

**Jill Benson**

MBBS, DCH, FACP_{PsychMed}, is Director, Health in Human Diversity Unit, Discipline of General Practice, University of Adelaide, and Medical Officer, Migrant Health Service, Nunkuwarrin Yunti and Parklands Medical Practice, South Australia. jill.benson@adelaide.edu.au

Asymptomatic schistosomiasis in a young Sudanese refugee

In 2004–2005, approximately 13 000 refugees settled in Australia, 70% of them from Africa.¹ Schistosomiasis is one of the many illnesses endemic in Africa and approximately 40% of refugees have been found to be infected by this parasite.² It has the potential to cause serious morbidity and mortality in those who are infected and after malaria is the second most prevalent tropical disease worldwide.³ Australia is not known to have an appropriate snail vector and so schistosomiasis is unlikely to be a public health problem.⁴ This article presents a case that demonstrates one of the sequelae of schistosomiasis – pipe stem cirrhosis – with associated splenomegaly and oesophageal varices.

Case study

AM, 17 years of age, arrived in Adelaide with his family as a refugee from Sudan in August 2005. He had spent the previous 5 years in Kakuma refugee camp in northern Kenya where over 82 000 people are living in an area of 25 km² with limited safety, health facilities, food or sanitation.¹ As part of an initial health assessment in Adelaide he was screened for illnesses endemic in Africa as well as for haemoglobinopathies, nutritional deficiencies, and torture and trauma issues.

Abnormalities found were: a positive titre (1:640) for schistosomiasis antibody, eggs of *Hymenolepis nana* (dwarf tapeworm) in his stool, sickle cell trait (38.2% HbS), low platelets, low white cell count and high bilirubin (*Table 1*). He had no evidence of hepatitis B or C, no malaria, normal liver function tests, no lymphadenopathy and no other specific health concerns. He was treated with praziquantel (Biltricide) at a dose of 40 mg/kg in two divided doses (with food), 4 hours apart.⁵ No follow up was arranged as he had no schistosome ova in his stools and cure rates are high using the recommended dose (*Figure 1*).⁵

AM returned in March 2006 complaining of 2 weeks of watery diarrhoea and lower abdominal pain. He had no abdominal tenderness and no change in weight but was noted for the first time to have a palpable spleen. A presumptive diagnosis of *Giardia lamblia* was made and he was given tinidazole 2 g by mouth. Repeat stools showed only *Hymenolepis nana*, which may have been responsible for the presenting symptomatology in March,⁶ and he was given a repeat dose of praziquantel.⁷ Blood tests revealed a rising bilirubin and falling platelet count, and ultrasound demonstrated an 18 cm spleen. A computerised tomography (CT) scan confirmed portal hypertension with prominent portal veins presumed by the radiologist to be from 'pipe stem cirrhosis' due to the schistosomiasis. He was referred to the local teaching hospital where further tests showed a normal autoimmune screen, negative leishmaniasis

immunofluorescent antibody, and no atypical cell populations on bone marrow. Endoscopy revealed 'sizeable oesophageal varices with high risk features' which had not been evident on the scan. His abdominal symptoms settled with the tinidazole and praziquantel, his blood tests improved slightly (*Table 1*) and his spleen decreased in size to less than 17 cm as measured by repeat ultrasound.

Discussion

Schistosomiasis (bilharzia) infects humans when their skin comes into contact with fresh water contaminated with certain types of snails that act as the vector for the parasite. Hundreds of eggs are laid each day. These eggs hatch, infect water snails, which in turn release schistosomal cercariae into the water. The cercariae penetrate the skin and migrate through the tissues and vascular system of the definitive host.⁸ Humans are the definitive

host for the most common schistosomes seen in travellers from endemic countries and in refugees to Australia. Adult worm pairs are found 'in copulo' in venous complexes either in the bladder in the case of *S. haematobium* which causes urinary schistosomiasis, or in the

portal venous plexuses surrounding the bowel in *S. mansoni* and *S. japonicum* which cause mainly gastrointestinal disease.

