# MEDICAL HELMINTHOLOGY

This part deals with the study of helminths (worms) that parasitize man.

They belong to two main groups:

| 1- Platyhelminths (flat worms) | 2- Nemathelminths (round worms) |
|---|---|
| a- Class: Trematoda<br>b- Class: Cestoda | Class: Nematoda |

# PLATYHELMINTHS

## Class: Trematoda (Flukes)

### General characters:

-The members of this class are commonly known as flukes.

-Adults are leaf-like, pear-shaped or elongated worms.

-All trematodes possess two **suckers** as organs of attachment.

-Covered externally by a cuticle that may be smooth, spiny or tuberculated.

- The body is made up of systems:

| | |
|---|---|
| **Digestive system** | Starts by the mouth opening, found at the bottom of the oral sucker. The mouth leads to a pharynx, then a short oesophagus, which bifurcates, into two long intestinal caeca. |
| **Excretory system** | Starts by a definite number of excretory cells called (flame cells). Waste products pass from the cell to the excretory tubules➔excretory duct ➔excretory bladder which discharge its contents through a pore situated at the posterior end of the fluke. |
| **Nervous system** | Is simple. It consists of a ring of nerve ganglia, around the pharynx, from which nerve fibers ramify |
| **Reproductive system** | All the parasitic trematodes are hermaphrodites except *Schistosoma* worms.<br>*The **male reproductive organs** consist of two or more testes.<br>*The **female genital organs** consist of a single ovary situated in front of the two testes. |
| **Nutrition and respiration** | Parasitic trematodes feed on blood, intestinal contents, biliary secretions and tissue juices depending on their habitat. Adult flukes are essentially anaerobic |

# Hepatic or Liver Flukes

## *Fasciola gigantica* (Large liver fluke)

**Geographical distribution:** Human infection has been reported from many regions including Egypt, Africa and Far East.

**Morphology:**

1. Large Fleshy leaf- like worm, measures 3 -7x1 cm.
2. Body formed of small anterior conical part (cephalic cone), shoulders with parallel borders and posterior round end.
3. Suckers: small oral anterior sucker and large ventral sucker.
4. Digestive system: mouth leads to oesophagus with muscular pharynx, two long intestinal caeca with lateral compound branches and medial T or Y-shaped ones.
5. Genital system (reproductive system):
   a. Common genital pore: anterior to the ventral sucker.
   b. Testes: two highly branched, one behind the other, about the middle third of the body
   c. Ovary: branched at the right side in front of the testis.
   d. Uterus: short and convoluted.
   e. Vitelline glands: highly branched and extend along the lateral fields.

**Life cycle:**

-**Habitat**: adult worms live in the bile ducts and gall bladder.

-**Definitive host**: man.

-**Intermediate host:** snail *Lymnaea cailliaudi.*

-**Reservoir hosts:** herbivorous animals as cattle, sheep, goat and camels.

-**Infective stage:** encysted metacercaria in water and on aquatic vegetations.

-**Stages in the life cycle**: egg → miracidium → sporocyst → 1st and 2nd generation redia → cercariae →encysted metacercariae→ adult.

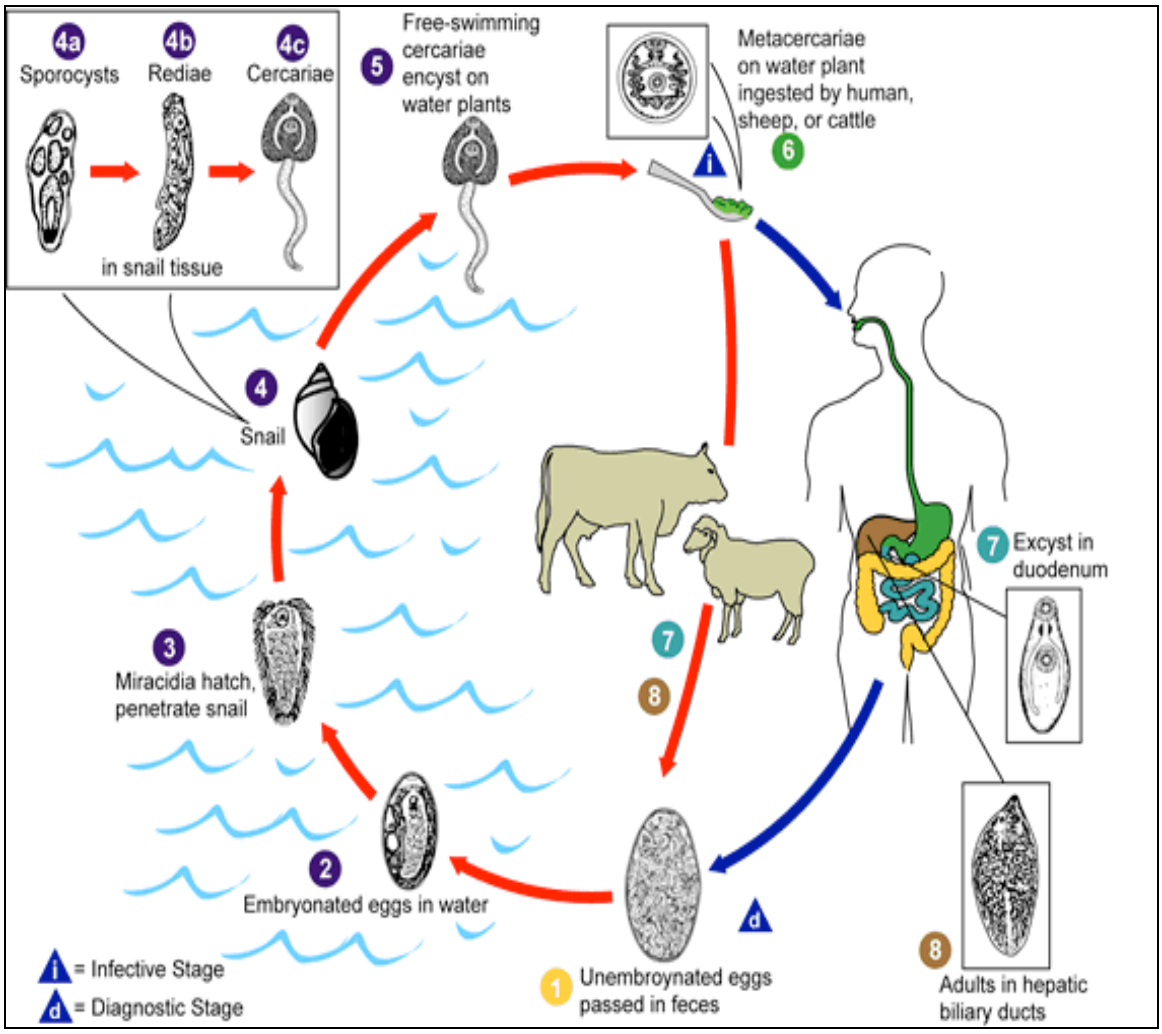
**Egg:** Size: 160x80 µ. Shape: oval.

Shell: thin. Color: light yellowish brown (bile stained).

Contents: immature (yolk cells).

Special character: operculated.

- Eggs are discharged with feces of infected host, in fresh water of canals, drains and River Nile, hatch within 2 weeks into miracidium.




**The life cycle of *Fasciola spp.***

**Miracidium**: a phototropic pyriform ciliated organism that can swim in water and penetrates the snail intermediate host.

**Sporocyst**: Simple elongated sac in the snail.

**Redia:** Cylindrical larva in the snail.

**Cercaria:** ***Leptocercous cercaria*** formed of body (0.3mm) and simple tail (0.7mm).

-Body with 2 suckers (oral and ventral), primitive gut, excretory system of flame cells, and cystogenous glands that secrete the cyst wall.

-Cercaria comes out from the snail and moves in water, gets attached to aquatic vegetations, loses its tail and changes into→encysted metacercaria.

**Encysted metacercariae:**

-Spherical 0.25mm. -Thick white cyst walls.

-They need about 12 hours after encystation to cause infection, and they live in water for 6- 10 months.

**Mode of infection:**

- By eating raw water vegetations or vegetables washed in infected water and by drinking infected water polluted by the **encysted metacercaria**.

• **In the duodenum**, the cyst wall dissolves and the metacercaria penetrate the wall of the intestine to reach peritoneal cavity.

• Metacercariae pass to the liver through it's capsule → through the liver tissue to their final habitat in the bile duct, where they maturate to adult in about two months after infection, eggs appear in the stool 3-4 months after infection.

