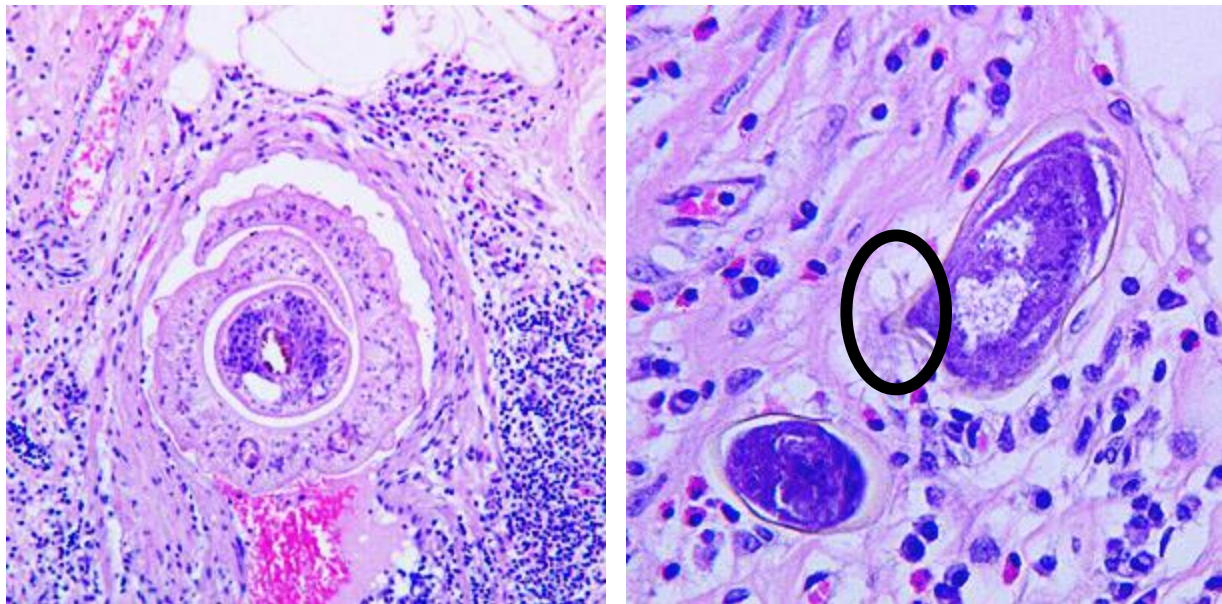


## PARASITOLOGY CASE HISTORY 11 (HISTOLOGY)

(Lynne S. Garcia)

A 37-year-old man from the United Arab Emirates was admitted to the hospital for complaints of upper and lower abdominal pain. The greater omentum and appendix were biopsied and sent to pathology for sectioning and staining. The following images from the omentum were seen (H&E routine staining). Images courtesy of Dr. Munaf Desai, Al Qassini Hospital, Shatjah, UAE (CDC).

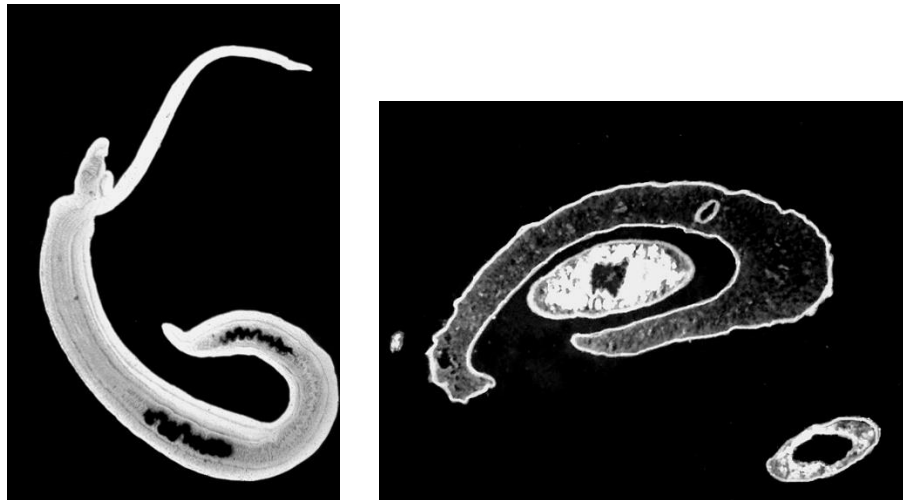


- Based on these images, what is your diagnosis?

