Index

SUPERFAMILY - SCHISTOSOMATOIDEA :-

General characters

(1.) The schistosomes are dioecious trematodes i.e the sexes are seperate

(2.) Males are shorter and stouter than females; lateral margins of male are folded ventrally to form a gynaecophoric canal in which the females are received.

(3.) Suckers are armed with delicate spines.

(4.) The muscular pharynx is lacking.

(5.) Intestine caeca reunite behind the ventral sucker to form a single canal; the length of the reunited intestine intestine varies in different species.

(6.) The number of testes in the male varies from 4 to 8.

(7.) Intestinal caeca reunite behind the ventral sucker to form a single canal; the length of the reunited intestine varies in different species.

(8.) Egg are non-operculated and when laid are fully embryonated containing a ciliated embryo, miracidium).

(9.) Cercariae have bifid tails and penetrate into the definitive host through the unbroken skin. There is no encysted metacercarial stage

(10.) Adult worms lives in the lumen of the portal

vein and its radicles.

3 Species Infecting Man :-

- Schistosoma haematobium
- Schistosoma mattheei
- Schistosoma intercalatum
- Schistosoma malayensi

Immunology :-

The antigen of living adult schistosome worm provides the major stimulus for provoking acquired immunity. In experimental animals a partial acquired immunity can be produced and in man a similar phenomenon can occur. In endemic areas schistosomiasis is mainly a disease of the young and the immunity develops gradually, taking several years to become pronounced with advancing age the resistance develops and there is decreased passage of eggs with lessening of association symptoms. The host thus acquires age the resistance develop and there is decreased passage of eggs with lessening of association symptoms. The host thus acquired some degree of resistance against reinfection, but it is unable to kill off the established population of the worms from primary infection. The adult worms which provoke immunity are not attacked by host's immune system because the worms living in the host incorporated host's material (antigen) into their cuticular tissues which are not attacked by host's antibodies.

Different Features of Schistosomes :-

	S. haematobium	S. mansoni	S. japonicum
Male :-			
Size	1 to 1.5 cm by 1 mm	1 cm by 1 mm	1.2 to 2 cm by 0.5 mm
Cuticula	Finely tuberculated	Grossly tuberculated	Non-tuberculate.
Testes	4 to 5; in group	8 to 9 in a zigzag row	6 to 7; in a single file
Female :-			
Size	2 cm by 0.25 mm	1.4 cm by 0.25 mm	2.6 cm by 0.3 mm
ovary	Behind the middle of the body	Ant. to middle of body	In the middle of body
Uterus	Contains 20 to 30 eggs	Contains 1 to 3 eggs	contains 50 or more egg
Recurved :-	long (recurving about the middle of body)	Longest (recurving in the ant. half of body)	Short (recurved in the post. fourth of the body)
Intestine	150 by 50 um; terminal spines, 3 pair basophilic	150 by 60 um; lateral spines, 2 pairs oxyphilic & 4 pairs basophilic	100 by 65 um; lateral knob. 5 pairs of oxyp-hilic (no basophilic).
Egg :- (Cephalic gland)			
Intermediate :-	Bulinus (Physopsis) and	Biomphalaria and Australorbis	Oncomelania.
Snail host :-	Planorbarius	—	—
Definitive host :-	Man	Man	Man
Geographical Distribution :-	Africa, Near East & Middle East	Africa & south America	Far East (Oriental)
Habitat :-	Vesical & prostatic venous plexus	Mesenteric plexus of sigmoido-rectal area (inferior mesenteric vein & its radicles)	Mesenteric plexus of ileocaecal area (superior mesenteric vein and its radicles

5 Schistosoma haematobium

Common name :- The vesical blood fluke.

Geographical distribution :- Various parts of Africa and Middle East and few cases in India from Ratnagiri in Maharastra state.

Habitat :- Adult worm live in copula, in the pelvic venous plexus - vesical, prostatic, and uterine plexuses of veins.

Morphology :-

In general, the three species of adult worms resemble each other closely; the peculiarities and differentiating features are however shown in table.

These are long living worms, having a life span of 20 to 30 years.

