بِسْمِ اللَّهِ الرَّحْمَانِ الرَّحِيمِ
Schistosomiasis or Bilharziasis

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Introduction

• Schistosomiasis, also known as bilharziasis, snail fever, and Katayama fever.

• It is a major parasitic disease in the world, with about 237 million infections.

• It is caused by parasitic worms *Schistosoma*.

• The disease is most commonly found in Africa, Asia and South America.
Human beings are infected with 3 major species of schistosomes:

- *Schistosoma haematobium*: causing urinary schistosomiasis.
- *Schistosoma mansoni*: causing intestinal schistosomiasis.
- *Schistosoma japonicum*: causing oriental schistosomiasis.
Blood Flukes
Schistosomes
(Schist = split, soma = body)

Blood flukes differ from other trematodes in:

• Separate sexes.
• Parasitic in vascular system.
• Female *cylindrical*, longer than male and carried in its *gynaecophoric canal*.
• Testes are more than 2.
- No pharynx.
- Intestinal caeca reunite to form a single caecum.

- **Eggs are not operculated and are provided with a spine or a knob.**

- No redia stage.
- **Forked cercaria is the infective stage.**
Urinary schistosomiasis

*Schistosoma haematobium*
Overview

- *Schistosoma haematobium* inhabits the vesical and pelvic venous plexuses.
- Infection is by direct skin contact with water contaminated by cercariae.
- Egg granuloma is the main pathologic lesion.
- Squamous cell bladder carcinoma is a major sequelae of urinary schistosomiasis.
Schistosoma haematobium

Geographical distribution:

• **Africa**: scattered areas & **in Egypt it is prevalent all over the Nile Valley**.

• **Asia**: Syria, Palestine, Iraq, Iran, Saudia Arabia, Yemen, India.
Geographical distribution of *Schistosoma haematobium*
**Adult morphology:**

**Male:**

- **Size:** 1 – 2 cm x 1 mm.
- **Shape:** flat, margins are folded to form gynaecophoric canal.
- **Cuticle:** fine tubercles on the dorsal surface.
- **Suckers:** oral sucker and a larger ventral sucker.
- **Digestive system:** intestinal caeca unite in the posterior 1/2.
Testes: 4-5 separate large testes.
Female: deposits 20-200 eggs/day.
Size: 2 - 2.5 cm × 0.25 mm.
Shape: cylindrical.
Cuticle: smooth.
Digestive system: intestinal caeca unite in the posterior 1/3.
Ovary: oval, in front of intestinal union.
Uterus: long, contains one row of 20 – 30 ova.
Vitelline glands: extend from behind the ovary.
*S. haematobium* female

*S. haematobium* male

*S. haematobium* couple
**Egg:**

- Size: $120 \times 60\mu$.  
- Shape: oval.  
- Shell: thin with *terminal spine*.  
- Colour: *translucent*.  
- Contents: *mature miracidium*.  
- Eggs sweep out with urine and rarely feces.
Miracidium:

- In fresh water, it can survive for 24 h.
- It is distributed homogenously in water.
Cercaria:

- **Body:** 200 µ, with 2 suckers, primitive gut and 5 pairs of penetration glands.
- **Tail:** 300 µ, biforked (Furcocercous cercaria).
- **Cercaria survives in canal water for 48 hours.**
**Life cycle:**

**Habitat:** vesical and pelvic venous plexuses.

**Definitive host:** man.

**Intermediate host:** snail *Bulinus truncatus*.

**Reservoir host:** no reservoir host.

**Infective stage:** furcocercus cercariae.

**Stages in the life cycle:** egg→ miracidium→ sporocyst→ furcocercous cercariae→ adult.
Mode of infection:

1) Skin penetration by cercaria, after bathing, washing or playing in infected canals.

