SCHISTOSOMA MANSONI INFECTION: INTESTINAL SCHISTOSOMIASIS

BY

Zoheir Farid

U.S. NAVAL MEDICAL RESEARCH UNIT NO. 3
(CAIRO, ARAB REPUBLIC OF EGYPT)
PSC 452, BOX 5000
FPO AE 09835-0007
Schistosoma mansoni infection: intestinal schistosomiasis

Zoheir Farid

Schistosoma mansoni is a trematode (fluke) which, like S. japonicum, inhabits the mesenteric venules. The key differences between S. mansoni and S. japonicum are listed on page 4567.

Life cycle

Man is infected by working in infested water containing living cercariae. The cercariae penetrate the skin or mucous membrane, develop into schistosomules and travel through the peripheral venules to the pulmonary vessels, from where they enter the general circulation. Those reaching the portal venous system develop into male and female adult worms in 6–12 weeks; most adult S. mansoni then migrate to their final habitat in the tributaries of the inferior mesenteric veins. Most eggs laid in the small venules around the colon pass into the tissues, but some are eliminated in the stools. Eggs which are passed in the stools hatch immediately in fresh water and liberate free-swimming miracidia, that penetrate a specific snail host (genus Biomphalaria) within 24 hours. Cercariae develop in 4–6 weeks and are passed into the water where they remain infective for 2–3 days. The cycle is then repeated when man enters fresh water containing the cercariae and becomes infected.

Pathology

Complications develop as a result of granulation and fibrous tissue formation around ova in the tissue, and spread of the granulomatous lesions to involve the colon, liver and, occasionally, the lungs and CNS. The severity of the disease is related to the host reaction to the eggs in the tissues — a form of delayed hypersensitivity reaction.

The inflammatory reaction, which may be diffuse or localized in a pseudotuberculoid pattern, is rich in eosinophils in the early phases, together with macrophages, lymphocytes, plasma cells and scattered foreign body giant cells surrounding the egg. In time, the lesions progress to marked fibrosis with less cellularity and degenerated, calcified eggs. The fibrosis following the schistosomal reaction is the cause of serious complications, specifically periportal hepatic fibrosis.

Clinical features

Cercarial dermatitis: itching with erythema and a papular rash may develop within minutes of penetration of the cercaria (swimmer’s itch). Symptoms usually last 2–3 days and are believed to be due to a hypersensitivity reaction to cercarial proteins.

Acute schistosomiasis usually develops 4–6 weeks after initial exposure and coincides with early egg-laying. This acute toxemic phase of the infection, essentially a form of serum sickness, is seen mainly in city-dwellers or young patients exposed for the first time. Patients present with a fever, arthralgia, myalgia, hepatomegaly and hypereosinophilia. The fever may, in some cases, last for 4–6 weeks and patients are often referred to hospital for investigation of PUO. Children may deny a history of water exposure, and the first indication of acute schistosomiasis is marked eosinophilia and tender hepatomegaly.

Eggs are not usually present in the faeces in the acute phase, but the diagnosis can be inferred by detection of a greatly elevated specific IgM, using enzyme-linked immunosorbent assay (ELISA) or rapid counterimmunoelectrophoresis. It is important to recognize that acute schistosomiasis and acute fascioliasis have similar clinical presentations; ELISA or counterimmuneelectrophoresis can demonstrate the rise in the specific schistosomal IgM or Fasciola IgG.

Chronic schistosomiasis: the chronic phase occurs from approximately 3 months to several years after exposure. Gastrointestinal stage — eggs are laid in the terminal venules of the inferior mesenteric veins of the large bowel, and symptoms are usually related to the intensity of infection. Some patients are asymptomatic, while others complain of abdominal pain and diarrhoea, which may be accompanied by blood, pus, and mucus in the stools. In Egypt and Sudan, schistosomal tubercles, nodular submucosal thickening, ulceration and polyp formation commonly develop in the rectosigmoid colon (Figure 1). Occasionally, extensive involvement of the colon with polyps leads to a protein-losing enteropathy. Rarely, a large mass will develop in the descending colon as a result of the inflammatory response to egg deposition. Ultrasonography is helpful in diagnosing these patients.

Portal fibrosis — if there is massive or repeated infection, the number of eggs carried back up the portal
vein to the liver may become enormous. This eventually results in portal fibrosis, with the development of portal hypertension and chronic passive congestion of the spleen. With further increased fibrosis and shrinkage of the liver, ascites with or without dependent oedema develops (Figure 2). Oesophageal varices may lead to haematemesis. Finally, hepatocellular failure with jaundice, hepatic coma and death may occur. Patients with advanced hepatosplenomegaly have an increased susceptibility to chronic Salmonella septicaemia. Some

Schistosomal colonic polyposis

- a Rectosigmoid colon (arrowed).
- b Transverse colon.
patients develop massive proteinuria and present with the nephrotic syndrome.

Other systems - eggs may lodge ectopically in many tissues and cause damage. Schistosomal cor pulmonale develops when eggs reach the pulmonary circulation through portocaval anastomoses. If eggs reach the CNS (most commonly the spinal cord in S. mansoni infection), transverse myelitis and various forms of palsy may result.

Diagnosis

A history of exposure to infection, combined with abdominal pain, diarrhoea or dysentery, should arouse suspicion of S. mansoni infection, and stools should be examined repeatedly for laterally spined eggs. Eosinophilia is usual in acute, but not chronic, schistosomiasis. Signoidoscopy may reveal involvement of the rectum and sigmoid colon (schistosomal tubercles and polyps), and rectal biopsy may be necessary to locate the eggs. Barium enema air-contrast radiographs are usually necessary to show the extent of colonic involvement. Immunodiagnosis, though useful in acute schistosomiasis, is of little value in the differential diagnosis of chronic disease in endemic areas. Ultrasonography is proving to be a useful technique for the demonstration of schistosomal periportal fibrosis and enlargement of the portal vein.

Treatment

All infected patients should be treated. The two main oral drugs available for the treatment of all stages of S. mansoni infection are praziquantel and oxamniquine. There is little to choose between the two drugs:

- oxamniquine, 20–30 mg/kg/day, is given over a 3-day period
- praziquantel is administered either as a single dose, 30–45 mg/kg, or in heavy infections as a total dose of 60 mg/kg in three divided doses over the course of 1 day.

Either drug results in a reduction in the number of eggs of over 90% and a cure rate of 70–90%, and can be given to patients with schistosomal hepatosplenomegaly.

Praziquantel and oxamniquine have relatively few side-effects, which are usually mild, transient and related to the gastrointestinal system.

Acute schistosomiasis is best treated with praziquantel, 75 mg/kg in three divided doses 4-hourly. Corticosteroids (prednisone, 5 mg t.d.s.) are used for 2–3 days to control the fever and toxemia before starting specific therapy.

ACKNOWLEDGEMENTS

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Figures 1 and 2 are reproduced by courtesy of Dr Nabil Ayad El Masry, US Naval Medical Research Unit 3, Cairo. The map of the world distribution of S. mansoni infection is redrawn courtesy of Mims Magazine.

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