the fish.
* The Procercoid larvae penetrate the intestine of fish and transformed into 3️⃣rd stage larvae called plerocercoid or sparganum.
- 3rd stage larva called plerocercoid or sparganum.
- 3rd stage larva (Plerocercoid) is the infective stage to man.

**Mode of transmission:** Orally, eat unproper cooked fish infected by the plerocercoid.

The worm may live for about 10ys.

Fig. 16: Mature segment of *D. latum.*

Fig. 17: Life cycle of *D. latum.*
Pathogenicity, clinical and complications

Diagnosis
* Eggs in stool.
* Proglittoides crawl out of anus.

Control and prevention
* Infection can be prevented by proper cooking of fish.
* Eradication of the 1st intermediate host by filtration and boiling the water.
* Proper disposal of sewage.

Human sparagnosis
Definition: Infestation of the human tissue by the larval stage of *Diphylobothrium mansoni*, which is called sparganum (Spirometra).
It is identical with plerocercoid, irregular, it has tendency to multiply by budding giving lateral branches.

*Diphylobothrium mansoni*: Inhabits the small intestine of dogs, and not in man.
1st intermediate host: Cyclop.
2nd intermediate host: Frog, fish, bird.

Mode of transmission
* Eating of uncooked salted fish, frog infected with sparganum stage, which migrate inside the body of man and settle in any organs (Cannot develop to adult).
* Ingestion of infected *Cyclops* containing Procercoid.
Applying the infected flesh of frogs, snakes which contain sparganum to ulcerated parts of the body or eye.

Sparganum directly migrates to human tissue

Skin, eye causing subcutaneous sparagnosis and ocular sparagnosis

**Clinical picture:** According to involved organs.

**Diagnosis**

*Serology may helpful.
*Surgical removal.

**Order Cyclophyllidae**

**Genus: Taenia**

*Taenia saginata*

(Beef tapeworm) Unarmed tapeworm

**Geographical distribution:** Cosmopolitan.

**Habitat:** Small intestine, commonly in jejunum.

**Final host:** Man.

**Intermediate host:** Cattle.

**Disease:** Taeniasis.

**Morphology and life cycle**

**Adult:** 4-6 meters in length, may be much longer 25 meters or more(The largest helminth causing infection to man).

**Scolex (Head):** 1-2 mm in diameter, quadrate in cross section, bearing 4 hemispherical suckers, situated at its four angles.

*Scolex has no rostellum.
*Suckers act as an organ for attachment.

**Neck:** It is long and narrow.
Cestoda

Strobila
*The gravid segments are nearly four times as long as they are broad (20 mm long and 5 mm broad). It has 15-30 lateral branches.
*Mature segments consist of male and female reproductive structures, testes, 300-400, and bilobed ovary.
*The gravid segment break away and are expelled singly.
*Eggs escape from the uterus through its ruptured wall.

Eggs → Spheroid shape
   (30-40 µm in diameter)

Eggs has → A thin hyaline embryonic membrane
   Thick outer wall which is radially striated

*Contents of eggs: hexacanth embryo (onchosphere)

Cystocercus bovis: (Bladder worm)
*A bladder – like structure lined with a germinal layer enclosing a cavity containing fluids.
*From the germinal layer develops an invaginated scolex with 4 suckers. It measures 1-2 cm.

Fig. 18: Scolex of *T.saginata.*
Life Cycle
* Eggs deposited in soil remain viable for several weeks.
* Eggs infective to cattle, which ingest the eggs while grazing.
* When ingested by cattle, the egg-shell ruptures and onchosphere hatches out in the duodenum of cattle reach to mesenteric venules or lymphatic, enter circulation filter out to striated muscles (Tongue, shoulder, ham, myocardium), the onchosphere after 60-70 days develop into mature larva *Cystocercus bovis* which is the infective stage.
* When such infected cattle is eaten raw or inadequately cooked by man, cysticerci are digested out of the meat in the stomach in upper part of small intestine, the head envaginated out of cysticerci, by gradual strobilation develops into adult worm in 2-3 months.
* Adult worm has life span of 10 yrs.
* Infection is usually oral.
(The larva stage not present in human.)
Cestoda

Pathogenesis

→ Asymptomatic

Gravid segment

Crawling out the anus

cause alarm or itching

Intestinal disturbance

Abdominal discomfort

Diagnosis

*Microscopic examination of feces shows eggs (Salt floatation is not suitable for concentrating eggs in feces).

*Formal – ether sedimentation method is useful.

*Gravid segment notices by patient, then examine by a hand lens, the gravid segment press between two slides.

Prevention and control

*Inspection of beef or pork for cysticerci at slaughter houses.

*Thorough cooking of meat or freezing at – 10C for 5-10days.

Fig. 20: Life cycle of *Taenia.*
**T. solium**

(Pork tape – worm)

Pork tapeworm, present where the pork meat consumed.

Adult present in small intestine (jejunum), 2-3 meters in length.

 Scolex has four suckers and a conspicuous rounded rostellum armed with double rows (20-50).

The mature segment has 200 testis and triple ovary (Accessory lobe).

Gravid segment is thick and with 5-10 lateral branches, expelled in chain. The infection transmitted by ingestion of undercooked pork meat.

The final host is man.

The intermediate host are man and pig. Infective stage is the larva *Cysticercus cellulosae*.

**Cystocercosis**

**Definition:** This is the invasion of human tissues by the larval stage of *T. solium* (*Cysticercus cellulosae*).

**Intermediate host:** Man.

**Mode of transmission**

*By ingesting the eggs with contaminated water and food.*

*A man harboring an adult worm may auto – infect himself either by unhygienic personal habits or by reverse peristaltic movements of the intestine whereby the gravid segments are thrown into the stomach, equivalent to the swallowing of thousands of eggs.

![Fig. 21: Scolex of T.solium.](www.wjpr.net)
The *Cysticercus cellulosai* is.
*An ovoid shape, bladder like.
*It surrounded by fibrous capsule.
*It contains a thick fluid, rich in protein and salt.
*It remain viable for several months.
*It measures about 5-10 mm but can be much larger.
*It occurs in brain or subarachnoid space.
*It can develop in human or pig.
*Human is a dead end and the larvae die without further development.
*It can solitary or multiple.
*Most common organs involve are: eyes, brain, heart, liver, lung and muscles, subcutaneous tissue.
*The larva evoke a cellular reaction starting with infiltration of neutrophils, eosinophil's, lymphocytes, plasma cells and at time giant cells.
*This is followed by fibrosis and death of the larva with eventual calcification.

Fig. 22: Life cycle of Cystocercosis.
Cestoda

Clinical
* Epilepsy
* Behavioral disorders.
* Paresis and hydrocephalus.
* Ocular: Blurring of vision, iritis, blindness.

Diagnosis
* Definitive diagnosis of Cystocercosis is biopsy of the lesion and its microscopic examination.
* CT scan (Computerize Tomography).
* Ophthalmoscopy.
* Serology.
* Cystocercosis in muscle and subcutaneous tissue particularly in the buttocks and thighs diagnosed by.
Radiological examination of calcified larvae.

*T. Multiceps: (Multiceps multiceps)*

Morphology
Adult: 40-60 cm in length.
The scolex is pyriform in shape it has four suckers and rostellum armed with double rows of 22-30 hooklets.
Gravid segments are 8-10 mm in length and 3-4mm in width.
Eggs are 31-36 µm in diameter.

Final host: Dogs, wolf, fox.
Intermediate host: Sheep, cattle, horses; Man accidently may infected with larva stage.

Mode of transmission
* Human become infected by ingested food or water contaminated with dog's feces containing T. multiceps eggs.
* Onchosphers hatch in the intestine penetrate the intestinal wall, settle in various organs of the body (Central nervous system where the migrating larvae develop into a coenurusis).
Coenurus
Bladder worm with multiple scolices, but no broad capsules or daughter cysts:
Each scolex is capable of developing into an adult worm if eaten by definitive host.

Coenurosis
Definition: Infestation of human C.N.S by Coenurus cerebralis (Larva stage of Multiceps multiceps).

Clinical
*C.N. S is affected, rarely spine cord.
*Headache, aphasia, seizures, hemiplegia, paraplegia.

Diagnosis
*X- Ray.
*Serology.
*After surgical removal of the larva and histologic recognition of the Coenurus

T. serialis: (Multiceps serialis)
*Similar to Multiceps multiceps, larva stage is Coenurus serials.
*It differs from Coenurus cerebralis in having.

Scolices arranged in streaks or serials.
*This larva may develop external or internal daughter cysts.
*Man infected by ingestion of the eggs of the Multiceps serials
*Extra intestinal cestoda
*Man: Intermediate host.
*Eggs are the infective stages.
*Larvae present inside man and responsible for the clinical picture.
* Man is the dead end for the parasite.
*No adult in human.

-Examples
Sparganum or plerocercoid larva of Diphylobothrium mansoni(Sparagnosis).
Cysticercus cellulosae of Taenia solium(Cystocercosis).
Hydatid cyst of Echinococcus granulosus (Hydatidosis).
Coenurus cyst of Multiceps multiceps (Coenurosis).
**Dipylidium caninum**  
(Double pored dog tape worm)  
**Host:** Dogs, cat and human mainly children.