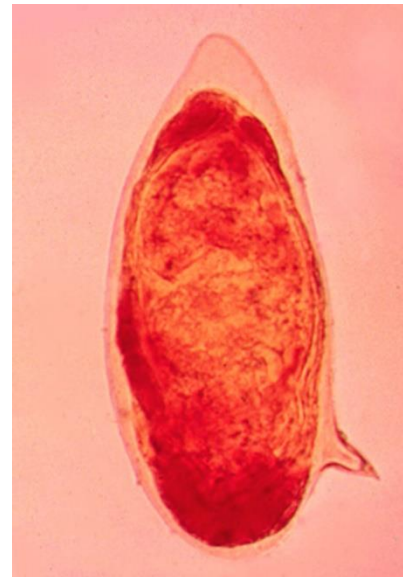
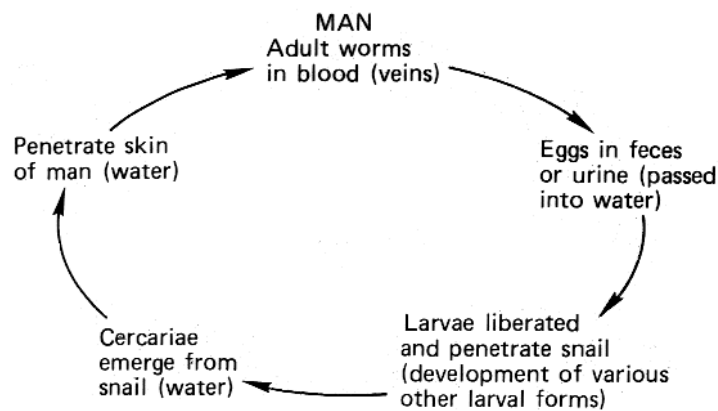
***Scroll Down for Answer and Discussion***

**Answer and Discussion of Histology Quiz #11**

This was a case of schistosomiasis caused by *Schistosoma mansoni*. In the images below the female worm is residing in the gynecophoral canal of the male. The male/female worms mate for life.



Female worm lying in male gynecophoral canal, whole mount, cross-section.



Schistosome life cycle; *Schistosoma mansoni* egg showing large lateral spine. Note the original pathology images above; the large lateral spine can be seen within the oval.

**Life Cycle.** Humans are the only important definitive hosts of *S. mansoni*. Fully embryonated eggs without an operculum (114 to 180  $\mu\text{m}$  by 45 to 73  $\mu\text{m}$ ) are passed in the stool. The eggs are light yellowish brown and elongate, and they possess a large lateral spine projecting from the side near one end

of the egg. The eggshell is acid fast when stained by a modified Ziehl-Neelsen technique. On reaching water, the eggs hatch, liberating miracidia, which must penetrate a suitable snail host (*Biomphalaria* spp.). Cercariae liberated from the infected snail infect humans when the latter come in contact with cercaria-infested water. On infection, the cercariae lose their tails, and the body (now called a schistosomulum) migrates through the lungs to the liver, where it develops to a mature adult in the mesenteric veins of the large bowel. Some worms are also found in the superior mesenteric veins, vesical plexus, and intrahepatic portion of the portal veins. Other veins have also been reported.

The male worm's body, which is flattened behind the ventral sucker, appears cylindrical as it curves to form the gynecophoral canal to clasp the female worm. The female worm is long, slender, and cylindrical in cross section. While held in the gynecophoral canal of the male, the female ingests 10 times more red blood cells than does the male. After mating, the female leaves the male and migrates against the flow of blood to the small venules of the intestine. Initial egg production begins 4 to 7 weeks after infection. *S. mansoni* mature females produce 100 to 300 eggs per day.

The eggs are immature when first laid and take approximately 8 to 10 days to develop a mature miracidium. They are nonoperculate and contain a lateral spine. Egg deposition takes place intravascularly. Many of the eggs laid are swept away and become lodged in the microvasculature of the liver and other organs. About half of the eggs swept away by the bloodstream become embedded in the mesenteric venule wall. The presence of eggs in the tissues stimulates granuloma formation; the eggs die, calcify, and are eventually absorbed by the host. The eggs that are not trapped in the tissues will continue the normal life cycle. The process of maturation and tissue penetration takes about 8 to 10 days; the eggs work their way through the tissues into the lumen of the intestine to be released from the body in the feces.

**Clinical Disease.** Disease syndromes associated with schistosomiasis are related to the stage of infection, previous host exposure, worm burden, and host response. Syndromes include cercarial dermatitis, acute schistosomiasis (Katayama syndrome), and related tissue changes resulting from egg deposition.