**Pathogenicity and clinical picture:** Four Symptomatic Patterns

- **Acute Phase**
- **Chronic Phase**
- **Halzoun**
- **Ectopic Infection**

**Acute Phase**

- Rarely seen in humans.
- Fever, tender hepatomegaly, and abdominal pain are frequent symptoms.
- Vomiting, diarrhea, and anemia may be present**.**

**Chronic Phase**

- **Symptoms include:** Irregular fever ,biliary colic, abdominal pain, tender hepatomegaly, and jaundice.
- **In children:** severe anemia and high eosinophilia are common.
- Inflammation of the bile ducts leads to fibrosis and a condition called **"pipe-stem liver".**
- **Liver rot : ** mechanical and toxic destruction of liver tissue by passage of large number of immature worms through the liver tissue leads to necrosis , fibrosis , hepatitis, and hepatomegaly
- Severe infections can lead to death.

**Halzoun:**

-Occurs when an individual consumes infected raw liver.

-The living *Fasciola* adult worm attach to the mucosa of the pharynx by its suckers. This causes oedematous congestion of the pharynx and larynx resulting in dysphagia and suffocation.

The case is treated by:

a) Gargling with alcoholic drink.
b) Giving emetic drugs.
c) Picking up of the worm by forceps.

- Tracheostomy in suffocation

- **Ectopic Infection :** In frequent, but can occur in peritoneal cavity, intestinal wall, lungs, subcutaneous tissue, and very rarely in other locations.

**N.B.:**

- **False fascioliasis:** it is due to eating of infected animals liver and passage of eggs in the stool. This must be excluded by repeated stool analysis one week after liver free diet.

**Diagnosis:**

| | |
|---|---|
| **Clinical** | fever, hepatomegaly, habit of green salad consumption |
| **Laboratory** | **1-Stool examination:** for detection of **eggs,** after asking the patient to stop eating liver for a few days before examination.<br>**N.B.:Flukes do not begin to produce eggs until about 4 months after infection. Prior to 4 months: serological tests can be used**<br>**2-Serological tests:** are of value during the migratory stage of the worms and ectopic infection for estimation of specific antibodies, as ELISA, IHA.<br>**3-Examination of sample of aspirated duodenal contents.**<br>**4-Eosinophilia.**<br>**5-Ultrasound and CT** |

**Treatment**:

1. Bithionol (Bitin).
2. Triclabendazole (Fasinex).
3. Surgical removal of ectopic flukes.

**Prevention and control**:

1. Mass treatment of infected man and animal reservoir.
2. Snail control.
3. Sanitary disposal of feces.
4. Protection: - Pure filtered water supply.
   -Proper washing or cooking of aquatic vegetation.

## *Fasciola hepatica*

**Pathogenicity:**

-The adult worm can live in sheep for 5 year and cause liver cirrhosis and ascitis.

- In man; the young adults burrow through the liver tissue feeding on its cells causing inflammation, necrosis **(liver rot**) and marked eosinophilia.

- The other pathological findings are similar to *F. gigantica*.

It is similar to *Fasciola gigantica* but differs in:

| ***Fasciola gigantica*** | ***Fasciola hepatica*** |
|---|---|
| l) Size:3-7x 1cm. | -Smaller, 2-3x1.3 cm. |
| 2) Anterior cephalic cone: Is rather small. | -Relatively big. |
| 3) Lateral sides: parallel. | -Converging. |
| 4) Ventral sucker: bigger. | -Oral and ventral suckers are equal. |
| 5)Medial intestinal caeca :T or Y shaped. | - Rudimentary. |
| 6)Snail host : | |
| In Egypt: *Lymnea cailliaudi*. | -In Europe: *Lymnea truncatula*. |
| 7) Reservoir host: Herbivorous animals as cattle & buffalos. | - Sheep. |

**Diagnosis, treatment, prevention and control are similar to *F. gigantica*.**

## *Opisthorchis viverrini*
## (Southeast Asian liver fluke)

**Geographical distribution:** It is endemic in Southeast Asian countries including Thailand, Laos, Vietnam and Cambodia.

- Although opisthorchiasis is not endemic in Egypt, imported cases may occur.

**Morphology:**

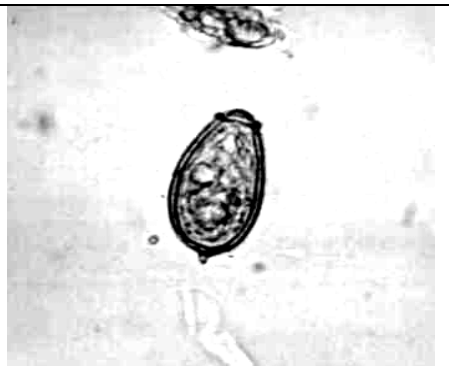
**1-Adult**

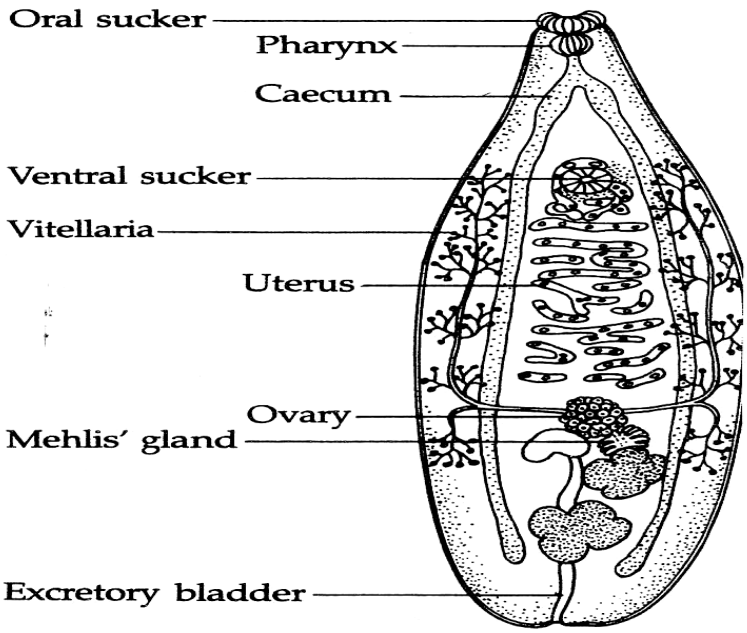
It is dorsoventrally flattened, lancet shaped, thin and transparent.

- Average size is 7 x 1.5 mm.
- Suckers: 2 suckers (oral sucker and the ventral sucker ).
- Testes: 2 in number, diagonal, deeply lobed, located near the posterior extremity.
- Ovary: single, multilobed in front of the anterior testes.
- The vitellaria consist of numerous follicles disposed in the lateral fields between ventral sucker and testes.

**2- Eggs :**

**Size** : 27x15µ
**Shape** : oval
**Shell** : thick
**Special character**: opercular shoulder and terminal knob
**Colour** : yellowish brown (bile stained)
**Contents** : mature (miracidium)






**Opithorchis adult morphology**

**3. Cercaria (pleurolophocercus):** formed of body and tail

- Body: have 2 suckers, primitive gut, 2 eye spots, 5 pairs of penetration glands**,** cystogenous glands and excretory bladder.
- Tail**:** simple, longer than the body, provided with cuticular sheath .

