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SCHISTOSOMIASIS IN PATIENTS IN THE SULTANATE OF OMAN

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Background: Following an intensive public health eradication program, the focus of schistosomiasis mansoni at Salalah in Southern Oman (the only focus of schistosomiasis in Oman), appeared to have been eradicated in 1994. Since the vector snails for *Schistosoma mansoni* (and also for *S. haematobium*) were widely endemic in freshwater springs at Salalah, and reintroduction of infection (or introduction in the case of *S. haematobium*) was possible, we undertook a prospective study in 1995 for schistosomiasis in patients at the Sultan Qaboos University Hospital (SQUH), and to obtain evidence of visits by these patients to Salalah.

Patients and Methods: From May 1995 to May 2000, Omani and expatriate patients presenting at SQUH were evaluated for possible schistosomiasis (residence in an endemic region, suggestive clinical features) by a search for ova in stool, urine, or in tissue biopsies, or when ova were absent, by serodiagnosis. A retrospective survey of schistosomiasis patients diagnosed in SQUH from 1990 to April 1995 was also performed. Ministry of Health (MOH) data on reports of schistosomiasis (a notifiable disease) in Oman from 1991 to 2000, were obtained.

Results: Thirty patients with schistosomiasis were identified, 10 retrospectively and 20 in the prospective study. Patients were aged 9 to 60 years, and 25 of them (10 Omanis, 14 Egyptians, 1 Sudanese) contracted the infection in Africa, while 5 Omanis became infected in Salalah (3 in 1999). Twelve patients had schistosomiasis mansoni, 6 had schistosomiasis haematobia, and in the rest, infection by both parasites was confirmed or possible. The most common presentation (23%) was hepatosplenic schistosomiasis with portal hypertension. No patient admitted to contaminating freshwater with excreta in Salalah. MOH data revealed 3 to 14 cases of schistosomiasis were reported annually between 1991 and 2000, and that all schistosomiasis cases reported from 1994 to 1998 originated from Africa.

Conclusion: Schistosomiasis remains an important health problem in Oman. Many doctors are unfamiliar with the clinical features and methods of diagnosis, thereby frequently causing delays in diagnosis. Because vector snails are endemic in Salalah, schistosomiasis patients must be detected and treated early. At infested freshwater sites at Salalah, mollusciciding should continue indefinitely, and notices should warn visitors not to contaminate the spring water.

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Key Words: Schistosomiasis mansoni, Haematobia, snail vectors.

The recent re-emergence of schistosomiasis mansoni in Salalah, Southern Oman,¹ has emphasized the need for constant vigilance at foci where vector snails exist. In May 1999, unauthorized individuals removed the warning signs which forbade contaminating the water with excreta and swimming at the freshwater springs of Wadi Sahanout near Salalah, a popular picnic spot and a well-known locality for *Biomphalaria arabica* snails, the vectors for *Schistosoma mansoni*.² The water was polluted by defecation, and three individuals who subsequently went swimming contracted schistosomiasis.¹ Prior to this

continuing, together with monthly snail-density counts, as well as annual stool examination of agricultural workers and schoolchildren living near the springs. No positive stools were obtained in 1999.⁴

In 1995, in the knowledge that *Biomphalaria arabica* and *Bulinus contortus* snails (potential vectors for *S. haematobium*, although no case of transmission had been reported) were widely distributed in Salalah,² and that schistosomiasis could be reintroduced (or introduced, in the case of schistosomiasis haematobia), we undertook a prospective study of patients presenting with schistosomiasis at the Sultan Qaboos University Hospital (SQUH). We also carried out a retrospective review of clinical records of patients diagnosed to have schistosomiasis from 1990 to April 1995.

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incident, no case had been reported since 1994.³ Since 1994, mollusciciding of all major freshwater sites (including Wadi Sahanout) where snails were present has been

TABLE 1. *Clinical data on 30 schistosomiasis patients.*

Presentation	Pathology	No (%)	Parasite
Abdominal pain, hematemesis, hepatosplenomegaly	Hepatic fibrosis, portal hypertension, esophageal varices	7 (23%)	SM/SH

Hepatomegaly	Hepatic fibrosis	1 (3%)	SM
Anemia, thrombocytopenia	Splenomegaly, hypersplenism	1 (3%)	SM
Bloody diarrhea	Enteritis	3 (10%)	SM
Acute abdominal pain	Acute schistosomal appendicitis	1 (3%)	SM
Acute flaccid paraplegia	Katayama syndrome with conus medullaris myelopathy	1 (3%)	SM
Cystitis, hematuria	Granulomatous cystitis, ureteric stenosis and hydronephrosis	4 (14%)	SH
	Cancer of bladder	1 (3%)	
Vaginal discharge	Cervical granuloma; ova present	1 (3%)	SH
Cor pulmonale	Pulmonary fibrosis	1 (3%)	SM
Eosinophilia	Enteritis	1 (3%)	SM
Asymptomatic (seropositive)		5 (16.5%)	SM/SH
		3 (10%)	SM

SM=*Schistosoma mansoni*; SH=*Schistosoma haematobium*.