**Schistosome Cercarial Dermatitis.** Cercarial dermatitis follows skin penetration by cercariae, and the reaction may be due partly to previous host sensitization. Few clinical manifestations are associated with primary exposure, but both humoral and cellular immune responses are elicited on

subsequent exposure. After cercarial skin penetration, petechial hemorrhages with edema and pruritus occur. The subsequent maculopapular rash, which may become vesicular, may last 36 h or more. Cercarial dermatitis is common with *S. mansoni* infections.

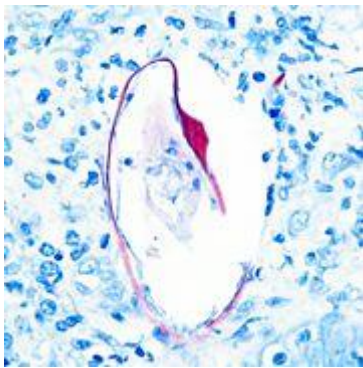
**Acute Disease: Katayama Syndrome.** Acute schistosomiasis (Katayama syndrome) is an early clinical manifestation of schistosomiasis that occurs several weeks post-infection with *Schistosoma* spp (trematode) worms and is associated with heavy primary infections and the initiation of egg production throughout areas of high transmission risk. Because of this time delay and its non-specific presentation, it is most likely to be misdiagnosed by travel medicine physicians and infectious disease specialists in non-endemic countries. The signs and symptoms resemble those of serum sickness. Characteristic symptoms include high fever, hepatosplenomegaly, lymphadenopathy, eosinophilia, and dysentery. Diffuse pulmonary infiltrates are seen radiologically, and almost all patients have eosinophilia and a history of water contact 14-84 days before presentation of symptoms.

**Chronic Schistosomiasis.** After production of eggs by the adult worms (Figure 20.6), the eggs become trapped in the fine venules and are able to pass through the tissues, escaping into the intestine or, less commonly, the bladder. The eggs liberate a number of soluble antigens, evoking minute abscesses, which facilitate their passage into the lumen. The passage of eggs through the wall of the intestine or bladder leads to symptoms that correlate with the worm burden of the host, including fever, abdominal pain, liver tenderness, urticaria, and general malaise. In *S. mansoni* infection, blood and mucus are detected in the stools and the patient may have diarrhea or dysentery.

**Diagnostic Procedures.** Specific diagnosis of schistosomiasis by detection of eggs in stool or urine specimens is possible only after egg production has begun. Eggs may be found in feces as early as 5 weeks after infection. The ease of egg detection depends on the worm burden and the duration of the infection. Patients with a low worm burden or old (chronic) infections may have very few eggs in the feces or urine, and the infections may not be confirmed due to insensitive diagnostic methods.

*Biopsy Specimens.* Rectal biopsy specimens have been particularly useful in detecting eggs in patients with light, chronic, or inactive infections. The biopsy tissue can be crushed between two glass slides; the tissue can also be stained without routine sectioning. This technique is more effective than histologic examination and allows assessment of the species and viability of

the eggs prior to staining. The viability of the eggs can be determined by closely observing the miracidium for flame cell activity. Each miracidium contains two pairs of flame cells, and these are actively beating in live miracidia. *S. mansoni* eggshells can be stained acid fast with a modified Ziehl-Neelsen stain. This technique has been used in tissue sections to differentiate *S. mansoni* eggs from *S. haematobium* eggs, which are not acid-fast positive. The shell of *Schistosoma mansoni*, *Schistosoma intercalatum* and *Schistosoma japonicum* eggs also stain positive unlike the eggs of other *Schistosoma* species. It is also important to remember that while the egg shell may not be acid-fast, the miracidium larva within the shell may be acid-fast (*S. haematobium*).



*Schistosoma mansoni* egg shell (acid-fast positive).

**Epidemiology and Prevention.** *S. mansoni* infections occur in western and central Africa, Egypt, Madagascar, and the Arabian Peninsula. The geographic distribution of the disease depends on the distribution of the intermediate snail hosts and the opportunity to infect humans and snails. Schistosomiasis is transmitted from infected people defecating or urinating in or near water where the appropriate snail host resides. Infections can persist indefinitely in humans. Most infected individuals have low worm burdens, but a few have very heavy infections. Studies indicate that there was little transmission of *S. mansoni* in Puerto Rico during the first half of the 1990s and confirm physician anecdotal data that no new infections have been seen during the past few years. Recent trends indicate that human schistosomiasis is disappearing from Puerto Rico.

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