**Geographical distribution:** Austria, Japan.

**Habitat:** Small intestine.

**Intermediate host:** Flea (*Ctenocephalus canis, C.felis*).

**Disease:** Dipylidiasis

**Morphology**

**Adult:** 10-70cm long.  
Scolex has 4 prominent suckers and retractile rostellum with up to 7 rows of spines.  
The proglottides segment has two genital pores one on either side, hence the name dipylidium (dipylos – two entrance).

**The gravid segment:** The uterus is sac – like, break up into a number of egg capsules, enclosed in an embryonic membrane, contains 8-15 eggs in each.

The eggs are spherical (25-40um) in diameter, they have thin, hyaline, brick – red tinged shell.

**Life cycle**

*Eggs in capsules or proglottides are ingested by the larva stage of dog flea, cat flea or human flea.  
*The Onchosphers are liberated in the intestine of these fleas and migrate with their body cavity, they develop into cysticercoid larvae.
Cestoda

Fig. 23: Life cycle of *D. caninum*.

*When fleas ingested by dogs or cat, the larvae develop into adult worm, which become sexually mature in 3-4 weeks.

**Pathogenesis**
*Human infections are rare and normally restricted to young children. Infected flea hosts may be crushed in the dog's mouth, resulting the cysticercoids, and these may be transmitted to children in the dog's saliva.

**Clinical pictures:**
- Asymptomatic
- Actively motile proglottides passed in stool cause alarm
- Intestinal disturbance (Pain, loss of appetite)

**Diagnosis**
*By finding characteristic gravid segments.
*Egg capsules may be seen in stools.
Cestoda

Prevention and control
* Insecticide dusting of pet dogs and cats to kill fleas.
* Treatment of infected persons, pet dog.

_Hymenolepis nana (H. nana)_
(Dwarfism tapeworm)
* It is the smallest tapeworm infecting human. The name Hymenolepis refers to.
(Thin membrane covering egg, (Hymen – membrane, lepis – covering).

_Nanos:-_ Dwarf or small.

_G. Disrtribution:_ Cosmopolitan

_Host:_ Human, rat and mice.

_Habitat:_ Small intestine (Upper 2/3 of ileum).

_Infective Stage:_ Cysticercoid(Without tail).

_Disease:_ Hymenolepiasis nana.

_Morphology_
_Adult:_ Small (4-5 cm) in length.

_Scolex:_ Globular four – cupped shaped suckers, 0.3mm in diameter.
Retractile rostellum with a single row of 20-30 hook lets.

_Strobila:_ It consists of 200 proglottides.

_Mature segment:_ 0.5x0.15mm long.Broader than longer.

_Male genitals:_ 3 testes, one on the side of genital pore and two on the a polar side.

_Female genitals:_ as in _Taenia._
(Testes and ovary close together in the middle of the mature segment).
Genital pores are marginal and are situated on the same side.
Gravid segment is a transverse sac, broader than long, full with eggs.
Egg:—* It is spherical 35-40 um it has a smooth, thin and colorless outer shell and an inner membrane (Embryophore), containing hexacanth embryo.
*The space between two membranes is filled with yolk granules and 4-8 polar filaments (At either end of embryophore).
*It is non – bile stained and floats in saturated solution of common salt.

Life cycle
* It is the only cestoda which is capable of completing its life cycle in a single host.
Direct and indirect cycle.

Direct cycle
*Eggs and proglottides with eggs are passed in the feces of infected humans and rodents.
*Man acquires infection by ingestion of food and water contaminated (Fecal oral rout).
*In lumen of small intestine a free onchosphere is liberated from the egg.
Cestoda

*It penetrates into a villus of anterior part of small intestine and develops, into cysticercoid larva (Without tail).
*The villus ruptures and the cysticercoid becomes free in the lumen of the small intestine.
*It attaches by its scolex to another villus further down after 2 weeks, develop into adult worm.
*After month from infection, the eggs appear in stool.
*In heavy infection, the eggs may hatch in the intestine before passing out in the feces, resulting in auto infection. (No intermediate host is required).

**Indirect cycle**
* A different strain of *H. nana* infects rat.
* Insect is the intermediate host.
* The eggs passed in rodent feces, are ingested by rat flea (*Xenopsylla cheopis*), which act as intermediate host.
* The eggs develop into cysticercoids larvae in the haemocele of these insects.
* Rodent get infection when they eat these insects.
* The murine strain doesn't appear to infect man.
* The human strain may infect rodents.
* Man is infected by accidental ingestion of these infected fleas.

![Fig. 25: Life cycle of *H.nana.*](image-url)
Control and prevention

* Personal hygiene.
* Rodent control.

**H. Diminuta**
(Rat Tapeworm)

* It is a common parasites of rats and mice.
* It is larger than *H. nana* (10-60 cm in length).
* Rostellum without hooks.
* Mature segment similar to *H.nana* but bigger.
* It has two a polar testes and one polar testes with the ovary in between.
* Gravid segment, its internal structure similar to *H.nana*.
* Egg is larger than egg of *H.nana*, measuring 60-80um in diameter, the polar filaments is absent(There is polar thickening).
* Its life cycle is similar to that of murine strain of *H. nana*.
* Rarely, human infection follows accidental ingestion of infected rat fleas.
* Human infection is asymptomatic.
* Diagnosis by finding eggs in stool.
* Control by using anti-rat campaign
* Notes: Infective stage of *H.nana* and *H.diminuta* is cysticercoid.
* Cysticercoid in *H.diminuta* has tail.

**Genus: Echinococcus.**

*Echinococcus granulosus.*
(Dog tapeworm), (Hydatid worm).

**Host:** A carnivores predator (Dog, fox, wolf).
G. Distribution: Cosmopolitan.

Intermediate host: Prey host (Ruminants, accidently man).

Habitat: Small intestine.

Disease: Hydatidosis, Hydatid disease.

Morphology
Adult worm: Small (3 – 6 mm) in length.

Scolex: pyriform, it has four suckers and prominent rostellum bearing two circular rows of hooklets.
It has 3 proglottides only: Immature-Mature-Gravid.
*Eggs are indistinguishable from those of Taenia.

![Fig. 26: Adult of E.granulosus.](image)

Life cycle
*Eggs are passed in dog feces.
*Sheep and cattle ingest them while grazing.
*Egg- shell disintegrates in the duodenum.
*Hexacanth embryos penetrate the intestinal wall and enter portal venules to liver to portal circulation.
Cestoda

* Liver act as first filter for the embryos.
* Embryos escape, filter out to systemic circulation to the lung.
* Lung act as second filter.
* Few embryos enter systemic circulation and get lodged in various organs: Brain, kidney, eye and bone.
* At the site of deposition the embryo develop into bladder or cyst (Hydatid cyst).
* Man is a dead end host.
* Hydatid cysts in animals are usually thrown in slaughter houses to be eaten by dogs, whereas adult formed in their small intestine.

**Hydatid cyst**

**Types of hydatid cyst**

- Unilocular: Osseous, sterile, fertile
- Multilocular

**Morphology of unilocular hydatid cyst.**

**Size:** varying from tennis ball to man fist.

Spherical in shape

* The cyst wall secreted by the embryo consists of two layers.

**a) Ectocyst**

* It is outer layer
* Acellular
* Laminated
* Hyaline membrane up to 1mm in thickness.

**b) Endocyst**

* It is the inner layer or germinal layer.
* It measures 22-25um in thickness.
* It gives rise to Ectocyst on outside and brood capsules and Scolices on inside. It secretes hydatid fluid.
Cestoda

Hydatid fluid
* It is clear, colorless.
* It has specific gravity of 1.005 - 1.010.
* It is slightly acidic (PH 6.7).
* It contains protein salt (sodium chloride, sodium sulphate, sodium phosphate and calcium salt).
* It is antigenic
* It is use for Casoni test.

Hydatid Sand
* It consists of brood capsule and free scolices and hooklets.
Sterile cyst (Acephalocysts):
* It is never produce brood capsules.
* If ingested by final host don't lead to infection.

Osseous cyst
* In bone, it has no fibrous capsule, it is mostly sterile.

* Endogenous daughter cyst.
Sometimes a fragment of the germinal layer may detach and develop daughter cyst inside the mother cyst.
It also has both Ectocyst and endocyst with brood capsules and scolices.
Grand-daughter cysts may also formed.

Exogenous cyst

- In case of hydatid disease of bone, because of high intracystic pressure.

Herniation or rupture of germinal and laminated layer may occur, through some weaker part of bone cyst.
Result in formation of exogenous cyst
Clinical disease (Hydatid disease)

Definition
Infestation of the human tissue by the larva stage (Hydatid cyst) of *Echinococcus granulosus*.

Mode of infection
* Playing, kissing of infected dogs and ingestion of eggs of *Echinococcus granulosus*.
* Ingestion of food contaminated with the eggs of *Echinococcus granulosus*.

Pathogenesis: Depend on the location of the cyst.

a) Liver cyst: It is in right lobe, it may.
* Rupture (Anaphylactic shock).
* Obstructive jaundice

b) Lung cyst: It may be.
* Asymptomatic
* Cough, hemoptysis, chest pain and breathlessness

c) Kidney cyst: It may be lead to.
* Pain and hematuria.

d) Bone cyst: It may lead to:
* Pathological fracture

Diagnosis
* Clinical picture
* High eosinophilia
* Intradermal test, (Casoni’s test)
* Serology.
* Examination of fluid.
* Histological examination of surgically removed cyst.
* Radio diagnosis: X- Ray, ultrasound and CT scan.
**Prevention and control**

*It is essential to wash the hand after touching dogs.*

*Kissing of pet dogs should be discouraged.*

**Fig. 27: Life cycle of *E.granulosus*.**

*Echinococcus multiocularis*

*(Alveococcus multiocularis)*

Host: Foxes, and bears.