Mechanism of egg laying :-

Oviposition usually occurs in the small venules of vesical plexus. The female, held in the gynaecophoric canal of the male, extends its anterior end far into the smallest venules and deposits the egg immediately longitudinally, one at a time. Each time an egg is laid, the worm withdraws a short distance and lay another egg immediately behind the first. In this way, the venules are filled with egg pointing backwards; the worm in copula migrate to an adjacent venule. The egg are held in position by the spines and by the contraction of vessels resulting from the withdrawn of the parent worm. The eggs then work their way

through the vessels and the mucosa of the urinary bladder, enter into the cavity and escape with the urine, usually at the end of the micturition

Life Cycle :-

It passes it's life cycle in 2 hosts.

Definitive host :- Man. Adult worm living in Vesical and prostatic venous plexus.

Intermediate host :- Fresh water snail (Bulinus truncatus and other species throughout Africa, Planorbarius metidjensis in Morocco and Portugal, and Ferrissia tenuis in India)

The embryonated eggs are passed with the urine of the definitive host and gain access to water. Ciliated larval (miracidia), hatched out of the eggs, move freely in water in search of their intermediate host. The miracidium on entering the proper larval host penetrates into the soft tissues of the snail and ultimately makes its way into the liver. Here it losses its cilia and other organs and in the course of 4 to 8 weeks undergoes developmental changes. The miracidium is transformed into a tubular sporocyst; the latter multiplies and forms a second generation of sporocysts. Several weeks after the infection, when no further multiplication occurs, the daughter-sporocysts give rise to the final larval forms, the forked tailed cercariae which are injected to man. The cercariae break off from the sporocyst and escape from the snail into water.

Infection results when human beings bathing or wad-

-ing in this water are infected, the cercaria penetrating the unbroken skin directly. On entry the cercaria cast off their tail and gain access to a peripheral venule. From here, they are carried through the right heart into the pulmonary capillaries. It requires some days for the larvae to pass through the capillary bed in the lungs, whence they are carried through the left heart into the systemic circulation. The majority are shunted in the abdominal aorta and gain access to the mesenteric artery, pass through the capillary bed in the intestine and enter portal circulation. In the intrahepatic portion of the portal blood stream, the larvae grow into adult. After becoming sexually differentiated, they move out of the liver against the blood current, migrating into the inferior mesenteric vein, rectal venous plexus, pelvic vein, & eventually enter the vesicle plexus of vein. It takes about 1 to 3 months for the worms to reach the vesical and pelvic plexuses of veins after the initial exposure of the skin. When the worms are sexually mature, they copulate and the fertilised female lay eggs which are ultimately voided with the urine. The cycle is thus repeated.

Pathogenicity:-

An individual bathing in an infected pool or coming in contact with contaminated water is labile to be infected. The cercariae stick to the surface of the skin of the swimmer or sa-ther, by means of their ventral suckers and as the water begins to evaporate, penetrate the skin.

Infecting Agent :- Cercaria. These have a free-swimming existence and can live in this state for a maximum period of 3 days.

Pathogenesis :- Skin. (Portal of entry)

Site of localisation :- Vesical plexus of vein (urinary bladder)

The terminal-spined eggs of S. haematobium may erode blood vessels and cause haemorrhages. Schistosoma eggs, deposited in the tissues, act like foreign protein and have an irritative effect leading to round cell infiltration and connective tissue hyperplasia. The tissue reaction in these cases produces what is known as formation of a "pseudotubercle" around each egg. The early nodules are highly cellular and are composed of eosinophils, giant cells, monocytes and lymphocytes; later on, the cellular reaction tends to disappear and is replaced by a whorl of fibrous tissue, in the centre of which degenerated and calcified eggs may be found. Large and progressive granulomas are found only around the eggs & may cause a diffuse fibrosis

Clinical features :-

The disease caused by schistosoma haematobium is referred to as schistosomiasis haematobia. Evolution of this disease passes through 3 phases :-

(1.) By the cercariae at the site of entrance : Local reaction (dermatitis). This is particularly seen with the cercariae of non-human schistosomes.

(2.) By the toxic metabolites liberated during the growth of schistosomulae in the portal blood of the liver.

(ii.) General anaphylactic reaction characterised by fever, urticaria, eosinophilic leukocytosis, enlarged tender liver and palpable spleen. The symptoms appears b/w 4th & 5th week of infection

(iii.) At the time of laying eggs: They may be regarded as a localising symptom, generally occuring within 3 to 9 months of the infection. The characteristic manifestation is a painless terminal haematuria. In course of time, the adjacent structure of uro-genital apparatus are involved, at first by the reversible granulomatous inflammatory reaction to eggs and later by the irreversible fibrosis and calcification.

