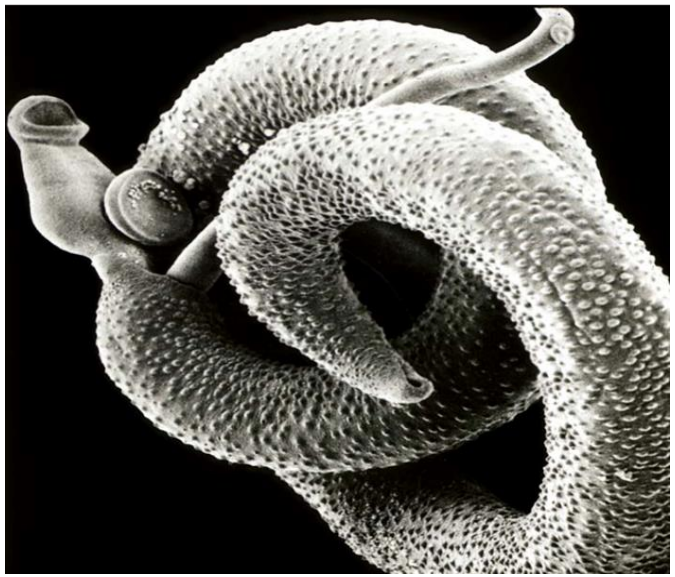
MEDICAL UNIVERSITY – PLEVEN
FACULTY OF MEDICINE
CENTER OF DISTANCE LEARNING

LECTURE № 4

SCHISTOSOMIASIS

(Bilharziosis, Morbus Mansoni, Katayama fever)



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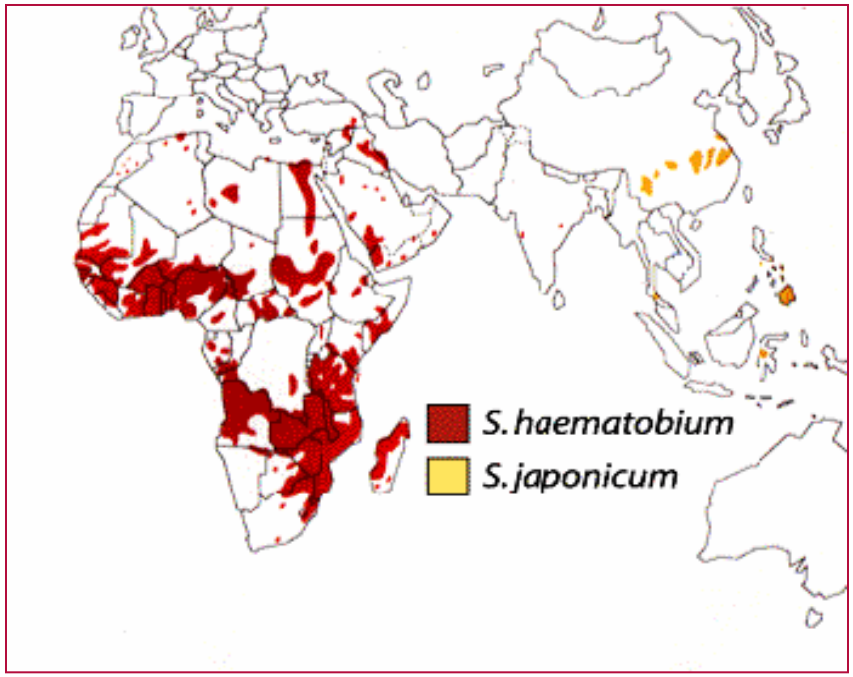
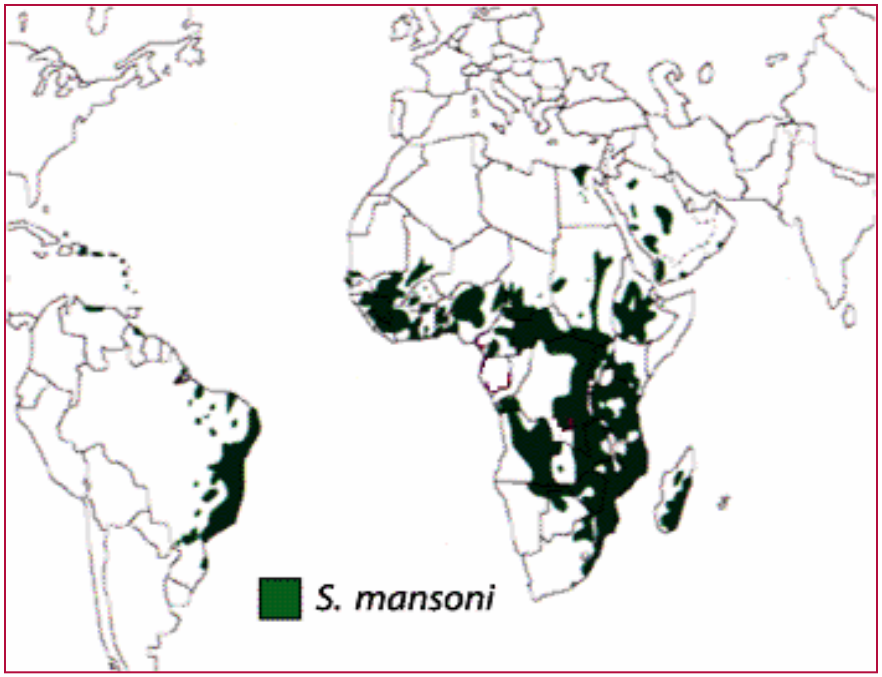
DEFINITION

Schistosomiasis (also called bilharziasis) is helminthiasis caused by trematodes of the genus *Schistosoma* and is characterized by a chronic course and damage of blood vessels of the genitourinary system, the gastrointestinal tract and the liver. Sometimes the lungs and other organs are affected.

SPREAD AND IMPORTANCE





Schistosomiasis is prevalent in tropical and sub-tropical areas, especially in poor communities without access to safe drinking water and adequate sanitation. It is estimated that about 90% of those requiring treatment for *schistosomiasis* live in Africa.

SPREAD AND IMPORTANCE

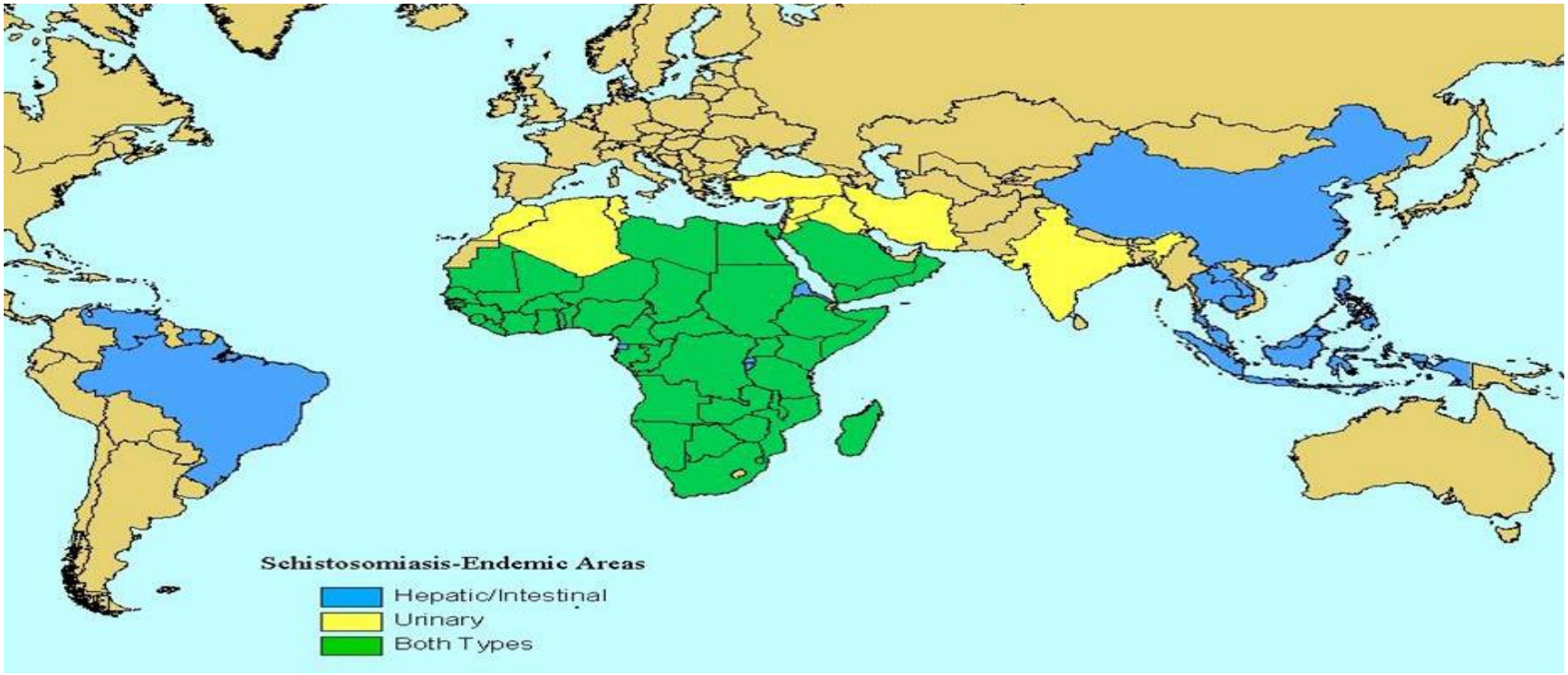
According to WHO data, about *700 million people live in endemic regions*, and are all the time exposed to the risk of infection.

Today, *schistosomiasis* is endemic in 78 developing tropical countries and 52 of these countries have a high level of endemicity.

Estimates show that at least 120.2 million people required preventive treatment for *schistosomiasis* in 2017, out of which more than 98.7 million people were reported to have been treated.

Schistosomiasis mostly affects poor and rural communities, particularly agricultural and fishing populations.

The number of deaths due to schistosomiasis is difficult to estimate because of hidden pathologies such as liver and kidney failure and bladder cancer. Estimates therefore vary widely between 20 000 and 200 000 deaths per year.



Schistosomiasis is common in equatorial regions, between latitudes of 38° north and 35° south.

The African countries most affected are Angola, Ghana, Mozambique, Egypt, Sudan; in South America – Brazil, and in South-West Asia – Yemen.

The importance of *schistosomiasis* is determined by their high prevalence in tropical countries, the severe and/or the permanent damages they inflict. They rank second, after malaria, as far as their health and economic importance in the tropical and subtropical regions are concerned.

ETIOLOGY

Schistosomes are bisexual trematodes of the family *Schistosomatidae*.

Five of these parasitize on humans:

S. mansoni
S. haematobium
S. japonicum
S. mekongi
S. intercalatum

The first three types are of greater importance for human health.

Species with limited distribution between the people:

S. mattheei
S. bovis
S. indicum
S. malayensis

S. mansoni lives parasitically in the mesenteric veins draining the large intestine. *S. haematobium* lives in the veins draining the urinary bladder plexus and *S. japonicum* – in the mesenteric veins draining the small intestines.

The female parasites are 7-17 mm x 0.25 mm in size, and the male ones - 6-13 mm x 1.0 mm.

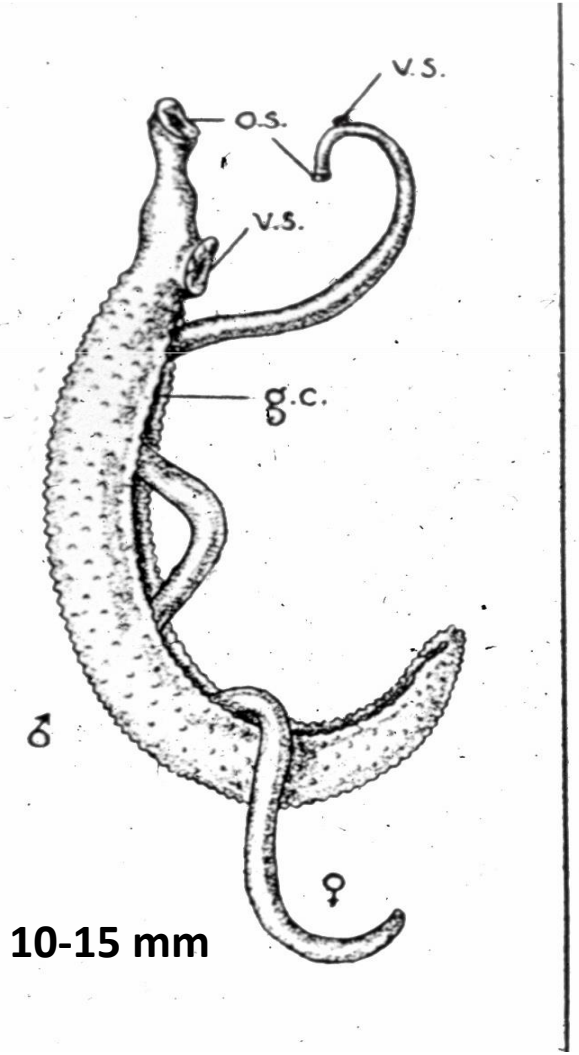
Along the body of the male there is a groove - canalis gynaecophorus, in which the female parasite is located. It is attached to the male by means of two cup-like formations, located at the anterior end.

The life span of the parasites is about 8 years, in some cases 20-30 years.

