CASE REPORTS

Interrelation of strongyloidiasis and tuberculosis

A D Dwarakanath, M Welton, C J Ellis, R N Allan

Abstract
A 31 year old man from Gambia, resident in the United Kingdom for two years, presented with a two month history of unproductive cough, malaise, weight loss, non-specific abdominal pain, and episodic diarrhoea. Acid alcohol fast bacilli were identified in his sputum, together with *Strongyloides stercoralis* larvae and *Giardia lamblia* cysts in his stools. This case illustrates that latent strongyloidiasis can become overt in the presence of tuberculosis, and the diagnosis of strongyloidiasis must be borne in mind in patients who have previously resided in endemic regions.

*Gut* 1994; 35: 1001-1003

Case report
Gambia is a small country on the western tip of Africa with a population of three quarters of a million people and an area of 11 000 square kilometres.

A 31 year old male heterosexual resident in the United Kingdom for two years was admitted with a two month history of generalised abdominal pain, anorexia, and a 10 kg weight loss. He also complained of episodic diarrhoea with mucus, but no bleeding, for several days each month.

On examination he was thin, with no lymphadenopathy or rashes. Examination of the abdomen showed ascites and there were persistent crepitations at the left base and right mid-zone on examination of the chest. A flexible sigmoidoscopy to the splenic flexure was normal, with a normal rectal biopsy.

On admission his haemoglobin was 12.9 g/l, white cell count 7.6×10⁹/l (no eosinophilia), erythrocyte sedimentation rate 56 mm/h, C reactive protein 177 mg/l, albumin 33 g/l, globulin 50 g/l, international normalised ratio 1.5 (which corrected to 1.0 with vitamin K). Over the next 10 days the alkaline phosphatase increased to 730 U/l (twice upper range of normal) and the γ-glutamyltransferase to 285 U/l (six times the upper range of normal). An ascitic tap was performed. Clear colourless fluid was obtained with a protein count of 65 g/l and lymphocytes 50%. Ziehl-Neelsen staining was negative. The chest x ray on admission showed two areas of patchy shadowing at the right mid-zone and at the left base (Fig 1).

He developed a swinging fever (Fig 2) and it was felt that the clinical picture was consistent with miliary tuberculosis, in view of the chest x ray and exudative ascitic fluid. Anti-tuberculous treatment was withheld at this stage because the diagnosis had not yet been confirmed. Stool microscopy subsequently yielded *Strongyloides stercoralis* larvae and *Giardia lamblia* cysts.

The patient was treated with thiabendazole orally for two days with initial resolution of his fever (Fig 3).

His temperature recurred when the thiabendazole was stopped, and he was treated with metronidazole after the isolation of *Giardia lamblia*. This did not relieve his symptoms and the fever persisted. The patient remained lethargic and his alkaline phosphatase and γ-glutamyltransferase increased. A liver biopsy was performed, which showed non-specific changes. There was no histological evidence of granulomas of *Strongyloides stercoralis* or tuberculosis. At this time despite the lack of firm evidence of acid alcohol fast bacilli he was given anti-tuberculous treatment and within two days his
symptoms resolved and his temperature returned to normal. Shortly after this, the microbiology laboratory reported that acid alcohol fast bacilli had been grown from his initial sputum cultures.

This confirmed the diagnosis of miliary tuberculosis together with Strongyloides stercoralis hyperinfection.

**Discussion**

*Strongyloides stercoralis* is a nematode that is endemic in the subtropics and tropics. Fifteen per cent of World War IIFar Eastern prisoners of war were still infected with *Strongyloides* 30 years after their return to the United Kingdom. Figure 4 summarises the 28 day life cycle.1

The symptoms of strongyloidiasis correspond to the three stages of infection: invasion of the skin, migration of larvae, and penetration of the intestinal mucosa by adults. Although about one third of the people with strongyloidiasis are asymptomatic, the remainder may have symptoms related to one or more of the stages of parasite migration in the host. The skin and pulmonary symptoms resemble those encountered in hookworm disease: pruritic papular erythematous rash and a Loeffer like syndrome with eosinophilia.1

The more characteristic clinical features are seen simultaneously with the intestinal phase of strongyloidiasis. Burning or colicky epigastric abdominal pain often occurs and is associated with diarrhoea and the passage of mucus. Some patients complain of nausea, vomiting, and weight loss with evidence of malabsorption or a protein losing enteropathy.4 Eosinophilia is often a prominent feature. In addition 5–22% of patients may develop a generalised or localised urticarial rash beginning perianally and extending to the buttocks, abdomen, and thighs.1

The disseminated hyperinfective syndrome, which occurs as a result of massive autoinfection is characterised by severe abdominal pain, diffuse pulmonary infiltrates, Gram negative septicaemia, meningitis, alveolar haemorrhage, and disseminated intravascular coagulation. Eosinophilia may be absent. Recognised causes of the hyperinfection syndrome are corticosteroids, neoplastic disease (in particular oat cell carcinoma, leukaemia or lymphoma), diabetic ketoacidosis, measles, leprosy, amoebiasis, immunosuppressive treatment, burns, HIV, and disseminated tuberculosis.4

Screening of patients for *Strongyloides stercoralis* larvae in stools before starting immunosuppressive treatment is recommended in patients who have resided in endemic areas.

Recommended treatment of strongyloidiasis is thiabendazole 25 mg/kg body weight for 48 hours. Some authors recommended treatment for 10 days. The dose of thiabendazole should be repeated at two and four weeks and further stools examined over the next three months.
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Gut 1994 35: 1001-1003
doi: 10.1136/gut.35.7.1001

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