Diagnosis :-

(a.) A microscopical examination of urine

(b.) Examination of stool

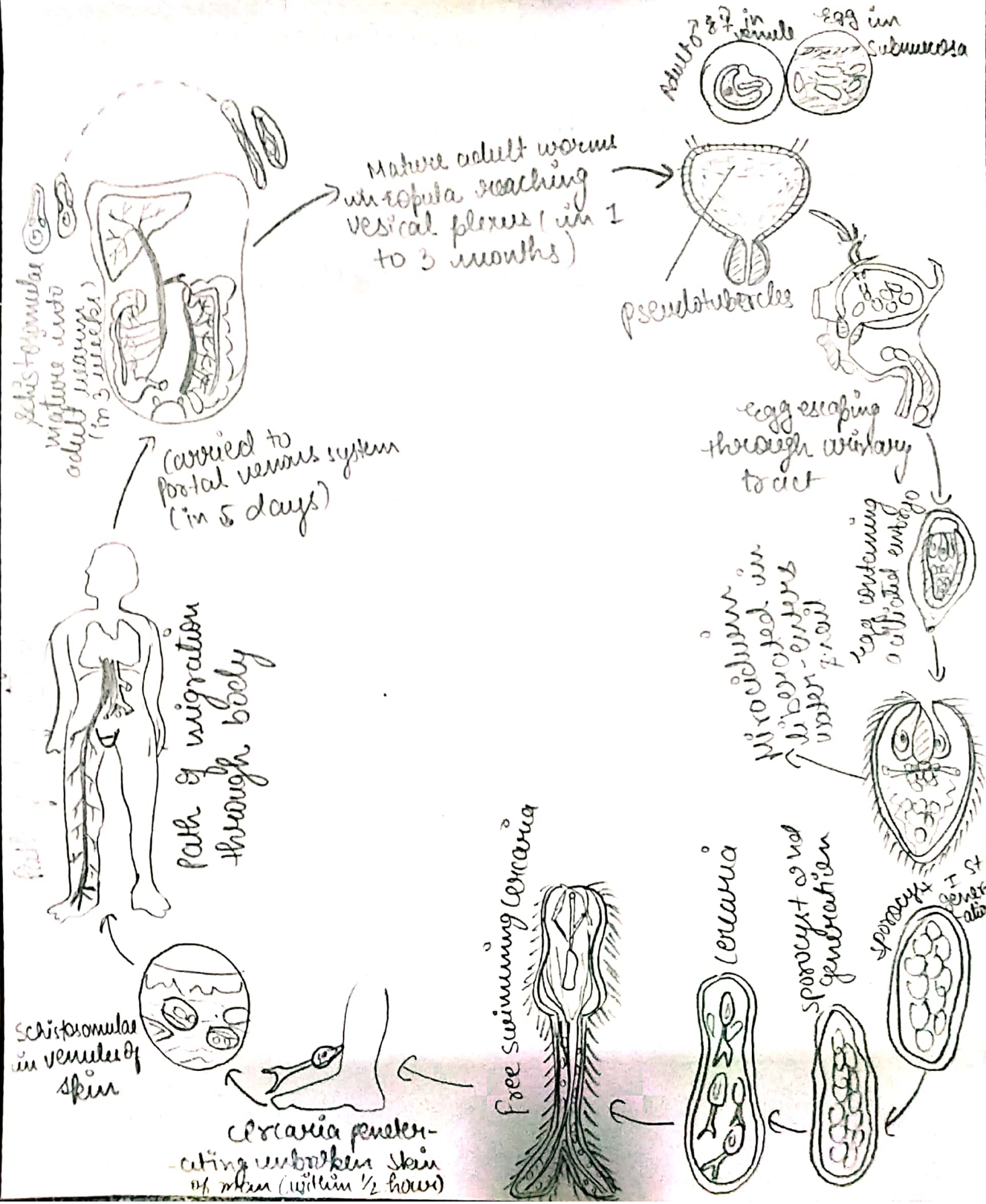
(c.) A piece of vesical mucosa is removed by cystoscopic biopsy.

Other Tests :-
- Eosinophillic count
- Aldehyde Test
- complement fixation test
- Intradermal skin Test

Treatment :- praziquantel (40 mg/kg/day in 2 divided doses for 1 day)

metrifonate (single dose of 7.5 mg to 10 mg/kg body weight, weekly for 3 week)

Praziquantel is more effective drug than metrifonate.



