Schistosoma Haematobium Infection in Malaysia — A Case Report

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Summary
An imported case of Schistosoma haematobium infection presenting with haematuria and proteinuria is described. This would constitute a first case of urinary schistosomiasis in Malaysia. The patient failed to respond to multiple antibiotic treatment and was successfully treated with praziquantel.

Key words: Urinary schistosomiasis, Schistosoma haematobium, haematuria, Malaysia, praziquantel

Introduction
Schistosomiasis, a chronic debilitating disease acquired by the cutaneous route, affects over 250 million persons living in rural and agricultural areas of the world. An estimated 1.5 billion human beings live in endemic areas\(^1\), with 600 million people being in constant threat of acquiring this protean disease\(^2\).

Intestinal schistosomiasis is caused by S. mansoni and S. japonicum and recently 2 new species causing the same clinical syndrome have been discovered and they are S. mekongi from Thailand\(^3\) and S. malayensis in Malaysia\(^4\). On the other hand, urinary schistosomiasis is caused by S. haematobium which is endemic in 52 African and Eastern Mediterranean countries\(^5\). The 5 principal schistosomes infecting man are transmitted by fresh water snails and the free swimming larvae, called cercariae, which are shed into the water and are able to penetrate host skin directly. After migration from the dermis through the lungs to the hepatic portal system, the schistosomes come to live in the small mesenteric or veins of the urinary plexus, where each female lays more than 100 eggs per day into the blood stream. The host’s immune reactions to the eggs, which become lodged in the tissues, is largely responsible for the chronic debilitating and often fatal disease.

Schistosomiasis in immigrants in Malaysia was first reported in 1904\(^6\). Between 1919 and 1933, 41 imported cases were reported in Tan Tock Seng Hospital in Singapore\(^2\) among Chinese immigrants. Indigenous schistosomiasis was found as an incidental finding in an orang asli in 1973\(^7\). Subsequently, a further 9 cases were reported\(^8\) and up to 1987, a total of 12 autopsy and 11 living cases of human schistosomiasis have been reported in Malaysia\(^2\). The etiological agent has been identified as S. malayensis, a new species within the S. japonicum complex.

While no new cases of indigenous schistosomiasis have been reported thus far, except for the ones reported, cases of intestinal schistosomiasis caused by S. mansoni and S. japonicum among foreigners and immigrants have been recorded in General Hospital and University Hospital, Kuala Lumpur, between periods 1964 and 1987\(^2\) and subsequently after that. All confirmations were by biopsy or autopsy based on the clinical presentation of hepatosplenoengaly and portal hypertension. So far, no cases of urinary schistosomiasis have been recorded in Malaysia among immigrants or foreigners admitted to our hospitals and this report would constitute the first of its kind.
Case Report

A 10 year old Ghanian girl was referred to the nephrology clinic in University Hospital in 1991 with 6 weeks history of gross haematuria, which occurred with every micturation associated with mild terminal dysuria. There were no blood clots or stones passed. She did not have any fever or cough nor abdominal pain or loin pain. Her appetite was normal. She had no easy bruising, alopecia or mouth ulcers. She had seen many general practitioners and had been treated for urinary tract infection with antibiotics with no response. The haematuria persisted. There was no family history or past history of haematuria or bleeding disorders. She is the third of 4 siblings. Except for her, all of the other family members are well. She had her BCG vaccination when she was 7 years old. The family moved to live in Malaysia in 1988.

Physical examination revealed a well child, weight 30 kg and height 131 cm. She was afebrile, blood pressure was 120/70 mmHg. There was no pallor or oedema. Review of systems was normal. In view of her country of origin and relative well-being, *S. haematobium* infection of the urinary tract was strongly suspected and her urine was sent for microscopic examination. Urine examination confirmed gross haematuria with 160 red blood cells and 40 white blood cells per microlitre of urine, proteinuria was 2+ and typical *S. haematobium* ova with well-preserved miracidia were seen. Urine culture for bacteria and screening for tuberculosis were negative. The renal profile was normal.

She was treated with oral praziquantel at a dose of 600 mg 6-hourly twice. On follow-up a month later she had no more haematuria and the urine was free from *S. haematobium* ova. She was then advised a 3 to 6 monthly urine examination to detect any recurrence.

Discussion

Vesicle schistosomiasis caused by *S. haematobium* is restricted to the African continent, except for a few pockets in India, Portugal and Mauritius. Being restricted in distribution, an establishment of an endemic foci in other countries is unlikely because of the absence of the snail hosts. Therefore, only imported cases such as the one described can thus be reported in other countries including Malaysia.

Haematuria and proteinuria are among the cardinal features of *S. haematobium* infection that have been well documented. One of the commonest causes of gross haematuria amongst Malaysian children is post-streptococcal glomerulonephritis. This child being not a Malaysian and having a residence history of being in Africa, and particularly in Ghana, with prolonged gross haematuria for 2 weeks and in the absence of any other features suggestive of post-streptococcal glomerulonephritis, a diagnosis of *S. haematobium* was strongly suspected and this was further confirmed by the presence of terminal spined ova in the urine sample examined.

Praziquantel has been found to be effective in the treatment without major haematological or biochemical changes. For *S. haematobium* infection, a single dose orally of 40 mg/kg body weight is effective and the cure rates have been found to range from 72% to 100%. In this case, the child was treated with 20 mg/kg body weight for 2 doses and the child responded and the follow-up after 2 months revealed its effectiveness. The only constraint was the availability of praziquantel in this country.

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References