2) Drinking water, when cercaria penetrates the mucous membrane above the gastric acidity.
Life cycle of schistosomes

1. Infective Stage
2. Eggs hatch releasing miracidia
3. Miracidia penetrate snail tissue
4. Sporocysts in snail (successive generations)
5. Cercariae released by snail into water and free-swimming
6. Penetrate skin
7. Cercariae lose tails during penetration and become schistosomulae
8. Circulation
9. Migrate to portal blood in liver and mature into adults
10. Paired adult worms migrate to:
    - Mesenteric venules of bowel/rectum (laying eggs that circulate to the liver and shed in stools)
    - Venous plexus of bladder

S. mansoni
S. japonicum
S. haematobium
Cycle of schistosomes inside the human body

1. Cercariae penetrates human skin
2. Schistosomula
3. Venous circulation
4. Vesical plexus
5. Inf. mesenteric plexus
6. S. haematobium
7. S. mansoni
8. Portal circulation
9. Systemic circulation
10. Liver
11. Aorta

Direction of venous blood flow
-Cercaria enters the skin or mucous membrane → **schistosomulum**.

-It is carried after 2 days by blood → Rt side of heart → lung → Lt side of heart → systemic circulation → mesentric-portal vessels → **intrahepatic branches of portal vein** → matures in 7 wk.
- Male carries female → migrate out of portal vein → vesical & pelvic plexuses → deposits eggs on endothelial lining of venous capillary walls.

- Eggs appear in urine 10 wk after infection.

- In fresh water, miracidium hatches → penetrates soft tissues of snail I.H. → sporocyst → cercariae → escape into water.

- Miracidium → 250,000 male or female cercariae.
Pathogenesis and clinical picture:

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

- There are four progressive stages:

1- **Stage of invasion:**

- Cercarial penetration of skin → local dermatitis, itching, irritation and papular rash.

![Image of cercaria penetration](image1.png)

![Image of papular rash](image2.png)
2-Stage of migration: by circulating schistosomula

a. **Lung**: verminous pneumonitis, haemorrhage, with cough, sputum & haemoptysis.

b. **Liver and spleen**: hepatosplenomegaly.

c. **Metabolic products** → toxic and allergic manifestations e.g. urticaria, fever, headache, cough, muscle pain, leucocytosis & eosinophilia.
3-Stage of egg deposition and extrusion (early or acute stage):

- Active egg deposition with escape of eggs in urine → tissue damage and haemorrhage.
  
a. Terminal haematuria.

b. Frequency of micturation.

c. Dysuria.

- Infection of seminal vesicle manifests by blood in seminal fluid.
4-Stage of tissue proliferation, repair and fibrosis (chronic or late stage):

- Trapped eggs stimulate immune response → **granulomas & fibrosis** with formation of sandy patches, bilharzial nodules & papillomata.

- Ulcerative lesions heal by fibrosis → hazards of fibrous tissue contraction.
a. **Urinary bladder**: polyps, ulcers, cystitis, contracted bladder, calcified bladder, diverticulosis, and **malignancy**.

b. **Ureters**: stricture, hydroureter.

c. **Kidneys**: hydronephrosis, pyonephrosis, and renal failure.

d. **Urethra**: stricture, and fistula.

e. **Genital organs**: pseudoelephantiasis of the penis, and granulomas formation.
f. **Embolic lesions:** eggs fail to be fixed to walls of venules are swept by blood → various organs → granuloma formation.

- Eggs swept from pelvic & vesicle plexuses → **pulmonary artery** → granuloma & fibrosis → obliteration of blood flow → pulmonary hypertension, Rt ventricular hypertrophy & Rt sided heart failure (**Bilharzial cor Pulmonale**).
Diagnosis:

1. Clinical:
- History of terminal haematuria & dysuria in endemic area is suggestive.

2. Laboratory:
   a. Direct methods:
      - Detection of eggs in urine: examination of last drops of urine sample.
      - Stool examination for eggs.
Eggs should be examined for viability by hatching test.