Schistosomiasis was introduced into South America when a local water snail proved to be a very efficient vector of *S. mansoni* brought

into the continent with African slaves.⁹ There is an ever present fear of the introduction and establishment in natural water bodies in Australia of snails accidentally imported with fish or plants from abroad, or on the feet of migratory birds.⁴ The absence of evidence however does not necessarily equate to absence of disease and the increasing incidence of schistosomiasis worldwide remains a concern.

Schistosomiasis is endemic in Africa, infecting some 200 million people.⁸ In 2001 it was estimated that 1.7 million disability adjusted life years (DALYs) were lost, not including the subtle effects of chronic infection from early childhood on growth, development and education.¹⁰ It is the most common cause of 'massive splenomegaly' (spleen >10 cm), others being hyper-reactive malarial splenomegaly, lymphomas, visceral leishmaniasis, haemoglobinopathies, chronic myeloid leukaemia and myelofibrosis.³

Schistosomiasis is asymptomatic in up to 80% of those infected, with tiredness being the most common symptom.¹¹ It is the most common cause of portal hypertension worldwide,¹² typically caused by *S. mansoni* and characterised by portal vein obstruction from intrahepatic periportal fibrosis, 'Symmers pipe stem fibrosis'.¹² Large numbers of eggs escaping from the lower mesenteric veins reach the periportal regions causing a granulomatous response leading to gradual occlusion of the intrahepatic portal veins. Schistosomal portal hypertension occurs in only a minority of those infected and is usually asymptomatic with normal liver function tests until very late in the course of the disease. The initial presentation, even in the young, may be with variceal bleeding. Co-infection with hepatitis B or hepatitis C and *S. mansoni* is associated with accelerated deterioration of hepatic function.¹²

Praziquantel in a dose of 40 mg/kg in two divided doses is the recommended regimen.^{6,12} It has minimal side effects but there may be some abdominal pain in those with heavy parasite loads. Resistance is rare and it is thought it may represent noncompliance, the invulnerability of immature worms, re-infection or the detection of dead eggs which may be passed in the stool for many months.⁵ In pregnant and lactating women praziquantel has

