

An unexpected pulmonary bystander

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ABSTRACT

A 30-year-old man from Eritrea was admitted with a pulmonary bacterial abscess. Unexpectedly, histopathology of the resected lobe also revealed an infection with *Schistosoma mansoni* with surrounding granulomatous tissue and fibrosis. Patients from endemic areas are often asymptomatic with blood eosinophilia being the only diagnostic clue. Early recognition is important as ongoing fibrosing inflammation may result in organ damage.

KEYWORDS

Schistosomiasis, lungs

INTRODUCTION

Recognizing an infection with *Schistosoma* is important to prevent severe complications. Patients from endemic areas with schistosomiasis are often asymptomatic with blood eosinophilia being the only diagnostic clue, as illustrated in the following case.

CASE REPORT

A 30-year-old male from Eritrea was referred to the University Medical Center Groningen because of a cavitary lesion in the left lower lobe, diagnosed on routine X-ray following immigration. His medical history was unremarkable. He had never been diagnosed with tuberculosis. Our patient had been coughing for several weeks, which was occasionally accompanied by haemoptysis. He had been experiencing fatigue, fever and weight loss. Laboratory results showed normocytic anaemia (haemoglobin 7.8 mmol/l, mean cell volume 87 fl), leukocytosis (leucocytes $12.3 \times 10^9/l$) with increased neutrophils ($7.31 \times 10^9/l$) and eosinophils ($1.14 \times 10^9/l$), and

What was known on this topic?

Schistosomiasis leads to granulomatous tissue in the infected organs which, unrecognised, may result in fibrosis and organ damage.

What does it add?

Patients who are infected with *Schistosoma* and grew up in an endemic area, rarely experience acute symptoms. Therefore, eosinophilia can be the only diagnostic clue in this patient category.

an increased C-reactive protein (68 mg/l). Laboratory tests including liver and kidney tests were in the normal range. A computed tomography scan of the chest confirmed a large cavitary lesion in the lateral region of the left lower lobe with a horizontal air-fluid level indicating either an abscess or empyema (figure 1). The fluid was aspirated by thoracocentesis. *Hemophilus influenzae* was cultured from the pleural fluid as well as the collected sputum. Tuberculosis, echinococcosis and strongyloidiasis were ruled out (with repetitive negative sputum cultures, polymerase chain reaction and Ziehl-Neelsen staining and with negative serology, respectively). Under the working diagnosis of a bacterial pulmonary abscess caused by *Hemophilus influenzae* our patient was treated with amoxicillin 500 mg x 4/day. The eosinophilia was explained at that time by an asymptomatic infection with *Giardia lamblia* isolated from the faeces, which was left untreated. During the next five months of adequate antibiotic treatment, our patient clinically improved and inflammatory parameters normalised; however, there was no radiological improvement. Therefore, left lower lobe lobectomy was performed (figure 2). Tissue culture showed *Hemophilus influenzae* resistant to amoxicillin. Unexpectedly, histology of the lung revealed a worm (figure 3a) and an egg with a lateral spine (figure 3b), compatible with the parasite *Schistosoma mansoni*. The surrounding tissue showed granulomas with fibrosing

inflammation. The diagnosis of schistosomiasis was confirmed with serology performed on serum at the time of presentation (*Schistosoma* worm antigen IgM 1:128 and IgG 1:64). Additional analysis, using a formalin-glycerine sedimentation method, also showed *Schistosoma mansoni* eggs in the faeces. Praziquantel 40 mg/kg/bodyweight/day was prescribed for one day. Our patient had an uneventful recovery from surgery and has been well during eight months of follow-up.

DISCUSSION

Schistosomiasis should be considered in all patients with an unexplained eosinophilia who originate from, or have travelled to, an endemic area for schistosomiasis. Schistosomiasis is endemic in most parts of Africa, where the prevalence can exceed 50% in some local communities.^{1,2} When exposed to contaminated water,

individuals become infected by skin penetration with the larvae (cercariae) of the *Schistosoma* worm. The cercariae enter the venous circulation and migrate first to the lungs and then to the liver, where they reside during the early stages of infection.^{1,2} During this acute phase, a febrile illness (Katayama fever) and pulmonary symptoms such as coughing, wheezing and shortness of breath may occur. The acute phase with pulmonary symptoms typically occurs 3-8 weeks after the initial infection.^{1,2} Imaging at this stage often reveals transient nodular lesions.^{3,4} However, as illustrated in the above case, patients who grow up in an endemic area rarely experience acute symptoms. Therefore, the acute phase of the disease often passes unnoticed in this patient category. Katayama fever mostly occurs in returning travellers who were exposed to a high worm burden and have little or no immunity against schistosomes. After the acute phase, the larvae mature to adult worms and descend through the venous system to their final habitat. Dependent on

