Blood Fluke: *Schistosoma haematobium*

**Introduction**

*Schistosoma haematobium* was the earliest blood fluke discovered. Theodor Bilharz, a German surgeon working in Cairo, distinguished the parasite as a causative agent of urinary infection in 1851. After the discovery, the infection (generally including all infections caused by schistosome) was called bilharzia or bilharziasis. Along with other helminth parasites *Opisthorchis viverrini* and *Clonorchis sinensis*, *S. haematobium* was explained as Group 1 (extensively demonstrated) carcinogens by the WHO International Agency for Research on Cancer (IARC) Working Group on the Evaluation of Carcinogenic Risks to Humans in 2009. The "urinary blood fluke" *Schistosoma haematobium* is a digenetic trematode parasite which belongs to a class of blood flukes (*Schistosoma*). It is generally documented in Africa and the Middle East and also the the major agent of schistosomiasis, the most widespread parasitic infection in human populations. It is the only blood fluke that shows infection of urinary tract, causing urinary schistosomiasis, and is the leading cause of bladder cancer. *Schistosomiasis* occurs by infection through eggs.

Adult worms are generally seen in the venous plexuses near the urinary bladder. The released eggs travel to the wall of the urine bladder causing haematuria and fibrosis of the bladder. The bladder becomes calcified, and there is increased pressure on ureters and kidneys known as hydronephrosis. Swelling of the genitals because of *S. haematobium* may enhance the propagation of HIV.

**Epidemiology**

*S. hematobium* is seen in Middle East and Africa, where newborns and small children are mainly infected. Infection is mainly prevalent in both the Nile Valley South Cairo and Nile Delta. The first epidemiological review in 1937 showed that infection rate was as high as 85% amongst people in the Eastern and Northern parts of the Delta.

**Morphology**

*Schistosoma haematobium* exists as both female and male, which are always found paired (a condition called as copula) and looks like an individual. The male measures 1 mm in width and 10–18 mm in length. It bears oral and ventral suckers near its anterior end. Its leaf-like flat body is nestled into the two sides to frame a channel or groove called gynaecophoric canal in which the female is wrapped up. Therefore, it gives the clear appearance of a cylindrical roundworm body. Only the terminal posterior and anterior ends of the female are exposed. In contrast to the male, a female exhibits each feature of a roundworm. It is elongated and cylindrical, measuring about 20 mm in length and 0.25 mm in width. The pathogenic form- "eggs" are oval in shape and
measure about $144 \times 58 \mu\text{m}$ in diameter, with a spine at terminal position. This is a significant diagnostic tool because co-infection with S. mansoni (having a lateral-spined eggs) is usual.

The miracidium measures about $55 \mu\text{m}$ wide and $136 \mu\text{m}$ long. The body is secured by anucleate epidermal plates separated by epidermal ridges. The epidermal cells radiate a number of hair-like cilia on the surface of body. Epidermal plate is absent only at apical papilla, or terebratorium, which bears numerous sensory organelles. Its internal body is totally loaded up with vesicles and glycogen particles.

**Host Range**

Definite host: Humans

Intermediate host: Fresh water snail
Life cycle

*S. haematobium* finishes its life cycle in people, as definitive hosts, and freshwater snails, as intermediate hosts. Nevertheless, not like all, other schistosomes that discharge eggs in the digestive tract, it discharges its eggs in the urinary tract and releases alongside the urine. In dormant freshwater, the eggs hatch in just 15 minutes into the larva called miracidia. Each miracidium is either male or female. Miracidia are covered by hair-like cilia with which they effectively swim searching for snails. Species of snail like *Bulinus*, including *B. nasutus, B. nyassanus, B. globosus, B. forskali, and B. truncatus*, can harbor the miracidia. The miracidia basically enter through the delicate skin of the snail and move to the liver. Inside the snail, their cilia is pushed off and extra-epithelial coverings is formed within 24 hours. At that point they change into sporocysts and experience dynamic cell division following two weeks. The mother sporocyst gives rise to numerous sporocysts. Every sporocyst frames new hatchlings called cercariae. One mother sporocyst delivers a large portion of a million cercariae. Following a month, the sporocysts break and cercariae are freed. Free cercariae infiltrate the liver and move out of the snail into water. Every cercaria has a biforked tail with which it swims to locate a human host. Again the cercariae are short lived and can live in water for 4–6 days except if they locate a human host.
Right when human interacts with a pervaded water, the cercariae connect themselves on the skin utilizing their suckers. After suitable direction, they begin penetrating the skin by discharging proteolytic chemicals that expand the skin pores (hair follicles). This procedure takes around 3–5 minutes and produces itching, at that point, they have infiltrated the skin. Their tails are ousted during the entrance with the end goal that only the head parts enters. Exactly when they enter the veins, they are known as schisotomulae. They enter the blood vessels to arrive at the heart and afterward the liver, in the way many are killed by the safe cells. Survivors enter the liver within 24 hours. From the liver they enter the portal vein to arrive at different sites of the body. The schistosomulae of S. haematobium arrive at the vesical vessels through anastomotic channels between radicles of the substandard mesenteric vein and pelvic veins. Subsequent to living inside little venules in the submucosa and mass of the bladder, they move to the perivesical venous plexus (a gathering of veins at the lower part of the bladder) to accomplish full development. To dodge identification by the host's safe framework, the grown-ups can cover themselves with have antigen.

Individuals sort out opposite sexes. The female body becomes encompassed inside the gynaecophoric canal of the male; in this way, turning out to be partners until the end of time. Sexual development is achieved 4–6 weeks of initial contamination. A female worm lays 500-1,000 eggs in a day. The female just leaves the male rapidly for laying eggs as it can enter the little and limited fringe venule in the submucosa so the eggs can be discharged into the bladder. The embryonated eggs penetrate the bladder mucosa utilizing proteolytic chemicals, supported by their terminal spines and by the contraction of the bladder.

The compound is a toxin explicitly for harming (putrefaction) the tissue. Under typical circumstance, the eggs discharged into the bladder don't cause obsessive side effects. In any case, eggs regularly neglect to penetrate the bladder mucosa and stay caught in the bladder wall; it is then these, which produce the sores by discharging their antigens and provoking granuloma development. Granulomata frequently ulcerate. This is the condition behind the obsessive injuries found in the bladder wall, ureter and renal; and furthermore tumor, both benign and malignant. The female constantly lays eggs for a staggering span. A normal life expectancy is 3–4 years.

**Diagnosis**

Conventionally, diagnosis has been made by assessment of the urine for detection of eggs. In chronic (severe) infections, or if eggs are difficult to locate, an intradermal injection of schistosome antigen to form a wheal is efficient in detecting infection. On the other hand diagnosis can be made by complement fixation tests. Commercial blood tests
including an **Indirect immunofluorescence test and an ELISA** have also proved to be helpful, but have less sensitivity ranging from 21% to 71%.

**Treatment**

The drug of choice is praziquantel, which is an quinolone derivative. However, it has low cure rate (only 82-88%).

**Prophylaxis**

1. The major reason of schistomiasis is the discarding of human waste into water supplies. Hygienic disposal of waste would be enough to eradicate the disease.
2. Water for bathing and drinking should be boiled in endemic regions.
3. Contaminated water should be avoided. However, agricultural practices such as fishing and rice cultivation engage long contact with water, producing avoidance impractical.
4. Efficient eradication of snails is an efficient technique.