Prophylaxis :-

(i.) eradication of this disease in man
(ii.) prevention of pollution of water with human excreta
(iii.) destruction of the snail vector in endemic area
(iv.) advance of swimming, bathing, wading, in infected water.

Schistosoma mansoni :-

Common name :- Manson's blood fluke

Geographical Distribution :-
various parts of Africa and south america

Habitat :- Adult worms in the mesentric veins of the sigmoid-rectal area; also in the branches of the portal vein in the liver.

Morphology :- Same as S. haematobium

Life Cycle :-

- Definitive host :- Man
- Intermediate host :- Fresh-water snail (Biomphalaria alexandrina in Africa and Australorbis glabratus in south America)

The cycle is from man to snail via water and from snail to man again via water.
The schistosomulae are carried to the liver (the route taken being the same) where they feed upon portal blood and develope into adult worms. Their subsequent behaviour differs and they migrate against the blood stream into the inferior mesenteric vein to reach the capillaries of the sigmoid-rectal area where the eggs are laid. These finally escape through the faeces.

Pathogenicity :- The disease caused by S. mansoni is designated as schistosomiasis mansoni; also known as intestinal bilharziasis, schistosomal dysentry. The visceral

form is known as visceral schistosomiasis or Egyptian splenomegaly.

The ectopic lesions include hepatomegaly, periportal cirrhosis portal hypertension, cor pulmonale & myelitis

Diagnosis :-

- A microscopical examination of faeces
- A pieces of rectal tissue removed by rectal biopsy