Major Species of Schistosomes Causing Disease

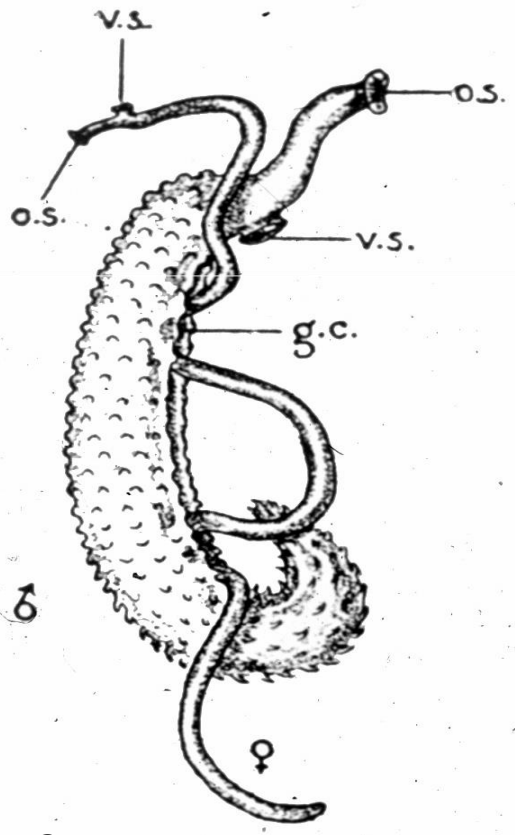
Species	Location in Host	Distribution
S. mansoni	Mesenteric veins draining the large intestine	Africa, West Indies, S. America (Suriname, Brazil, Venezuela)
S. haematobium	Veins draining the urinary bladder plexus	Africa, Middle East, India, Portugal
S. japonicum	Mesenteric veins draining the small intestine	China, Indonesia, Japan, Philippines

GS: Canalis gynaecophorus, OS: oral sucker; VS: ventral sucker



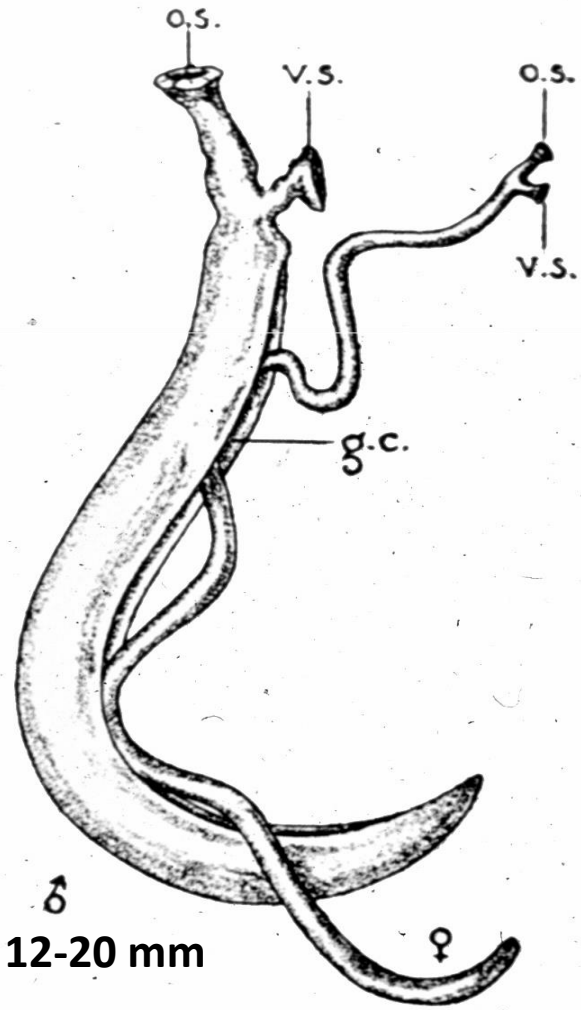
10-15 mm

S. haematobium



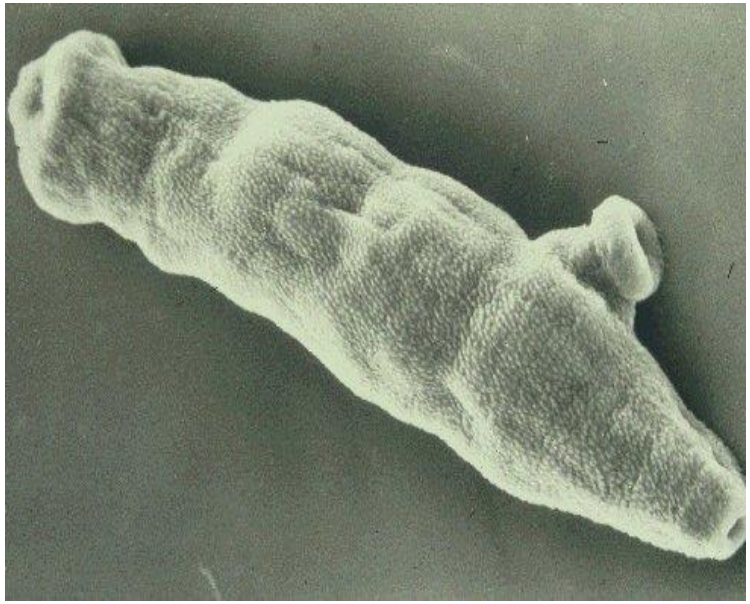
6-12 mm

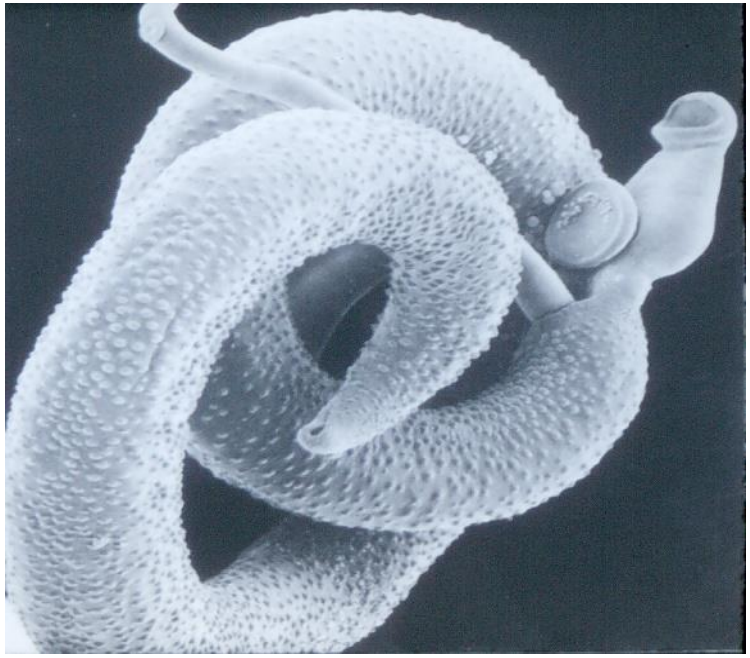
S. mansoni



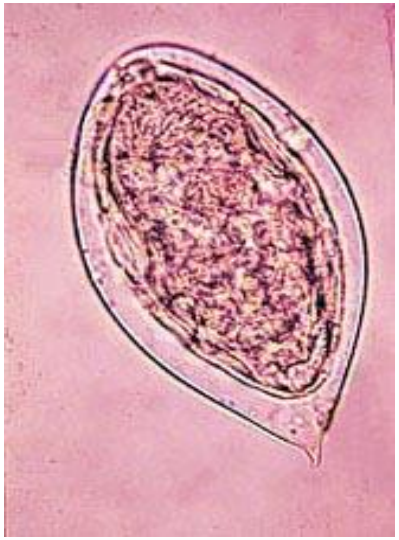
12-20 mm

S. japonicum



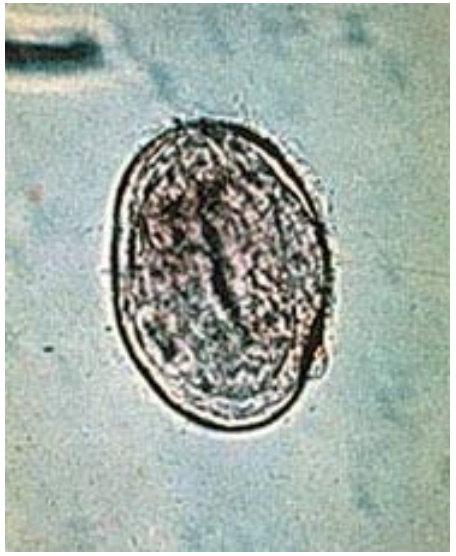


THE SCHISTOSOME EGGS



S. hematobium
~140μ

S. mansoni
~140μ



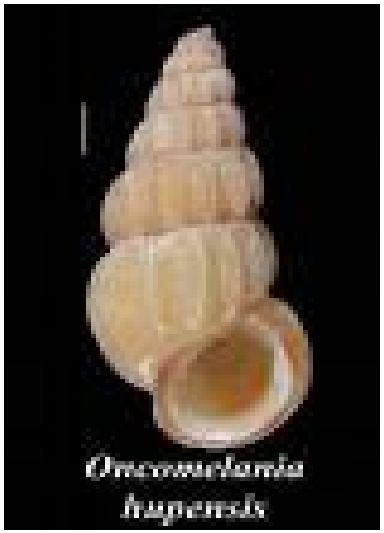
S. japonicum
~85μ

The eggs are yellow and oval, 85 – 140 μm in size. The one end is more rounded. The eggs of *S. haematobium* have at the one end a large terminal spine. The eggs of *S. mansoni* have a large lateral spine and these of *S. japonicum* – a small lateral spine, which is hard to see. The eggs contain the larval form of the parasite - miracidia.

The intermediate hosts of the schistosomes are mainly water snails of the genus *Bulinus*, *Planorbis*, *Ferrissia*, *Biomphalaria*, *Oncomelania* and *Tricula aperta*. These snails live in slow-moving water near shorelines – lakes, rivers, dams, irrigation systems, etc., with lush vegetation, as well as in paddy fields. The optimal temperature for the parasite to develop in the snails is 22-26°C.

Bulinus

(S. haematobium)



Oncomelania

(S. japonicum)

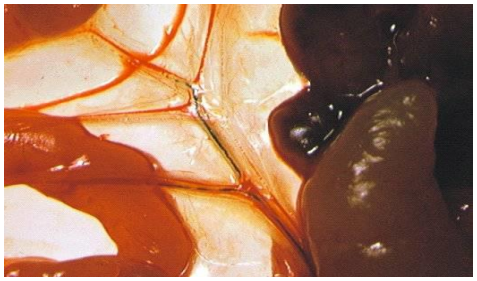
Biomphalaria

(S. mansoni)

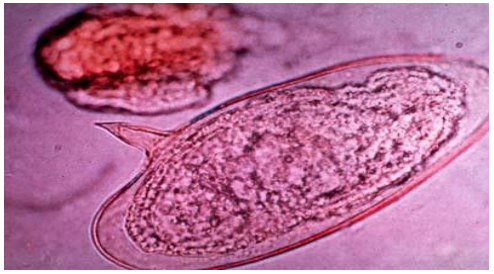
LIFE CYCLE

The biological cycle of the parasites include successive development stages in the *definitive host (human and animals)* and the *intermediate hosts (freshwater snails)*.

The sexually mature parasites are mostly found in the mesenteric veins, where the females lay their eggs. The eggs pass through the walls of blood vessels and the tissues of the urinary bladder and the intestines to be excreted with the urine or the feces.



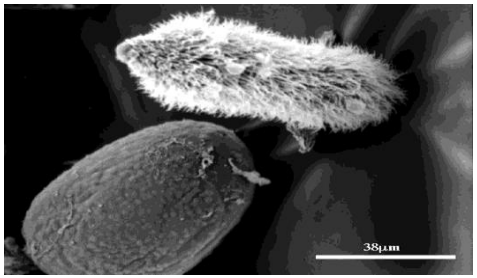
sexually mature parasite



egg

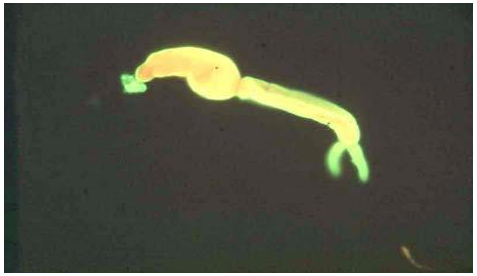
LIFE CYCLE

When eggs reach fresh waters, they produce the larval stage - *miracidia*, which swim by means of ciliae and actively penetrate the body of the intermediate host. Here, the miracidia develop and multiply, and become a *cercariae*.



miracidia

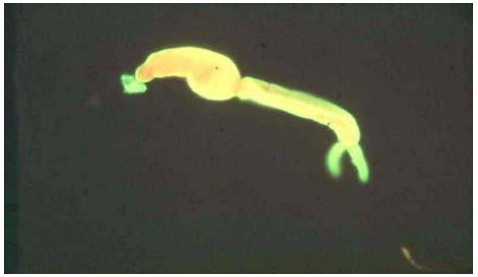
intermediate host



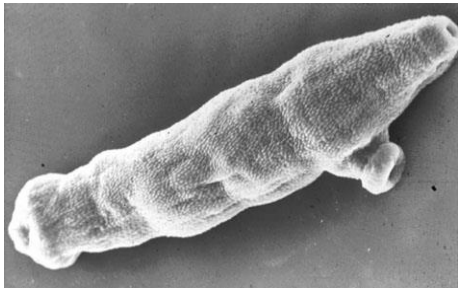
cercariae

LIFE CYCLE

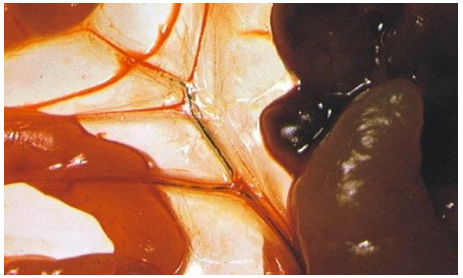
At this final stage of development, the cercariae leave the intermediate host, swim using its tail and when they come in contact with the definitive host, they enter the body of the host through the skin or mucosae. The parasites migrate via the lymph and blood vessels, pass through the lung capillaries and via the greater circulation reach the hepatic veins. It takes them 30-45 days to develop and mature into adults. Then they migrate to the veins of the mesenterium and the genitourinary system, where the females lay their eggs.



cercariae

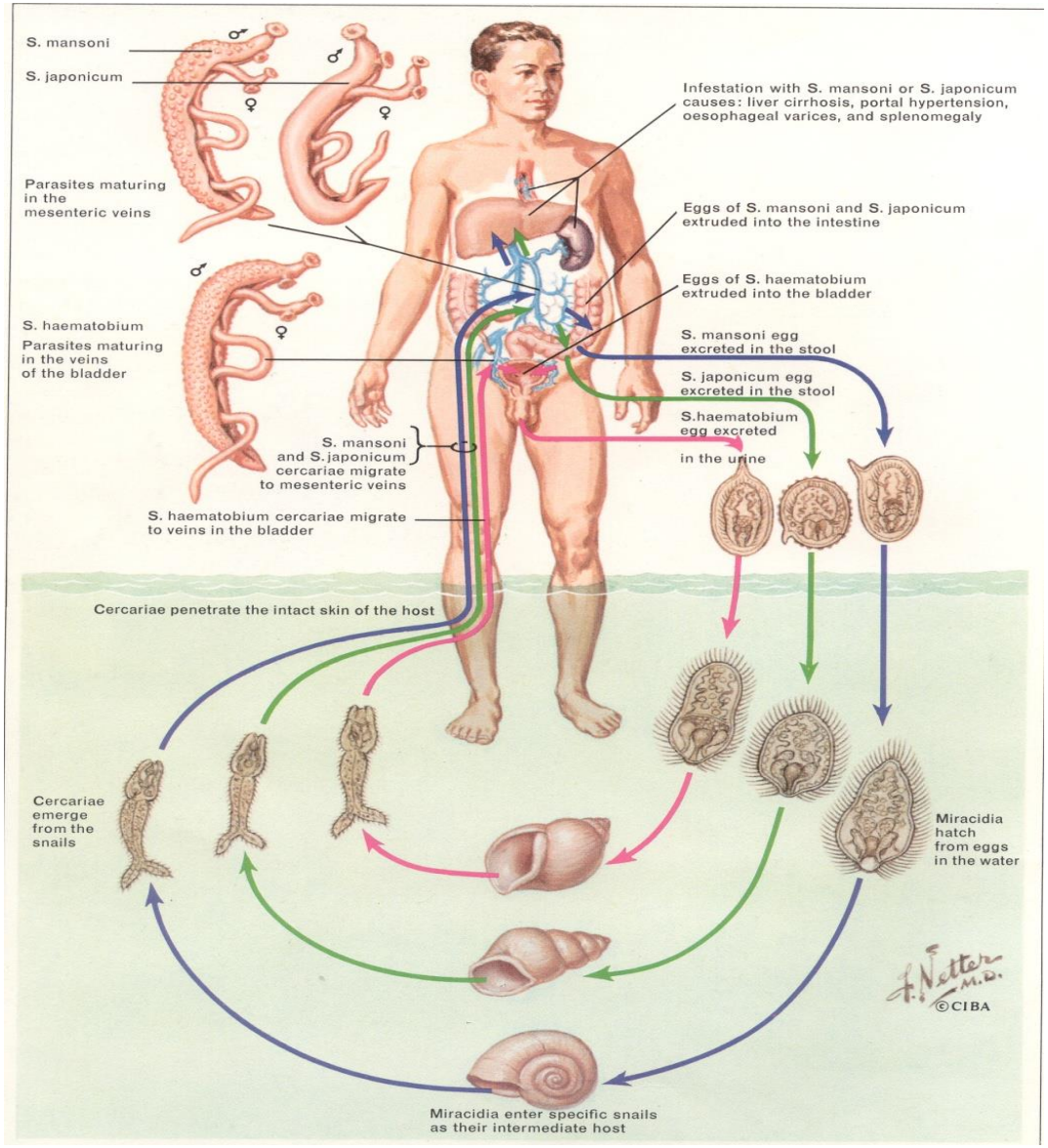


migrate parasite (schistosomula)



the parasite in the hepatic veins

THE ALL LIFE CYCLE



There are several types of schistosomiasis:

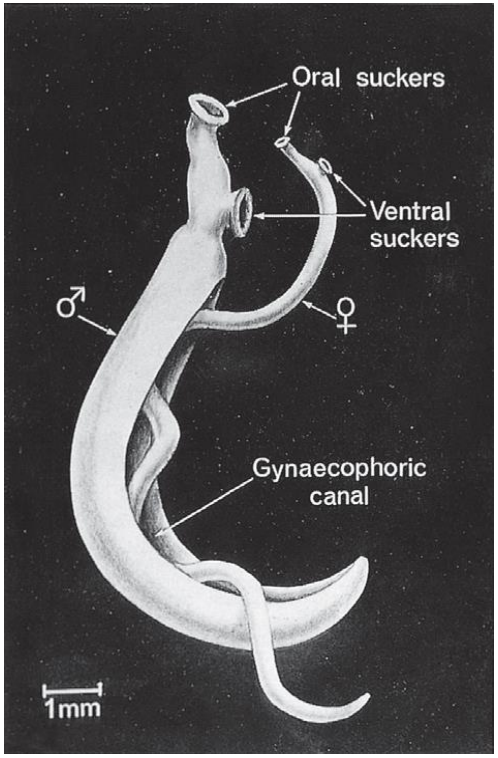
1. Urogenital schistosomiasis.
2. Intestinal schistosomiasis.
3. Japanese schistosomiasis.
4. Other types.

