Schistosomiasis among travellers returning from Malawi: a common occurrence

Sir,

In August 2001, 11 pupils accompanied by four teachers, from a secondary school in London, visited Malawi for 4 weeks. During their trip they swam in Lake Malawi, having been assured by local people that this area was free of schistosomiasis.

Five weeks later, one of the pupils, a 17-year-old boy, presented with a history of ‘flu-like’ symptoms, an itchy rash and swelling of the eyelids. On direct questioning, he remembered a generalized itchy rash that occurred immediately after swimming. Clinically, he was apyrexial with an extensive urticarial rash. The rest of the examination was unremarkable. He had an eosinophilia at $0.98 \times 10^9/l$ and elevated alanine transaminase, but otherwise investigations were normal. Stool and urine microscopy and schistosomiasis serology were negative. He was admitted and diagnosed with Katayama fever, an acute manifestation of schistosomiasis. He was given two doses of praziquantel, $20 \text{mg/kg}$ and prednisolone $20 \text{mg daily}$ for 5 days.

On the day of his discharge, his friend, a 16-year-old boy, presented with a 10-day history of ‘flu-like’ symptoms, fever and swelling of the lips, eyes and hands. He had wheals on his limbs, but examination was otherwise normal. Investigations revealed an eosinophilia of $2.21 \times 10^9/l$. Stool and urine examinations were negative, but his schistosomal serology was weakly positive. He was also diagnosed with acute schistosomiasis and treated with praziquantel and prednisolone.

We subsequently screened all 15 members of the party 8 weeks after their exposure using a structured questionnaire, clinical examination and laboratory testing. In all, 13 had acquired schistosomiasis; all 13 had an eosinophilia. Twelve had positive schistosomal ELISA and two had eggs of $S. \text{haematobium}$ seen in their urine. No cases of $S. \text{mansoni}$ were found. The two index cases displayed the most florid clinical picture of Katayama fever, but ten others complained of a range of symptoms (Table 1). Of the two that did not become infected, one spent less time in the lake than the others (3 h compared to an average of 8.5 h) and the other had applied copious amounts of mosquito repellent. Prophylactic DEET has been shown to reduce the risk of acquiring schistosomiasis infection.

Schistosomiasis has three distinct clinical syndromes. The first, ‘swimmer’s itch’, occurs in response to cercarial penetration of the skin. Katayama fever, the second clinical entity, classically occurs 4–6 weeks later, and is thought to be precipitated by the onset of ovulation by maturing schistosomulae. The syndrome is most marked in primary infections in patients who have not previously been exposed. Chronic schistosomiasis results from the deposition of schistosome eggs and subsequent fibrosis, classically involving the liver with $S. \text{mansoni}$ and $S. \text{japonicum}$, and the urogenital tract with $S. \text{haematobium}$. Significant morbidity may also be associated with ectopic disease at various sites. The high prevalence of infection among this group reinforces the frequency with which schistosomiasis is acquired in Lake Malawi.

### Table 1 Symptoms described by infected individuals

| Possible ‘swimmer’s itch’ | 2/11 |
| ‘Unwell’ at some point since return | 10/11 |
| Flu-like episode | 9/11 |
| Febrile episode | 2/11 |
| Fatigue | 8/11 |
| Myalgia | 2/11 |
| Dry cough | 8/11 |
| Rash | 0/11 |
| Subcutaneous tissue swelling | 0/11 |

The data exclude the two index cases, hence $n=11$. 

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range of symptoms they experienced were very non-specific, and could easily have been attributed to a viral infection; indeed several had consulted primary health care services and been given this diagnosis. The key to diagnosis is to suspect it in anyone with exposure to potentially infected waters 4–8 weeks previously, and the presence of a peripheral blood eosinophilia. A negative serology result and normal microscopy of stool and urine does not rule out schistosomiasis.

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Bilateral massive adrenal haemorrhage revealing coeliac disease

Sir,
We describe a case of coeliac disease that remained undiagnosed for many years and induced a hypocoagulant state and bilateral adrenal haemorrhage. This is the first reported case of bilateral adrenal haemorrhage in a patient with this disorder.

A 43-year-old woman presented with sub-acute back pain requiring morphine. She did not smoke, drink alcohol, or take medications or illicit drugs. Physical examination revealed severe cachexia (BMI 15 kg/m²). She was afebrile, with a blood pressure of 110/75 mmHg. She reported a 10-year history of progressive unexplained weight loss with lack of energy. There was no decrease in appetite until 6 months prior, when she began to limit her intake due to persistent diarrhoea. Laboratory investigation revealed potassium 3 mmol/l, albuminaemia 14 g/l, prealbuminaemia 0.086 g/l (N 0.21–0.41), calcium 1.2 mmol/l, phosphate 0.5 mmol/l, cholesterol 2 mmol/l and vitamin D 8 nmol/l (N 26–113). Haemoglobin was 11 g/dl with normal white blood cell and platelet counts. C-reactive protein was <7 mg/l. Prothrombin level was 16% (N 70–100%) with a severe decrease in vitamin-K-dependant cofactors (factor II 0.09, factor X 0.04 IU, with normal factor V). Thyroid function was normal. HIV test was negative.

Parenteral nutrition was initiated, with correction of vitamin K and D deficiencies. Given her past medical history, a diagnosis of coeliac disease was considered, and later confirmed by strongly positive antibody titres against both gliadin (ELISA: immunoglobulin A 1300 UR/ml and immunoglobulin G 1250 UR/ml, N<25) and endomysium (indirect immunofluorescence 1:80) and by subtotal duodenal atrophy. A gluten-free diet was initiated. Anti-nuclear antibodies, lupus anticoagulant, anticardiolipin and anti-beta2glycoprotein 1 were all negative. Thrombophilia screen, including protein S and C levels, anti-thrombin level, activated protein C resistance and factors V and II, was normal. Radiological evaluation of the lumbar spine revealed severe osteopenia, which was subsequently confirmed by bone mineral density (lumbar T score –4). Bone scintigraphy revealed multiple fissures on ribs without signs of vertebral lumbar fractures. A thoracic-abdominal CT scan revealed isolated enlarged adrenals, and led to the diagnosis of bilateral adrenal haemorrhage (Figure 1). Cortisol level and a cosyntropin stimulation test did not show adrenal insufficiency, but hydrocortisone replacement was prescribed, due to the high risk of loss of adrenal function. Two months later, the patient had gained 10 kg and was able to discontinue morphine. Six months later, CT scans showed atrophy of the adrenal glands.

Coeliac disease is a common condition (prevalence about 1:300 using serological tests) but frequently undiagnosed in adulthood, particularly when gastrointestinal symptoms are absent.1,2 In this patient, severe malabsorption led to vitamin K deficiency, and the resulting anticoagulated state predisposed her to massive bilateral adrenal haemorrhage.