**Life cycle:**

**Habitat**: adult worms live in the biliary tract of humans and other reservoirs, attached to the mucosa by suckers.

**Definitive host: man**

**Intermediate host (require 2 intermediate hosts) :**

-**1st I. H**. : aquatic snails of genus **Bithynia**

- **2nd I.H**. : freshwater fish (**Cyprinoid fish**)

**Reservoir hosts**: dogs, cats and other fish eating mammals

**Infective stage**: encysted metacercaria in muscles of fresh water fish

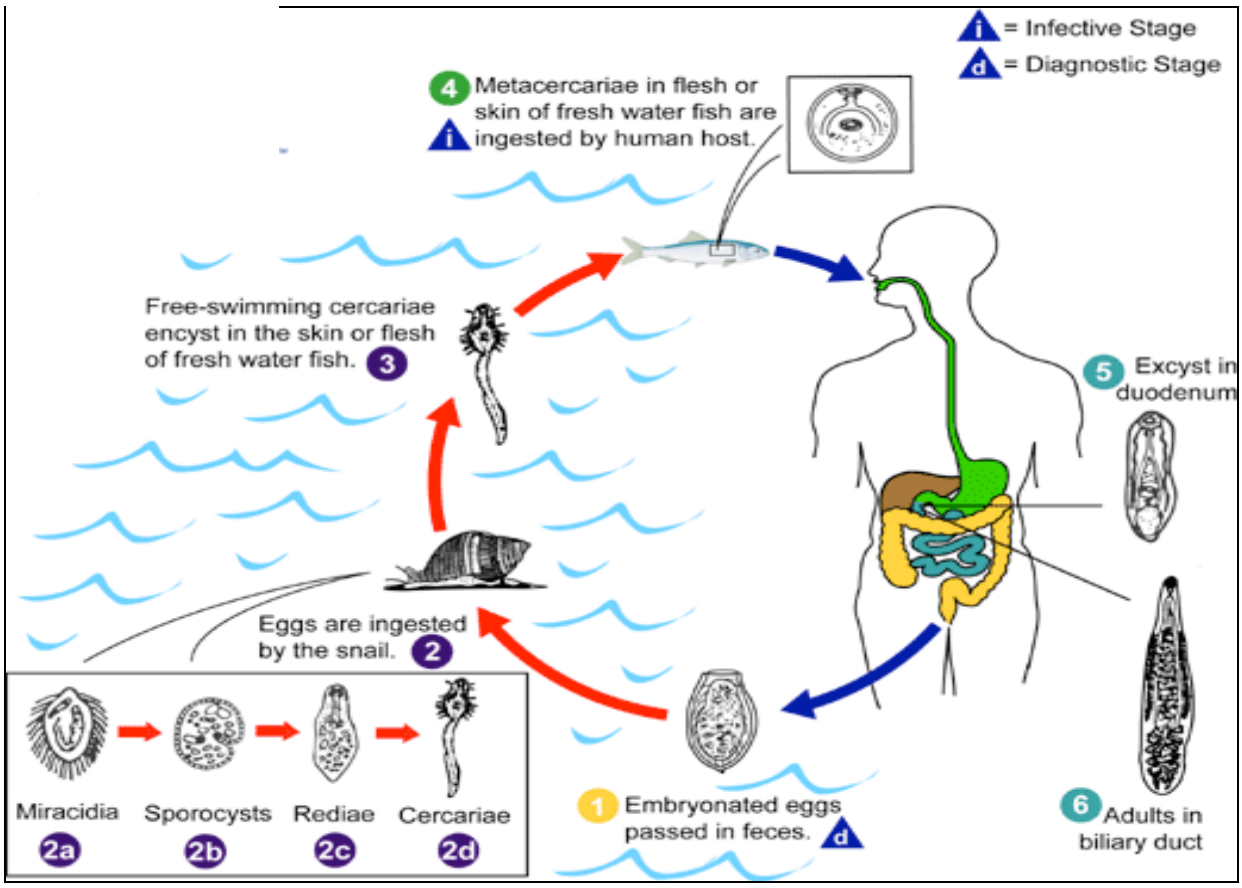
**Stages in life cycle**:

egg→ingested by the snail→miracidium→sporocysts→rediae→cercariae →encysted metacercaria in fish muscles →excysted metacercaria→adult

**Mode of infection:**

1- eating raw (dried or salted) or inadequately cooked freshwater fish.

2- infection can also occur through fingers or cooking utensils contaminated with the metacercaria during preparation of fish for cooking.




**life cycle of *Opisthorchis viverrini***

- Opisthorchis eggs are embryonated, pass out with faeces, ingested by the snail I.H. and hatch inside into miracidium which then develop into sporocysts then rediae stages to become cercariae.
- Cercariae then escape from the snail and swim in water waiting to get attached to the 2nd I. H., lose their tails and encyst under the scales or in the flesh of fish to become metacercariae in about 3 weeks.
-After ingestion of infected fish, metacercaria excyst in the duodenum, migrate through the **ampulla of vater** to the biliary tract, where they mature into adult in about 1 month.
- Eggs exit the bile ducts and are excreted in the feces.

**Pathogenesis and symptomatology:**

- The severity of the pathology appears to be associated with both intensity and duration of infection.

1- Most infections are asymptomatic.

2- Patients in the early stage have high fever, epigastric pain, diarrhea and tender hepatomegaly.

3- With chronic infection, the symptoms can be more severe, and hepatomegaly and malnutrition may be present.

4- In rare cases, cholangitis, cholecystitis, and **cholangiocarcinoma** (CCA) may develop.

**Opisthorchis viverrini is considered a group 1 carcinogen (known to cause cancer in humans).**

- The pathogenesis of O. viverrini-mediated hepatobiliary changes may be due to:
    1. Mechanical irritation caused by the liver fluke suckers
    2. Its metabolic products
    3. Immunopathological process

**Diagnosis**

**I. Clinical**

**II. laboratory:**

1. Detection of eggs in stool samples or aspirated bile.
2. Detection of fecal Opisthorchis antigen has been reported .
3. Several serologic tests for the diagnosis of opisthorchiasis have been reported.

**Treatment**:

1. Praziquantel is the drug of choice.
2. Surgical intervention may become necessary in cases with obstructive jaundice.

**Prevention and control**

Health education, avoid eating raw fish, proper cooking of fish, treatment of infected cases, snail control.

## Case study:

A 36-year-old man suffering from intermittent fever, diarrhea, indigestion and abdominal pain in the right hypochondrium. Upon examination, he had a slightly enlarged tender liver and yellow colouration of the sclera. When questioned regarding his eating habits, the patient admitted to having a fondness for un-cooked water-cress and raw vegetables. Stool examination for ova& parasites was ordered. Blood sample was collected for complete blood count and liver function tests. Haematology results showed evidence of anaemia and eosinophilia (60% eosinophils). The patient's liver enzyme levels were slightly elevated. The diagnosis was made microscopically after the observation of large, oval, 160X80 u, yellowish-brown, operculated eggs in the concentrated stool specimen.

**Questions:**

1. Which parasite might be causing this infection?
2. Which other helminth lays eggs indistinguishable from the eggs described in this specimen?
3. How does transmission of this parasite occur?
4. What are the usual symptoms of the disease in humans?
5. How the diagnosis of this infection is usually made?
6. How do you exclude false diagnosis?

# Intestinal flukes

## *Heterophyes heterophyes*

**Geographical distribution:** Common in Egypt in Nile Delta, especially around the lakes of Manzala and Borollos, Turkey and Far East (Japan, China, Korea, Philippine).

**Morphology:**

1. Size: 1.5-3mmx 0.5 mm.
2. Shape: Pear shape, the anterior end is more or less narrow, while the posterior end is broadly rounded, some spines cover the cuticle especially anteriorly.
3. Suckers: **three suckers**
   - Oral sucker: small around the mouth.
   - Ventral sucker: large about the middle of the body.
   - Genital sucker: postero- lateral to the ventral sucker.
4. Genital system:
   - Testes: two oval, smooth, and opposite each other in the posterior part of the body.
   - Ovary: One ovary, smooth in front of the testes.

**Life cycle:**

-Habitat: adult lives between the villi of the small intestine.

-Definitive host: man.

-Intermediate host: -first is a snail, called *Pirenella conica.*

-Second is fish, Tilapia (Bolty) and Mugil (Boury).

-Reservoir host: cat, dog, and any fish eating animals.

-Infective stage: encysted metacercaria in the muscles of the fish ($2^{nd}$ I. H.).

-Stages in the life cycle: egg → miracidium → sporocyst → 1st and 2nd generation redia → cercaria →encysted metacercaria → adult.

**Egg:** Size: 30xl5 μ.

Shape: Oval.

Shell: Thick with operculum at one pole and a small Knob at the other.

