

Strongyloides hyperinfection in immune compromised host

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ABSTRACT

Strongyloides stercoralis is an intestinal nematode that cause mild to moderate gastrointestinal symptoms and can persist in the host for long time due to internal autoinfection. In immunocompromised host the infection can result in overwhelming strongyloidiasis and affect multiple organ systems. We report a case of pulmonary hyperinfection syndrome secondary to *Strongyloides stercoralis* in an immunocompromised host. The diagnosis was made through examination of sputum specimens which demonstrated filariform larvae. Early diagnosis is essential to reduce the morbidity and mortality and it is important to have high index of suspicion especially in immune compromised host in endemic areas.

Keywords: Strongyloidiasis, pulmonary hyperinfection, pneumonitis, *Strongyloides stercoralis*

INTRODUCTION

Human sufferings and death due to parasite infections has been known for ages. The impact of parasitic infections on human health is significant across the globe. *Strongyloides stercoralis* is an intestinal nematode which can persist in human for long period because of indirect development and autoinfection. It can result in fatal hyperinfection syndrome in immunocompromised host. ¹⁻⁶ Hence early detection and treatment is essential for which high index of suspicion is essential.

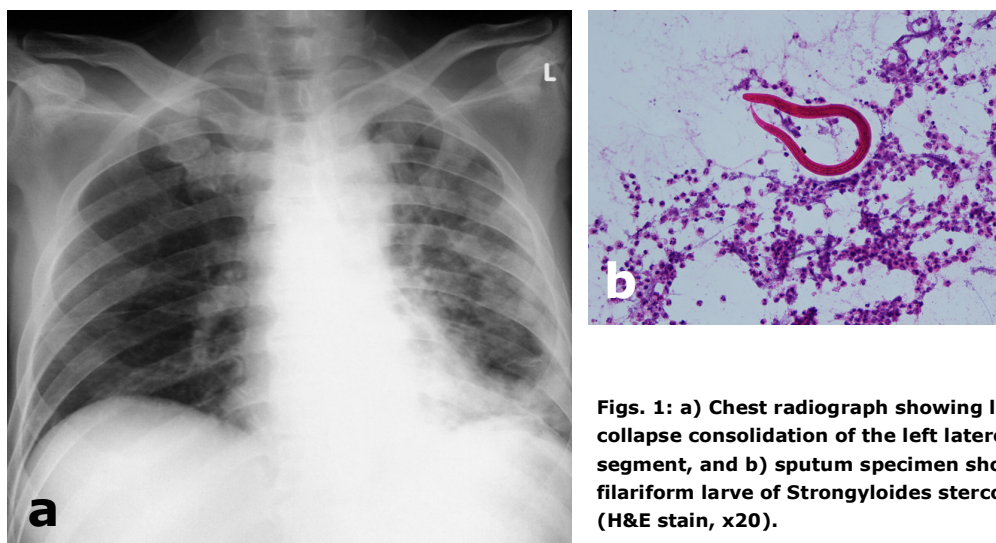
CASE REPORT

A 59-year-old man presented with progres-

sive shortness of breath and cough, productive of yellowish green sputum of two weeks duration. He was diagnosed with focal segmental glomerulosclerosis (FGS) in May 2000, and was treated with daily oral Prednisolone 60 mg that was tapered over eight months to 40 mg daily. Despite on this dose, he relapsed in July 2001 and restarted with same treatment and tapered on another eight months. He had a retroperitoneal myxoid liposarcoma excised in 2004 and a recurrence in 2007 that was successfully treated. In 2012 April he presented with another relapse of FSGS and restarted with steroids and tapered over five months.

He later presented with progressive shortness of breath and productive cough, the

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Figs. 1: a) Chest radiograph showing left collapse consolidation of the left laterobasal segment, and b) sputum specimen showing a filariform larva of *Strongyloides stercoralis* (H&E stain, x20).

the latest presentation.

Relevant laboratory investigations showed leukocytosis with neutrophilia, iron deficiency anaemia and positive for stool occult blood. Peripheral blood did not show any eosinophilia at the time of presentation. There were no parasite/ova in the stool. Imaging studies showed segmental collapse consolidation in laterobasal segment of left lower lung lobe and soft tissue shadows in peripheral part/subpleural part of left lung (Figure 1a). Abdomen showed recurrent liposarcoma in right retroperitoneal area. The sputum was sent for cytology as a part of routine investigations to exclude malignancy.

The sputum microscopy showed filariform larvae of parasite morphologically consistent with *Strongyloides stercoralis* (Figure 1b). The case was thus considered *Strongyloides* pneumonia in a patient with FSGS relapse and recurrent intra-abdominal liposarcoma who was on long term steroid. He was started on appropriate treatment.