<table>
<thead>
<tr>
<th>Living egg</th>
<th>Dead egg</th>
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<tr>
<td>Translucent</td>
<td>Opaque</td>
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<tr>
<td>Intact moving miracidium</td>
<td>Dead miracidium (non motile or silent)</td>
</tr>
<tr>
<td>Contracting and relaxing.</td>
<td></td>
</tr>
<tr>
<td>Surrounded by R.B.Cs.</td>
<td>No R.B.Cs</td>
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<tr>
<td>Hatches in fresh water (Positive hatching test)</td>
<td>Does not hatch (Negative hatching test)</td>
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• **Cystoscopy:** in chronic cases.

• **Urine precipitin test:**
  - For detection of schistosomal antigens excreted in patient’s urine.
  - Hyperimmune serum + patient’s urine → precipitate in + ve cases.

• **ELISA:** for circulating antigens.
b. **Indirect methods:** for detection of antibody.

i. Indirect haemagglutination test (IHA).

ii. Indirect fluorescent antibody test (IFA).

iii. ELISA.

iv. Circum-oval precipitin test (COPT):
   - Clean viable eggs + patients serum.
   - In +ve cases precipitates appear around the egg.

d. Blood picture.
Treatment:
- Early stage: antihistaminics.
- **Praziquantel** (Biltricide or Distocide):
  - It is given orally as 40 mg/kg body weight, in 2 divided doses for one day.
- **Metrifonate** (Bilarcil).
Intestinal schistosomiasis

Schistosoma mansoni
Overview

- *Schistosoma mansoni* inhabits the inferior mesenteric vein and portal venous system.

- The basic pathological lesion is egg granuloma in liver & colon.

- Acute schistosomiasis: fever, enlarged & tender liver, dysentery and eosinophilia.

- Chronic schistosomiasis: asymptomatic intestinal or symptomatic hepatosplenic.
Schistosoma mansoni

Geographical distribution:

- **Africa**: Wide spread.
  - In Egypt it is prevalent in Nile Delta.

- **Asia**: Saudia Arabia and Yemen.
- **South America**.
• **Morphology:** similar to *S. haematobium* with few differences:

• **Male:**
  
  – Size: **shorter**, 8-10mm \(\times\) 1mm.
  
  – Cuticle: **coarse tubercules**.
  
  – Digestive system: intestinal caeca unite at the anterior third.
  
  – Testes: **6-9 as a mass**.
• Female: deposits 300 eggs/day.
  – Size: shorter, 14-22mm × 0.15mm.
  – Digestive system: union of intestinal caeca occurs at the anterior third.
  – Ovary: at the anterior third.
  – Uterus: short with 1-4 ova.
S. mansoni in copula
• Egg:
  - Shape: oval.
  - Size: 140 × 70µ.
  - Shell: thin with lateral spine.
  - Colour: translucent.
  - Contents: mature miracidium.
  - Eggs sweep out with feces and rarely in urine.
• **Miracidium**: in the upper layer of water, fused penetration glands.

• **Cercaria**: provided with 6 pairs of penetration glands.
**Life cycle:** the same as *S. haematobium* but:

- **Habitat:** radicals of inferior mesenteric vein, and portal system.

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

- **Reservoir host:** monkeys and rodents.
Life cycle of *Schistosoma mansoni*
Pathogenesis and clinical picture:

Disease: schistosomiasis mansoni, intestinal bilharziasis.

1-Stage of invasion: (as in S. haematobium).

2-Stage of migration: (as in S. haematobium).

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

- Egg deposition mainly in pelvic colon & rectum → erosion of submucosa and villous tissue → inflammation, tissue damage & haemorrhage.
The patient suffers from:
- Dysentery with mucous and blood in the stool.
- Abdominal pain.
- Frequent stool.

4-Stage of tissue proliferation, repair and fibrosis (chronic or late stage):
- **Eggs trapped:** in the intestinal wall → formation of sandy patches, nodules and papillomata.
- The wall becomes thickened fibrosed & complicated with strictures, sinuses & prolapse.
• **Embolic lesions:** 50% of eggs are swept by blood → liver → block presinusoidal capillaries.