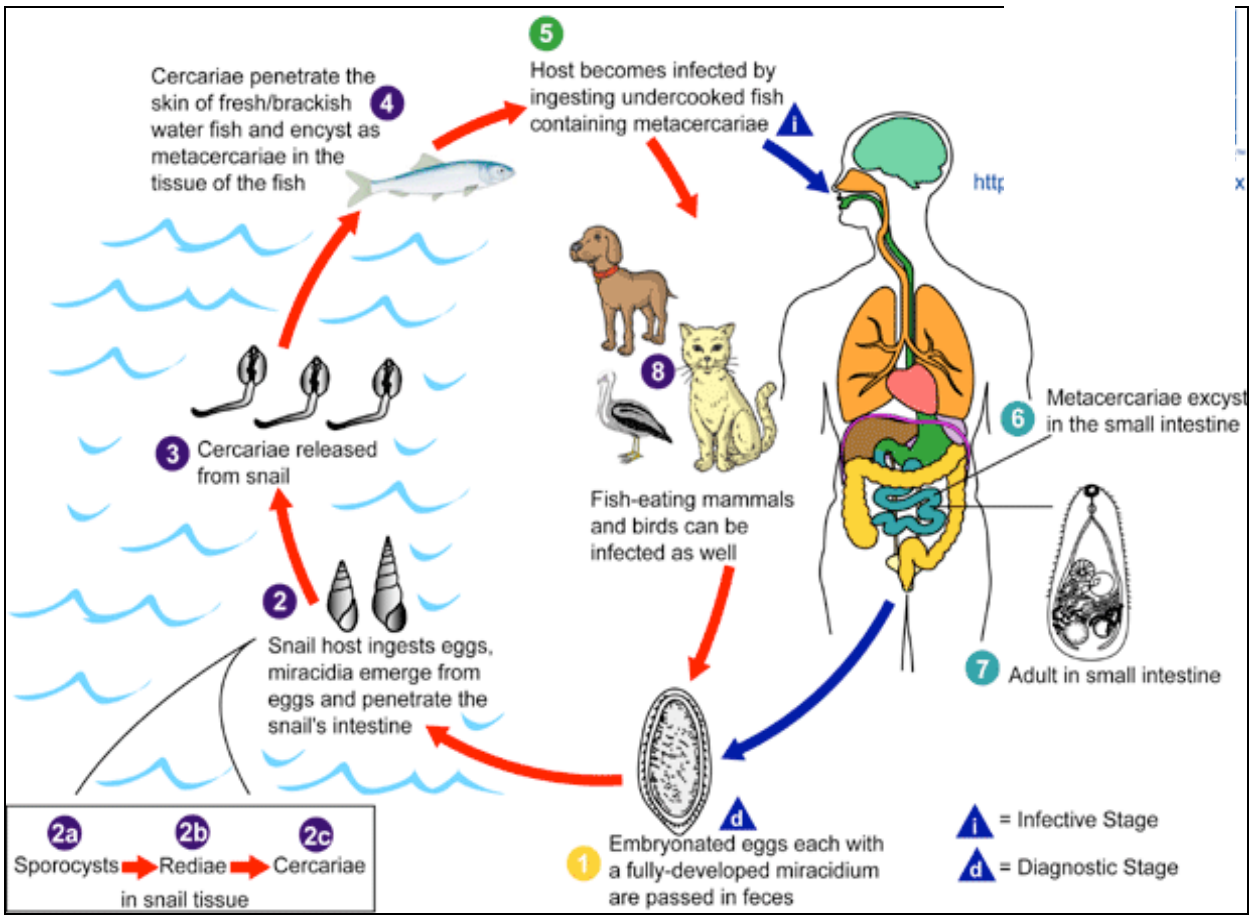
Colour: golden yellow.

Contents: Full mature embryo (miracidium).

-**Eggs pass with the stool, which must reach to brackish water**.

-In water, the eggs are ingested by the snail first intermediate host (*Pirenella conica)* common in lakes Manzala and Borollos in Egypt.

-In the snail, the miracidium hatches into a sporocyst, that gives rediae then cercariae, which escape from the snail in about 30 days and become free in water.




**Life cycle of *H. heterophyes***

**Cercaria**: **-** Consists of body and tail.

- The body**:** is oval, has 2 suckers, primitive gut, 2 dark eye spots and 7 pairs of penetration glands.
- The tail : is simple, covered from one side by membrane which reaches to the tip of the tail then ascend to cover the distal 1/3 of the other side, it is called **lophocercous cercaria** (pipe like).

- The cercaria in water searches for the second intermediate host, which is fish, *Tilapia* (Bolty) and *Mugil* (Boury).
- It penetrates the tissue of fish and becomes encysted metacercaria under the scales or in the muscles, and become infective within 20 days, it is spherical and 250 µ.

**Mode of infection**: By eating insufficient cooked, roasted or salted fish, staked less than ten days (sweet fesekh), containing the infective stage (encysted metacercariae).

-In the small intestine the cyst wall is dissolved, the metacercariae embedded between the villi, maturate and the eggs appear in the stool 2-5 weeks after infection.

**Pathogenicity and clinical picture:**

| | |
|---|---|
| **Intestinal** | Attachment of the parasite to the mucosal membranes; inflammation with superficial ulcers and necrosis occur.<br>*Mild infection with no symptoms<br>*Heavy infections cause:<br>-Abdominal colic. -Abdominal discomfort.<br>-Chronic intermittent diarrhea, sometimes with blood |
| **Extra-intestinal** | The eggs may reach the general circulation to different organs and form parasitic granuloma and fibrosis |

**Diagnosis**: Stool examination for the characteristic eggs.

**Treatment:** 1. Praziquantel (Biltricid).

2. Niclosamide (Yomesan).

**Prevention and control:**

1. Sanitary disposal of feces.
2. Avoid eating raw, insufficient cooked fish or salted fish, salted less than 10 days (sweet fessekh), and proper grilling of fish.
3. Fried fish is safe as temperature needed for frying is high enough to kill metacercariae.
4. Periodic examination of fishermen stool for *Heterophyes* eggs.
5. Mass treatment of infected cases.
6. Snail control.

## Lung fluke

## *Paragonimus westermani*

**Geographical distribution:** Heavily infected areas are found primarily in the Far East including Japan, Korea and Taiwan; it is also found in central Africa.

**Adult morphology:**

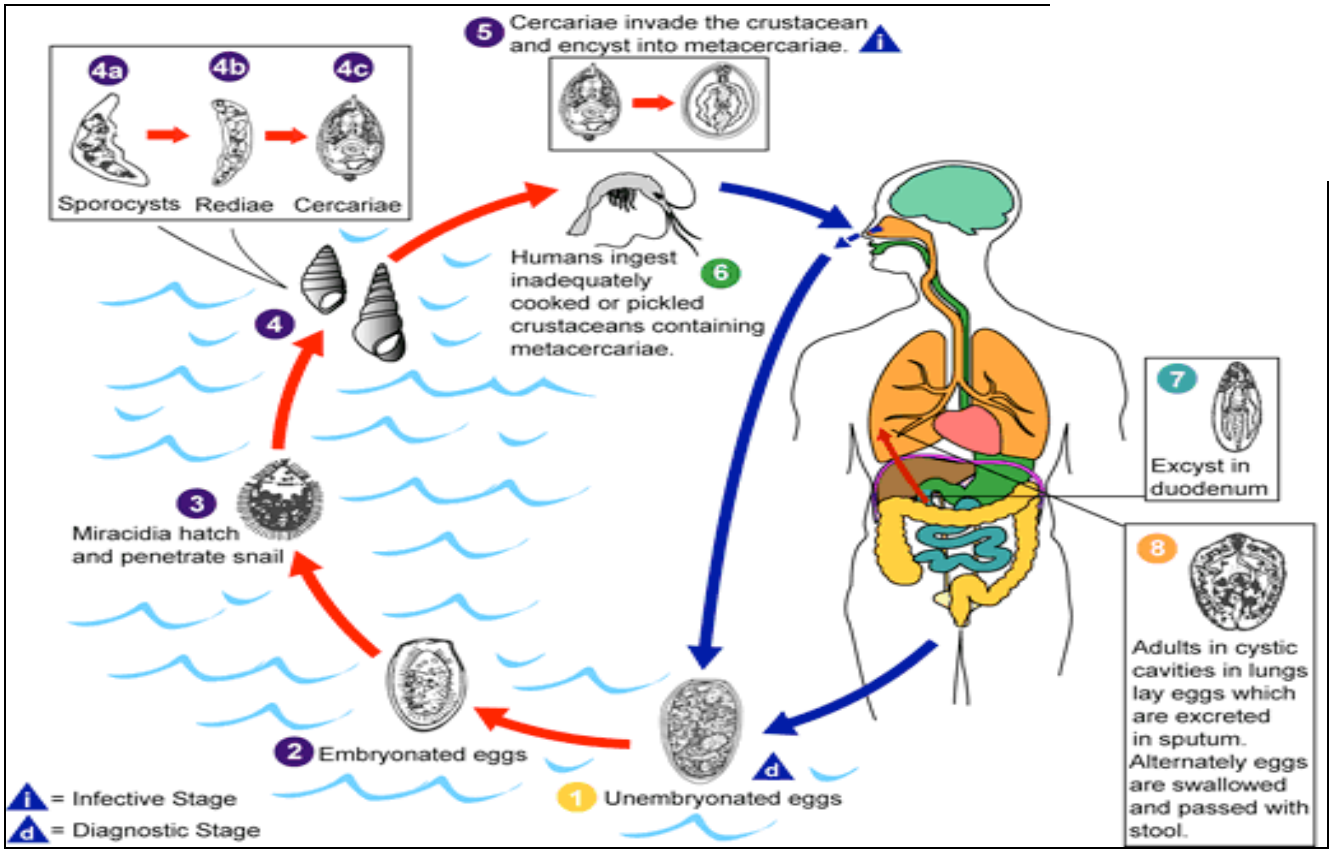
| | |
|---|---|
| **Shape** | Ovoid, thick, reddish brown, rounded anteriorly and tapering posteriorly. |
| **Size** | 0.5 - 1.5 cm in length |
| **Suckers** | the oral and ventral suckers are equal |
| **Genital system** | a) Testes: deeply lobed situated nearly side by side midway between the ventral sucker and posterior end.<br>b) Ovary: large, lobed and situated behind the ventral sucker, the uterus coils opposite to it |