Other Testes :-

Visceral schistosomiasis

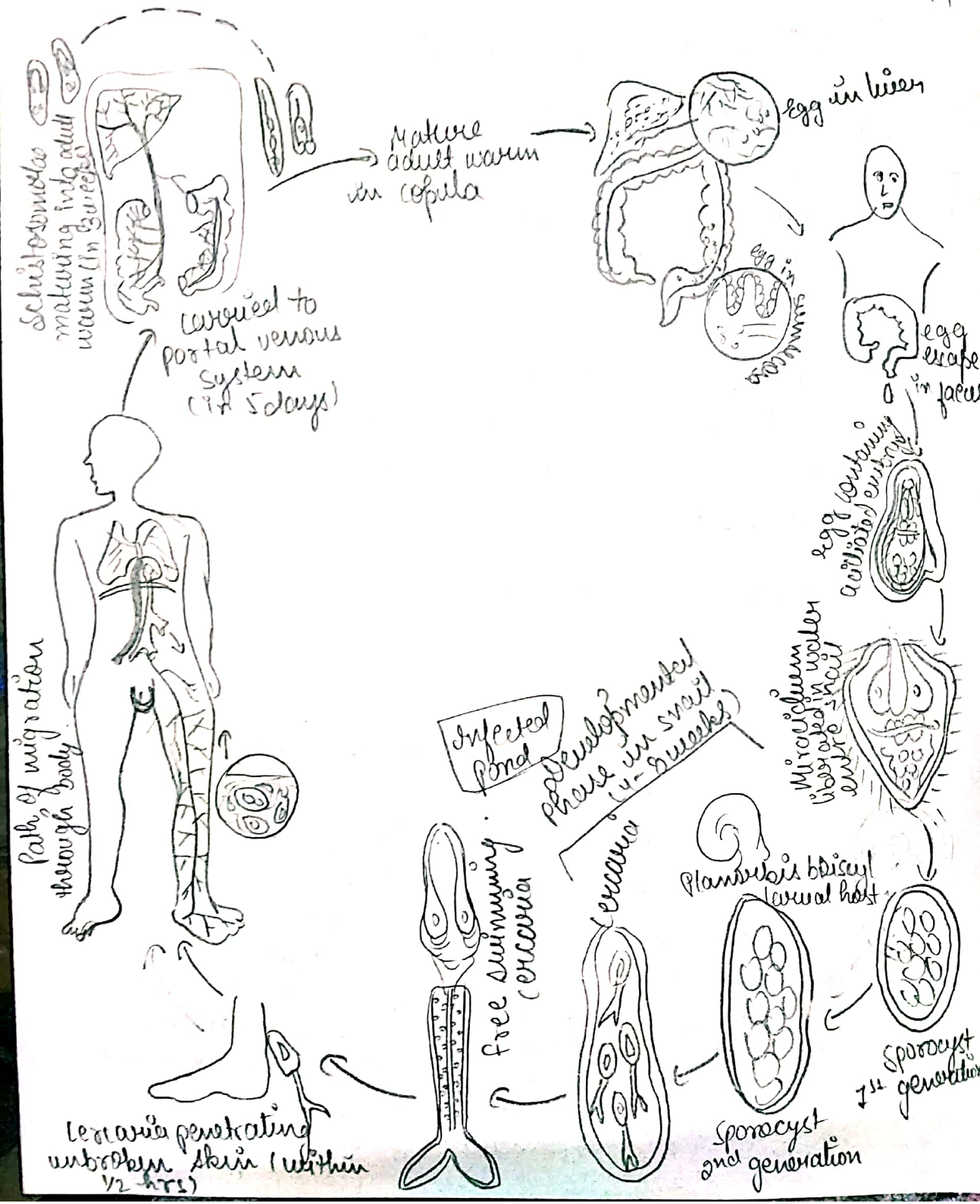
Treatment :-

- Praziquantel (40 mg/kg/day in 2 divided doses for 1 day)
- metrifonate (single dose of 7.5 mg to 10 mg/kg body weigh, weekly for 3 week)
- Parazipcantel is more effective drug than metri-fonate.

→ Prophylaxis :-

(i.) eradication of this disease in man
(ii.) Prevention of pollution of water with human excreta
(iii.) destruction of the snail vector in endemic area
(iv.) advance of swimming, bathing, in infected water

other drugs are :- Oxamniquine (15-20 mg/kg as single dose is also effective.



Schistosoma japonicum

Common name :- The Oriental blood fluke

Geographical Distribution :-
A parasite of the Far East - being found in China, Japan, Southern Formosa, Philippines, Celebs and Shan states of Burma.

Habitat :-
Adult worms are found in following places :-
(i.) Intrahepatic portion of the portal venous system
(ii.) Mesenteric veins draining the ileo-caecal region.
(iii.) Rectal plexus of vein

Morphology :- same as S. haematobium

Life cycle :-
Definitive host - Man Domestic animals (cat, dog, pig & cattle) & field mice serve as reservoir
Intermediate host - fresh-water snail of genus oncome-lania (Katayama or Blanfordia)

The schistosomulae of S. japonicum is carried to the liver exactly in the same way as those of S. haematobium. They grow into adult worms and becomes sexually mature inside the intra hepatic portion of the portal venous system but subsequently migrate against the blood stream into the superior mesenteric vein, down to the capillaries of the last part of the ileum, caecum and ascending colon. The egg finally escape

through the faeces.

Pathogenicity & Clinical Features :-

This disease also known as Katayama disease. An individual bathing in an infected pool or coming in contact with contaminated water is liable to be infected. The cercaria stick to the surface of the skin of the swimmer, by means of ventral suckers and as the water begings to evaporate, penetrate the skin.

NOTE :- The lesions produced in schistosomiasis japonica are much more pronounced than in S. mansoni, because the larger output of eggs, the localising symptom is chiefly intestinal, involving the ileo-caecal region and is manifested by dysentric attacks. Furthermore on account of the proximity of its location to the liver, the chances of the liver being involved are greater.

Periportal cirrhosis in liver, it is the granulomatous fibrosis pylephlebitis with terminal scarring developing round eggs with which lodge in the smallest portal vein/venules. As a result, the liver lobules are not affected & there is no nodular regeneration of the liver cells. A characteristic feature is the intense new formation of blood vessels in the portal field giving an "angiomatoid" appearance. Pigmentation due to deposition of regurgitated haematin pigment similar to malarial pigment, inside the Kupffer's cells may be found. Haematin pigment is regurgitated by the parent worm after digestion of blood in the its alimentary canal, because the intestine terminates blindly.

Schistosomal Hepatic Fibrosis:-

Chronic hepatic lesions in schistosomiasis is not a true portal cirrhosis and is thus, different from that of Laënnec's cirrhosis or other types of cirrhosis. It is a confluent progressive fibrosis which is the outcome of sclerosis of many egg granulomas.

Spleen:- Enlargement of the spleen is due to mechanical factor and results from pre-sinusoidal portal hypertension through intrahepatic block of small portal venules by fibrosis. Schistosomal lesions are not found in the spleen.

Oesophageal Varices:-

A consequence of portal hypertension; rupture leads to massive haematemesis.