- Egg antigen elicits T-cell dependent granulomas periportal → fibrosis & **portal hypertension** → splenomegaly, ascitis & oesophageal varices.
• Haematemesis.

• Melena.

• Eggs swept to pulmonary artery: by collateral circulation → pulmonary fibrosis & cor pulmonale.

• Renal envolement: due to precipitation of immune complexes in the glomerular vascular bed → end-stage renal failure.

• Iron deficiency anaemia.
Diagnosis:

1-Clinical:

- **Early:** diarrhea and dysentery with mucus and blood in stool.
- **Late:**
  - Anal fissures and perianal sinuses.
  - Bilharzial hepatic fibrosis manifestations.
2-Laboratory:

a. Direct methods:

i. **Stool examination:** detection of the characteristic eggs in stool or rarely in urine.

ii. **Rectal swab:** to palpate pathological lesion in rectum & fecal sample is then examined for eggs.
iii. **Sigmoidoscopy and rectal biopsy** from mucosa of sigmoid colon to see pathological lesions and *Schistosoma* eggs.

iv. **ELISA** for circulating antigens.

**b. Indirect methods:** as schistosomiasis haematobium (IHA, IFAT, ELISA, COPT).

c. **Radiology:** Ultrasound, CT and X-ray.

d. **Blood picture.**
Treatment:

• Praziquantel: a single oral dose is effective against all *Schistosoma* species infecting man.

• Oxamniquine (Vansil).

• Chemotherapy followed by surgical interference in portal hypertension.

- Urine & stool examination should be repeated 3 ms after treatment & viability test should be done to see if the patient is cured or not.
Oriental schistosomiasis

*Schistosoma japonicum*
Overview

• *Schistosoma japonicum* inhabits the superior and inferior mesenteric veins.

• This parasite has a very wide host range, infecting at least 31 species of mammals.

• It can be considered a true *zoonosis*.

• *S. japonicum* is the most pathogenic of the *Schistosoma* species.
**Schistosoma japonicum**

**Geographical Distribution:** common in Far East (Japan, China, Philippines, Formosa, Korea).
**Morphology:** similar to *other* schistosomes with few differences:

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

- Size: 12-26 mm × 0.3 mm.
- Intestinal caeca: unite at the posterior two fifths.
- Ovary: posterior.
- Uterine tube: long, contains 50 – 100 eggs.
**Egg:**

- **Size:** 70-100×50µ.
- **Shape:** rounded.
- **Shell:** thin, has curved tubercle-like projection in shallow depression.
- **Colour:** translucent.
- **Contents:** mature miracidium.
- **Eggs sweep out with feces.**

- **Miracidium:** similar to *S. mansoni*.
- **Cercaria:** with 5 pairs of penetration glands.
**Life cycle:** as other schistosomes but differs in:

**Habitat:** mesenteric veins of small & large intestine.

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

**Intermediate host:** snail *Oncomelania*.
Pathogenesis:

**Disease:** Oriental schistosamiasis.

- The disease 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 pathogenesis is more dangerous than other schistosomes due to the following:

1. It inhabits the small intestine.

2. Affection of superior mesentric veins with great approximity to liver → severe affection.

3. Many reservoir hosts.

4. I.H. is amphibian and operculated.

5. Female lays 3000 eggs/day.

6. Absence of eggs spine allow their dispersion.
Clinical picture:

- The clinical manifestations of *schistosomiasis japonica* are similar to that of *schistosomiasis mansoni* but:
  - The disease is serious and fatal.
  - Diarrhea is common due to the pathological lesions of the small intestine.
• **Katayama disease** (acute schistosomiasis): early clinical manifestations of schistosomiasis that coincide with the maturation of adult worms and beginning of oviposition.

• It appears between **2-7 weeks** after exposure to first schistosome infection or heavy reinfection.