SCHISTOSOMIASIS UROGENITALIS

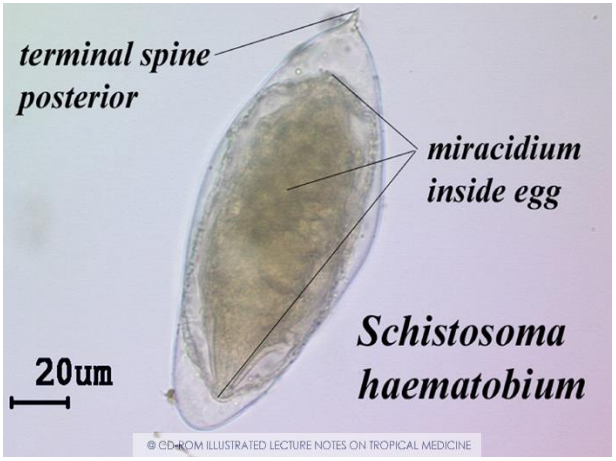
BILHARZIOSiS

ETIOLOGY

It is caused by ***Schistosoma haematobium***. The female parasite is 20-26 mm x 0.25 mm in size, and the male - 10-15 mm x 0.75-1.0 mm.



The eggs are pale, oval (83 -187 µm x 60 µm), with a spine at one end. They contain the larval form (miracidium). The females lay about 300 eggs a day.



PATHOGENESIS AND PATHOLOGY

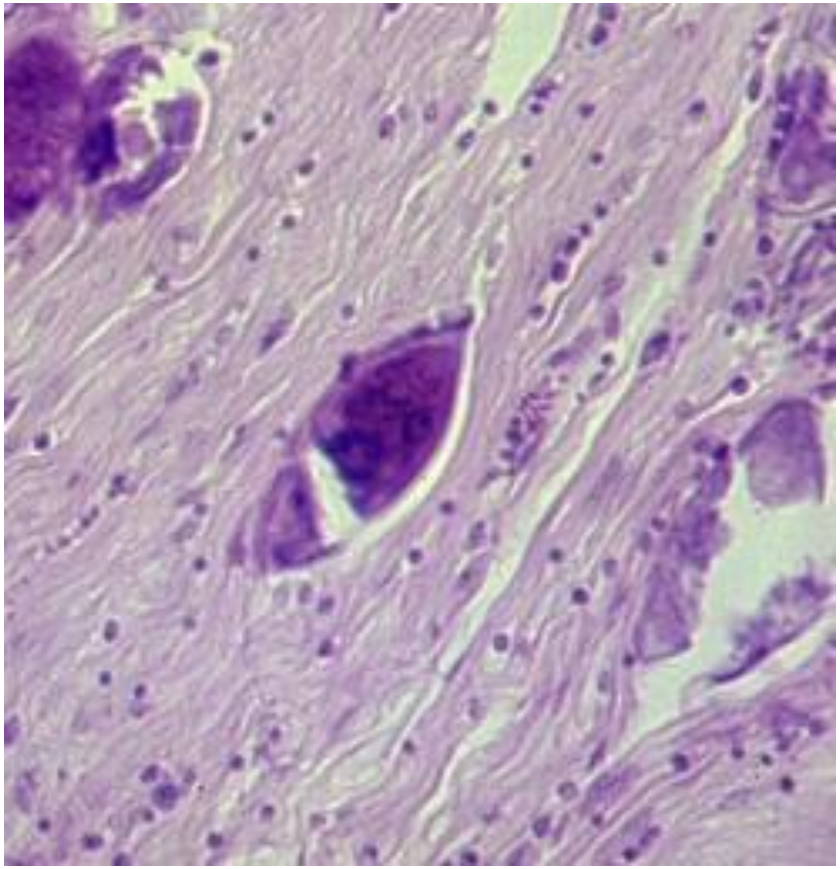
There are four stages in schistosomiasis: invasion into the host, migration with maturing of the parasite, acute and chronic. The cercariae penetrate through the skin or mucosa and stay at the site of invasion for 4-5 days. An erythematous swelling appears with infiltration of eosinophils and neutrophils. At the early migration stage of the disease the main pathogenic mechanism is connected with the sensibilization of the host's organism by the metabolic antigens of the schistosomes, as a result of which inflammatory infiltrates are formed in the lungs and other organs.

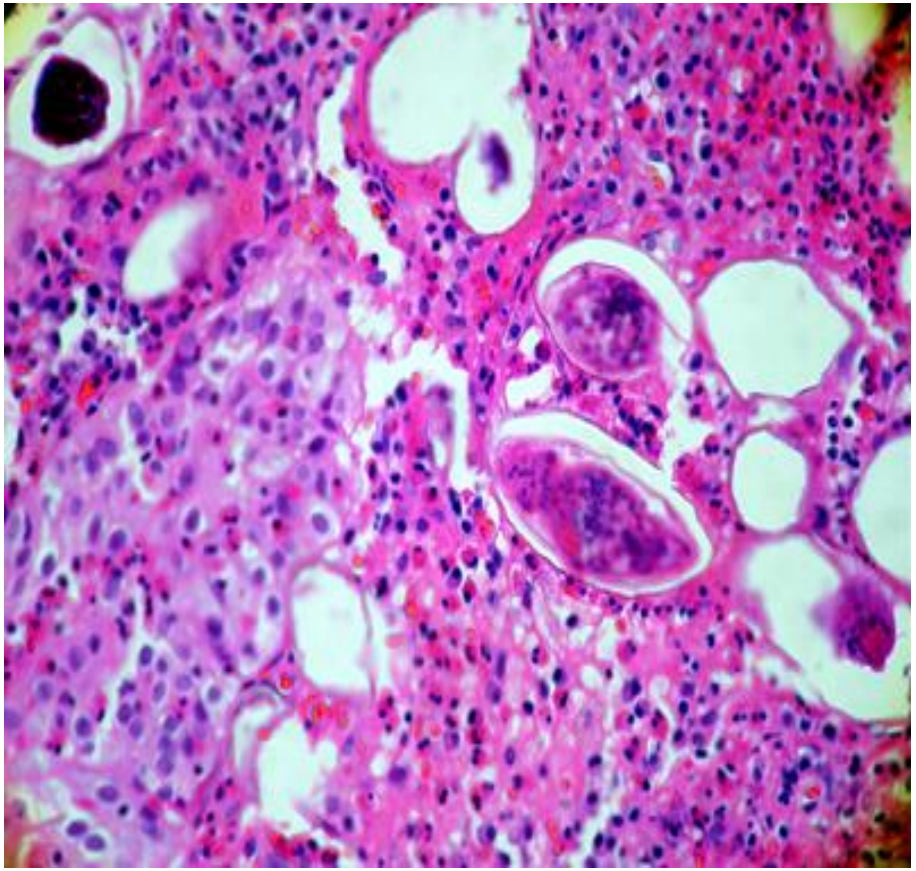
PATHOGENESIS AND PATHOLOGY

In the *chronic stage*, mechanic injury is added, especially during the migration of the eggs through the walls of blood vessels and the urinary bladder, as well as other organs (liver, lungs).

Inflammatory allergic granulomas – *"bilharziomas"* are formed around the eggs, located in tissues of the urinary bladder or the urethra. This results in ulceration or hyperplasia, which in turn lead to the formation of benign papillomata. The umbilical wall undergoes fibrosis and the bladder is deformed. Part of the eggs are not excreted in the lumen, get stuck and calcify in the wall. The clusters of eggs are visualized as yellowish-white sand-like spots *(sandy patches)* across the thinned mucosa. When the urethra or the ureter are affected, obliteration, strictures and hydronephrosis may occur.

The inflammatory granuloma around the eggs of *S. haematobium*





CLINICAL PRESENTATION

The severity of clinical signs varies and depends on the intensity of invasion (number of parasites that have entered the body).

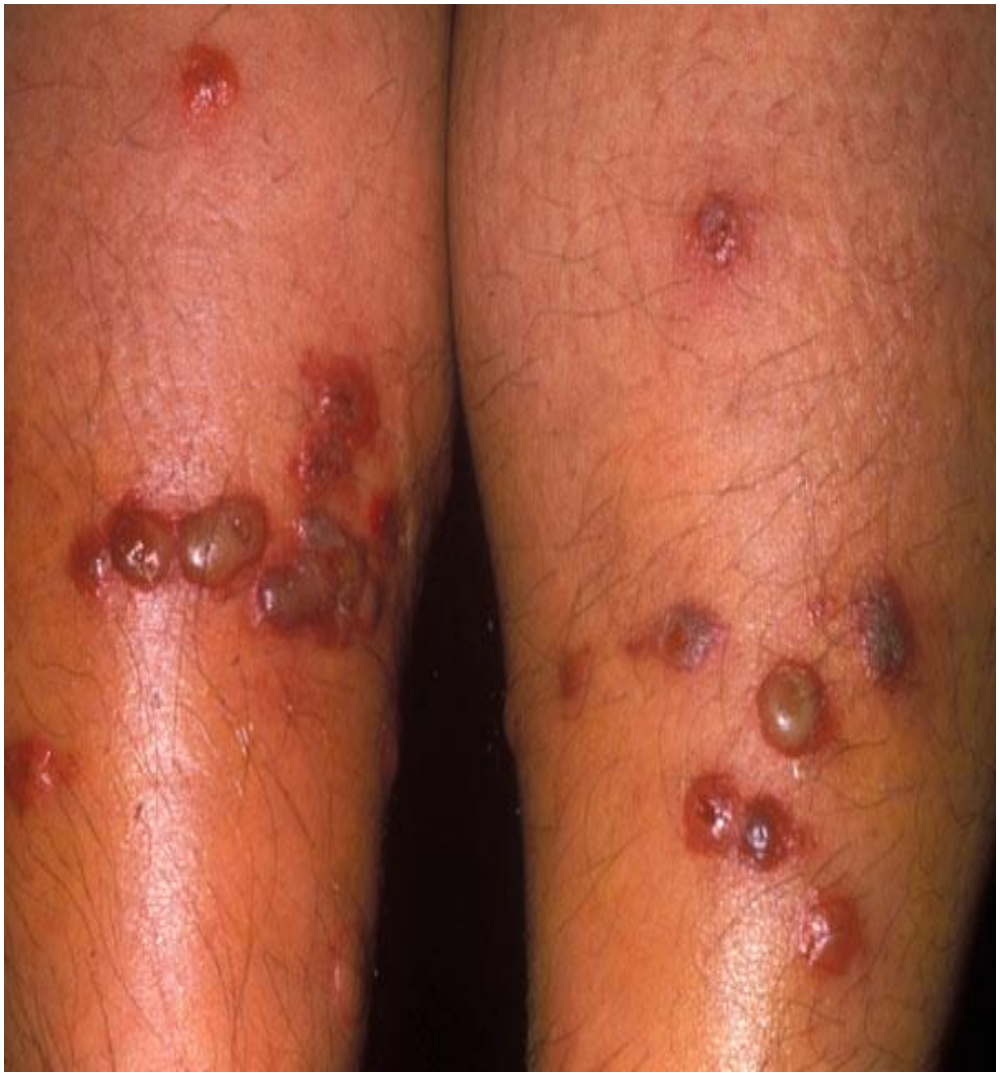
There are *asymptomatic*, *mild*, *severe* and *very severe* forms.

