PUBLICATION REPORT

1736

14/93

SCHISTOSOMA MANSONI INFECTION: INTESTINAL SCHISTOSOMIASIS

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Schistosoma mansoni infection: intestinal schistosomiasis

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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.

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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.

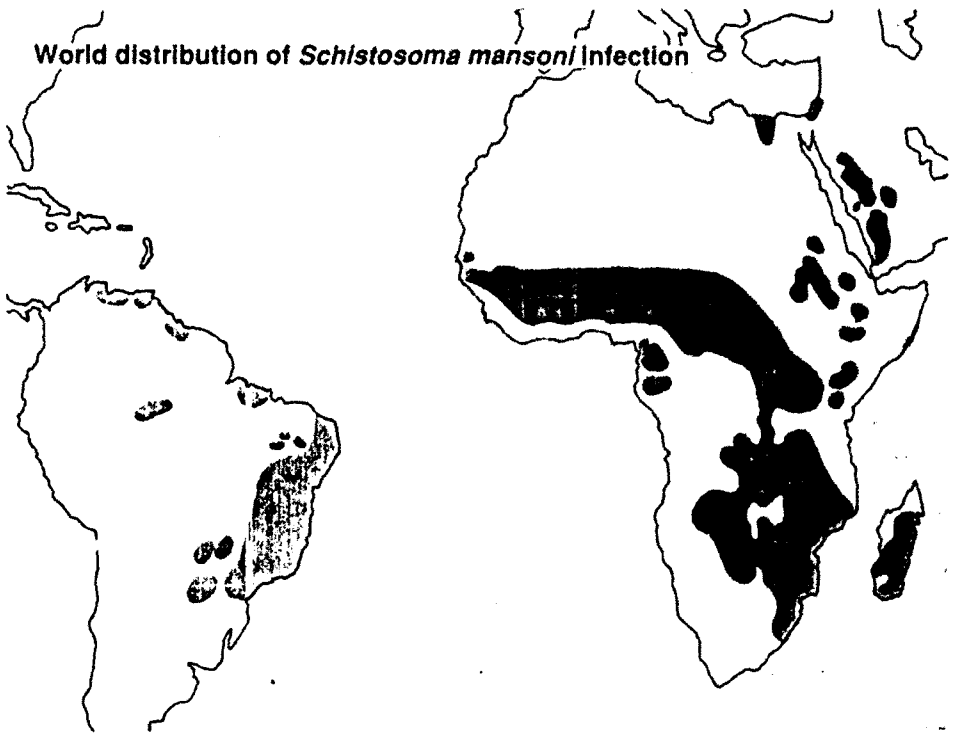
Acute schistosomiasis usually develops 4–6 weeks after initial exposure and coincides with early egg-laying. This acute toxaemic phase of the infection, essentially a form of serum sickness, is seen mainly in city-dwellers or young patients exposed for the first time. Patients present with a fever, arthralgia, 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 counter-immunoelectrophoresis. It is important to recognize that acute schistosomiasis and acute fascioliasis have similar clinical presentations; ELISA or counterimmuno-electrophoresis 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.

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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. Sigmoidoscopy 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.

2 Schistosomal hepatosplenomegaly with ascites.

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 toxaemia before starting specific therapy. ❑

ACKNOWLEDGEMENTS

This work was supported by Naval Medical Research and Development Command, NMC, NCR, Bethesda, Maryland, USA (Work Unit No. MR00001.01-3073). The opinions and assertions contained herein are the private ones of the author and are not to be construed as official or as reflecting the view of the Department of the Navy or the naval services at large.

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.

FURTHER READING

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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	1992	

4. TITLE AND SUBTITLE
Schistosoma mansoni Infection: Intestinal Schistosomiasis

5. FUNDING NUMBERS
PE- 61152N
WU- MR00001.01.3073

6. AUTHOR(S)
Farid, Zoheir

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
U.S. Naval Medical Research Unit No. 3
PSC 452, Box 5000
FPO AE 09835-0007

8. PERFORMING ORGANIZATION REPORT NUMBER
14/93

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
Naval Medical Research and Development
Command, National Naval Medical Center
Building 1, Tower 12
Bethesda, MD 20889-5044

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES
Published in: Med. Intrnl, pp. 4561-4563, 1992; Acc. No. 1736.

12a. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release;
Distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*
Please see attached.

Accesion For	
NTIS CRA&I	☒
DTIC TAB	☐
Unannounced	☐
Justification	
By	
Distribution /	
Availability Codes	
Dist	Avail and / or Special
A-1	20

14. SUBJECT TERMS
Schistosoma mansoni; Infection; Life cycle; Pathology; Clinical features; Diagnosis; Treatment; Patients; Egypt.

15. NUMBER OF PAGES
3

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
UNCLASSIFIED	UNCLASSIFIED	UNCLASSIFIED	