• It is frequently seen in *S. japonicum*, heavy infection with *S. mansoni* and rarely in *S. haematobium* infection.
It is characterized by:

- Acute onset of nocturnal fever, headache, weakness, cough (Flu like illness).

- Abdominal pain & diarrhea.

- Hepato-splenomegaly and involvement of the CNS (2-3%).

- Eosinophilia (40%).

- Diffuse pulmonary infiltrates.
Acute schistosomiasis (Katayama stage): There are increased *interstitial* and *vascular* markings in both lungs and mild *hilar lymphadenopathy*
Diagnosis:
- Stool examination for the characteristic egg.
- Other methods of diagnosis as in schistosomiasis mansoni.

Treatment:
- *Schistosoma japonicum* is resistant to treatment.
- *Praziquantel* is the only effective drug.
**Schistosoma intercalatum**

- Common in central and West Africa.
- Adults live in mesentric venous plexuses.
- I. H.: snail *Bulinus africanus*.
- **Eggs**: $170 \times 60\mu$ with terminal spine.
- These eggs are unique because they are Zeihl-Neelsen positive.
- Eggs are detected in stool.
- Cause mild disease, hepatomegaly is not marked.
Control of schistosomiasis

1-Prophylaxis:

- Health education.
- Personal prophylaxis for exposed ones by wearing boots and gloves, use of repellents e.g. dimethyl phthalate.
- Quick & thorough drying of exposed wet skin.
- Pure water supply.
- Treatment of canals water.
2- **Human cases:**

- Mass treatment by praziquantel: 40 mg/kg in a single oral dose.

- Sanitary disposal of excreta.

3- **Reservoir hosts:** should be controlled.
**4-Snail control:**

**a. Physical methods:**

- Dryness of canals or use alternate canals (Winter closure period).
- Periodic clearance of canals from vegetations and weeds.
- Pitching canal banks with concrete to prevent growth of aquatic plants.
- 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*).

– Certain toxic plants e.g. *Balanites aegyptiaca* and *Ambrosia maritima* (*Damsisa*).

– **Pathogens:** infection of snails by miracidia of avian schistosomiasis to decrease its vitality.
c. Chemical methods (molluscicides):

– **Copper sulphate**: 10-20 ppm.
– **Sodium pentachlonophenate**: 5- 10 ppm.
– **Baylucide**: 2 ppm.

• The first one is widely used in Egypt.
• It is not effective against eggs of snails, so it should be reapplied every 3 ms to kill newly hatched snails or applied as a chemical barrier at inlet of canal to give a concentration of 0.5 ppm.
Cercarial dermatitis
Swimmer’s itch, Bather’s itch

- It is produced by cercariae of non-human schistosomes (bird and animal schistosomes) in fresh water or marine water, worldwide.

- Cercariae penetrate the human skin, but cannot proceed beyond the germinal layer, and rapidly destroyed in the skin.
1. Eggs are passed in feces.

2. Eggs hatch and liberate miracidia.

3. The parasite develops in a molluscan intermediate host.

4. Cercariae penetrate the skin of the birds and migrate to blood vessels to complete the cycle.

5. Humans are exposed to the dermatitis-producing cercariae.
**Clinically:** dermatitis, irritation, itching, urticarial wheels and 2ry bacterial infection.

- Subsequent exposures cause severe itching & formation of macules & papules which last for 1 week.

**Diagnosis:** history of contact with water followed by skin rash.
Treatment:

- Local antihistaminies, antipruritis, antibiotics for 2ry bacterial infection.

- 5% copper sulphate to relieve itching.

- 2% methylene blue to prevent bacterial infection.
Prevention and Control:

- Applying any water repellent as petroleum jelly, sunscreen → reduce cercarial penetration.

- Avoid polluted water and swimming in shallow areas near shore.

- Rapid drying of the skin to avoid cercarial penetration.

- Snail control.
Thank you