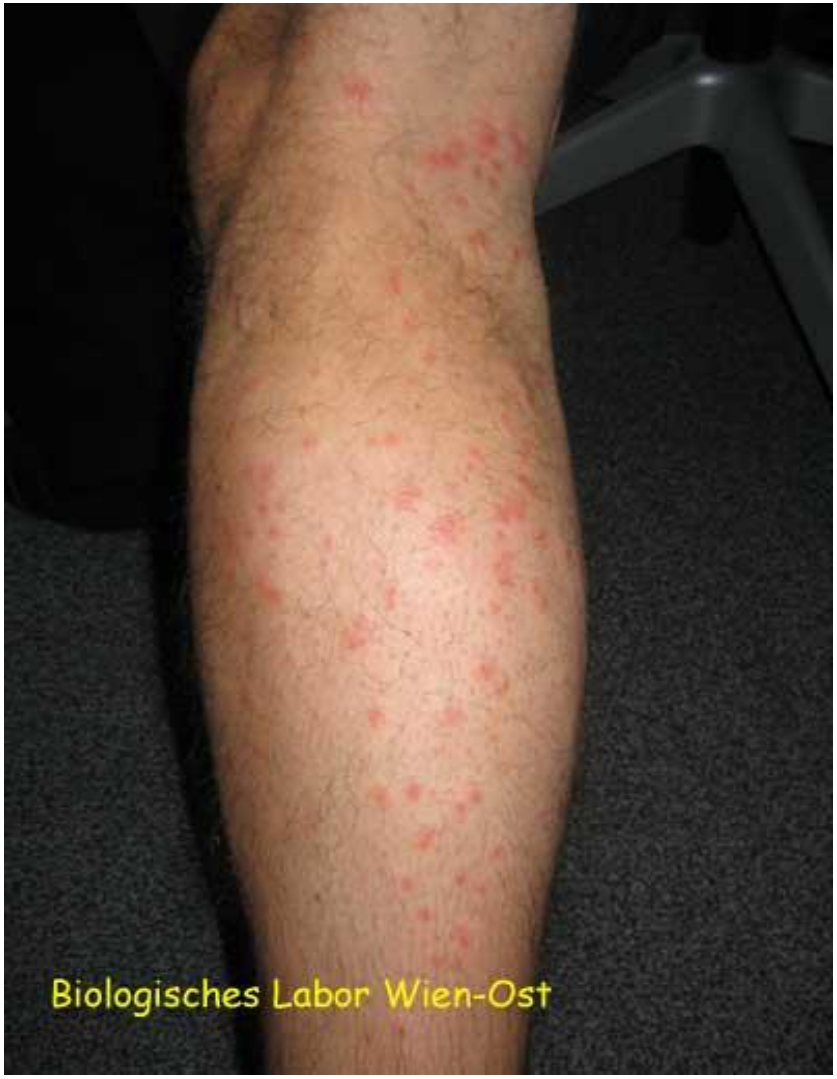
Incubation period is 30 days

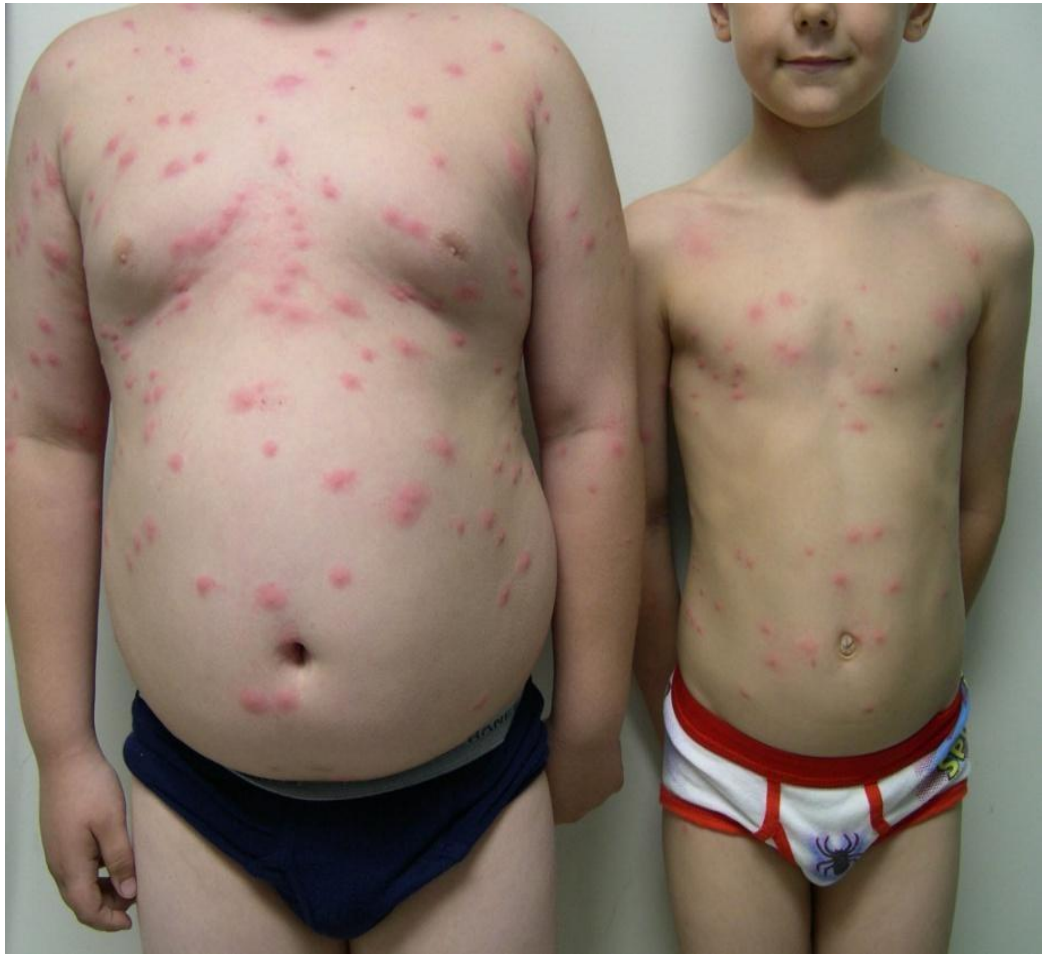
Stage of invasion into the host

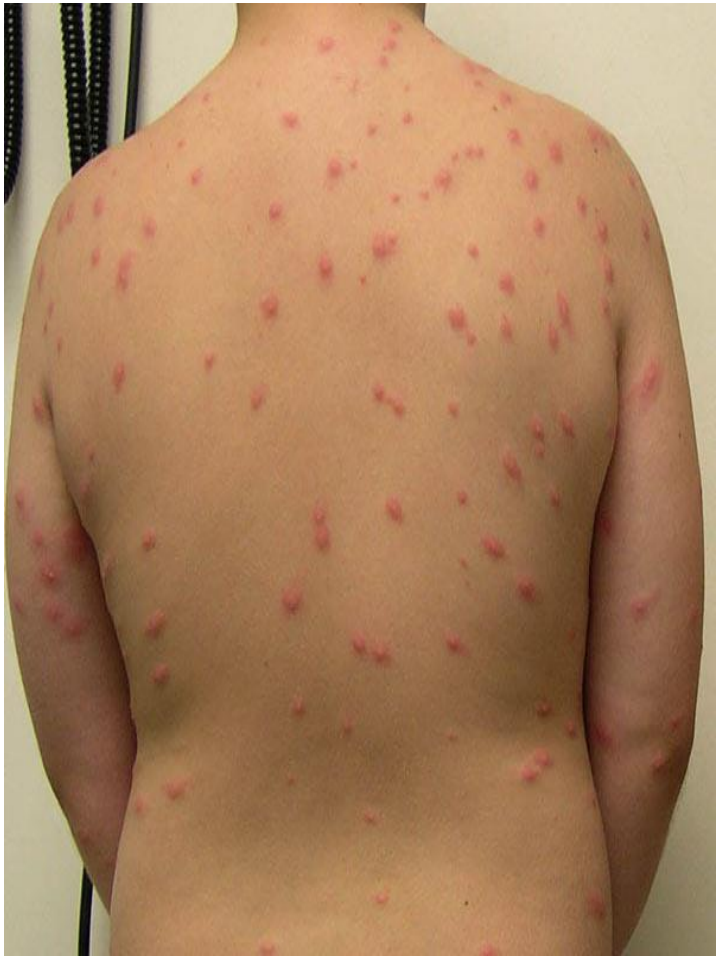
At the moment when the cercariae penetrate the skin, a sharp pain is felt, similar to a needle prick. Transient itching dermatitis is possible.

Cercarial Dermatitis (Swimmer's Itch)









Cercarial Dermatitis

Stage of migration and maturing of the parasite

In the *migration stage*, the most eminent symptoms are those of allergy (high fever, cough, sometimes asthma-like attacks, eosinophil infiltratates in the lungs, leukocytosis, eosinophilia – 20-60%). Patients complain of loss of appetite and general fatigue.

These early symptoms persist for about a month and a latent period follows, ranging from several weeks to 3 months.

Acute stage

After the eggs have been deposited, acute symptoms appear. The first and most characteristic symptom is *terminal hematuria*, but in some cases blood is detected in all urine samples. There is frequent urination, with possible burning pain. Patients also complain of dull pain in the region above the symphysis, the perineum and lumbar region. Eggs are detected in the urine. In cases of profuse hematuria, as a result of ruptured blood vessels, even adult parasites may be seen in the urine. In case there is no reinvasion, the hematuria gradually subsides.

CHRONIC STAGE

In *chronic schistosomiasis*, linear web-like and circulatory shadows are visualized on the wall of the bladder as a result of calcified eggs. Cystoscopy reveals characteristic changes – hemorrhages in the early stage, then yellowish formations that contain non-viable eggs. Later, ulcers and the typical *"sandy patches"* appear.

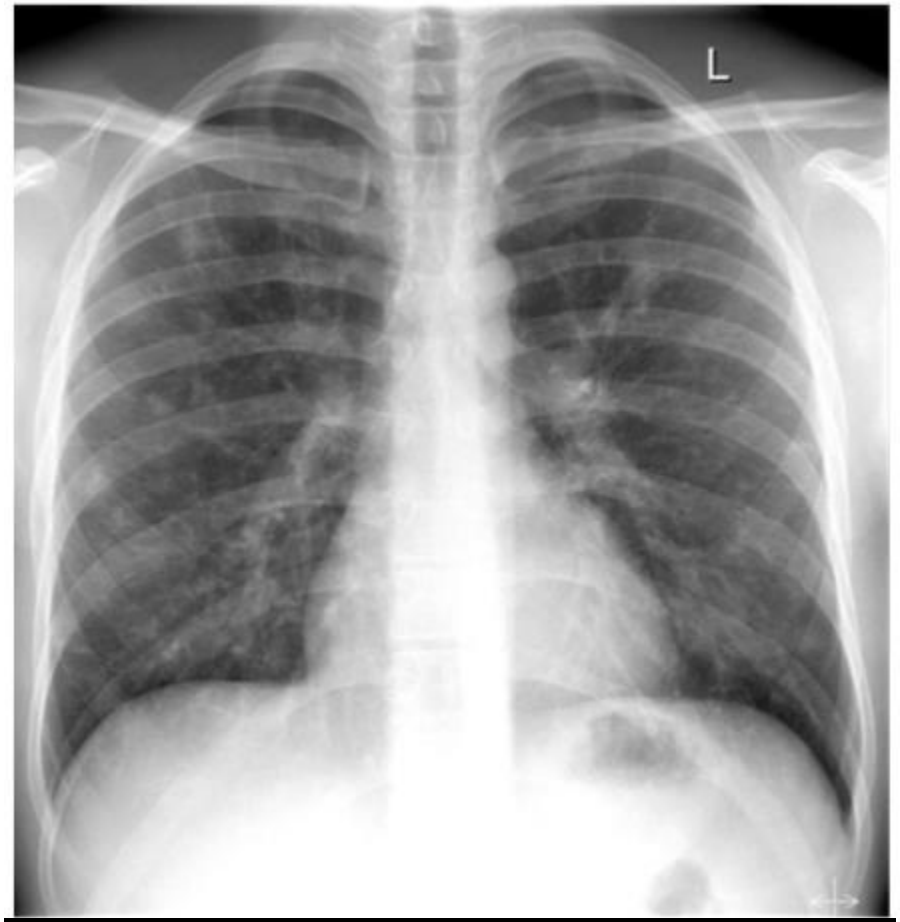
Damages of the reproductive organs are seen more rarely. Colpitis, endometritis, adnexitis, dysmenorrhea, etc. may occur in females. In males, possible damages include prostatitis, epididymitis, urethritis, and pseudoelephantiasis of sexual organs.

COMPLICATIONS

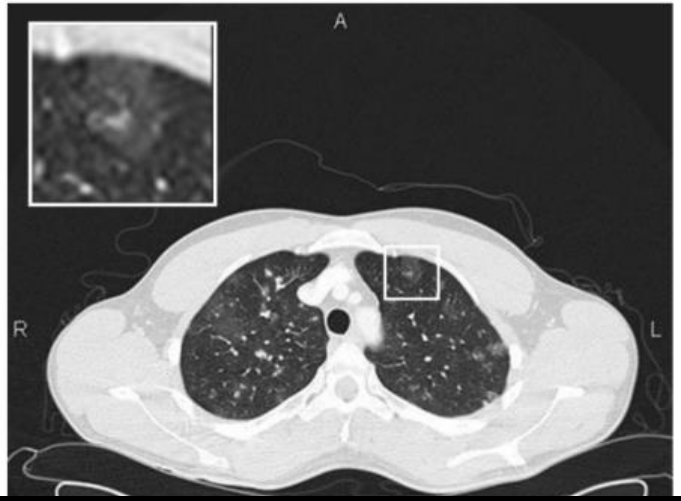
1. When the eggs are located in the liver, ***hepatitis*** develops.

2. ***Pulmonary embolism*** with *S. haematobium* eggs leads to lesions of the parenchyma and the arteries, which are followed by fibrosis pulmonary and cardiac failure (*Aertz disease*, most common in Egypt).

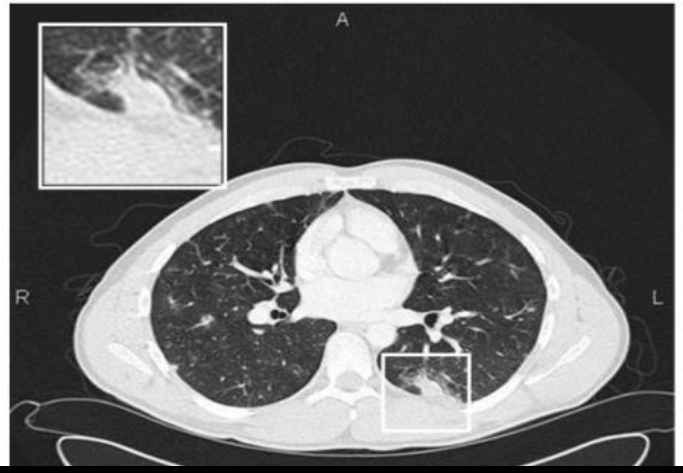
3. Eggs may reach other organs too – *brain*, *spinal cord*, *conjunctiva*, etc. The finding of eggs of *S. haematobium* in the CNS without clinical sequelae is not rare; eggs appear to produce minimal or no histological reaction, in contrast to the production of inflammatory responses when laid elsewhere. The spinal cord is affected more often than the brain.



28 y old male with pulmonary schistosomiasis. Chest radiography (pa) showing multiple small pulmonary nodules scattered over both lungs without obvious predilection.



28 y old male with pulmonary schistosomiasis. Chest CT of the upper lungs showing blurred ground glass nodules scattered over both lungs
(Siemens Sensation 16. 120 kV, 100 mAs, 16×1.5 mm collimation, slice thickness 3 mm).



28 y old male with pulmonary schistosomiasis. Chest CT of the middle lungs showing ground glass nodules and subpleural consolidation of the left superior lower lobe, both consistent with acute eosinophilic infiltration in the context of acute pulmonary ...

COMPLICATIONS

4. Common late complications include stenosis and strictures of the urethra, fibrous cystitis, papillomatosis, and in some cases - carcinoma of the urinary bladder.

A superimposed bacterial infection leads to cystitis, pyelonephritis, hydronephrosis, pyonephrosis, nephrolithiasis, urosepsis, etc.

All these complications, along with a worsening kidney insufficiency may result in death.

DIAGNOSIS

In the early stage of the disease, the diagnosis is based on clinical and epidemiological data. In the acute and chronic stage, parasitological (etiological) diagnosis is of the greatest importance. Cystoscopy provides reliable information, yet detection of eggs is the most reliable proof for the presence of parasitic invasion.

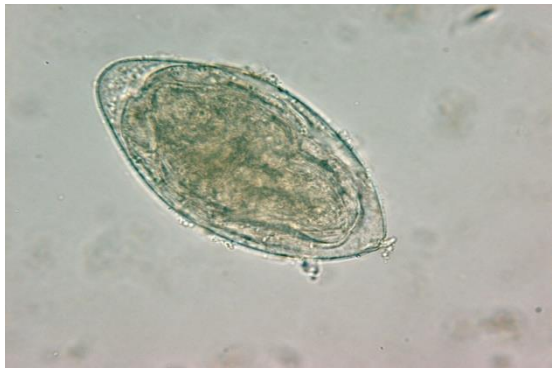
Materials for investigation. These are: urine, endovesicular biopsy sample, sputum (in pulmonary schistosomiasis), blood serum.