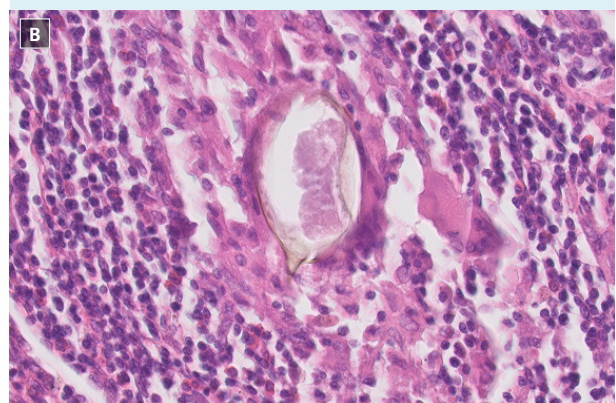
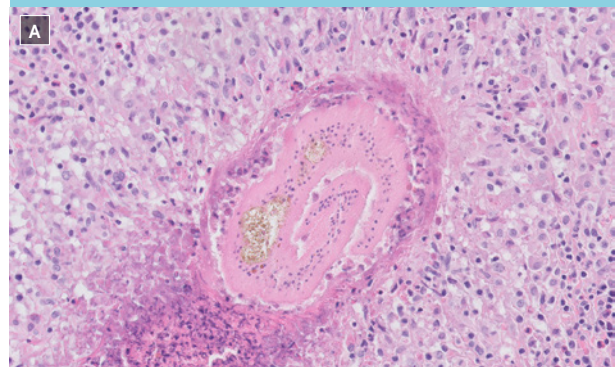
Figure 1. CT scan of the lungs shows a 7 x 11 cm large cavitory lesion in the left hemi-thorax, containing an air-fluid level compatible with an abscess or empyema



Figure 2. Left lower lobe after lobectomy, showing a 4 cm cavitory lesion



Figure 3A. Deceased *Schistosoma mansoni* worm with surrounding granuloma. The surrounding parenchyma show extensive infiltrate with eosinophils, plasma cells and lymphocytes. Digestive tract remnants can be seen as brown pigment in the centre of the worm. (Haematoxylin and eosin; 200x).
B. *Schistosoma mansoni* egg with in surrounding granuloma in the lung. Note the characteristic lateral spine. (Haematoxylin and eosin; 200x)



the type of *Schistosoma*, the worms usually settle in either the venous plexus of the bowel (*S. mansoni*, *S. japonicum*) or the urinary bladder (*S. haematobium*) and begin to lay eggs, which may occasionally be ectopically deposited throughout the body.^{1,2} The chronic phase of infection can last for several years wherein patients may not experience any symptoms. Due to the presence of eggs in organs, local inflammation and formation of granulomas occur, leading to fibrosis. In the case of *S. mansoni*, portosystemic shunting may also allow eggs to pass directly to the lungs.^{5,6} As in our patient, pulmonary infiltration results in pulmonary fibrosis. Roughly 5-10% of patients who are chronically infected develop pulmonary arterial hypertension which ultimately leads to right heart failure.^{5,6} Other complications of intestinal schistosomiasis are polyposis of the colon, colon carcinoma and portal hypertension due to liver fibrosis. Complications of bladder schistosomiasis include recurrent urinary tract infections and bladder cancer. Cerebral schistosomiasis occurs in roughly 5% of infected patients and can be due to an infection with *S. japonicum* as well as with *S. mansoni*. Because of these severe complications, recognising an infection with *Schistosoma* is important.⁷ Therefore, any patient originating from, or travelling from

an endemic area, who is found to have eosinophilia should be investigated for schistosomiasis.

DISCLOSURES

There are no conflicts of interests.

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