Habitat: Small intestine.

**Intermediate host.**

Rodents, some larger mammals and accidently man.

**Larval stage:** Multilocular or malignant hydatid cyst, it is mainly in liver rapid growing and metastasis to other organs, it has not daughter cyst.
CHAPTER-3
NEMATODES

Phylum Nemathelminths
Nematoda
General Characteristics
*Nematodes belong to the phylum nematoda.
Many species of nematodes are.
*Free living in fresh water, salt water, soil, mud.
*Parasitic in plants and animals.
*They are the most abundant and widespread animal group.
*The name nematode mean thread – like”.
*Nematodes are elongated, cylindrical, unsegmented worms with tapering ends.
*They are bilateral, symmetrical.
*The body is covered with a tough cuticle, which may be smooth, striated, bossed or spiny.
*The adults vary greatly in size from about a millimeter to a meter in length.
*Flesh – colored bodies.
*Sexes are separate, male is smaller and thinner than female.
*They Have digestive system.
*Male have ventrally curved posterior end, which may have papillae, copulatory spicules and bursa.
*Anterior end possess lips, teeth, hooks, cutting plates or papillae for attachment to final host.

The body wall has no epithelial lining and consists of.
*An outer layer, hyaline non- cellular cuticle.
**A subcuticular epithelium.
***A layer of muscle cells.

*The male reproductive system consists of a long convoluted tube which differentiate in to: testis, vas deferens, seminal vesicle and ejaculatory duct which open into cloaca.
*The female reproductive system consists of ovary, oviduct, seminal receptacle, uterus and vagina.
The female nematodes may be divided as follows.

**Oviparous** (Nematodes which lay egg)

*unsegmented.*

*Ascaris lumbricoides.*

*Trichuris trichiura.*

*With segmented ova*

*Ancylostoma duodenale.*

*Necator Americans.*

*Containing larva*

*Entrobious vermicularis*

**Viviparous** (Nematodes which give birth to larvae).

*Dracunculus medinensis.*

*Wuchereria bancrofti.*

*Brugia malayi.*

*Trichnella spiralis*

**Ovo – viviparous** (Nematodes laying eggs containing larvae which immediately hatched out): *Strongyloides stercoralis.*

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**Classification of nematodes**

a) According to habitat of adult worm:

- **Intestinal**
  - Not need intermediate host (Soil – Transmitted –Parasite)

- **Tissue (Somatic)**
  - Need intermediate host
General types of esophagus may be

1) Simple club-shaped
   * _Ancylostoma duodenale_ and _Ascaris lumbricoides_.

2) Double bulbed esophagus
   * Pin worm

3) Rhabditiform type
   It consists of an anterior cylindrical part and posterior pyriform part connected with each other by neck (Separated by constriction).
   * _Strogyloides stercoralis_

4) Cylindrical esophagus
   It Composed of two parts short muscular narrow anterior part and a long wide glandular posterior part (Filaria).

5) Cellular type
   The esophageal tube is surrounding by a raw of cells.
   * _Tichinella spiralis_

**_Trichinella spiralis_**
(Trichina worm)
Disease: Trichinosis
Distribution: Temperate regions.
Host: Rat, pig, man
Nematodes

Habitat: Adult worm in the small intestine (Duodenum and jejunum) (Larvae are embedded in the tissue of the same host).

Morphology

Adult

They are viviparous release 1st stage larvae into intestine mucosa.

*Female: 3-4 mm in length.

*Male: 1.4 – 1.6 mm in length. At its tail end it bear a pair of conspicuous papillae (claspers), that it uses to hold on to the female worm during mating.

*Life span of adult is very short.

Larvae

* Larvae measure 80um in length.

* The infective stage larva becomes encysted in the striated muscle fiber.

* It measures 1mm in length.

* The larva in the cyst is coiled and hence, the species name (spiralis).

Life cycle

*The whole life cycle is passed in one host: Man or Rat or Pig.

*Man acquires infection by ingestion of raw or inadequate cooked pork containing the viable larvae.

*Smoking, salting or drying the meat doesn't destroy the infective larval forms.

*The larvae are released in the stomach by the action of digestive enzymes, free larvae are transported to the duodenum and jejunum, they penetrate into epithelium, four mouls occur becomes mature (adult), after 5 days become sexually mature.

*After fertilizing the female the male die, the fertilized female discharges a large number of the first stage motile larvae into intestinal mucosa, the larvae are born continuously throughout the next 5 days.

*These larvae enter the lamina propria and from there penetrate the mesenteric lymphatic's and blood stream.

*The larvae go to general circulation become distributed to central nervous system, striated skeletal muscles, and other sites, while they die in most other situations.
Nematodes

*They grow and develop in the skeletal muscles.
*Deposition in the muscles occurs mostly during the second week of infection.
*Larval development in muscles takes place during the next three to four weeks.
*After this, they become encysted and remain as infective larva inside the cysts for many years.
*At the time of deposition in the muscle fibers, the larvae are about 100 um by 6 um in size.
*They grow in size, becoming about 1 mm long.
*They remain tightly coiled and enclosed within a fibrous capsule.
*The cyst is formed by the tissue reaction around the encapsulated larvae.

Cysts are usually
Ovoid.
400 um by 250 um in size.
Lying longitudinally along the muscle fibers.
They calcified in about 2 years, but the larvae often remain viable even inside calcified cysts.
Cysts develop preferentially in muscles relatively poor in glycogen.

(Diaphragm, intercostal, pectoral girdle, cervical, tongue, Jaw and extra ocular muscles).
*The deltoïd muscle being easily accessible chosen for taking diagnostic muscle biopsies.

Pathogenesis
The symptomatology are divide into three successive stages.

Stage of intestinal invasion (Enteric phase).
This occurs during the early stages of the infection and lead to.
*Inflammation of duodenal and jejunal mucosa.
*Gastrointestinal symptoms.

Stage of Larva migration: The invasion of muscle lead to.
*Fever, Myalgia and Periorbital edema.
*Eosinophilia is a constant feature.
Nematodes

*Myocarditis and encephalitis are serious.
*Respiratory symptoms may occur.
This stage appears usually one to four week after infection.

**Stage of encapsulation (Encystment phase).**
*This stage occurs only in striated muscles.
*In other tissues they degenerate and are absorbed.
*Larvae calcify.
*The calcified cysts can be seen grossly in exposed muscle fibers.

**Diagnosis**
*Detection of spiral larvae in muscle tissue.
(Deltoid, biceps, gastrocnemius) are usually selected for biopsy.
*Detection of adult worms and larvae in stool during diarrheal stages.
*Blood examination.
Total white blood cell count is slightly elevated (12,000-15,000/ul) (Eosinophilia 20-90%).
*Serology.

*Bachman intradermal test.
Uses a 1:5000 or 1:10,000 dilution of the larval antigen, an erythematous wheal appears in positive cases within 15-20 minutes.
The test remains positive for years after infection.
*Calcified cysts may be demonstrated in skiagrams.
*Xenodiagnoses: Biopsy bits are fed to laboratory rats, which are killed a month or so later, larvae can be demonstrated more easily in the muscles of such infected rats.

**Prevention and control**
*Properly cooking pork and its products.
*Inspection of slaughtered pigs.
*Preventing pig from eating raw meat and offal.
Nematodes

Trichuris trichiura
(Trichocephalus trichiurus)
(Whip worm)
Disease: Trichuriasis.

Distribution: Cosmopolitan

Host: Man only (Host specific).

Habitat: Caecum, appendix, colon, rectum.
(No reservoir hosts), (No intermediate host).

Infective stage
Egg with 2\textsuperscript{nd} stage larva.

Morphology
Adult worm
*Whip shape.
*Anterior three – fifth is very thin and hair like and the posterior two – fifth is thick.
*The male: 30 – 45mm in length and have coiled posterior end.
*The female worm are longer (35-50 mm) in length and their posterior extremity is comma or arc-shaped.
*The worm has a lifespan of 5 -10years.

Fig. 28: Adult of whip worm.
Nematodes

**Eggs**
*Barrel – shape with a mucous plug at each pole.
*Shell is yellow to brown (bile – stained) and plugs are colorless.
*They measure 50 – 54 um.
*They float in saturated salt solution.

**Life cycle: (Without migration)**
*The entire life cycle can be passed in one host.
*The egg passed in feces contains an unsegmented ovum, at this stage it is not infective for humans.
*The egg undergoes development in soil (warm, moist).
*The infective Rhabditiform larva develops within the egg in 3-4 weeks.
*At lower temperatures this may be delayed for 3 months or more.
Infection occurs when the mature embryonated eggs containing the infective larva are swallowed in food or water.
*The eggs hatch in the small intestine and the larva which emerges through the pole of the egg passes down into the caecum, In about 2-3 months, they become mature adult.
*It embedded on the caecal wall with the thread like anterior portion piercing the mucosa and the thick posterior end projecting out.
*Eggs start appearing in feces usually about 3 months after infection.

**Pathogenesis and clinical**
*Asymptomatic
*Mechanical and allergic effect.
*Chronic dysentery
*Oozing of blood may occurs at site of attachment, lead to iron deficiency anemia.
*Mechanical blockage of the appendiceal lumen by masses of worms may cause acute appendicitis.
*Young children may develop rectal prolapsed (Coconut cake).