Brain (space occupying lesion) and lungs (cor pulmonale) may also be involved

Diagnosis:-

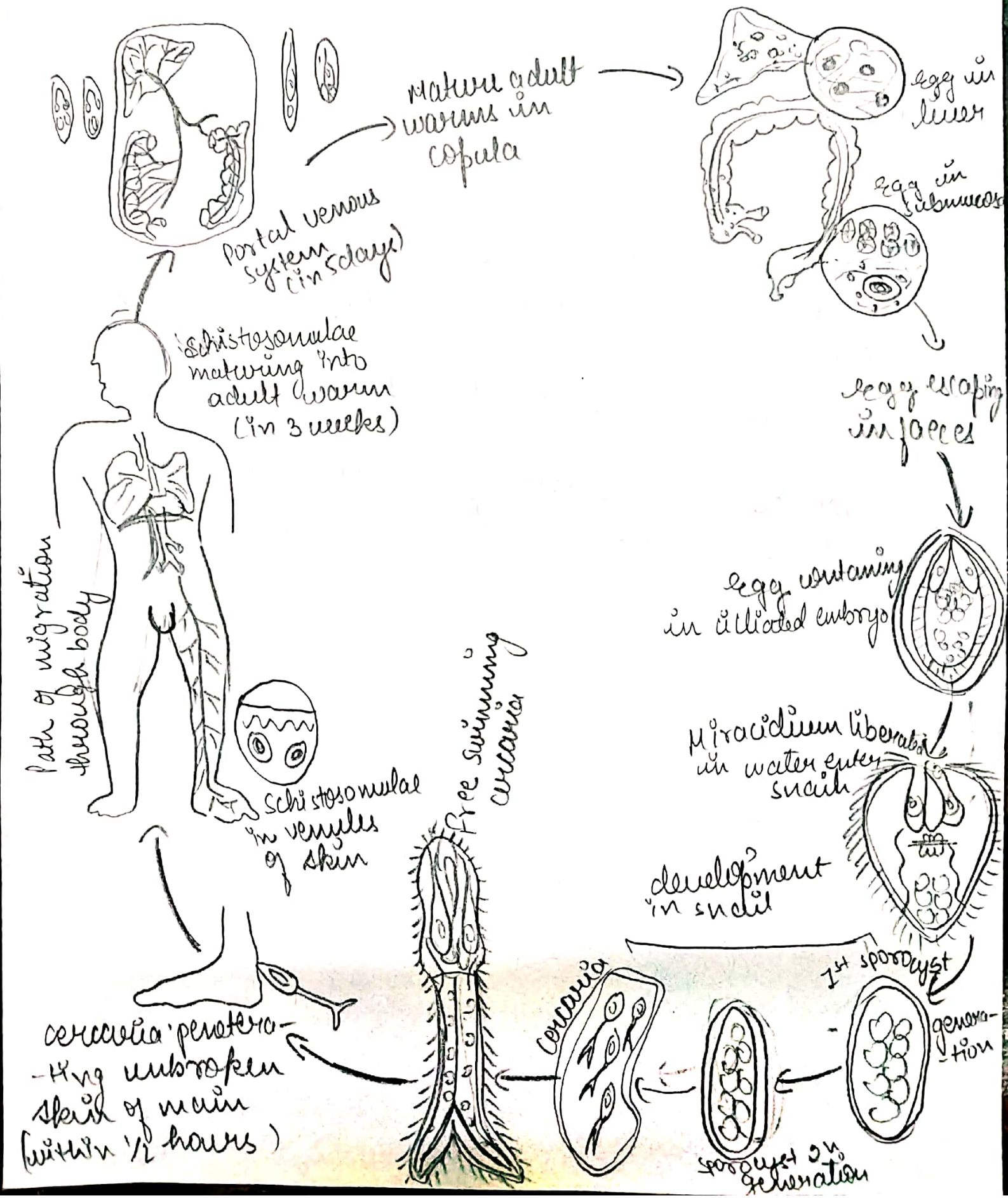
- A microscopical examination of faeces.
- A pieces of rectal tissue removed by rectal biopsy

Other methods:-

- Portal venography, USG and computed tomography are helpful in indirect diagnosis of disease.

Treatment:-

- Praziquantel (40mg/kg/day in 2 divided doses for 1 day)



- metrifonate (single dose of 7.5 mg to 10 mg / kg body weight, weekly for 3 weeks)
- Praziquantel is more effective drug than metrifonate.

Prophylaxis :-

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