Patients and Methods

The prospective study from May 1995 to April 2000 evaluated patients with a history of residence in an endemic region, clinical features suggestive of schistosomiasis, seropositivity, or with a confirmed diagnosis. These patients included Omanis from Salalah, East African-born Omanis, and Egyptian and Sudanese expatriate workers. Evaluation included history of occupational or recreational exposure to infection, documentation of clinical signs of acute or chronic intestinal or urinary schistosomiasis, and examination of stool, urine, or biopsy material (e.g., rectal snips) for ova. Immunodiagnosis included indirect hemagglutination assay (IHA) for antibody levels to schistosoma in sera, and performed in the Marcel Merieux Laboratories in Lyon, France, but schistosoma antigen detection tests were not available. Other investigations included chest x-ray (e.g., for signs of cor pulmonale), ultrasonography of the liver for evidence of periportal fibrosis, endoscopy (gastroscopy, proctoscopy, sigmoidoscopy, cystoscopy), or laparoscopy to obtain tissue biopsies, and magnetic resonance imaging (MRI) when neuroschistosomiasis was suspected (1 case).

The retrospective study of case records in the hospital Medical Records Department identified patients with schistosomiasis from 1990 to 1995. Some unreported cases were identified from histopathology records. The total number of cases reported (schistosomiasis is a notifiable disease in Oman) from 1991 to 2000 was obtained from the Department of Surveillance and Disease Control, Ministry of Health (MOH). Data prior to 1991 were unavailable.

Results

TABLE 2. *Schistosomiasis cases reported by nationality in Oman:1991-2000.*

Nationality	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000

From January 1990 to April 2000, 30 patients were diagnosed at the SQUH to have schistosomiasis, 10 in the retrospective, and 20 in the prospective surveys. Patients were aged 9 to 60 years (mean 36 years), and 62% were males. Twenty-five patients (14 Egyptians, 1 Sudanese, and 10 Omanis of East African origin) had contracted infection in Africa. Most African-born Omanis were from Zanzibar. Five Omanis were infected in the Salalah region, including three between May and August 1999.¹

Table 1 lists the clinical data recorded on the patients. The most common symptomatology present in 7 patients (23%) was abdominal pain with hepatosplenic schistosomiasis, and signs of portal hypertension including esophageal varices. Bloody diarrhea occurred in three patients and genitourinary symptoms in six patients, one of whom had cancer of the bladder. Less frequent presentations are listed in Table 1. One Egyptian woman, aged 26 years, presented in labor as a primigravida with severe pulmonary hypertension, right ventricular failure, and underlying hepatic fibrosis and portal hypertension caused by *S. mansoni*.⁵ She was seronegative for hepatitis C and HbsAg. Acute flaccid paraplegia resulting from Katayama syndrome with myelopathy occurred in a 9-year-old Salalah boy six weeks after an episode of bloody diarrhea following exposure to fresh water at Wadi Sahanout. The stool contained abundant *S. mansoni* ova, and MRI revealed generalized edema of the spinal cord from the mid-thoracic level to the conus. He responded rapidly to high-dose prednisolone treatment with praziquantel, and was able to walk with assistance after 2 weeks.¹

Twenty-two patients had evidence of active infection, e.g., ova present in stool, urine or in tissue biopsy, or markedly elevated IHA serum antibody levels to *Schistosoma*, exceeding a titer of 1:320 (reference range for laboratory $\geq 1:80$), accompanied by signs of persisting infection, and 8 were seropositive in the IHA test with titers $\geq 1:80$ -1:1280 (reference range $\geq 1:80$), but without conclusive evidence of active infection. Twelve patients had schistosomiasis mansoni, 6 had schistosomiasis haematobia, and in 13 Egyptian, Sudanese, and East African-born patients, infection by both parasites was confirmed or possible.

With regards to relevant co-existent disease, nine patients (7 Egyptians and 2 Omanis), were seropositive for hepatitis C virus infection, and 4 had hepatitis Bs antigen (HBsAg) seropositivity. Two patients with hepatosplenic schistosomiasis died, and one who was seropositive for HBsAg developed hepatoma.