**Diagnosis**
*Eggs in stool.
*Proctoscopy.
*Leyden crystals are usually abundant in stool.
Blood Test: Anemia (Iron deficiency anemia).

**Prevention and control**
*Sanitary disposal of feces.
*Avoiding consumption of raw vegetables.
*Treatment of infected person.

![Life cycle of Whip worm.](image)

**Fig. 29: Life cycle of Whip worm.**

**Genus Capillaria**
There are three species of zoonotic importance, *C. hepatica, C. philippinensis* and *C. aerophila*.

Adult worms of Capillaria are similar to Trichorus, but are smaller and delicate. *C. hepatica.*
*It is a common parasite of rats.*
*It is a parasite of the liver of rats, but it has been found in a wide variety of mammals including humans.*
*Adult female measures 20mm long and about 100um wide.*
*Adult male is about half as long. The eggs resemble those of *Trichuris trichiura*, but the poles are less tapered and measure 51-68 um X 30-35 um.*
*The female worm lays eggs in the liver.*
Nematodes

*When the eggs contained within infected liver are eaten by animals during cannibalism, the eggs liberated by the digestion of the tissues are passed in the feces and develop to the infective stage in damp soil.
*When the infective stages are ingested by humans with contaminated food or drink, they hatch in small intestine produce larvae, larvae migrate through the portal system to the liver they mature in about one month and the female worms lay eggs.

Clinically
a) Acute and sub-acute hepatitis
b) Hyperesinophilia

Diagnosis
Demonstrated eggs or adult in liver biopsy.

C. Aerophila
*It is a worldwide parasite of cats, dogs, foxes end other carnivores.
*It lives beneath the mucosa of:

Upper and lower respiratory tract.
*Male measure up to 18mm x 70um.
*Female up to 20mm x 105 um.
*Female worm lays eggs that measures 59-80um x 39-40um with characteristic polar plugs in tunnels in the mucosal epithelium.
*They are later sloughed into the air passages cough up, swallowed and discharged in the feces, eggs passing out with the feces embryonated in the moist soil, embryonated eggs enter a new host via contaminated food or water.

Clinically
a) Tracheobronchitis
b) Productive cough

Diagnosis
*Demonstration of
Eggs in the sputum or feces or both.
Adult worm in the lung biopsy.
**C. Philippinensis**

*It is an intestinal parasite.

*The original animal host remains unknown.

*Fish eating birds are the natural definitive hosts.

*In man it lives burrowed into the mucosa of the small intestine (jejunum).

*Adult males measures 1.5mm-3.9mm in length.

*Adult females measure 2.3mm-5.3mm in length.

*Female produce ova which are barrel – shaped, smaller (36-45um x 20um), more cylindrical in shape, and have less prominent bipolar plugs and have a thin shell which shows radial lines.

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**Eggs in feces of infected person**

[Diagram: Eggs in feces of infected person → Embryonated in water in 5-10 ds. → Develop further If swallowed by small freshwater and brackish water fish → Man acquires infection by ingestion of raw or undercooked fish containing larvae, then become adult in small intestine of man.]

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*The female adult is sometimes larviparous or can produce thin – wall egg that hatches within the intestinal tract, the larva can moult and reinvade the intestinal mucosa (Auto-infection and hyper infection).

**Clinical**

*C. philippinensis* causes intestinal capillariasis.

*Malabsorption, loss of electrolytes specially potassium and plasma protein.

*Patient complains of diarrhea, abdominal pain, vomiting.

*Losing enteropathy characterized by dehydration.

**Diagnosis**

*Detection of eggs in stool.
Nematodes

*Larvae and adult may also be seen in stool.

Fig. 30: Life cycle of *C. hepatica*.

Fig. 31: Life cycle of *C. philippenensis*.
**Enterobius vermicularis**

*(Pin worm. Thread worm. Seat worm)*

**Disease:** Entrobiasis, oxyuriasis.

**Distribution:** Cosmopolitan.

**Host:** Man specially children.

No migration.

**Habitat:** Perianal region, caecum colon, rectum and appendix.

Not need intermediate host (S.T.P).

**Morphology**

**Adult worms**

* Small, white, spindle in shape.
* At anterior end both male and female worms possess a pair of wing – like expansions, known as cervical alae.
* The male measures 2-4mm in length.
* The posterior one – third of the body is curved and carries a prominent copulatory spicule.
* The female is longer, 8-12 mm in length, its posterior extremity is straight and drawn out into a thin pointed pin – like tail.
* Males live for 7 weeks and females live for 5-13 weeks.

**Eggs**

* Colorless
* Not bile – stained.
* Flattened on one side (Plano convex).
* They are surrounded by a thin smooth, transparent shell and usually contain fully developed larvae.
* They float in saturated solution of common salt.

**Life cycle (No migration)**

* Pin worm is monoxenous, passing its entire life cycle in the human host.
* It has no intermediate host.
Nematodes

*Male dies after fertilization, while the gravid female go down to anus to lay its egg in the peri-anal region.
*A single worm lays from 5000-17,000 eggs.
*The egg contains mature larva, and infection occurs in few hours.
*Under cool moist conditions, the egg remain viable for 2 weeks.

Man is infected by swallowing the eggs by one of the following ways:
*Autoinfection (hand to mouth) in food or drink.
*Handling contaminated clothing or bedding.
*Inhalation of air-born eggs in dust.
*Retro infection: eggs hatch in the per anal region and larvae migrate back through the anus to the large intestine.

Clinical
*Infection occurs mostly in children.
*It is more common in female than males.

Irritation, itching, scratching of perianal area, this is lead to.
*Sleeplessness
*Nervous symptoms
*Secondary bacterial infection
*The worm may migrate to the vagina, uterus, fallopian tube, sometimes reaching the peritoneum, they may causes symptoms of salpingitis.
*Pruritic ani especially at night due to nocturnal migration of gravid female to deposit eggs in per anal region.

Diagnosis
*History and clinical picture.
*Demonstration of the adult and the egg are seldom found in feces.
*NIH (National Institutes of Health).
*Scotch adhesive tape swab.

Prevention and control
*Personal hygiene.
*Wearing of gloves.
Nematodes

* Frequent changing and washing of bed – covers and night clothes.
* Keeping the bedrooms clean and dust free.
* Treating the infected persons. If infection is confirmed in one individual in a household, other family members should be examined and treated.

Fig 32: Life cycle of Pin worm.

Fig 33: Adult of Pin worm. A-male, B-female.
**Ascaris lumbricoides**  
*(Round Worm)*  
**Disease:** Ascariasis.  
**Distribution:** Cosmopolitan.  
**Host:** Man.

**Migratory worm.**  
**Habitat:** Small intestine, particularly jejunum.  
No intermediate host (S.T.P). Man acquires infection orally, air-born way.  
The round worm, *Ascaris lumbricoides* is the largest nematoda parasite in the human intestine.

**Inf ective stage:** Egg with Rhabditiform larva (2nd stage larva).

**Morphology**  
* Cylindrical worms, with tapering ends, the anterior end being more pointed than the posterior.  
* Flesh colored.  
* The mouth at the anterior end has three finely denticulate lips, one dorsal and two Ventro – lateral.  
* The digestive and respiratory organs of the worm float inside the body cavity possessing a toxic fluid known as ascaron.  
* Male worm (15-30 cm) in length, the posterior end is curved ventrally to form a hook, and carries two copulatory spicules.  
* Female worm: it is larger than male (25-40cm) in length, its posterior extremity is straight and conical.  
* A single worm lay up to 200.000 eggs per day.

**Eggs:** Fertilized, unfertilized and decorticated.  

**Fertilized eggs:** It is spherical or ovoid, bile stain.  
It measures 60-75um.  
It enclosed in a stout translucent shell contains of three layers.  
a) Outer coarsely mamillated Albuminoid layer.  
b) Thick transparent middle layer.  
c) Inner lipoid Vittaline membrane.
Decorticated eggs
Some eggs are found in feces without the outer mamillated coat, they are called the decorticated egg.

In the middle of the egg is a large unsegmented ovum containing a mass of coarse lecithin granules. It nearly fills the egg, except for a clear crescentic area at either pole.

Unfertilized egg
It is Longer, up to 90um and more elliptical.
The shell is thinner with the outer mammillary coat scanty and irregular.
The ovum is atrophic and contains numerous disorganized, highly retractile granules of various sizes.

Fig 34: Eggs of Round worm.

Fig 35: Life cycle of round worm.
Nematodes

**Life cycle**
*The adult worm is in the small intestine of man (Jejunum).*
*Fertilized eggs contain unsegmented ovum are passed in feces.*
*They are not immediately infected man.*
*They remain in soil (Temperature 22-30°C + Humidity), first moult occurs within the egg shell in 10-14 days and become embryonated egg with second stage larva.*
*When man take orally food or water contaminated with embryonated egg with second stage larva, in small intestine of man (Duodenum) the ingested eggs hatch to liberate the larvae, which burrow their way through mucous membrane of small intestine and by portal circulation go to liver and via hepatic veins, inferior vena cava reach right heart and by pulmonary artery reach the lung.*
*In lung, the larvae grow in size and moult twice, then larvae break through the capillary wall reaching the alveoli, and migrate up to bronchi, trachea and larynx, crawl over the epiglottis to the pharynx and are swallowed.*
*They pass down the esophagus and stomach, then localize in upper part of small intestine.*
*After 1 month, the larvae undergo another moulting and transform into adult.*
*After 6-10 weeks, the adult worms become sexually mature, the gravid females begin in discharge eggs in the stool and the cycle is repeat.*

**Pathogenicity and clinical**

**Clinical pictures in ascariasis can be caused by**
*Adult worm*
*Migratory larvae*
*Ectopic lesion*

**Loeffler's syndrome**
The migrating larvae may lead to inflammatory and hypersensitivity reactions in the lung. There is formation of granuloma and eosinophilic infiltrates. It is leads to fever, cough, dyspnea, urticarial rash and eosinophilia. Sputum may be blood – tinged and may contain Ascaris larvae and Charcot – Leyden crystals.
Allergic inflammatory reactions to migrating larvae may involve other organs such as liver, kidneys. Larval and adult worm secrete allergen which elicit the production of IGE by the host, leading to hypersensitivity and histamine release. (IGE: Immunoglobulin E.)