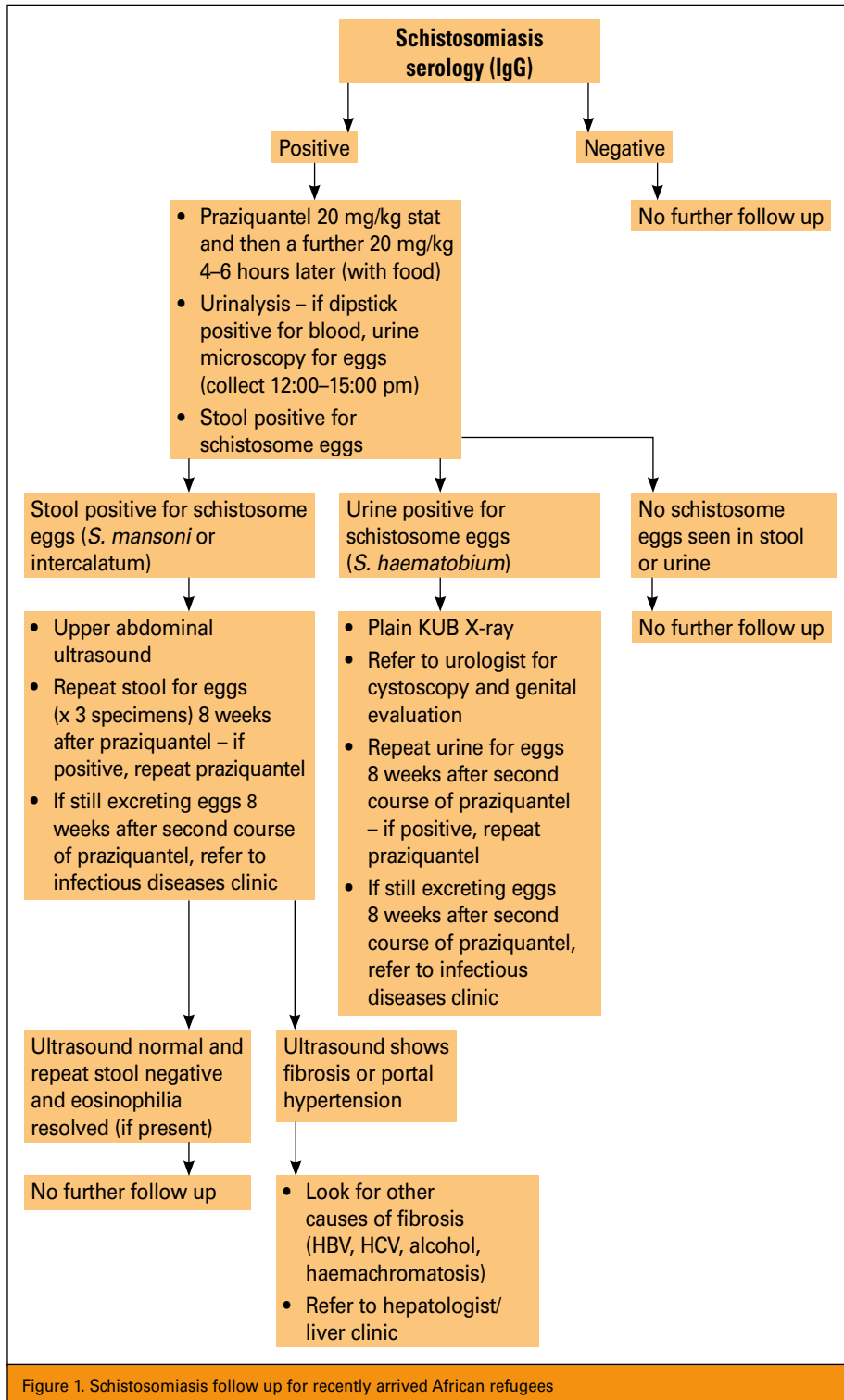


Figure 1. Schistosomiasis follow up for recently arrived African refugees

Table 1. AM's test results

	11/10/2005	8/3/2006	5/5/2006	6/7/2006
Platelets x10 ⁹ /L (150–400)	75	48	54	61
White cell count x10 ⁹ /L (4.00–11.00)	3.56	1.8	2.67	2.71
Bilirubin mcmmol/L (6–24)	25	43	30	31

not been shown to have any teratogenic effects, and in many circumstances it is deemed that the benefits outweigh the potential harm.⁸ When all worms are killed and egg production ceases, the granulomatous response to existing eggs subsides and associated hepatic changes and portal hypertension may significantly diminish or partially resolve over a period of months.⁸

The main risks of *S. mansoni* liver disease are thrombocytopenia with its risk of bleeding; splenomegaly with the risk of trauma; and the development of oesophageal varices because of the portal hypertension.¹³ Splenectomy is unlikely to be part of the treatment as it has been shown to be of only limited benefit and carries many risks.¹³ If there is haemorrhage of varices and banding is unsuccessful or contraindicated, portosystemic shunts or one of the newer shunt procedures may be needed to reduce portal pressure.⁴

The lack of subsidised payment for praziquantel on the Pharmaceutical Benefits Scheme (PBS) in Australia means that general practitioners need to refer patients to specialised refugee services or hospital for treatment. Schistosomiasis was not mentioned in the list of recommended investigations for the new Medical Benefits Schedule (MBS) item number 714 for Health Assessments for Refugees,¹⁴ but it is obviously important to be aware of this potentially severe disease in those who come from endemic areas.

Conclusion

This case of schistosomiasis illustrates just one of the many asymptomatic 'exotic' illnesses that may have serious, long term implications if not detected and treated promptly and appropriately. As community GPs are now being encouraged to be the first point of contact for refugees, there needs to be support and education about diseases that are uncommon in Australia. Similarly, as the source countries for refugees vary, hospital doctors need to

be conscious of the changing need for unusual tertiary investigations and treatments. Screening, treatment, and follow up and a high index of suspicion may prevent morbidity and mortality in patients such as refugees who settle in Australia.

Conflict of interest: none declared.

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