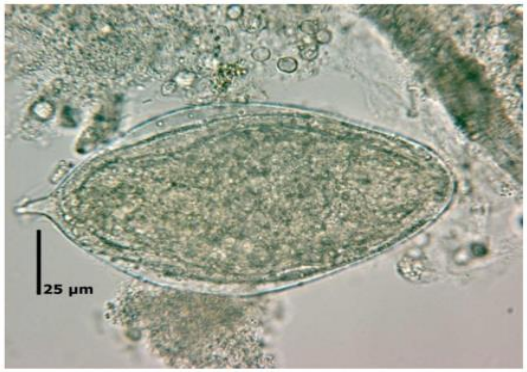
Strict observation of requirements in collecting urine samples is of great importance for effective diagnostics. It is recommended that samples be collected between 11 am and 2 pm, after some physical exertion, or 24-hour urine be collected.

DIAGNOSIS

Methods of investigation.

Microscopic. Microscopy of urine should be performed after concentration methods have been applied (sedimentation in conic mugs, centrifuging – 1500 revolutions for 5-10 min). Lately, filtration through Nucleopore® membrane has been introduced. Slices of biopsy materials are stained with *hematoxylin - eosin*. A sputum sample is analyzed after processing with 2% sodium hydroxide (NaOH) and centrifugation.





Eggs of schistosoma haematobium showing the characteristic terminal spine.
(Magnification factor 500)

DIAGNOSIS

Methods of investigation.

Serological. These are useful in patients who live or have traveled to endemic regions and their results from parasitological investigations are negative. High-sensitivity and high-specificity tests (ELISA, Western blotting, etc.) are used.

DIFFERENTIAL DIAGNOSIS

It is made by ruling out diseases of the genitourinary system with similar symptoms and different etiology:

- cystites;
- urethrites;
- hydronephrosis;
- hematuria;
- haemoglobinurias;
- cancer of the urogenital tract;
- renal tuberculosis, etc.,

in individuals living in or traveling to endemic countries.

TREATMENT

Admission to hospital is advisable for patients with more severe forms of the disease

Etiological treatment

Praziquantel, tabl. 600 mg, 40 mg/kg b.w. is administered in a single dose (for children and adults), or

Metrofunate, tabl. 100 mg., 7.5 mg/kg b.w. for children and adults, three times, at intervals of 2 weeks.

EPIDEMIOLOGY

Source of infection. It is assumed that the source is an infected person, who excretes eggs of the parasite.

S. haematobium has been detected in some animals too – monkeys (in East and West Africa), rodents (in Kenya and South Africa, swine (in Nigeria). However, their role as sources is insignificant.

EPIDEMIOLOGY

Mechanisms, factors and routes of transmission. Humans get the infection on direct contact with cercariae released by water snails and are viable for 24 hours. The *intermediate hosts* of urogenital schistosomiasis are mainly water snails of the genus ***Bulinus***, and more rarely, ***Planorbis*** (Portugal) and ***Ferrissia*** (India).

EPIDEMIOLOGY

Susceptibility. All people are susceptible, but the most vulnerable are rural populations, especially people working in the fields or fishing in fresh waters. Highly intensive invasion is reported in children aged 10-16.

PREVENTION AND CONTROL

Complex measures are taken in endemic regions:

-early diagnosis and treatment of definitive hosts;

-extermination of intermediate hosts - water snails (biological methods, treatment with molluscicides), hydromelioration measures;

-improvement of better hygiene and sanitation;

-health education, etc.

Schistosomiasis control is one of the major WHO programs.

SCHISTOSOMIASIS INTESTINALIS

MORBUS MANSONI

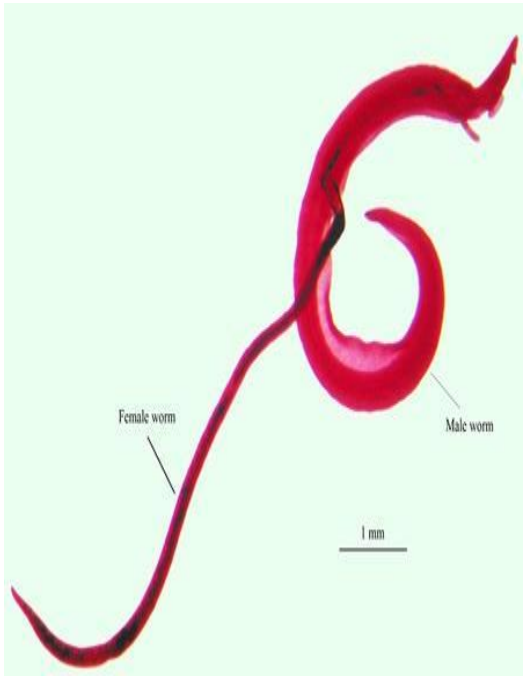
ETIOLOGY

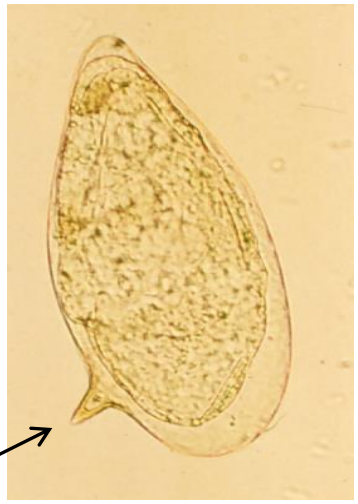
It is caused by ***Schistosoma mansoni***.

The female parasites are 7-17 mm x 0.25 mm in size, and the male ones - 6-13 mm x 1.0 mm.

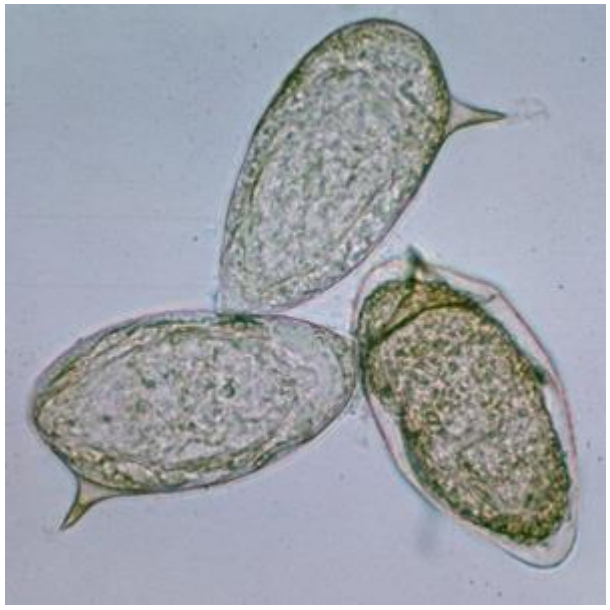
The eggs are yellow, oval (112-175 μm x 45-70 μm), one end is more rounded, with a *big triangular laterally located spine*.

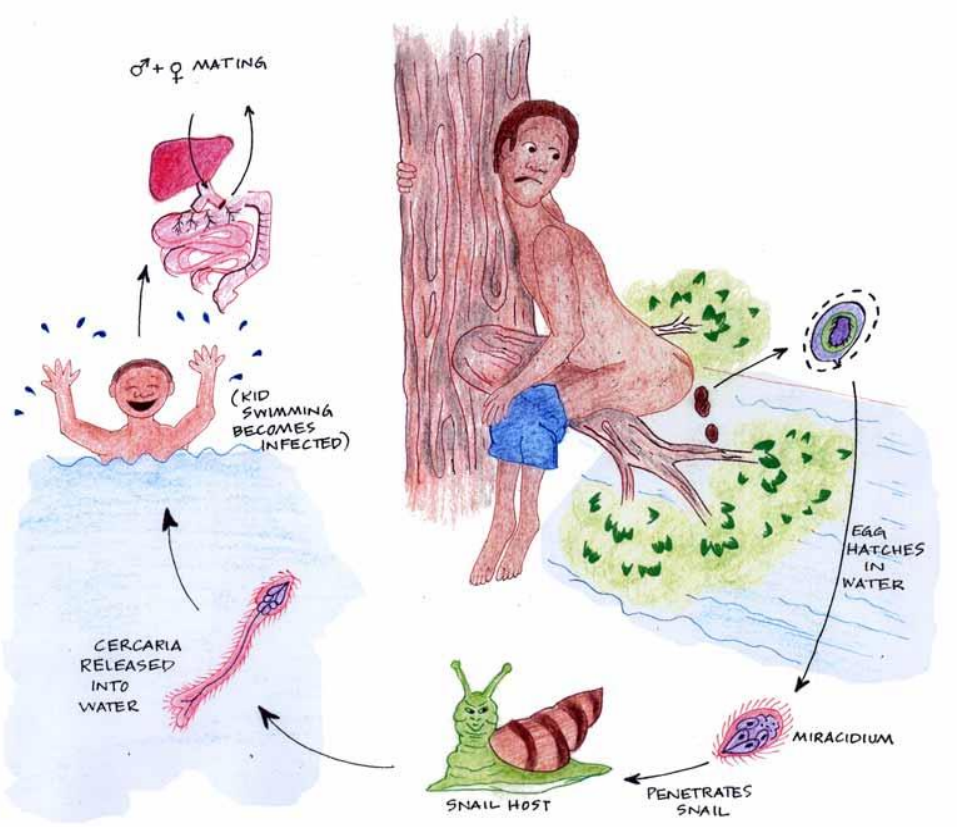
The eggs contain miracidia.





large lateral spine





The adult parasites live in the mesenteric veins. After copulation the female parasites lay their eggs in the venules of the large intestine and the rectum. The eggs pass through the intestinal mucosa, get into the lumen and are excreted with the stools.

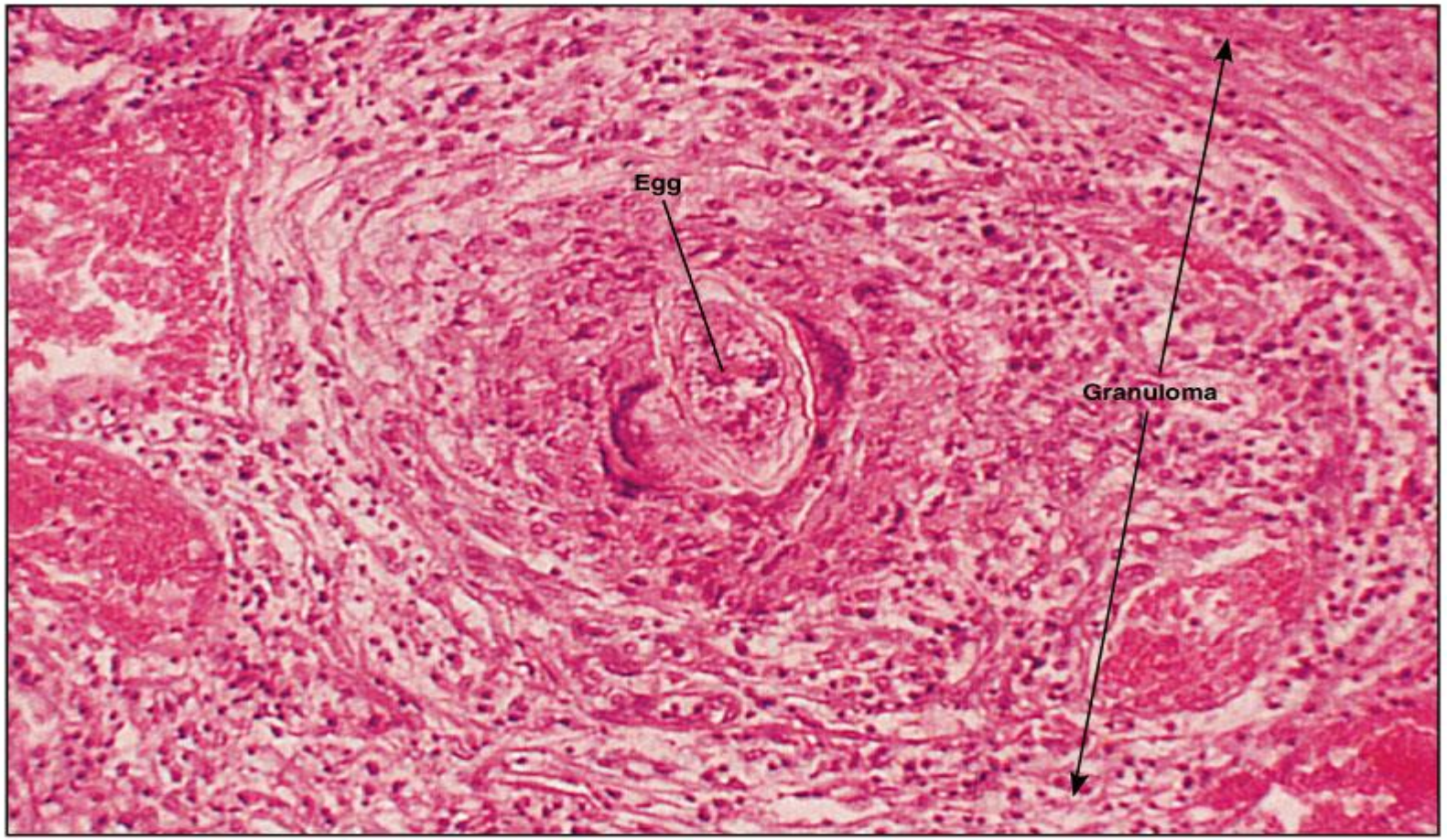
The life span of the parasites is about 8 years, in some cases 20-30 years.

Each day about 300-500 eggs are produced.

PATHOGENESIS AND PATHOLOGY

Pathogenesis and pathological changes are the same as in urogenital schistosomiasis.

1. Mechanical damage and proteolysis of the tissue during the passage of eggs and parasites through the lung, liver and colon wall.
2. Cytotoxic and sensitizing effect of parasitic secretions and excretions.
3. Immunopathogenetic reactions of the organism with the formation of inflammatory granulomas, papillomas and fibrosis changes in the colon wall.
4. Chronic blood, protein and micronutrients loss.
5. The superimposition of secondary infection.



In the chronic stage, granulomas, pseudopolyps and pseudotumors, and sandy patches are formed in the large intestine.