**Pathological effects**

a) Nutritional effects.
b) Toxic effects.
c) Mechanical effects.

**a) Nutritional effect.**

*The worms may be present in enormous numbers, sometimes exceeding 500, in small children, occupying a large part of the intestinal tract, interferes with digestion and absorption of food. This is lead to Protein–energy malnutrition and vitamin A deficiency (Night blindness).*

**Growth retardation.**

Toxic effect: It is due to.

Hypersensitivity to the worm antigens, lead to: Fever, Urticaria, wheezing and conjunctivitis.

**b) Mechanical effect**

*Abdominal pain.*

*Intestinal obstruction.*

**The worm may reach to different organs and lead to.**

* Lung abscess.
* Coming out from nose and Eustachian tube.
* Pancreatitis.
* Bile duct obstruction.
* Perforation of abdominal wall and peritonitis
* Liver abscess.

**Diagnosis**

Nematodes

Eosinophilia.
Detection of eggs in feces.
Detection of adult in feces, vomits.
Serology (Not useful in diagnosis).

**Confirm test:** Barium contrast radiography of the abdomen (Tramway sign).

*Update diagnosis*
Molecular diagnosis is highly sensitive and specific.
Antibody-based diagnostic may be sensitive.
Kato-Katz thick smear are standard detection method for ascariasis diagnosis but less sensitive.

**Prevention and control**
*Proper washing of raw vegetables.
*Washing hands before meals.
*Mass treatment, personal hygiene.

**Visceral Larva Migrans: (VLM).**
*It is produced.
when man accidentally ingested embryonated eggs of.
a)*Toxocara cani*(Ascarid of dog)
b)*Toxocara cati*(Ascarid of cat)

*The eggs hatch in small intestine of man and the larvae penetrate the wall of the gut and enter the lymphatic or blood stream.
* Man is unnatural host for dog and cat Ascarid.
* The larvae of animals cannot complete its cycle through the lung and back to the intestine.
*The larvae migrate through viscera.

**Granulomatous lesion formed in.**
a)Lung
b)Liver
c)Eye(Ophthalmic larva migrans)
d)Other viscera
Nematodes

Clinical:
- Fever
- Hepatomegaly
- Cough
- High eosinophilia
- Hyperglobulinaemia

Diagnosis
* Eosinophilia
* Serological tests.
* Biopsy from suspected organs if possible.

Prevention and control
* Personal hygiene.
* Avoid playing with contaminated soil.
* Periodic examination of dogs and cats.
* Treatment of infected animals.

Fig. 36: Life cycle of Toxocara.
**Nematodes**

*Strongyloides stercoralis*
*(Dwarf thread worm)*

**Host:** Man, dogs

**Distribution:** Tropical and subtropical areas.

**Disease:** Strongyloidosis, Cochin – China diarrhea.

**Habitat:** Small intestine (Duodenum and jejunum).

Migratory worm (S.T.P).

** Infective stage:** Filariform larvae.

**Mode of transmission:** Penetration.

**Morphology**

**Adult worm**

*Parasitic female is: Small, thin, 2-3mm in length.*

*It has cylindrical esophagus occupying the anterior third of the body and the intestines in the posterior two-thirds of the body.*

*The caudal extremity is pointed.*

*Free – living adult female measures 1mm in length.*

*The female is ovoviviparous.*

*Parasitic males are shorter and broader than the female, they don't invade the intestinal wall.*

*Free living – males is slightly smaller than the free – living female.*

*They have 2 spicules and gubernaculum.*

**Eggs**

*In gravid female, the eggs are conspicuous within the body, lying antero-posteriorly in a single file (5-10 eggs in each uterus).*

*The eggs measure 50-58 um x 30-34 um.*

*They are thin – shell, transparent and oval.*

*They contain larvae ready to hatch.*

*As soon as the eggs are laid, the rhabditiform larvae start hatching and bore their way out of the mucus membrane into the lumen from where they are passed in feces (The eggs are not detected in the feces).*
Rhabditiform larvae
* These are the most commonly seen in stool specimens.
* They measure 200-250um in length.
* They have short mouth and double –bulb esophagus.

Filariform larvae
* These are long, slender.
* They measure 600-700um in length.
* They possess short mouth, long cylindrical esophagus and notched tail.
* They are highly infectious.
* They are non-feeding.

Life Cycle
* The adults (Parasitic males and females) live in small intestine of man.
* After fertilization, the male die and the females burrow deeply in the mucosa.
* Eggs are laid in the mucosa, hatch into rhabditiform larvae which migrate to the lumen of intestine and pass in feces.

In the soil, the rhabditiform larvae either
a) Develop into filariform larvae infective to man(Direct cycle).
b) Or give rise to free –living generation (Indirect cycle).

a) Direct cycle (Like hook worm)
When the environmental conditions are unfavorable for free existence, the rhabditiform larvae moult and become filariform larvae in 2-3 days.

b) Indirect cycle
When environmental conditions are favorable for free – living, the rhabditiform larvae moult and give free – living male and female within 2 days.

Worms mate and females lay eggs in the soil. Egg hatch giving rhabditiform larvae which either,
- Repeat the free-living generation.
- Give filariform larvae.
Man is infected when the filariform larvae penetrate his intact skin or mucous membranes.

*Larvae enter the venous circulation to right heart to lung to alveoli to bronchial tree to epiglottis to small intestine.
*Moult twice during migration to become adult.
*Rhabditiform larvae appear in stools within 4 weeks of infection.

Autoinfection also occurs if.
The rhabditiform larvae change into filariform larvae in the lumen of intestine before expelled in stools penetrate the mucosa (Endo – autoinfection) or the change occur at perianal area (Exo –auto infection).

Autoinfection results in hyper – infection.

Fig. 37: Comparison between 3th stage larva of hookworm and Strongyloides.
Pathogenesis and clinical pictures

The clinical disease may be classified as

a) Cutaneous (Invasion stage): As in Ancylostoma.
b) Pulmonary (Migratory stage): (As in Ancylostoma).
c) Intestinal phase:

In heavy infection may lead to.

* Epigastric pain.
* Chronic inflammation of intestinal mucosa, necrosis and fibrosis.
* Dysentery
* Malabsorption syndrome

Others

* Eosinophilia
* The Filariform larvae are penetrating the perianal skin and cause linear urticarial lesion over the thigh, trunk (Larva currens).
Nematodes

*In hyper infection that occurs in immune deficit hosts, the clinical pictures depend on the site affected.
  a) Brain abscess
  b) Meningitis
  c) Peritonitis
  d) Septicemia (If circulating larva e carry intestinal bacteria).

Diagnosis
*Rhabditiform larvae in stools, sputum, duodenal aspiration.
*Agar plate culture for stool.
*Eosinophilia.
*Serological tests.

Prevention and control
Prevention of soil contamination with feces.
Avoiding contact with infective soil.

Cutaneous Larva Migrans: (CLM)
(Creeping eruption)
*It caused by non-human species of hookworms, *Ancylostoma brazilienes* and *A. caninum*, when their larvae infect man by skin penetration.
*Infection with these hookworms of dogs and cats are acquired from soil contaminated with excreta of these animals.
*The larvae produce itching papules which develop into serpigenous tunnels in the epidermis.
*Larvae remain localized.
*Sites most commonly affected are the feet, space between the toes, hands, knees and buttocks.
*The disease is self-limiting. Humans are an accidental and dead-end host so the larvae eventually die.

Treatment: Topical and oral anthelminthic drugs are used.
Nematodes

Hook worm

*Ancylostoma duodenale* (The old world hook worm)

**Disease:** Ancylostomiasis.

*Ancylo* = bent stoma = mouth

**Distribution:** Tropical and subtropical areas.

**Host:** Man.

**Habitat:** Small intestine, mostly in the jejunum.

**Mode of transmission**
- Penetration
- Trans placental
- Trans mammary
- Rarely orally

**Infective stage:** 3rd stage larvae (Filariform larvae).
* Migratory worm.
* Not need intermediate host (S.T.P).

**Morphology**

**Adult**
* They are cylindrical worm.
* They are pale pink, but may appear reddish brown due to ingested blood.
* The body is curved.
* The anterior end is somewhat constricted and bent dorsally.
* The mouth is not at the tip but directed dorsally.
* A large mouth cavity (buccal capsule) with two pairs of teeth at the anterior margin (ventral or upper), two plates at the posterior margin (dorsal or lower) and two sub ventral lancets in its bottom.
* A club – shaped esophagus (1/6 the length of the worm).