DISCUSSION

Strongyloides stercoralis is an intestinal nematode having unique ability to replicate in a human host. The larvae were first described in 1876 in the stool of French troops with severe diarrhoea, returning from the Indochina.⁷ Their ability to replicate by parthenogenesis and cycles of autoinfection allow the infection to persist for long period without symptoms and undetected. *Strongyloides stercoralis* is common in South-East Asia, sub-Saharan Africa and Brazil. With reference to the life cycle (Figure 2),^{1,3} it is the filariform larvae which is infective to human. They can enter the circulation by penetrating the skin or by penetrating the gut wall or perianal skin, and then enter the pulmonary circulation and alveoli. Pulmonary symptoms may arise during migration from the alveolar spaces to the bronchial tree and then to the oesophagus. In uncomplicated cases, pulmonary symptoms are rare. However, if the host is immunocompromised, in particular altered cell mediated immunity, a hyperinfection syndrome may result. In our case, the cause of paras-

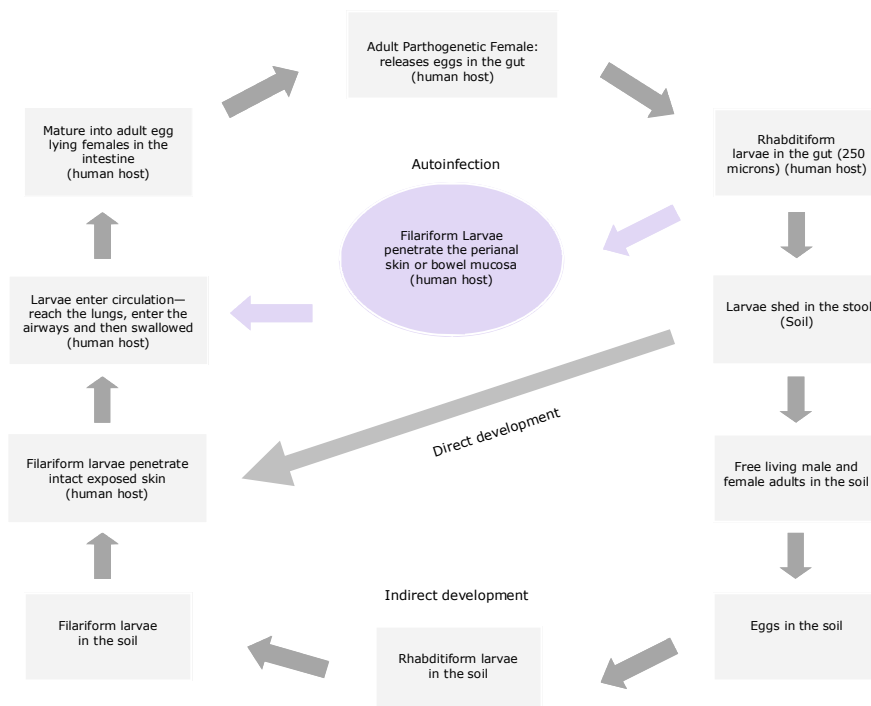


Fig. 2: Life cycle of *Strongyloides stercoralis*; infection by *Strongyloides* can occur through several modes: Autoinfection in infected hosts, Direct involvement or Indirect involvement of the host.

ite persistence or reinfection was likely due to the prolonged steroid treatment.

The term parasite is derived from a Greek word which means 'one who eats at the table of others',⁸ and if the table is offered without resistance the invaders gain the upper hand. There are various immune effector mechanisms against parasites in the host; antibody production, antibody dependent cell mediated cytotoxicity, activation of macrophages, production of CD8+ cytotoxic T cells and natural killer cells.⁸ Antibody dependent cell mediated cytotoxicity is the most important. This involves the helper T-cells which produce different cytokines and activate the eosinophils and mast cells against the parasites. However, the parasites have a variety of mechanisms to evade the host defense system; antigenic variation, suppression of

complements and inflammatory mediators, prevention of phagolysosome formation, immune suppression and breaching the defense barriers. Malignancies, autoimmune diseases, malnutrition and being on immunosuppressed from medications or diseases can weaken the immune system, and increase the risk of hyperinfection syndrome.¹⁻⁵

Steroids have very complex anti-inflammatory and immunosuppressive effects. Steroids suppress the activation and clonal proliferation of the T-helper cells, decreased antibody dependent cell mediated toxicity, reduction of interleukins production, expression of cyclo-oxygenase 2, reduced concentration of the complement components and levels of nitric oxide. Steroids also cause acute suppression of eosinophilia and reduced lymphocytic activation in response to parasit-

ic infection.^{5,10} Our case also did not have peripheral eosinophilia. Steroids have direct effect on parasites by accelerating transformation of rhabditiform larvae to the invasive filariform larvae or rejuvenating reproductively latent adult females.⁴ Overall, steroids suppress the host's inflammatory response against the parasites and results in a fulminant disease. Our case had been on long term steroid therapy and had been immunosuppressed.

The clinical manifestations of strongyloides infection can occur from 20 days post infection to as late as several years. In asymptomatic cases, the infection can persist for decades without further exposure of the host to exogenous infective larvae.⁶ Hyperinfection syndrome or disseminated infection is associated with high mortality rate. Therefore, a high index of suspicion is important in the management of hyperinfection syndrome. Patient may present with recurrent pneumonia, multiple pulmonary infiltrates, skin lesions and malabsorption in endemic areas.¹⁰

Definitive diagnosis is through demonstration of the larvae in the stool. Serial stool examination is more sensitivity than single stool examination.^{2,9} Examination of a single stool sample may miss 70% or more cases, and the diagnostic sensitivity increases to 50% if three consecutive stool samples are examined. Diagnostic rate of over 90% has been reported when seven samples were examined. The other diagnostic modalities are demonstration of larvae in duodenal aspirates (especially in children), sputum, bronchioalveolar lavage fluid, bronchial washings and brushings and even in pleural fluid. Serum antibodies to strongyloides antigens (ELISA)

is a sensitive method.

In conclusion, *strongyloides stercoralis* in immunocompromised individuals may result in disseminated infection and hyperinfection syndrome which is associated with a high mortality. Early diagnosis necessitates a high index of suspicion in endemic areas. Patients may present with recurrent pneumonia, malabsorption or skin lesions.

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