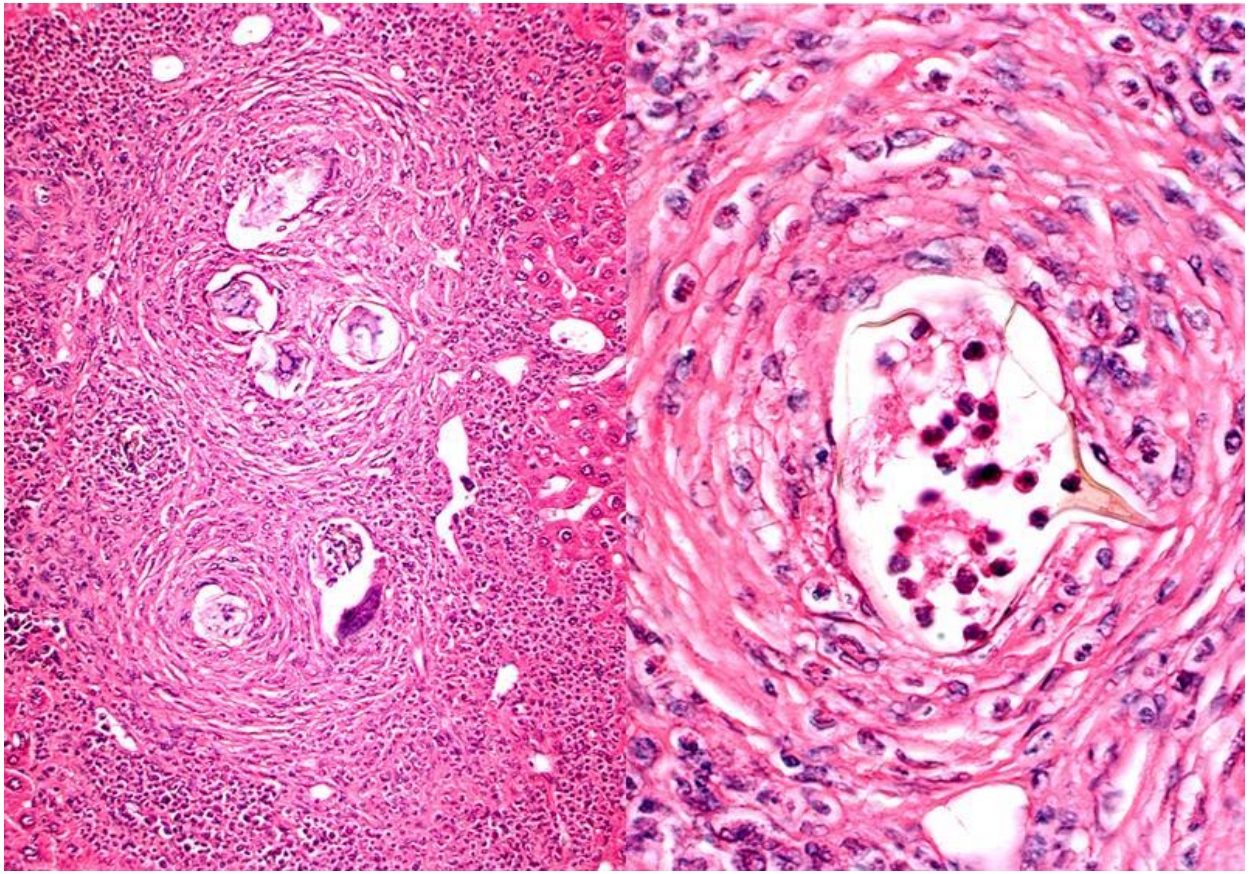
CHRONIC GRANULOMA (PSEUDOTUBERCLE)

In the center:

dead eggs (eggshell breaking or calcification)

Surround:

epithelioid cells, eosinophiles and foreign body giant cells



CLINICAL PRESENTATION

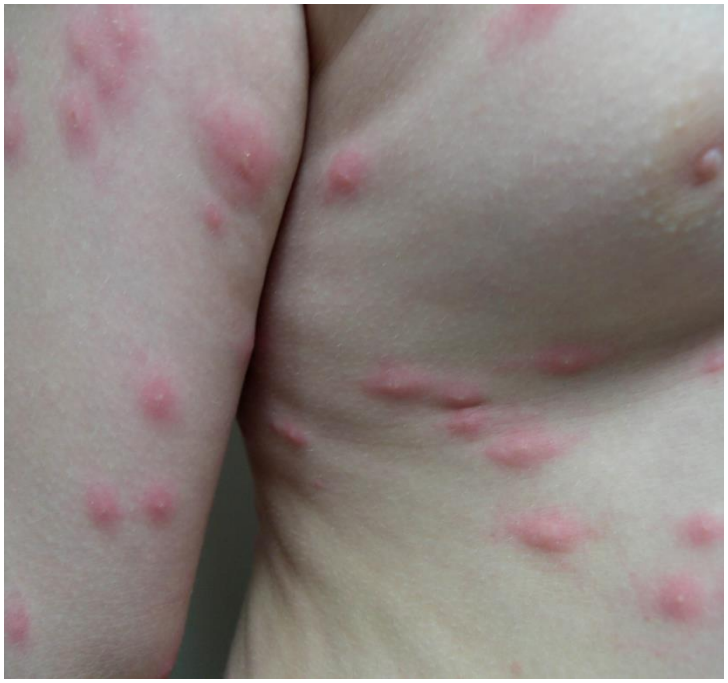
Different clinical forms are seen, ranging from asymptomatic to severe, the latter usually seen in cases of primary infection.

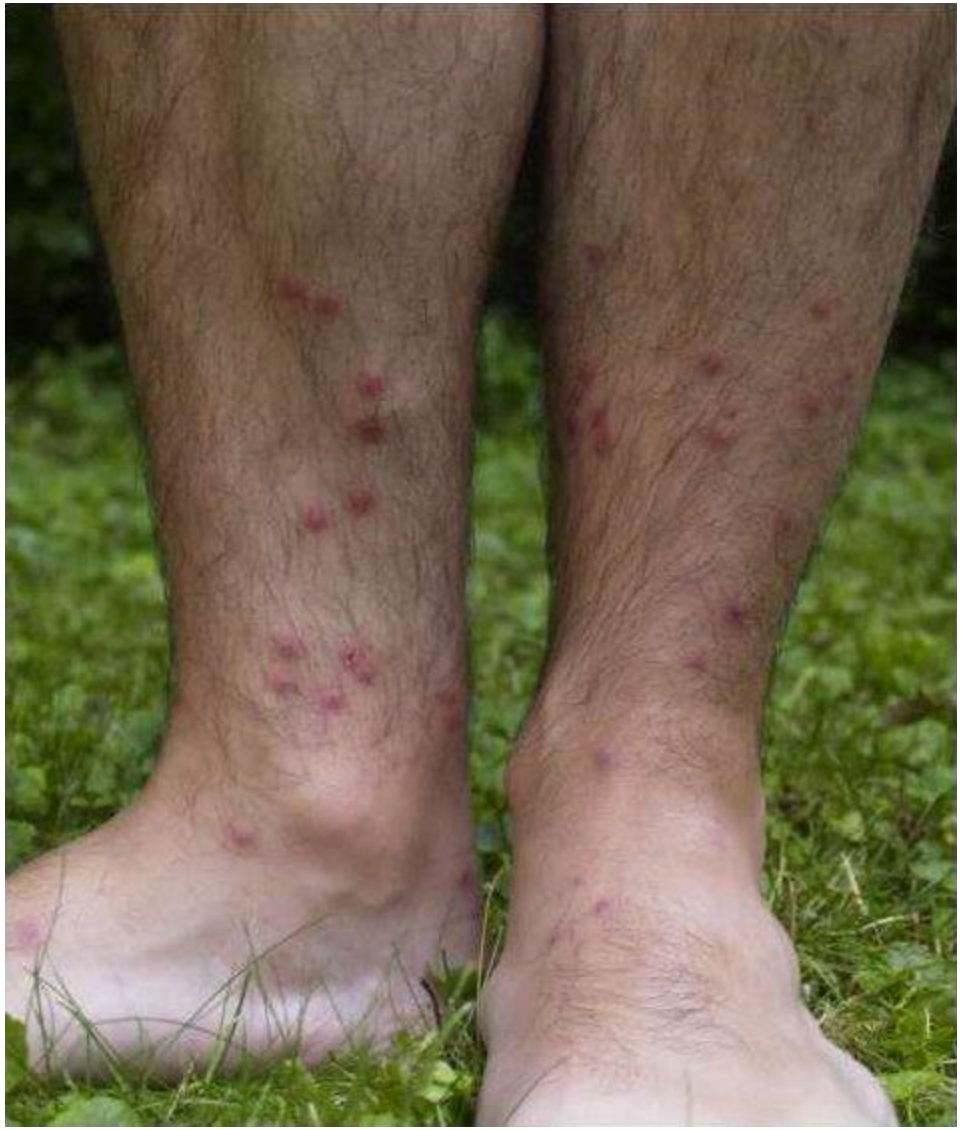
Five main stages may be defined:

1. Invasion stage;
2. Hypersensitive stage;
3. Acute intestinal stage;
4. Chronic stage;
5. Irreversible stage (chronic, hepatic, cardiopulmonary), as well as rarely seen damages of the CNS, heart and skin.

Incubation period – 3 weeks to 2 months

During the ***first stage***, a transient itching cercarious dermatitis is seen up to 24 hours following invasion.





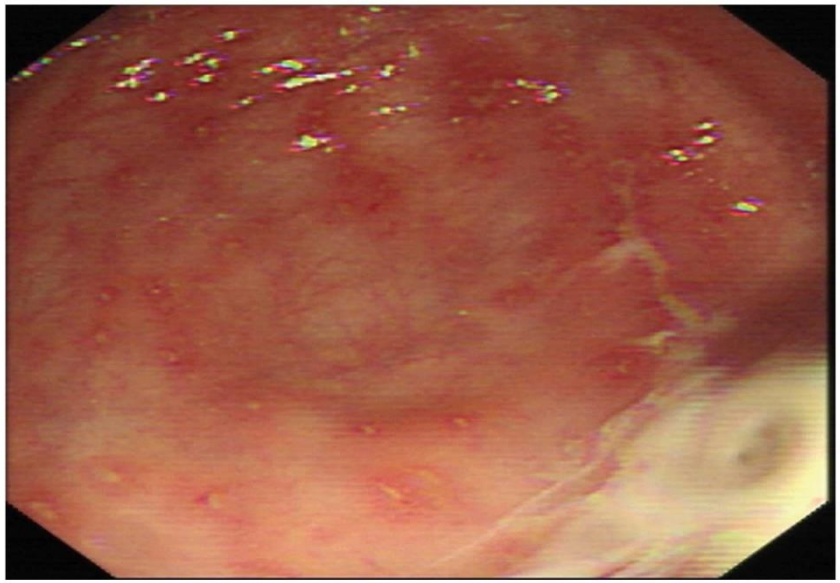
The symptoms of the ***hypersensitive stage*** appear 5-8 days after infection. The hypersensitive stage shown by:

- weakness;
- headache;
- temperature;
- asthma-like cough;
- urticaria;
- articular and muscular pain;
- loss of appetite;
- reduce in body mass.

The ***acute intestinal stage*** is connected with the deposition of eggs in the intestinal mucosa. The symptoms of the intestinal stage appear 6-8 weeks after infection.

This stage is characterized by fever, abdominal pains, dysentery syndrome (feces contain blood and mucus), tenesmus, loss of appetite and weight, hepatomegaly, spelnomegaly and lymphadenitis.

This stage lasts 6-12 months. Gradually, acute symptoms subside.



Endoscopic manifestation of acute intestinal schistosomiasis: Mucosal hyperemia and edema, mucus exudation, vague vascular striation and scattered small ulcers and yellow nodules.

Chronic stage:

During the ***chronic stage***, alternation of constipation and diarrhoea is seen, as well as rectal bleeding, rectal prolapse, hemorrhoids, intestinal polyposis, papillomatosis, and ulceration, followed by malignization, etc. In patients with intense invasion, liver fibrosis, hepatosplenomegaly, portal hypertension, and cirrhosis of Symmers develop. The cardiopulmonary form occurs when the eggs enter the lung arterioles, thus causing obstruction of the capillaries, endarteritis and thickening of the intima.

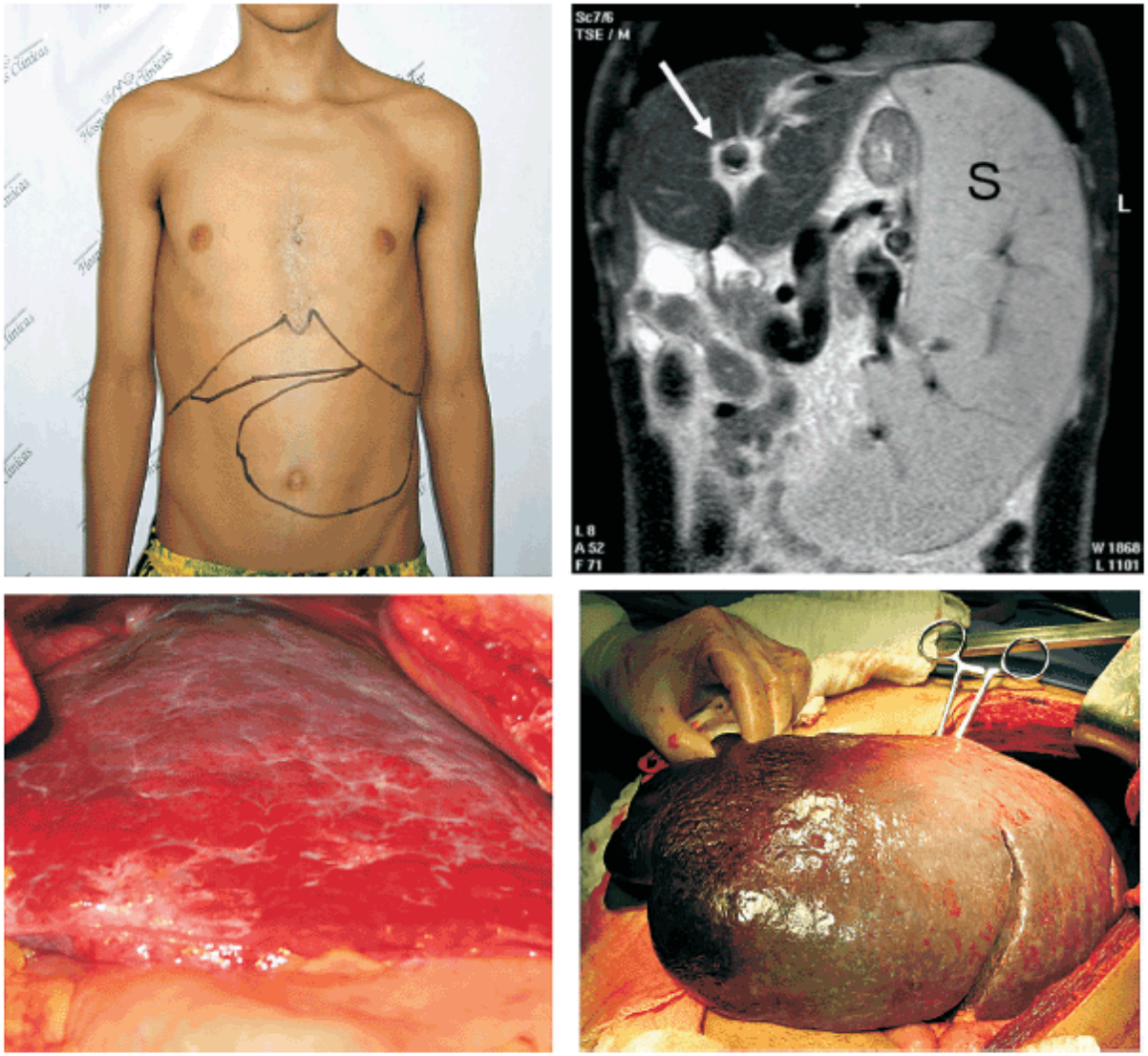
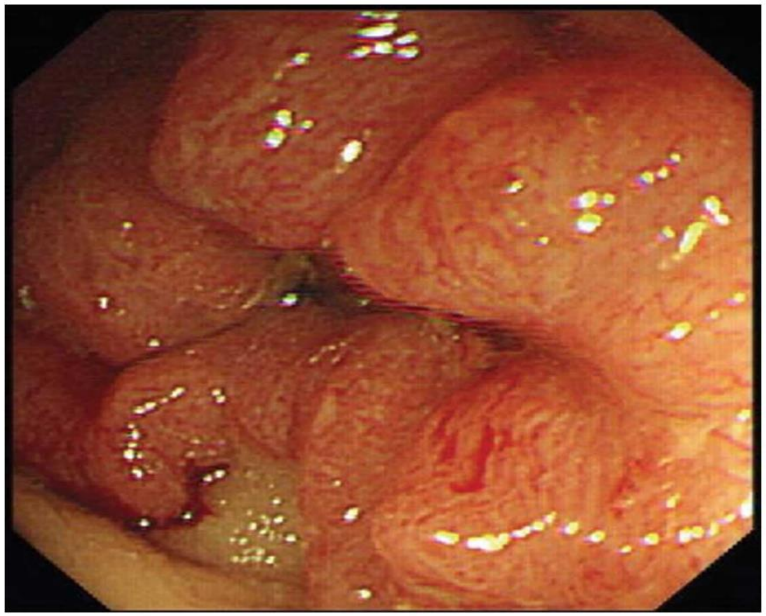
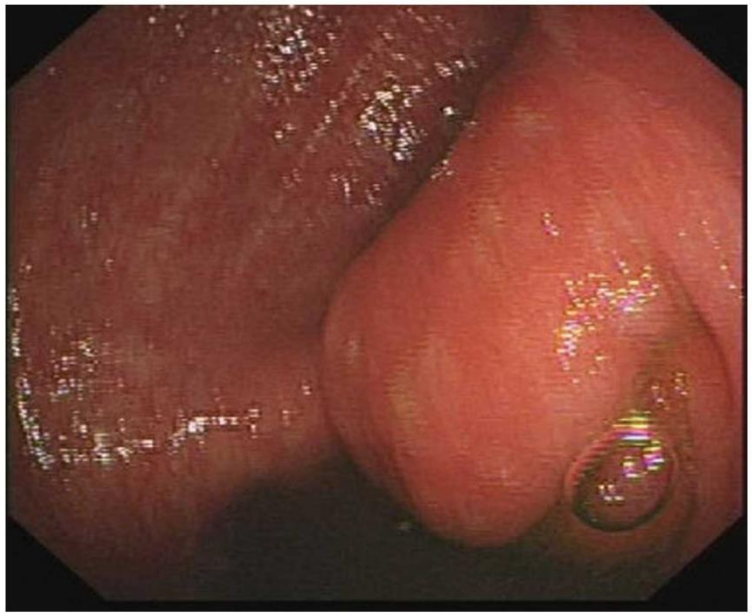