**Male:** 1 cm in length, has one set of genitalia provided with 2 spicules and copulatory bursa.
Nematodes

**Female:** 1.5 cm in length, has two set of genitalia.

**Copulatory bursa**

Copulatory bursa are present in the male worm for: attachment with the female during copulation.

*They consists of three lobes:
One dorsal and two lateral.
These lobes are supported by 13 chitinous rays:
Five each in lateral lobes and three in dorsal lobe: one dorsal and two extra dorsal rays.
The dorsal ray is partially divided at the tip and each division is tripartite.
*The life span of the adult worms in the human intestine is 3 -4 years.

**Eggs**

*Eggs are oval or elliptical measuring 60 um in length.
*They are colorless (Not bile- stained) and are surrounded by a thin transparent hyaline shell.
*They possess a segmented ovum with usually four blastomeries.
*There is a clear space between the segmented ovum and the egg shell.
*The eggs float in saturated salt station.

**Table 2: The difference between Larvae.**

<table>
<thead>
<tr>
<th>Rhabditiform Larva</th>
<th>Filariform larva</th>
</tr>
</thead>
<tbody>
<tr>
<td>About 250-500u in length long buccal cavity.</td>
<td>About 600-700u in length.</td>
</tr>
<tr>
<td>Rhabditiform esophagus (1/3 body length)</td>
<td>Cylindrical esophagus (1/4 body length).</td>
</tr>
</tbody>
</table>

**Life cycle**

*Eggs freshly passed in feces are not infective for man.
*When deposited in the soil, embryo develops inside the eggs.
*Its development takes place in sandy loamy soil with decaying vegetation under a moist warm, shady environment.
*In about 2days, a rhabditiform larva, hatch out of the egg.
*It feeds on bacteria and other organic matter in the soil, grows in size and mount twice to become filariform larva with sharp pointed tail.
*Filariform larvae are non-feeding.
*They can live in the soil for about 5 weeks.
*Direct sunlight, drying or salt water can kill the larvae.
*When a person walks barefooted on soil containing the filariform larvae, they penetrate the skin and enter the subcutaneous tissue.
*The larvae enter the venules to circulation to right heart to lung to alveoli, then, migrate up the respiratory tract to the epiglottis, they crawl over the epiglottis to pharynx and are swallowed.
*During their migration they mount for the third time.
On reaching jejunum, they mount for the fourth time and become adult.
*There is no multiplication in the host, one infective larva develops into the a single adult.

Fig. 39: Life cycle of Hook worm.

Fig. 40: Adult, larva and egg of Ancylostoma duodenalea.
Pathogenesis and clinical
The damage caused by hookworms depends on the following factors.
* The size and age of the patient.
* The nutritional condition of the patient.
* The presence of complicating disease (causing anemia).
* The numbers of worms.

Clinical picture
A, B caused by larva.

A) Invasive stage
Skin penetration by filariform larva is lead to itching (Ground itch).

B) Migratory stage
In the lung the larva leads to pneumonitis, cough, eosinophilia.
(The Loeffler syndrome, commonly seen in ascariasis is rare in hookworm infection).

c) Intestinal stage (Caused by adult worm).
* The worms attach themselves to the gut by their buccal capsules.
* They suck into their mouth apportion of intestinal villi.
* They utilize gut epithelial cells and plasma for their food.
* The worms sucks blood, which passes out undigested and unutilized, through its intestines causes of anemia in ancylostomiasis.
An adult Ancylostoma can sucks about 0.2ml of blood a day, while the smaller Necator sucks in about 0.03 ml per day.

As the secretions of the worm contain anticoagulant activity, bleeding from the site may continue for some time, this add in blood loss. This chronic loss of blood over a period of time lead to Iron deficiency anemia.

Toxic substance produced by the worm lead to:
- shortening of Red blood cells life span.
- Hookworm infection may cause an intestinal syndrome resemble peptic ulcer.
- Hypoproteinemia.
- Severe Hookworm anemia may lead to cardiac failure.

**Diagnosis**

- Direct method: demonstration of eggs in feces.
- Adult worm may also be detected in the soil.
- Aspiration of duodenal contents by Ryle's tube may reveal eggs or the adult worm.
- Blood examination microcytic hypochromic anemia and eosinophilia.
- Stool examination show occult blood and Charcot – Leyden crystals.

**Baerman test:** Testing of soil for the presence of larvae.

**Necator americanus**

| Table 3: Differences between the adult worm of *A. duodenale* and *N. americanus*. |
|--------------------------------|---------------------------------|
|                                | *A. duodenale*                  | *N. americanus*               |
| **Size**                       | Larger and thicker              | Smaller and thinner           |
| **Buccal capsule**             | Six teeth, four hook-like on ventral surface and two-knob-like on dorsal surface | Four chitinous plates, two each on ventral and dorsal surface |
| **Copulatory bursa**           | Dorsal ray is single. Total number of rays is13. Two separate spicules. | Dorsal ray is spilt from the base. Total number of rays is14. Two spicules fused at the tip. |
| **Posterior end of female**    | A posterior spine is present    | No posterior spine is present |
| **Vulval opening**             | Situated behind the middle of the body. | Situated infront of the middle of the body. |

Prevention and control

- wearing shoes, gloves.
- Treatment of patients carrier.
- Sanitary disposal of human feces and education of peoples.
Tissue nematodes

1) Guinea worm (Medina worm), *Dracunculus medinensis*.

**Disease:** Dracunculosis.

**Distribution:** Nile valley, Saudia Arabia, Yemen, India and areas where wells are used for water supply.

**Host:** Man, dogs, horse.

**Habitat:** The adult worm occurs in the subcutaneous, tissue, particularly those of the ankle and foot, arms and shoulder.

**Intermediate host:** Cyclops.

It distributed in areas where wells are used for water supply.

**Table 4: The differences between intestinal and tissue nematode.**

<table>
<thead>
<tr>
<th>Intestinal</th>
<th>Tissue nematode</th>
</tr>
</thead>
<tbody>
<tr>
<td>-not need intermediate host (soil –Transmitted parasite)</td>
<td>- Need intermediate host.</td>
</tr>
<tr>
<td>-Transmission : orally or penetration.</td>
<td>- Bite or penetration.</td>
</tr>
<tr>
<td>-Infective stage: 2(^{nd}) stage larva.</td>
<td>3(^{rd}) stage larva.</td>
</tr>
<tr>
<td>3(^{rd}) stage larva.</td>
<td></td>
</tr>
<tr>
<td>Ex: Ascaris, Ancylostoma</td>
<td>Madina worm, filarial.</td>
</tr>
</tbody>
</table>

**Morphology**

**Adult worms**

Mature female is a slender, long worm, measuring 50-80cm x 2mm.

*The body is cylindrical, the anterior end is bluntly rounded and the posterior extremity is tapering and is bent to form a hook.*

*The worm is viviparous, and discharges embryos in successive batches for a period of 3 weeks until the gravid female completely empties its uterine contents.*

*The body fluid is toxic and causes a blister if it escapes into the tissues.*

*Male, which is but rarely seen, much smaller, 10-40mm long and 0.4 mm thick.*

*The posterior end of the male is coiled on itself one or more times.*

*The life span of the female is about 1 year and that of male is not more than 6 months.*
Embryos (larvae)
* These are coiled bodies with rounded head and long slender tapering tail (1/3 body), it is comma shape.
* They measures 500-750 um in length, the cuticle shows prominent striations.
* Larva swims about with a coiling and uncoiling motion.

Life cycle
The life cycle take place in two host.
1) Final host: Man.
2) Intermediate host: Cyclop.

* After fertilization, male die in the tissue and the female migrates to the superficial layers of the skin, especially regions liable to come in contact with water.
* The worm secretes a substance, which cause a blister on the skin (Over its anterior end).
* The blister bursts and a small ulcer is formed.

On contact with water
* The uterus is projected out of the ulcer and hundreds of active larvae are released (When the host leaves the water, the exposed end of the uterus dried and blocks the release of further larvae).
* The larvae move about with a stiff motion, briskly coiling and uncoiling the body.
* In water they can live for only a few days.
* If these larvae are ingested by cyclop, they moult twice and are infective in about 2 weeks.

Mode of Transmission and life cycle
* Human become infected by drinking water containing infected Cyclops.
* On reaching the stomach the Cyclops are ingested by gastric juice and the larvae liberated.
* They then migrate through the digestive tract and reach the loose connective tissue probably via lymphatic's.
* There they mature to male and female adult worms in about 6 months.
* Male fertilizes the female and dies.
Nematodes

*In another 6 months the fertilized female migrates to subcutaneous tissue of those parts of the body which are likely to come in contact with water and discharges the embryos in water.

Pathogenesis
*Migration of adult female under the skin causes allergic reactions in the form of rash, nausea.
*Formation of reddish papule in skin, which rapidly become considerably inflammation.
*Lesion may be single or multiple.

These lesions are most common
*Between metatarsal bones of the soles of the feet.
*On the ankle.
*Hand or arm.
*Trunk, buttock, scrotum.
*Knee joint, calf, thigh and shoulder.
*On rupture of the blister leading to ulcer formation.
*The fluid in the blister is bacteriologically sterile and yellow in color, it contains polymorphic cells and monocytes.