All patients with active infection received praziquantel, 40 mg/kg. Three of the asymptomatic seropositive cases in the retrospective study had not been given praziquantel, but

Omani*	2	5	10	5	4	4	5	8	6	1
Egyptian*	0	1	3	2	1	3	5	2	1	1
Tanzanian	7	0	1	0	0	0	0	0	0	1
Others*	0	0	0	0	1	0	0	1	0	0
Total	9	6	14	7	6	7	10	11	7	3

*From 1994-1998, all patients contracted infection in Africa; three of the Omani 1999 cases are described in this article.

in the case of five similar patients in the prospective study, praziquantel was given, since the presence of living worms could not be excluded. The total number of cases of schistosomiasis reported to the Ministry of Health from 1991 to 2000 is presented in Table 2.

Discussion

Schistosomiasis mansoni was first reported in Salalah in 1979,⁶ although *Biomphalaria arabica* snails had been observed in the district as long ago as 1896.⁷ The disease had apparently been introduced by Egyptian or Sudanese farm workers, or East African-born Omanis.⁶ Following a comprehensive public health program (patient identification and treatment, mollusciciding of freshwater sites), by 1994, it appeared that the disease had been eradicated.³

An important strategy to prevent reintroduction of infection was the placing of notices in Arabic and English at the 8 major freshwater sites where vector snails existed, prohibiting contamination of water with excreta, or bathing. However, as the 1999 outbreak demonstrated, it takes only one careless person to reintroduce schistosomiasis to a suitable environment. The warning notice has now been replaced at Wadi Sahanout, and a search has been undertaken to identify other infected individuals.¹ (At the time of preparing this report, none have been detected). Apart from migrant workers, Salalah residents who escaped detection during the eradication program, and who still excrete eggs, are obviously potential sources of reintroduction of infection (*S. mansoni* can survive for up to 37 years⁸). Detection and treatment of any such individuals is clearly of paramount importance. None of our patients who had visited or lived in Salalah, which included a Salalah resident still excreting eggs 10 years after exposure, admitted to passing stool or urine in freshwater locations.

During the period 1990-1999, we identified 30 SQH patients with schistosomiasis, most cases being diagnosed during the prospective study. Table 1 demonstrates the wide spectrum of clinical disease encountered. It was notable, especially in the retrospective series, that diagnosis was frequently delayed. Case records often neglected to mention that the patient had resided in an endemic region. Our Salalah patient with acute myelopathy was referred as a case of suspected poliomyelitis, and the preceding history of bloody diarrhea following bathing at Wadi Sahanout had not been elicited.

Many doctors were apparently unfamiliar with the

clinical features of schistosomiasis and the syndromes resulting from ectopic ova deposition. A common mistake

was attributing liver disease solely to cirrhosis caused by hepatitis B or C viruses, and co-existent schistosomal periportal fibrosis (well-demonstrated on ultrasonography), was not recognized. One Egyptian patient with chronic hepatitis C infection had worked as a farmer in Oman for 10 years, without schistosomiasis being suspected. His IHA antibody titer to *S. mansoni* was >1:8192 (reference range \geq 1:80).

When schistosomiasis was suspected, methods of diagnosis were often inappropriate, e.g., a single search for ova in stool or urine in advanced disease. It is essential that the correct investigation is performed at different stages of the disease, in both schistosomiasis mansoni and haematobia.⁹ The emphasis is on recovery of ova, and especially the use of rectal snips,^{10,11} but in cases where they cannot be recovered, e.g., in early or very late infection, immunodiagnosis is helpful. Serology does not distinguish well between past and current infection, but single high titers in the IHA test, e.g., \geq 1:320 (reference range for laboratory \geq 1:80), suggests active infection. Antigen detection, if available, identifies living worms.

It was notable that some seropositive but asymptomatic patients in the retrospective study were not treated. Our practice is to give praziquantel to all seropositive individuals, whether or not ova are recovered, even if they are asymptomatic, to ensure that all worms, including any in an ectopic location, are eradicated. Praziquantel is cheap, safe and effective, and is given as a single dose.

Table 2 shows that schistosomiasis remains an important diagnosis in Oman, and 3-14 cases are reported annually to the Ministry of Health. It is likely that many cases are undiagnosed or unreported. We estimate that the reported figures represent perhaps 50% of actual cases.

In conclusion, schistosomiasis is an uncommon but important cause of morbidity in Oman, as it is in many other countries of the Arabian Peninsula.¹²⁻¹⁵ It is important that doctors in this part of the world be familiar with the main features of the disease and appropriate diagnostic methods.⁹ All untreated patients, including seropositive, asymptomatic individuals, should receive praziquantel, to ensure destruction of all worms, including those in ectopic locations. In Arabian Peninsula countries where vector snails are endemic, surveillance for human schistosomiasis, and mollusciciding of freshwater habitats, should be continued indefinitely.

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