Fig. 6: a 21-year-old patient with hepatosplenic schistosomiasis. On the upper right hand side, a coronal section of the abdomen captured by magnetic resonance imaging (MRI), showing periportal fibrosis (white arrow) and a huge spleen (S). It is shown below, during surgery, the nodular surface of the liver (on the left) and the spleen (on the right).



Endoscopic manifestation of chronic intestinal schistosomiasis. The colon mucosae present with hyperaemic edema and stenosis of the colon cavity combined with colon cancer.



Endoscopic manifestation of chronic intestinal schistosomiasis, mucosal thickening and cicatrix with polypoid protrusion.

Clinical laboratory investigations reveal hypoproteinemia, increase in total IgM, elevated transaminases and other enzymes, that are typical of liver dysfunction. Signs of hypersplenism, anemia, leukopenia, thrombocytopenia are found on investigating peripheral blood.

CLINICAL SYMPTOMS OF 96 CASES OF INTESTINAL SCHISTOSOMIASIS

Clinical symptom	No. of cases (n=96)
Diarrhea	64
Mucous stool	47
Abdominal distention	39
Abdominal pain	75
Low fever, emaciation and asthenia	46
Splenomegaly	61
Bloody purulent stool	21
Constipation	23
Alternation of diarrhea and constipation	9
Abdominal mass	17
Intestinal obstruction	2

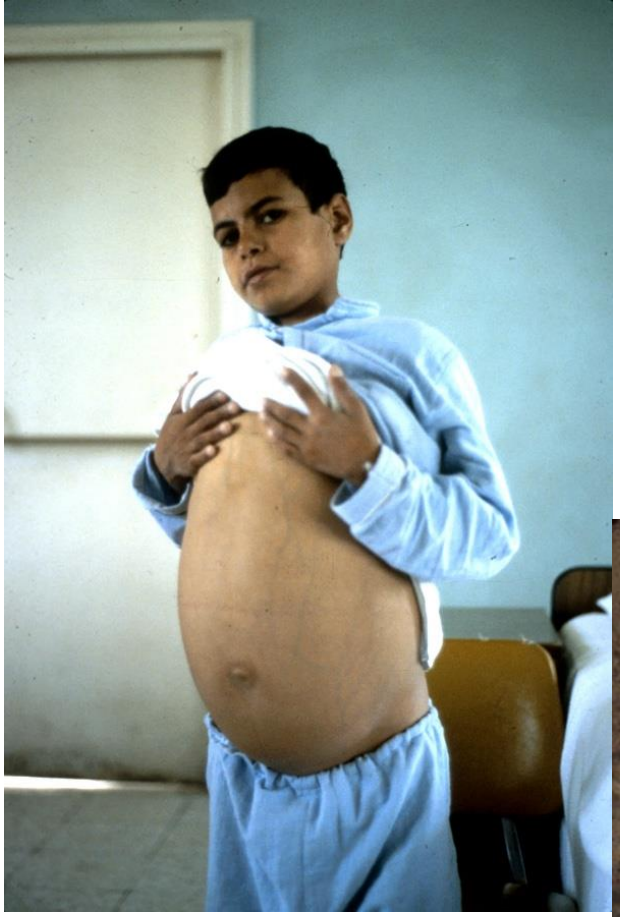
LOCATION OF PATHOLOGICAL CHANGE

Location of pathological change	No. of cases (n=96)
Rectum	47
Sigmoid colon	55
Descending colon	11
Transverse colon	8
Ascending colon	6
Cecum	9

Irreversible stage is characterized by:

- liver fibrosis;
- portal hypertension;
- fibrosis of Symmers (fibrosis of portal vein);
- esopahgeal varices;
- cor pulmonary;
- renal insufficiency;

The 2 faces of schistosomiasis

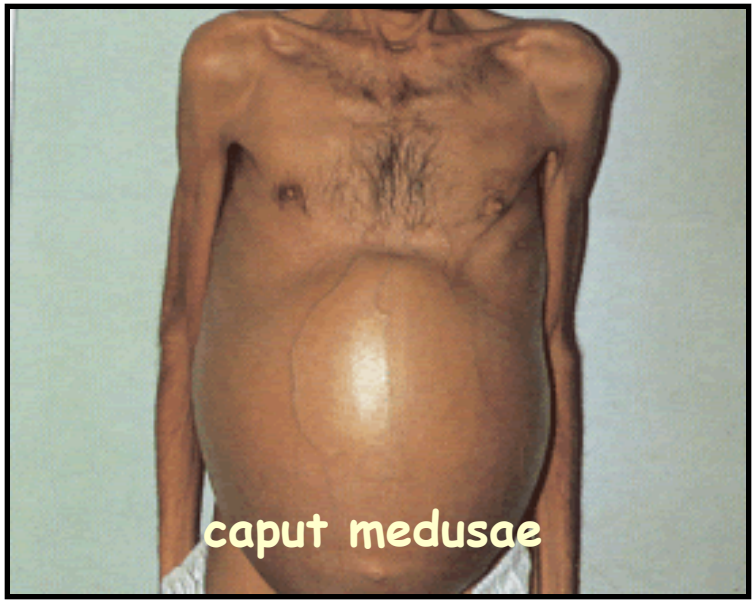


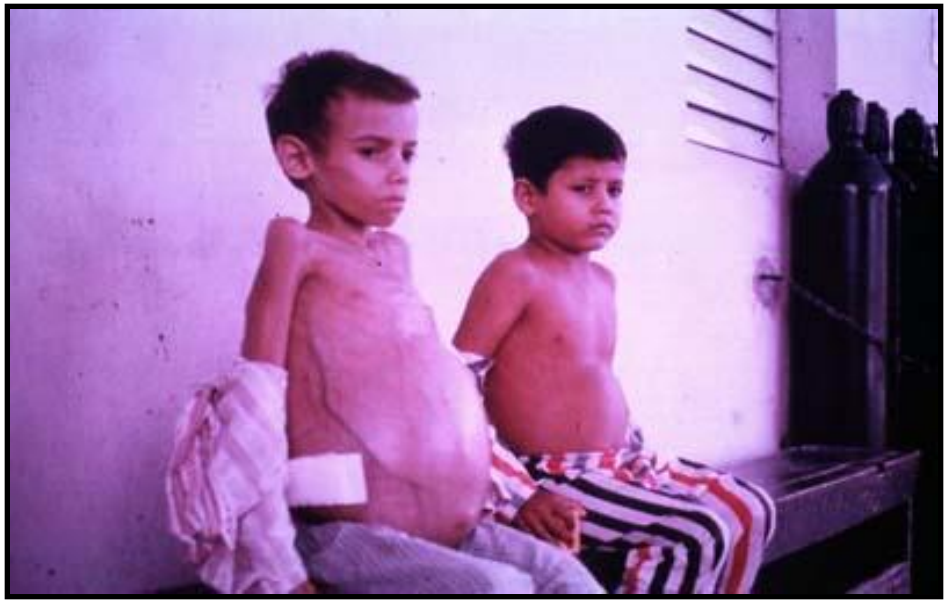
Egyptian boy with hepatosplenomegaly, ascites fluid build-up and superficial collateral circulation

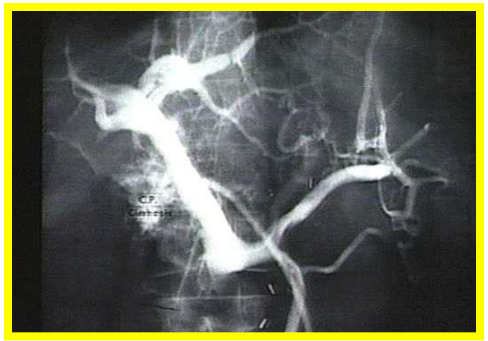
(NAMRU-3 clinical ward in Cairo)

'Intestinal' asymptomatic schistosomiasis at the Egyptian village level

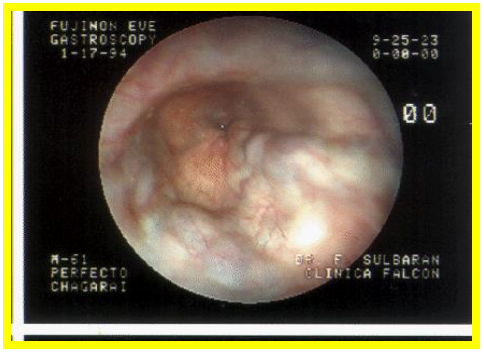
Portal hypertension in schistosomiasis intestinalis







collateral circulation in patients with schistosomiasis mansoni.



esopahgeal varices

DIAGNOSIS

The basic approaches are those applied in urogenital schistosomiasis. Rectoscopic examination is important.

Materials for investigation are stool samples, biopsy material from intestinal mucosa.

Methods of investigation.

Microscopic: applied to stool samples – sedimentation (in cases of less intensive invasion), method of Cato, native fecal smear (in highly intensive invasion), staining with hematoxylin-eosin of biopsy samples.

Serological - ELISA, Western blotting, etc.

TREATMENT

Treatment should be given to all persons with the parasitic invasion, regardless of the clinical form.

Etiological therapy. The drug of choice is ***Praziquantel*** (40 mg/kg b.w.) in a single dose. The drug is effective in the acute stage and in patients in whom the liver and spleen are affected.

Oxamiquine (caps. 250 mg; syrop 250 mg/5 ml) in doses of 15-30 mg/kg b.w., twice a day, and for children at a dose of 20 mg/kg b.w., twice a day.

EPIDEMIOLOGY

Source of infection. The source is the definitive host. In most cases, this is an infected individual. However, some animals such as cattle, rodents, dogs and other mammals play a certain role in the transmission of the parasitosis.

Mechanisms, factors and routes of transmission.

They are not very different from those in urogenital schistosomiasis. Humans get the infection through the skin when in contact with cercariae. The intermediate host is a water snail of the genus ***Biomphalaria***.

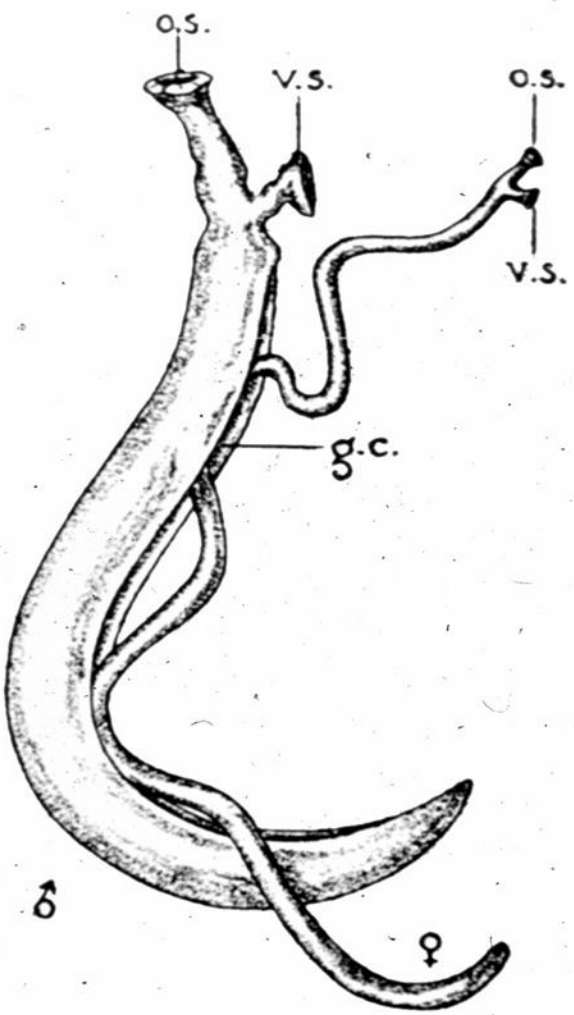
Susceptibility. All people are susceptible.