**Life cycle:**

| | |
|---|---|
| **Habitat** | worms generally live in pairs encapsulated in pockets of the lungs |
| **Definitive host** | Man |
| **Intermediate hosts** | 1st I.H.→ snail *Melania& Semisulcospira.*<br>2nd I.H.→ crabs and crayfish |
| **Reservoir host** | Carnivores as dog, fox, wolf, tiger and pig |
| **Infective stage** | Encysted metacercaria in muscles, gills, legs and viscera of crabs and crayfish. |

**Stages in the life cycle**: egg → miracidium → sporocyst→ redia→ cercaria → metacercaria →adult.

• The life cycle is completed in 6-8 months.




***Paragonimus westermani* life cycle**

**Egg:**

| | | | |
|---|---|---|---|
| **Size** | 100 x 50 µ | **Color** | Golden brown |
| **Shape** | Ovoid | **Content** | Immature miracidium |
| **Shell** | Thick shell | **Special characters** | With flat operculum |

-The eggs escape from the pulmonary pockets through the bronchioles and are coughed out with sputum, or swallowed and pass immature with feces.

-Eggs require from 15 days to several weeks in water to complete embryonation then

hatch and miracidia escape.

**Miracidium:** enters the snail first I.H. then develops into sporocysts → rediae → cercariae in 3-5 months.

**Cercaria: Microcercous with a knob like tail**. The released cercariae penetrate the crustaceans 2nd I.H. then develop into metacercariae.

**Metacercaria**: requires 6-8 weeks to become infective.

**Mode of infection:**

- Human and mammals' infection occur by eating raw or insufficiently cooked crabs or crayfish infected with the encysted metacercariae.
- Metacercariae excyst in the small intestine pass through the intestinal wall, grow for about one week into young flukes, penetrate the diaphragm and pleural cavity and come to rest in the lung, forming cystic cavities then get mature.

**Pathogenicity and clinical picture:**

1. The worms provoke granulomatous reactions leading to fibrotic encapsulations of the parasites with a picture of generalized or localized diffuse fibrosis, pneumonia and tubercles like abscesses.
2. Clinically: The disease is insidious in onset.

   -There may be initial episodes of chills and fever with persistent cough and haemoptysis.

   -The sputum is viscous and flecked with dark golden brown particles and may be bloody.
3. Eosinophilia (20 - 25 %).
4. Pleural effusion may occur.

**Diagnosis:**

1. By finding the characteristic eggs in sputum, feces or in aspirated pleural effusion.
2. Plain x-ray chest and computerized tomography show nodular shadows *&* cavities.
3. Immunodiagnostic tests as ELISA to detect early & chronic infection.

**Treatment:** Praziquantel.

**Prevention and control:**

1. Avoid eating raw, inadequately cooked or freshly salted crabs or crayfish.
2. Treatment of cases.
3. Snail control.

# Blood flukes

# (Schistosomes)

Human beings are infected with three main species of schistosomes:

1. ***Schistosoma haematobium*:** causing urinary schistosomiasis. (present in Egypt)
2. ***Schistosoma mansoni*:** causing intestinal schistosomiasis. (present in Egypt)
3. ***Schistosoma japonicum*:** causing oriental schistosomiasis. (not present in Egypt)

## *Schistosoma haematobium*

## (Urinary schistosomiasis)

**Geographical distribution:**

Africa: scattered areas. In Egypt, it is prevalent all over the Nile Valley.

Asia: Syria, Palestine, Iraq, Iran, Saudi Arabia, Yemen, India.

Europe: Cyprus and South Portugal.

**Adult morphology:**

| | **Male** | **Female** |
|---|---|---|
| Size | 1 -2cm x l mm | long (2 - 2.5 cm x0.25mm) |
| Shape | flattened, lateral margins are folded ventrally to form the gynaecophoric canal | cylindrical (round in cross section) |
| Tegument | provided with fine tubercles on the dorsal surface | smooth. |
| Suckers | sub-terminal oral sucker around the mouth and a larger ventral sucker some distance behind | weakly developed |
| Digestive system | mouth, oesophagus without muscular pharynx, two simple intestinal caeca that unite in the middle into single blind caecum | like male but union of intestinal caeca occurs at the posterior third. |
| Genital system | **Testes** :4-5 separate testes, smooth, globular arranged in one line postero-lateral to the ventral sucker.<br>**Male genital pore** :behind the ventral sucker. | **Ovary** :oval, smooth lies just in front of the intestinal union.<br>Uterus: long, straight, terminates at the genital pore, contains one raw of 20 - 30 ova<br>**Vitelline glands**: extend from behind the ovary till the posterior end. |

**Life cycle:**

-Habitat: *Schistosoma haematobium* adults live in the vesical and pelvic venous plexuses in man surrounding the kidney, pelvis, urinary bladder, urethra, prostate, seminal vesicles, lower 1/3 of uterus and vagina.

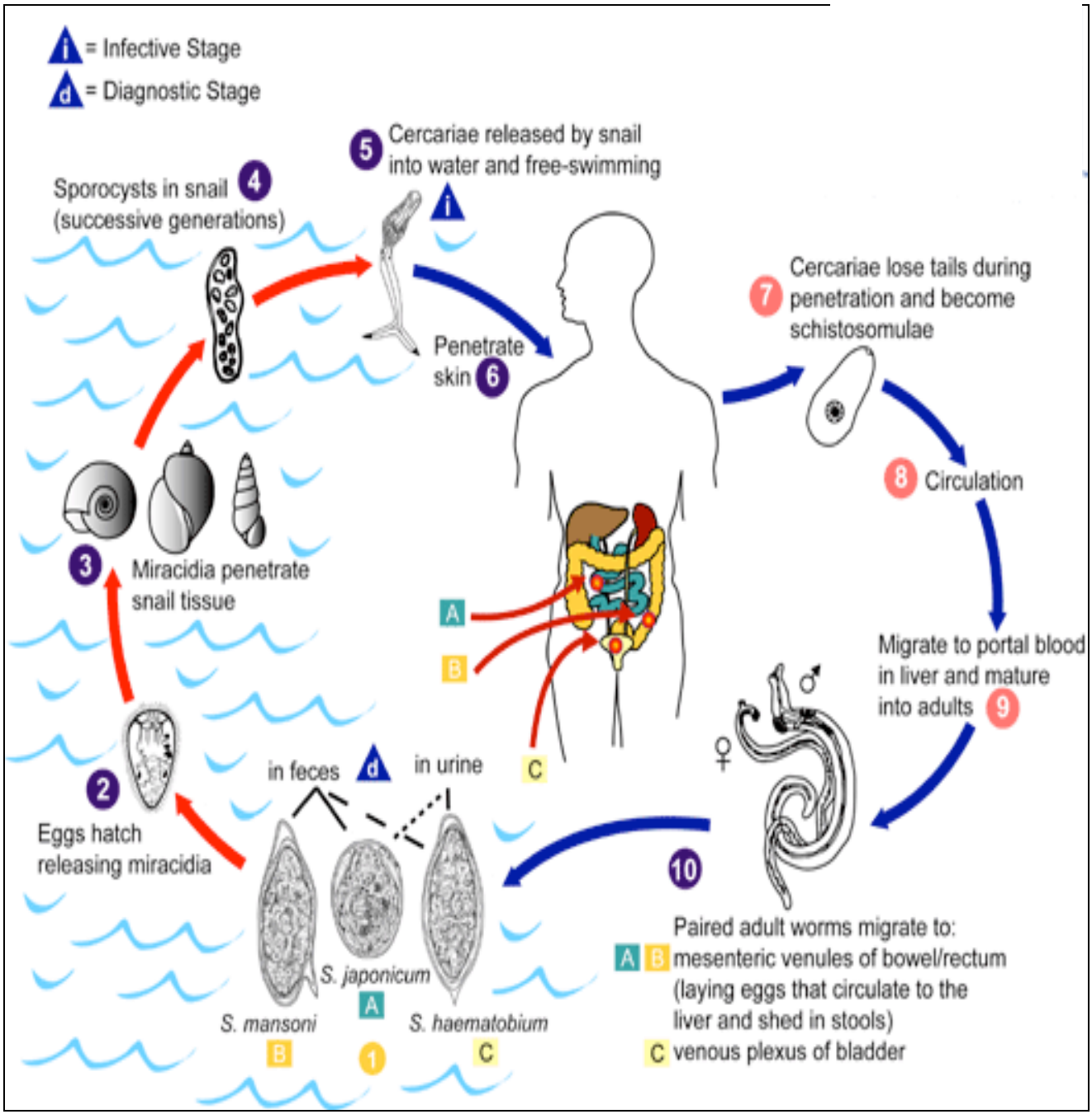
-Definitive host: man.