Serious complications from introduction of bacteria under the skin is lead to
*Tetanus spores
*Abscesses
*Synovitis
*Arthritis

Diagnosis
*Clinical picture (Papule, blister, and ulcer).
*Observation of an adult worm under the skin.
*Detection of calcified worm by X-Ray.
*Serological tests.
*Eosinophilia in blood.
*Intradermal test.
*Detection of larvae (embryo).
Cold water is placed on the ulcer on contact with water a large number of motile larvae are discharged which can be examined under microscope.

**Control and prevention**

*Filtering or boiling drinking water.

*Preventing infected persons with on emerging worm from entering the water sources.

*Treat water sources by chemical of low toxicity.

![Fig 42: Life cycle of Madina worm.](image-url)
2) Filarial worms

Filarial nematodes belong to the
Phylum: Nematode
Class: Secernentea.
Superfamily: Filarioidea

General characteristics
*They are slender thread-like worm.
*They need intermediate host (Blood – sucking insects) for transmission.
*Adult female viviparous, laying larvae termed microfilariae.
*Microfilariae are found in blood or skin of the final host, they are the diagnostic stage of filarial worms.

Adult worms inhibit
The circulatory and lymphatic channels, muscles, connective tissue, serous cavities.
They measure 80-100 mm x 0.25-0.3mm.
The worm has a simple lipless mouth, small buccal cavity, a cylindrical esophagus without bulb.
The tail of male worm has no caudal bursa, but carries perianal papillae and unequal spicules.
The microfilariae, which appear in blood are easily seen and provide a good basis for identification of parasite.
They measure 180-300 um in length.

They can be differentiated on the basis of
*The size.
*Periodicity of their appearance in peripheral blood.
*Present or absent of sheath.
*Distribution of nuclei in caudal region of larva.

Microfilarial Periodicity
*When largest number of microfilariae occur in the blood at night, it is known as the nocturnal periodicity.
*The microfilariae circulate in the peripheral circulation between 9p.m and 2 a.m.
Nematodes

*When largest number of microfilariae occur in the blood during day, it is known as diurnal periodicity.
*Non – periodic microfilariae, in this case the microfilariae circulate at somewhat constant levels during the day and night.
*Sub-periodic or nocturnally sub – periodic microfilariae are those that can be detected in the blood throughout the day but detected at higher level during the late afternoon, at night.

**A according to the normal habitat of the adult worm, human filarial infections can be classified as follow**

1-lymphatic filariasis.

* W. bancrofti
* B. malayi
* B. timori

2-Subcutaneous filariasis

* Loa loa
* Onchocercus volvulus
* Mansonella streptocerca

3-Seros cavity filariasis

* Mansonella ozzard
* Mansonella PERSTAN

**Wuchereria bancrofti**

Bancrofts filarial worm.

**Disease:** Wuchereriasis or bancroftian Filariasis.

**Distribution:** It occurs in Asia, Africa, Australia and South America.

**Habitat:** adult worm occurs in the major lymphatic ducts of the lower half of the body(Rarely upper arms).

**Final host:** Man.
Nematodes

Intermediate host: Female Mosquitoes of various species, The major vector is *Culex fatigans*. In Egypt *C.pipiens*.

Morphology
* Adult worms are transparent, creamy white, long, hair like structure.
* They are filiform in shape with both ends tapering.
The male worms measure 2.5-4cmx0.1mm and the female worms measure 8-10cmx0.2-0.3mm.
The posterior end of the female is straight, while that of male is curved ventrally and contains two spicules of unequal length.
* Both male and female worms remain coiled together and it is difficult to separate them.
* The female is viviparous and liberates sheathed embryos(microfilariae) into lymph from where they find their way to blood.

Life Cycle
* The adult female releases the microfilariae into the lymph.
* The microfilariae circulate In the blood stream.
* They show a nocturnal periodicity In peripheral blood, being seen in large numbers in peripheral blood only at night between 10 pm and 4 am.
* Nocturnal periodic microfilariae are believed to spend the day time mainly in the capillaries of the lung and kidney or in the heart and great vessels.
* Microfilariae do not multiply or undergo any further development in the human body.
* If they are not taken up by a female vector mosquito, they die. Their lifespan is believed to be 2-3 months.
* When a vector mosquito feed on a carrier, the microfilaria are taken in with the blood meal and reach the stomach of mosquito.
* They cast off their sheaths, penetrate the stomach wall and within 4-17 hours migrate to the thoracic muscles where they undergo further development.
* They metamorphose into the first-stage larva which is sausage-shaped form with spiky tail, measuring 125-250x10-15um.
* Within a week, it moult one or twice, increase in size and becomes the second stage larvae, measuring 225-325x15-30um.
*In another week it becomes the elongate third stage filariform larva.
*This filariform larva migrate to the labium of the vector.
*When the mosquito sucks blood, the filariform larva (Infective stage) coming out of the labium and invade the human body through the bite wound.
*Then enter the lymph system and changed into adult worms after one year.

![Life cycle of W. bancrofti.](image)

**Clinical manifestations**
Filariasis leads to a wide spectrum of clinical manifestations, ranging from carrier state to chronic picture.
Filariasis may cause great suffering disfiguration and disability.

**The main clinical features are**
1) **Inflammatory manifestations**
Due to toxic products of living or dead adult worms, there may be.
*Lymphangitis(Orchitis-epididymitis….etc.), lymphadenitis (Enlarged of lymph node).
*Fever, chill, headache, vomiting and malaise.
*Leukocytosis and eosinophilia.
2) **Obstructive manifestations**

Due to fibrosis following the inflammatory process and also due to the coiled worms inside lymphatic's.

**This may result in**

* Lymphorrhagia: Rupture of lymph avarices leads to the release of lymph or chylus. (Chyluria, chylus ascites and chylothorax).
* Lymphangiovarix: Dilatation of lymph vessels commonly occur in the inguinal, scrotal, testicular and abdominal site.
* Hydrocele.

This is a very common manifestation of filariasis. Accumulation of fluid occurs due to obstruction of lymph vessels of the spermatic cord and also, by exudation from the inflamed testes and epididymis.

3) **Elephantiasis**

This is a delayed sequel to repeat lymphangitis, obstruction. Soft edema of the affected part followed by hypertrophy of the skin and subcutaneous connective tissue. The part becomes thickened, rough and fissured. Elephantiasis affects the legs and genitalia (Scrotum, penis and vulva).

![Microfilaria of wuchereria](image_url)
**Nematodes**

**Diagnosis**
Clinical pictures, laboratory tests.

**Laboratory Tests**
a) Demonstration of microfilariae in peripheral blood. Microfilariae may also be detected in other specimens such as chylus urine or hydrocele fluid.
b) Demonstration of the adult worm in biopsy specimens
c) Skin test with filarial antigens
d) Serological tests
f) Indirect way such as eosinophilia
(Demonstration of microfilariae in peripheral blood is the diagnostic test most commonly employed).

**Control and prevention**
a) Eradication of mosquito.
b) Detection and treatment of carrier.

**Occult filariasis**
This is the hypersensitivity to Microfilarial antigens.
It manifests by: Eosinophilia + generalized lymph node enlargement, hepatosplenomegaly and pulmonary symptoms.

*The adult worm produces the microfilariae continuously but they don't reach the peripheral blood because they are destroyed in the tissues.

**Tropical pulmonary eosinophilia TPE**
This is a manifestation of occult filariasis characterized by.
Malaise-fever-weight loss and respiratory symptoms such as.
Dry nocturnal cough, dyspnea and asthmatic wheezing with marked increase in blood eosinophilia.
TPE is associated with marked increase in total serum IGE.
It is associated with *W.bancrofti* and *B.malayi* infection.

**Brugia malayi**
**Common name:** Malayan Filaria.
Nematodes

**Distribution:** Far East.

**Habitat:** The adult worm found in lymphatic vessels of distal extremities

**Intermediate host**
Mosquitoes of genera *Mansonia*, *Anopheles* and *Aedes*.

**Life cycle:** Similar to *W. bancrofti*.

**Clinical manifestations**
Similar to *W. bancrofti*, mild elephantiasis may occur.
There is no Chyluria, it may also cause tropical pulmonary eosinophilia.

**Diagnosis**
Same as *W. bancrofti*

*Burgia timori*
It is limited to timor and some other islands of eastern Indonesia.
The vector is *Anopheles barbirosties* a night feeder.
No animal reservoir.
The microfilaria is larger than *B. malayi*.
The sheath of *B. timori* fail to take giemsa stain.

**Subcutaneous Filaria**

*Loa loa*

**Common name:** The eye worm

**Disease:** Loasis.

**Distribution:** Tropical Africa.

**Habitat**
*Adult worms live in subcutaneous tissue of man.*
*They also may occur in the conjunctival tissues of eye.*
*The sheathed microfilariae are found in blood, they appear in peripheral circulation only during day (Diurnal periodic).*
-Infection is transmitted through the bite of infected female mango fly (*Chrysops* species).
Nematodes

**Life cycle:** Similar to *W.bancrofti.*

**Clinical manifestations**
The clinical pictures of loasis depend on.
The migratory habit of adult worm.
The adult worms live in subcutaneous connective tissues and wander round the body causing.

*Painless edematous swelling known as*  
**Calaber swellings.**  
The swellings measure 5-10cm in diameter, they are allergic in origin, probably to excreatory products of adult.  
Last for a few hours to few days and reappear in elsewhere, 
they may occur anywhere on body but most common on:*The back of hand or arm.  
-Sometimes are accompanied by: Itching, erythema, fever, eosinophilia.

**Ocular manifestations**
The adult worms may wander slowly across the conjunctiva, causing some discomfort and edema of the eyelid.