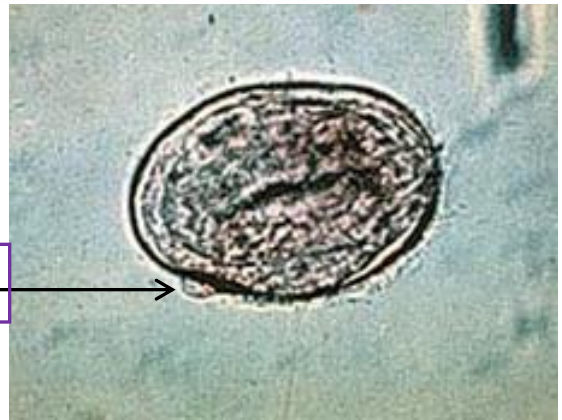
SCHISTOSOMIASIS JAPONICA

KATAYAMA FEVER

ETIOLOGY

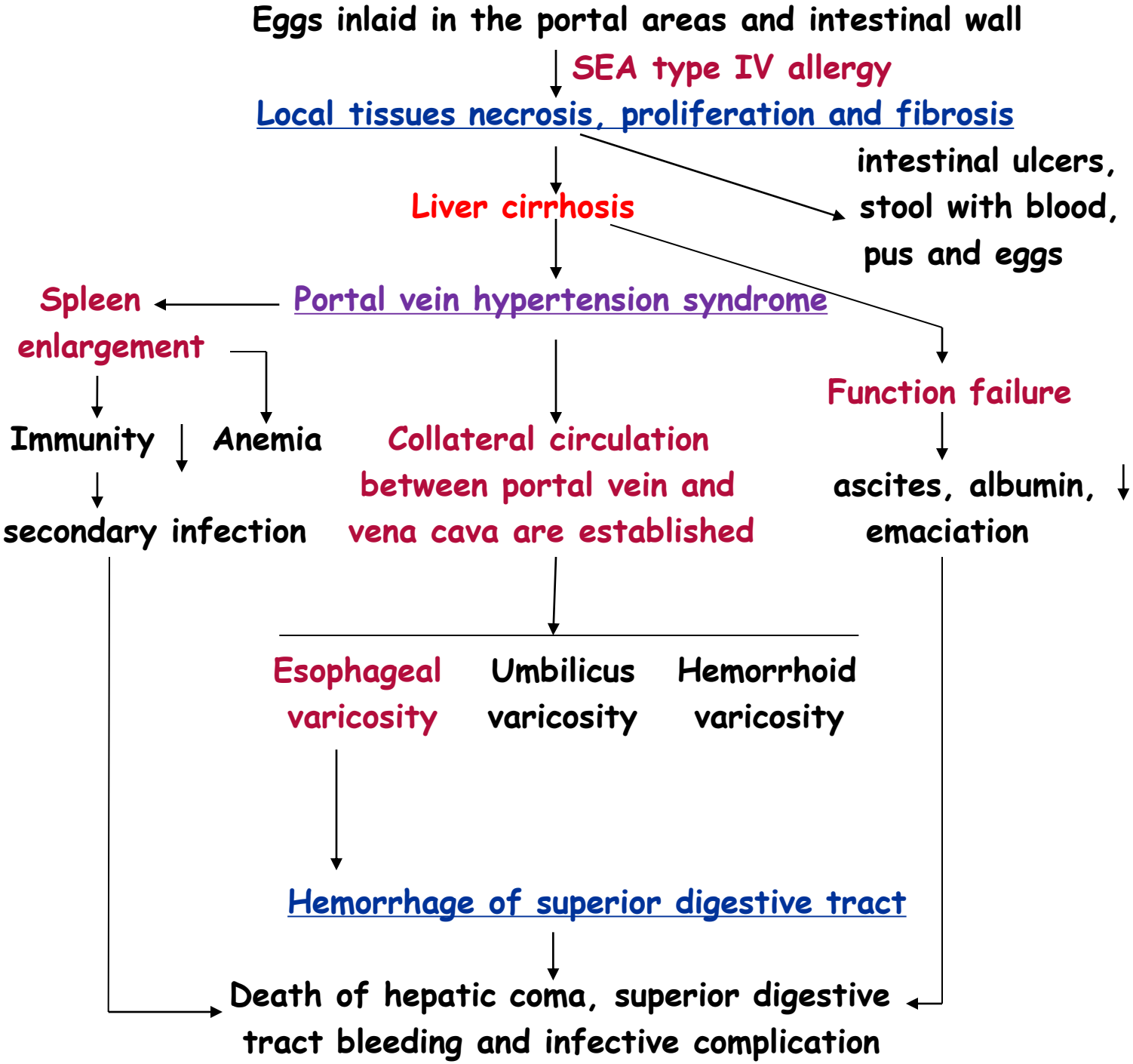
It is caused by ***Schistosoma japonicum***. The female parasites are 12-28 mm x 0.3 mm in size, and the male ones - 12-20 mm x 0.5 mm. The eggs (70-100 μm x 50-65 μm) are round, but less elongated than those of the other schistosomes. They are pale yellow, with a small delicate lateral bud and contain a ciliary embryo. The female parasite produces 3000 - 5000 eggs a day.





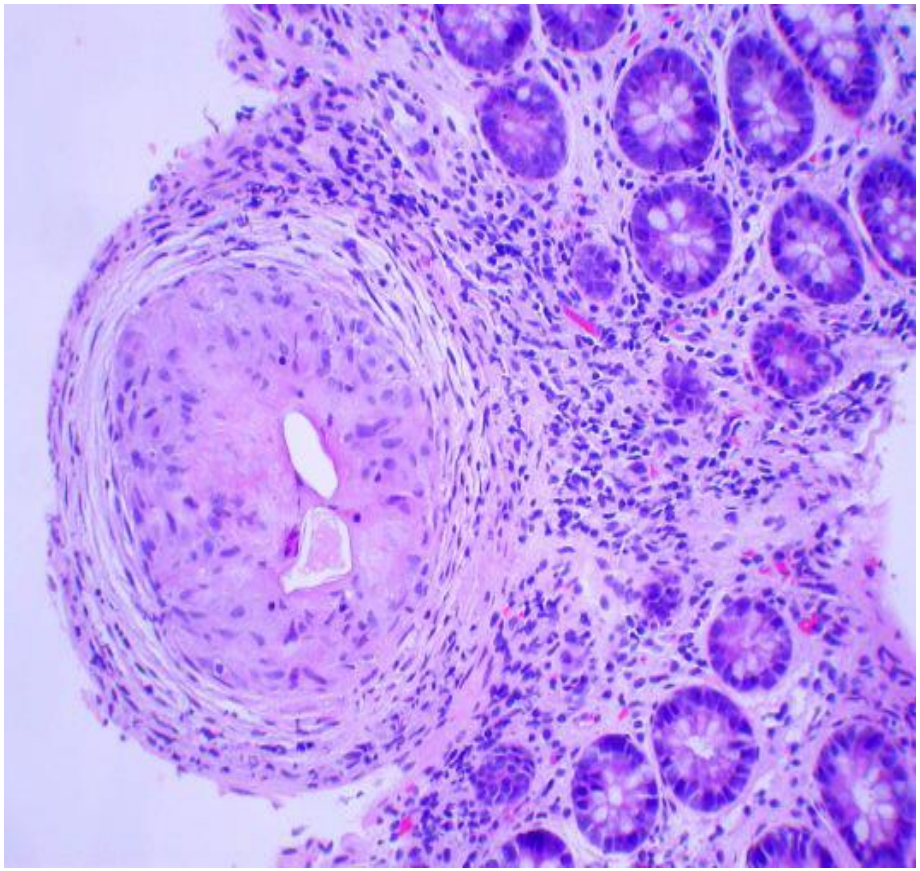
PATHOGENESIS AND PATHOLOGY

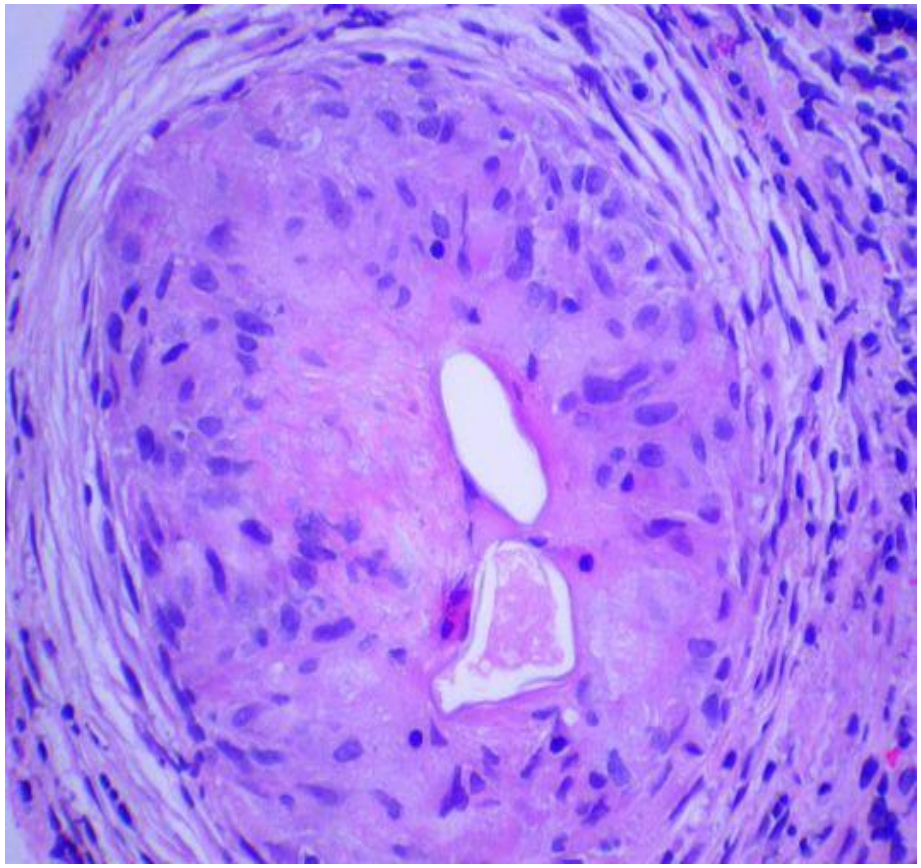
These are similar to those in intestinal schistosomiasis. However, because of the large numbers of eggs produced and probably the more intensive antigenic irritation, the pathological changes in the intestinal tract and the liver appear earlier and are more expressed. The acute and chronic disease is characterized by a granulomatous reaction as a result of the eggs, whose presence leads to fibrosis in organs supplied only by the mesenteric-portal system. Fibrosis (fibrosis of Symmers) of the portal vein is seen. Portal hypertension causes splenomegaly and disturbance of collateral circulation with formation of esophageal varices, etc.


Eggs inlaid in the portal areas and intestinal wall
SEA type IV allergy
Local tissues necrosis, proliferation and fibrosis
intestinal ulcers, stool with blood, pus and eggs
Liver cirrhosis
Spleen enlargement
Portal vein hypertension syndrome
Function failure
Immunity
Anemia
Collateral circulation between portal vein and vena cava are established
ascites, albumin, emaciation
secondary infection
Esophageal varicosity
Umbilicus varicosity
Hemorrhoid varicosity
Hemorrhage of superior digestive tract
Death of hepatic coma, superior digestive tract bleeding and infective complication

Chronic granuloma (pseudotubercle) surround the egg of

Schistosoma japonicum





CLINICAL PRESENTATION

Clinical manifestations are similar to those in Mansoni disease. However, as compared to the rest of intestinal schistosomiasis, the *schistosomiasis japonicum* has the most dramatic course.

There are *asymptomatic*, *mild*, *severe, very severe and fulminant* forms.

Three clinical stages may be defined: invasive stage, acute and chronic stage.

The penetration of cercariae into the skin causes localized inflammation and pruritus known as *cercarial dermatitis* or *"swimmer's itch".*

CLINICAL PRESENTATION

More commonly, successful cercarial entry to the body and development of larvae in the bloodstream brings about a self-limiting febrile illness known as *"snail fever"* or *Katayama fever* – acute stage.

This can be accompanid by *more severe symptoms* : fever, cough, eosinophilic lung infiltrates, abdominal pains, marked eosinophilia, dysentery syndrome (diarrhoeal stools, mixed with blood and mucus), arthralgias, myalgias, lymphadenopathy, well-expressed hepatosplenomegaly etc .

CLINICAL PRESENTATION

Schistosoma japonicum causes chronic pathology when the adult worms find their way into the portal circulation. A mated pair of worms produces 300 to 3,000 eggs per day, which are released into the capillaries and portal veins.

Bleeding and polyp formation in the bowel wall are thus common complications of egg transfer from the venule to the bowel lumen. The process of local inflammation can lead to protein loss, iron loss, anemia of chronic disease, diarrhea and in some cases, intestinal obstruction.

CLINICAL PRESENTATION

More than half of the worm eggs become permanently trapped in host tissues, where they each evoke an immune-mediated inflammatory granuloma. This is especially apparent in the liver, where a classic pattern of periportal *"pipestem" fibrosis* is manifested, which leads to portal hypertension and its sequelae of varix formation, ascites and splenomegaly.

As portal hypertension increases, eggs are shunted into the pulmonary circulation, where local fibrosis causes pulmonary hypertension, which may lead to cor pulmonary.

CLINICAL PRESENTATION

Eggs reaching the central nervous system lead to local granuloma formation, which can cause seizures, paresis and paralysis of the extremities, diffuse encephalitis, meningitis or meningo-encephalitis, etc.

Lethal outcomes are reported resulting from hepatic and renal insufficiency, bleeding from esopahgeal varices, damages to CNS, etc.

Fulminant forms have been described with lethal outcome.

DIAGNOSIS

The symptoms, signs and history of living in endemic areas only give a presumptive diagnosis. The definitive diagnosis depends on the pathogen examination.

1. Stool examination

(1) Direct fecal smear for acute stage - method of Cato, native preparation (in highly intensive invasion)

(2) Concentration method: sedimentation (in cases of less intensive invasion).

2. Biopsy can be done by proctoscope for terminal stage.

3. Immunological tests are subsidiary for reference only.

TREATMENT

Treatment should be given to all persons with the parasitic invasion, regardless of the clinical form.

Etiological therapy: The drug of choice is ***Praziquantel*** (40 mg/kg b.w.) in a single dose.

Alternatively - *Oxamniquine* (**Vansil**) caps. 0,250
15-30 mg/kg once or twice.

EPIDEMIOLOGY

Sources of infection. These are some wild and domestic animals – rodents, dogs, cats, cattle, swine, horses, sheep and infected humans. In some regions, e.g. the Philippines, wild animals are a major reservoir of infection.

Mechanisms, factors and routes of transmission.

As in the rest of the schistosomiasis. The intermediate host is a water snail of the genus *Oncomelania*.

Susceptibility is universal.

OTHER SCHISTOSOMIASIS

Several other schistosomas are known that cause disease in human that are less spread.

Schistosoma mekongi. The female parasite (12 mm x 0.23 mm) and the male one (15 mm x 0.4 mm) live in the mesenteric veins. The eggs are ellipsoid (40-45 μm in diameter), with a small button-like hook at one end.

Data on pathogenesis, pathology and clinical presentation are still scarce but they are like those in *S. japonicum*.

So far, *S. mekongi* has been detected in humans and dogs as definitive hosts and sources of invasion. The intermediate host is a water snail of the genus *Tricula aperta*, living in the main tributaries of the Mekong river in Laos and Cambodia.

OTHER SCHISTOSOMIASES

Schistosoma matthei. The eggs of the parasite are elongated in shape, 210-240 μm x 40-70 μm in size, with a hook-like formation at one end. They are excreted in the environment with the stools and urine. The adult parasites inhabit the mesenteric veins. *S. matthei* is a parasite mainly detected in sheep, cattle, buffaloes and wild African animals. It is relatively rarely seen in the local population of South Africa, Zimbabwe and Zambia. The intermediate host is a water snail of the genus ***Bulinus***.

OTHER SCHISTOSOMIASES

Schistosoma bovis. This parasite is a representative of the *S. haematobium* complex. The eggs (230-380 μm x 70-90 μm in size) are more elongated and narrower than those of the *S. haematobium* in the central part, and possess a big hook-like appendage at one end. An intermediate host is a water snail of the genus ***Bulinus***. *S. bovis* is often detected in sheep, cattle, goats and horses in South Europe, Israel, Africa and Iraq. Isolated cases of human disease have been reported with detection of eggs in urine and stool in South Africa, Zimbabwe and the Democratic Republic of the Congo.

Media from various sources available online, has been used in this presentation. They belong to their respective authors.