-Intermediate host: snail *Bulinus truncatus* in Egypt.

-Reservoir host: no reservoir host.

-Infective stage: **furcocercus cercaria**.

-Stages in life cycle: egg →miracidium→ sporocyst→ furcocercous cercaria→adult.




**Life cycle of *Schistosoma* species**

**Egg**: Eggs sweep out in urine and rarely with feces.

| | |
|---|---|
| Size | 120 x 60 μ. |
| Shape | oval. |
| Shell | thin with terminal spine. |
| Colour | translucent. |
| Contents | mature miracidium. |

**Miracidium:** -In fresh water the miracidium hatches.

- It is distributed homogeneously in water.
- It penetrates the soft tissue of the snail intermediate host **(*Bulinus truncates*)** where it develops into first and second generation sporocysts then cercariae that escape into water. Each miracidium gives rise to 250.000 cercariae.

**Cercaria:** (**furcocercus cercaria**)

a. Body: 200 u in length with 2 suckers, primitive gut and 5 pairs of penetration glands.

b. Tail: 300 μ in length, bifid or bi-forked **Forked cercaria is the infective stage**.

- The cycle inside the snail takes 1-2 months.

-It survives in canal water for 48 hours and is attracted to man by the body temperature.

**Mode of infection:**

1. Infection occurs by skin penetration within minutes up to half an hour as water begins to dry, after bathing, washing or playing in infected canals. Penetration is helped by the penetration glands and mechanically by the tail activity.
2. Drinking water may lead to infection when cercaria penetrates the mucous membrane above the gastric acidity that kills it.

- The body of cercaria enters the skin or mucous membrane leaving the tail (schistosomulum). It is carried by the blood → left side of the heart → systemic circulation → intestinal capillary bed → intra-hepatic branches of the portal vein where it matures in 7 weeks.
- Then male carries the female in the gynaecophoric canal and migrates out of the liver in the portal vein **against the blood stream** to reach the vesical and pelvic plexuses to deposit the eggs.
- Eggs appear in urine 10 weeks after infection.

**Pathogenicity and clinical picture:**

**Disease**: **schistosomiasis** haematobium, vesical or urinary bilharziasis:

There are four progressive stages:

| | |
|---|---|
| **Stage of invasion** | Skin reaction due to cercarial penetration in the form of local dermatitis, itching (**bather's itch),** irritation and papular rash. |
| **Stage of migration** | Due to circulating schistosomules<br>a- Lung: **verminous pneumonitis** (small patches of inflammation) & hemorrhage, with cough, sputum & heamoptysis.<br>b- Liver and spleen: **hepatosplenomegaly.**<br>c- Metabolic products of maturing parasites → toxic and allergic manifestations e.g. urticaria, fever, headache, cough, wheezes, muscle pain, leucocytosis & eosinophilia. |
| **Stage of egg deposition and extrusion (early-acute stage)** | Active egg deposition with escape of eggs in urine → tissue damage and hemorrhage that manifest with:<br>**a- Terminal haematuria** (blood in the last part of micturation) which is due to increased contraction of bladder → injury of venules by egg spine → drops of blood in urine.<br>**b-** Frequency of micturation.<br>**c-** Dysuria (burning pain during micturation). |
| **Stage of tissue proliferation, repair and fibrosis (chronic-late stage)** | -Eggs trapped in the wall of blood vessels stimulate both humoral and cellular immune response to miracidial antigen→aggregation of inflammatory cells around eggs (granulomas) and fibrosis with the formation of sandy patches, bilharzial nodules, papillomata which may ulcerate.<br>- Inflammatory reaction heals by fibrosis :<br>a- Urinary bladder: polyps, ulcers, cystitis, contracted bladder, calcified bladder, diverticulosis, malignancy (due to parasite toxic secretions).<br>b- Ureters: stricture, hydroureter.<br>c-Kidneys: hydronephrosis,2ry infection(pyonephrosis)&renal failure.<br>d-Urethra: stricture, fistula.<br>e-Genital organs: pseudo-elephantiasis of the penis, granuloma in prostate, seminal vesicle, spermatic cord, ovaries, uterus and vagina.<br>-**Embolic lesions**: *Schistosoma* eggs are swept by blood to reach various organs (liver, lungs, brain or other organs).<br>Eggs swept from the pelvic and vesical plexuses to→ the pulmonary artery branches produce granulomata and fibrosis with obliteration of blood flow → pulmonary hypertension, right ventricular hypertrophy and right sided heart failure **(Bilharzial cor-Pulmonale).** |

**Diagnosis:**

I. **Clinical**: history of terminal haematuria & dysuria in endemic area is suggestive. In mild infection, haematuria manifests only after muscular activity. Infection of seminal vesicle manifests by blood in seminal fluid.

II. **Laboratory**:

**a. Direct methods**:

1. **Detection of eggs in urine**: microscopical examination of last drop of urine sample for the eggs after sedimentation or centrifugation (concentration method).
2. **Cystoscopy**: in chronic cases when eggs cannot be detected in urine, for histopathological lesions as well as eggs.

*Eggs should be examined for viability by the **hatching test** to differentiate between living and dead eggs: fresh water is added to the urine sediment and examined after 30 minutes by a hand lens to demonstrate swimming miracidia.

| Living egg | Dead egg |
|---|---|
| Translucent | Opaque |
| Intact moving miracidium Contracting and relaxing. | Dead miracidium (not motile or silent) |
| Surrounded by R.B.Cs. | No R.B.Cs |
| Hatches in fresh water (Positive hatching test) | Does not hatch (Negative hatching test) |

3. **Urine precipitin test**: for detection of schistosomal antigens excreted in patient's urine. Hyper immune serum + patient's urine → precipitate forms in +ve cases.
4. **ELISA** for circulating antigens.

**b- Indirect methods:** Serological tests for detection of antigen antibody reaction.

1. Indirect haemagglutination test (IHA).
2. Indirect fluorescent antibody test (IFA).
3. Enzyme-linked immunosorbent assay (ELISA): for antibodies detection.

**Treatment:**

1. Praziquantel (Biltricide or Distocide).
2. Metrifonate (Bilarcil).

## *Schistosoma mansoni*

## (Intestinal schistosomiasis)

**Geographical distribution:**

It is widespread in Africa.

In Egypt, it was prevalent in the region of the Nile delta, but after construction of the high dam, it invaded upper Egypt.

Also *S. mansoni* is found in Saudi Arabia, Yemen and Tropical America.

**Morphology**: similar to *S. haematobium* with few differences listed below:

1. **Male:** -size: shorter, 8-10mm x l mm.

   -Cuticle: more coarsely tuberculated.

   -Digestive system: intestinal caeca unite at the anterior third.

   -Testes: 6-9 as a mass.

2. **Female:-**size: shorter, 14-22mm x0.15mm.

   -Digestive system: union of intestinal caeca occurs at the anterior third.

   -Ovary: at the anterior third.

   -Uterus: short with 1-4 ova.

3. **Egg:** size: 140 x 70 µ.

   Shape: oval.

   Shell: thin with lateral spine.

   Colour: translucent.