It may cause granulomata in the bulbar conjunctiva, and proptosis.

**Diagnosis**
*High eosinophilia is common.*
*Microfilariae may be shown in peripheral blood collected during the day.*
*The adult worm can be demonstrated by removal from the skin or conjunctiva.

**Onchocerca volvulus**
**Common name:** The convoluted Filaria, blinding Filaria.

**Disease:** Onchocercosis.

**Distribution:** Tropical Africa and Central America.

**Habitat:** The adult worms are located in nodules in subcutaneous tissue of man.

**Intermediate host:** Day –biting female black Fly of the genus *Simulium*
**Nematodes**

**Life cycle:** Similar to *W.bancrofti.*

**Clinical manifestations**

**a) Skin lesion**

Adult worms causing subcutaneous onchocercomas, dense fibrous nodules, painless ranging from 5-25mm in diameter.

These nodules tend to occur over anatomic sites where the bone is superficial, such as Knee, hips, iliac crests, sacrum, ribs, scapulae, elbows and scalp.

The nodules are fully movable.

Microfilariae also cause skin lesions such as: Dermatitis, pigmentation, atrophy and fibrosis.

**b) Ocular lesion**

Microfilariae may wander through the adjacent skin and reach other tissues, including the eye.

They cause corneal and retinal lesion lead to blindness.

It is known as river blindness as it occurs in people living near fast running rivers where *Simulium* flies breed.

**Diagnosis**

* Clinically.
* Demonstration of microfilariae in skin snips.
* Biopsy of nodules :Section of adult worms surrounded by fibrous tissues.
* Excision of an onchocercoma and identification of adult worms.
* Immunological tests.
* Mazzotti test.
Serous cavity filaria

Mansonella ozzardi

(New world Filariasis).

Distribution: South America

The vector is the midge Culicoides.

Microfilariae can be found in the blood and skin.

Fig. 45: Life cycle of *Onchocerca volvulus*.

Fig. 46: Microfilaria of Filaria species.
Mansonella Perstans

The vector is the Culicoides.

Microfilariae can be found in blood.

Distribution: Africa and South America.

Table. (5): Differentiation of microfilariae.

<table>
<thead>
<tr>
<th>Microfilaria</th>
<th>W.bancrofti</th>
<th>B.malayi</th>
<th>Loa loa</th>
<th>O.volvulus</th>
<th>M.perstan</th>
<th>M.ozzardi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheath</td>
<td>Loose</td>
<td>-Loose</td>
<td>-Tight</td>
<td>300x8u-</td>
<td>100x5u-</td>
<td>200x5u-</td>
</tr>
<tr>
<td>Size</td>
<td>-250x8u-</td>
<td>200x6u-</td>
<td>250x8u-</td>
<td>250x8u-</td>
<td>250x8u-</td>
<td>250x8u-</td>
</tr>
<tr>
<td>Appearance</td>
<td>-Smooth curves</td>
<td>-Kinky curves</td>
<td>-Kinky curves</td>
<td>-Kinky curves</td>
<td>-Kinky curves</td>
<td>-Kinky curves</td>
</tr>
<tr>
<td>Nuclei at tail</td>
<td>Free- 2nuclei</td>
<td>Full Blood</td>
<td>Full Blood</td>
<td>Full</td>
<td>Full</td>
<td>Full</td>
</tr>
<tr>
<td>Habitat</td>
<td>Nocturnal</td>
<td>Nocturnal-</td>
<td>Diurnal-</td>
<td>-Tight</td>
<td>-Tight</td>
<td>-Tight</td>
</tr>
<tr>
<td>Periodicity</td>
<td>-Nocturnal</td>
<td>-Nocturnal</td>
<td>-Diurnal</td>
<td>-Non.P</td>
<td>-Non.</td>
<td>-Non.P</td>
</tr>
</tbody>
</table>

*Dirofilaria immitis (Heart dog worm)*

It is a common parasite of dogs.

These worms inhibits the right ventricle and pulmonary artery of dogs.

It is transmitted by mosquitoes.

In human immature worms may be found in pulmonary artery and the lower lobe of right lung.

In man the condition is called A microfilaraemic filariasis(Tropical pulmonary eosinophilia syndrome).

The dead worm becomes as embolus blocking a small branch of pulmonary artery, produce pulmonary infarct. The healed infarct appear as a (Coin lesion) on chest x-ray.
Important Questions

1) Describe the pathogenicity and laboratory diagnosis of
   Taenia saginata
   Echinococcus granulosus
   Schistosoma haematobium
   Ascaris lumbricoides
   H.nana

2) Discuss geographical distribution and habitat of
   T.solium
   H.heterophyes
   L.loa

3) Mention
   -Classify of nematodes.
   -General characteristic of blood trematoda.
   -The differences between intestinal cestoda and extra intestinal cestoda.

4) Give the main complications of the following parasites
   A.duodenale
   P.westermani
   Enterobius vermicularis

5) What is the other name for the following parasites
   E.granulosus
   D.caninum
   L.loa
   H.nana

MCQs

1) Best site for taking biopsy for diagnosis of trichinellosis is
   a) Deltoid muscle
   b) Diaphragm
c) Pectoralis major
d) Liver

2) In which of the following parasites sexes are separate
a) *T.saginata*
b) *H.heterophyes*
c) *F.hepatica*
d) *Schistosoma mansoni*

3) Eggs are passed in sputum in case of infection with
a) *Clonorchis sinensis*
b) *A.lumbricoides*
c) *H.nana*
d) *P.westermani*

4) All of the following parasites may cause iron deficiency anemia except
a) *A.duodenale*
b) *Diphyllobothrium latum*
c) *T.trichiura*
d) *N.americanus*

5) NIH swab is used by detection of eggs of
a) *A.lumbricoides*
b) *N.americanus*
c) *E.vermicularis*
d) *A.duodenale*

6) What is the most organ involved in Hydatidosis
a) Liver
b) Lung
c) Spleen
d) Kidney

7) Cyst of *E.multilocularis* differs from that of *E.granulosus* in having
a) Multiple locules
b) Little or no fluid
c) Hyperplastic germinal layer
d) All of above

8) **Common name for** *D.latum* **is**
   a) Fish tapeworm
   b) Beef tapeworm
   c) Pork tapeworm
   d) Rat tapeworm

9) **Which of the following stages of** *A.duodenale* **is infective to human beings?**
   a) Rhabditiform larva
   b) Filariform larva
   c) Eggs
   d) Adult worm

10) **Which is the final host of** *E.granulosus*?
    a) Dogs
    b) Man
    c) Fox
    d) Wolf

7) **Fill the space**
   - The common name for *D.caninum* is-----------.
   - Casoni test is used for diagnosis of-----------------.
   - The intermediate host in indirect life cycle of *H.nana* is--------.
   Coenurus is the larva stage of -----------.
   -(Larva currens) is the name given to the migratory larvae of ---------------

8) **Put in tables the infective stage and diagnostic stage of the following parasites.**
   a) *H.nana*
   b) *T.saginata*
   c) Dog tapeworm
   d) Whip worm

9) **Draw the Eggs of the following parasites.**
   a) *A.lumbricoides*
Questions and References

b) *H. nana*

c) *T. saginata*

d) Species of *schistosoma*

10) Cases

a) A 30-years – old patient reported in the medical outpatient department with chief complains of fatigue, weakness, diarrhea and numbness of extremities.

The peripheral blood film revealed megaloblastic anemia and stool examination showing yellowish-brown Operculated eggs, 70mm length.

Which parasite is likely to be the cause of the disease?

b) A 50-year-old patient reported in surgery out-patient department with multiple subcutaneous nodules over the right iliac crest. They measured 5-15mm and fully movable. Adult worm and microfilariae detected inside the excited nodules and skin snips that taken from the lesions.

-Which is the nematoda likely to be in the case?
-What is the intermediate host?

11) Discuss about the control and prevention steps in case of Pin worm?

12) Migratory worms, they are passing to lung during their life cycle. Explain the reasons for migration?

13) Mention the name of parasites that transmitted by

a) Penetration

b) Orally by eating contaminated food and water

c) Orally by eating improper cooked pork meat

d) Air-borne inhalation.

14) Why the geographical distribution of parasite is important in practical medical fields?
15) Mention the main important parasite spread in Yemen, according to the following classification.
- Blood trematoda.
- Intestinal cestoda.
- Extra intestinal cestoda.
- Soil-Transmitted-Parasite.
- Tissue nematode.

16) What is your opinion about Zoonotic disease?

17) Give the habitat of.
* Rat tape-worm
* Beef tape worm
* Eye worm

18a) Explain briefly the life cycle of T. saginata?
18b) Give in table the different between the gravid segment of Beef tape worm and Pork tape worm?

19) Cases.
Case 1: In poly clinic in Taiz, mother comes and need your advice, because her kids complain of itching during night, and she saw a rice like-worm in their underwear's. What are your advices to her? And why?
Case 2: In Taiz schools, what are your advices to students if they want to go to Warazan region for swimming?
Case 3: From Mawia region in Taiz, woman comes complain of difficulty in breathing, by investigation the specialist see cyst like, fill with fluid in Chest x-Ray, he diagnosed the case as hydatid disease and refer the case to the surgeon. What is the name of parasite that cause this disease? What are the other investigations should be done by doctor? What is the mode of transmission in the case? What is the infective and the diagnostic stage in this case?
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