   Contents: mature miracidium.

   Eggs sweep out with feces and rarely in urine.

4. **Miracidium:** found in the upper layer of water, has fused penetration glands.

5. **Cercariae:** provided with 6 pairs of unicellular penetration glands.

**Life cycle:**

-Habitat: radicals of the inferior mesenteric vein draining the large intestine, and in the portal system.

-Definitive host: man.

-Intermediate host: snail *Biomphalaria alexandrina* in Egypt.

-Reservoir host: monkeys and rodents.

**Pathogenicity and clinical picture:**

**Disease:** schistosomiasis mansoni, intestinal bilharziasis.

Pathogenicity of ***Schistosoma mansoni*** is similar to that of *Schistosoma haematobium* with the following variations:

-**Stage of egg deposition and extrusion** (early or acute stage):

Active egg deposition especially in the pelvic colon and rectum leads to erosion of submucosa and villous tissue followed by inflammation, tissue damage and hemorrhage. The patient suffers from:

a. Dysentery with mucous and blood in the stool.
b. Abdominal pain.
c. Frequent stool.

**-Stage of tissue proliferation, repair and fibrosis** (chronic or late stage):

a. Eggs trapped in the wall lead to formation of sandy patches, nodules and papillomata. The wall becomes thickened, fibrosed and may be complicated with strictures, sinuses, fistulae and prolapse.
b. **Embolic lesions**: female *S. mansoni* produces about 300 eggs / day. 50% are swept by blood and reach the liver. They block the presinusoisal capillaries and the soluble egg antigen (SEA) elicits T-cell dependent granulomas around each egg → periportal fibrosis and portal hypertension → splenomegaly, ascitis and oesophageal varices due to opening of the porto-systemic shunts at the cardiac end of the oesophagus.
c. **Haematemesis**: due to ruptured oesophageal varices.
d. **Melena**: digested blood after ruptured oesophageal varices.
e. Eggs directed to the lungs by collateral circulation lead to **cor-Pulmonale.**
f. Renal involvement occurs due to precipitation of immune complexes in the glomerular vascular bed leading to **end-stage renal failure**.

**Diagnosis**:

**I. Clinical:**

a- Early: diarrhea and dysentery with mucus and blood in stool.
b- Late:

- Anal fissures and perianal sinuses.
- Bilharzial hepatic fibrosis causing:
  - Portal hypertension.
  - Splenomegaly &ascitis.

- Hepatic dysfunction.

- Portal hypertension, haematemesis and melena.
- Blood loss, leading to iron deficiency anaemia.

**II**. **Laboratory:**

**a. Direct methods:**

1. Stool examination: detection of the characteristic eggs in stool (lateral spine).
2. Rectal swab using a gloved finger lubricated with soap to palpate the pathological lesion in the rectum and the fecal sample are then examined on a slide for *Schistosoma mansoni* eggs.
3. Sigmoidoscopy and rectal biopsy to visualize the mucosa of sigmoid colon for pathological lesions and *Schistosoma* eggs.
4. ELISA for circulating antigens.

**b. Indirect methods**: as schistosomiasis haematobium.

**Treatment:**

1. Praziquantel: a single oral dose is effective against all *Schistosoma* species infecting man.
2. Oxamniquine (Vansil).
3. Chemotherapy followed by surgical interference in portal hypertension.

N.B.: urine and stool examination should be done after 3 months of treatment for *Schistosoma* eggs. The viability test should be done to decide whether the patient is cured or not.

## *Schistosoma japonicum*

## (Oriental schistosomiasis)

**Geographical distribution:** in Far East (Japan, China, Philippines, Formosa, Korea).

**Morphology:** similar to other Schistosomes with few differences.

**1. Male:**

-Size: 9-22 mm x 0.5 mm.
-Cuticle: smooth.
-Intestinal caeca unite very late posteriorly.
-Testes: 6-8 small testicles in a single column.

**2. Female:**

-Size: 12-26 mm x 0.3 mm.
-Intestinal caeca unite at the posterior two fifths.
-Ovary: posterior.
-Uterine tube: long, contain 50 - 100 eggs.

**3. Egg:** sweep out with feces.

| | |
|---|---|
| Shape | rounded. |
| Size | 70-100x50 µ. |
| *Shell* | *thin, spineless but has curved tubercle-like projection in shallow depression* |
| Colour | translucent. |
| Contents | mature miracidium. |

**4. Miracidium: similar to *S. mansoni* miracidium.**

**5. Cercaria:** provided with 5 pairs of penetration glands.

**Life cycle:**

-Habitat: *Schistosoma japonicum* lives in mesenteric veins of large & small intestine.

-Definitive host: man.

-Reservoir hosts: cats, dogs, cattle, horses, pigs, rodents, sheep and goats.

-Intermediate host: snail *Oncomelania.*

**Pathogenicity:**

**It causes oriental schistosomiasis**.

The disease caused by ***Schistosoma japonicum*** is similar to that caused by ***Schistosoma mansoni*** with the following differences:

- The mesenteric lymph nodes are affected. Adhesions and thickening of mesentery and omentum occur.

- Liver fibrosis and splenomegaly are common; the condition is called "**Katayama Syndrome".**

**The pathogenesis is more dangerous than other schistosomes** due to:

1. It inhabits the small intestine, affecting the absorption process, leading to retarded growth in children.

2. Many reservoir hosts makes control difficult.

3. Intermediate host is amphibian (lives in mud for long time).

4. Female lays 3000 eggs/day, with high metabolism and more antigens excreted.

4. Absence of a spine and the rounded contour of the eggs allow their dispersion in the body with CNS and spinal cord affection.

**Clinical picture:** The clinical manifestations of **schistosomiasis japonica** are similar to that of **schistosomiasis mansoni** (intestinal schistosomiasis) but:

1. The incubation period is short.
2. The disease is serious and fatal.
3. Diarrhea is common due to the pathological lesions of the small intestine.

4. **Katayama disease** is more sever and is characterized by :
   a. Acute onset of fever, headache, weakness, cough, abdominal pain, diarrhea, eosinophilia (40%), hepato-splenomegaly and involvement of the CNS (2-3%).
   b. Symptoms of katayama disease coincide with the maturation of the adult worms and beginning of oviposition i.e. 5-7 weeks after infection.
   c. The disease is frequently seen in heavy infection with ***S. mansoni*** and rarely occurs in ***S. haematobium*** infection.

**Diagnosis:**

1. Stool examination for the characteristic eggs**.**
2. Detection of circulating antigens and antibodies by different immuno-diagnostic assays.

**Treatment:**

**- *Schistosoma japonicum*** is resistant to treatment.

-Praziquantel is the only effective drug for this infection.

## *Schistosoma intercalatum*

- Common in central and West Africa.
- Adults are found in intestinal venous plexuses of man.
- Intermediate host: snail *Bulinus africanus.*
- **Eggs** : 170× 60µ
  -With terminal spine as Schistosoma haematobium eggs and **are Zeihl-Neelsen positive.**
  -Eggs are detected in stool.
- Usually cause benign disease and hepatomegaly is not marked.

**Control: (for all species of schistosomes)**

**(1) Protection (prophylaxis from infection)**:

| | |
|---|---|
| A) Health education | -should be directed mainly for school children.<br>-prevention of urination or defecation in or near water streams. |
| B) Personal prophylaxis | By wearing of boots and gloves, use of repellents e.g. dimethyl phthalate. |
| C) Quick and thorough drying of exposed wet skin | |
| D) Pure water supply and Sanitary disposal of excreta | |
| E) Treatment of canals water to become safe | e.g. storage more than 48 hours, boiling or addition of chemicals like chlorine to render it free from living cercariae. |

**(2) Treatment of cases:** mass treatment by Praziquantel.

**(3) Reservoir hosts:** should be controlled.

**(4) Snail control:**

| | |
|---|---|
| **a) Physical methods** | - Dryness of canals e.g. double canalization system (one used and one dried).<br>- Periodic clearance of canals from vegetations and weeds.<br>- Pitching canal banks with concrete to prevent growth of aquatic plants.<br>- Wire screens at inlets of canals to prevent & collect snails |
| **b)Biological methods** | - Natural enemies of snails e.g. birds (ducks, geese) or snails (*Marisa*).<br>- Certain toxic plants e.g. *Balanites aegyptica* and *Ambrosa maritima (Damsisa)* whose leaves and fruits are toxic to snails.<br>- Pathogens**:** infection of snails by miracidia of avian schistosomiasis to decrease its vitality. |
| **c)Chemical methods** | chemical substance used to kill snails (**molluscicides**)<br>- **Copper sulphate** : 10-20 p.p.m., widely used in Egypt, but it is not effective against eggs of snails, should be reapplied every 3 months to kill the newly hatched snails or applied as a chemical barrier at the inlet of canal to give a concentration of 0.5 p.p.m.<br>- **Sodium pentachlorophenate** : 5- 10 p.p.m.<br>- **Baylucide:** 2 p.p.m. |

## Cercarial dermatitis
## Swimmer's itch, Bather's itch

- It is caused by cercariae of non-human schistosomes (**bird and animal schistosomes**) in fresh water or marine water.
- Cercariae penetrate the human skin, but cannot proceed beyond the germinal layer, and rapidly destroyed in the skin by the host defense mechanism.

| | |
|---|---|
| **Clinically** | - Dermatitis,itching, urticarial wheels and 2ry bacterial infection.<br>- Formation of macules & papules occur by subsequent exposures |
| **Diagnosis** | History of contact with water followed by skin rash |
| **Treatment** | - Local antihistamines, antipruritis, antibiotics for 2ry bacterial infection. |
| **Prevention and control** | - Rapid drying of the skin to prevent cercarial penetration.<br>- Snail control. -Avoid polluted water |

## Human schistosomes

| | ***S. haematobium*** | ***S. mansoni*** | ***S. japonicum*** |
|---|---|---|---|
| **Male:** Length | 1-2 cm | 0.8-1 cm | 0.9-2.2 cm |
| Breadth | 1 mm | 1 mm | 0.5 mm |
| Surface | Fine | Coarse | Non tuberculated (smooth) |
| Union of intestinal caeca | At the middle | At the anterior third | Very late posteriorly |
| Testes | 4-5 separate | 6-9 as a mass | 6-8 compressed |
| **Female:** Length | 2-2.5 cm | 1.4-2.2 cm | 1.2-2.6 cm |
| Breadth | 0.25 mm | 0.15 mm | 0.3 mm |
| Union of intestinal caeca | At posterior third | At anterior third | At posterior two fifths |
| Ovary position | Posterior | Anterior | Posterior |
| Uterine tube | Long, contains 20-30 eggs | Short, contains few eggs | Long, contain 100 eggs |
| Vitelline glands | Occupy the posterior third | Occupy the posterior two third | Occupy the posterior two fifths |
| **Egg** : Size | 120 × 60 $\mu$ | 140 × 70 $\mu$ | 100 × 50 $\mu$ |
| Spine | Terminal | Lateral | Lateral, short, curved tubercle |
| Exit | Urine, rarely feces | Feces, rarely urine | Feces |
| **Miracidium** | Found in all levels of water | Found in the upper layer of water. | As *S. mansoni* |
| **Cercaria** | 5 pairs of penetration glands | 6 pairs | 5 pairs |
| **I.H.** | *Bulinus truncatus* | *Biomphalaria alexandrina* | *Onchomelania* |
| **R.H.** | No | Monkeys & rodents | Horses, cattle, dogs, cats and rodents |

**Case study:**

A 13-year-old male, from a village near Mansoura, presented to the Out-Patient Clinic of Mansoura University Hospital with complaints of painful urination, the presence of blood in his urine, fatigue, fever and general body aches. Upon examination, the physician ordered a urine analysis and urine culture to rule out a urinary tract infection.

Culture results were negative for pathogenic bacteria. Microscopic examination of the urine sediment revealed proteinuria, many RBCs (haematuria) and few white blood cells. Oval, translucent eggs with prominent terminal spines were also detected.

**Questions:**

1. Which parasite is the cause of this patient's infection?
2. How is this infection transmitted?
3. Mention the complications of this parasitic infection.
4. Describe the detected egg.
5. Compare this egg with those of other members of this genus.
6. Which types of specimens should be collected for diagnosis?
7. How is this infection diagnosed?
8. Describe the "hatching test" and mention its value.
9. What is the association of this infection with bladder cancer?
10. How is this infection treated?
11. How is infection with this parasite prevented and controlled?

**Case study :**

A 22-year-old Egyptian woman was visiting American relatives and developed fever, malaise, dysentery and abdominal pain. Her relatives brought her to the family doctor for examination. Upon examination, she was noted to have liver tenderness. Blood was drawn for complete blood count and liver enzyme analysis. Three stool specimens were submitted for examination for ova and parasites.

The patient was noted to be mildly anaemic and had slightly elevated liver enzyme levels. Two of the 3 stool specimens revealed a small number of eggs. Each is oval, translucent, 140X70 p with a prominent lateral spine.

**Questions :**

1) Which parasite is causing this patient's infection?
2) Give an account on Katayama syndrome?
3) What are the possible complications of this parasitic infection?
4) How is this infection diagnosed?
5) Do bird species in this genus cause human disease?
6) How is this infection treated?

# Snails (Mollusca)

- Snails may be aquatic, amphibians or land snail.
- Their life span ranges from few months up to 2 years.

**The snail consists of:**

| | |
|---|---|
| **(1) Shell:** the hard part of the snail, and each shell composed of: | **- Whorls** (segments): separated by sutures (grooves).<br>**- Apex**: the top of the snail.<br>**- Body whorl**: the big whorl above the aperture<br>**- Siphon:** the outside eversion of the lower margin of snail.<br>**- Umbilicus:** the margin of the opening may be inverted towards the body forming a groove called umbilicus.<br>**The aperture:** may be right-sided (dextral) or left-sided (sinistral). |
| (**2) Body** | It is the soft part of the snail, which gets its shelter inside the snail. It is composed of head, foot and viscera |

**To identify a snail the following points should be considered:**

**1.** Apex: whether sharp or blunt.

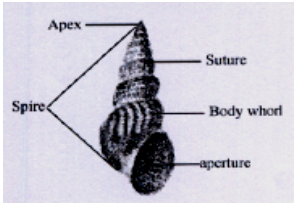
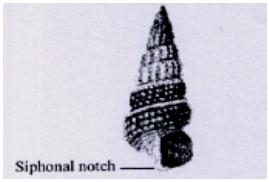
**2.** Aperture: whether directed to the right side (dextral) or to the left sides (sinistral).

**3.** Shell: its shape, color, presence of tubercles & the depth of the sutures.

## Classification of some Egyptian snails:

### I- Dextral (right- sided snails)

### 1. Snails with conical spire (conical):

| ***Melania tuberculata*** | ***Pirenella conica*** |
|---|---|
|  <br>No medical importance. |  <br>1st I.H. of *H. heterophyes*. Resembles *Melania* except in: |
| 1- Dextral, long spire<br>2- Shell ornamented with fine tubercles arranged in numerous rows.<br>3- No siphonal notch.<br>4- Sharp apex.<br>5- Oblique whorls. | 1- Whorls less oblique provided with coarse tubercles arranged in 1-4 rows.<br>2- The presence of siphonal notch. |

**2.Snails with medium spire:** all are of no medical importance

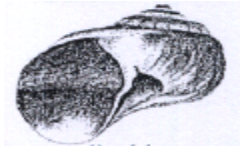
| ***Cleopatra bulimoides*** | ***Cleopatra cyclostomoides*** | ***Vivipara unicolor*** |
|---|---|---|
|  |  |  |

**3. Fragile snails:**

| ***Lymnea cailliaudi***  I.H. of *Fasciola gigantica* | ***Lymnea truncatula***  I.H. of *Fasciola hepatica* |
|---|---|
| - Dextral, prominent spire.<br>- Thin fragile shell.<br>-Oval aperture half the length of the snail.<br>-Large body whorl (first whorl above the aperture). | - Resembles *L. cailliaudi* except in smaller size. |

## II - Sinistral (left - sided snails)

| ***Bulinus truncatus***  I.H. of *Schistosoma haematobium* | ***Physa acuta***  No medical importance |
|---|---|
| -Sinistral, short spire.<br>-Small or medium sized.<br>-Blunt apex and shoulder.<br>-Deep sutures.<br>-Comparatively large body whorl. | - Resembles *B. truncatus* except in:<br>- Sharp apex.<br>- Sutures less deep. |

| ***Biomphalaria alexandrina***  I.H of *Schistosoma mansoni* | ***Lanistes bolteni***  No medical importance |
|---|---|
| -Sinistral.<br>-Discoidal shell (button shape).<br>-Flat upper surface. | -Large with short spire.<br>-Blunt apex.<br>-Large umbilicus. |