

# *Strongyloides Stercoralis* Infection in a Case of Ankylosing Spondylitis

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## ABSTRACT

A 69 year old man was admitted in the hospital with chief complaints of fever of 2 days duration, swelling and pain of lower limb and knee joint for 4 days. He was a known case of ankylosing spondylitis for last 10 years with chronic intake of corticosteroids. On admission the patient was diagnosed with right lower limb cellulitis and right lung consolidation and pleural effusion. Blood culture and synovial fluid culture showed growth of *Staphylococcus aureus*. 2 days later patient complained of diarrhoea. Routine stool examination showed plenty of rhabditiform *Strongyloides Stercoralis* larva. The patient was treated with Ivermectin for *Strongyloides* and was discharged after total resolution of infection with advice of regular follow-ups for any dissemination or hyperinfection.

**Keywords:** *Strongyloides stercoralis*; Hyper infection; Spondylitis; Immunodeficient

## INTRODUCTION

*Strongyloides stercoralis* is a soil transmitted nematode endemic in tropical and subtropical regions including south east Asia and far east like Myanmar, Thailand, Vietnam and Malaysia [1]. Its prevalence is seen in temperate regions but is more common in the hot and humid regions. It is commonly transmitted by the filariform larvae that penetrates the skin and enter the systemic circulation. It remains asymptomatic in immune competent patients but can cause hyper infection, disseminated infection that can prove fatal in immunosuppressed patients [1-4].

The worm passes its life cycle in one host and a change of host is not essential [1-3]. Man is the optimum host. The parasite has a unique ability to undergo auto-infective cycle due to which it can persist for years. Hyper infection is seen only when immunity of the host wanes and the autoinfection cycle accelerates leading to increased number of filariform larva [5]. The adult *Strongyloides* worm lives in the mucous membrane of the small intestine of man especially in the duodenum and jejunum [1]. As soon as the eggs are laid, the rhabditiform larvae start hatching and bore their way out of the mucous membrane into the lumen from where they are passed in the faeces [1-3].

Alternatively, rhabditiform larva can directly transform into filariform larva inside the gut and penetrates the perianal skin or colonic mucosal wall and enter the systemic circulation to repeat the migration and form an internal cycle of auto infection [6]. This characteristic allows the worm to persist in human host for decades [3].

A high index of suspicion is needed to detect infection and thereby protect those on steroid and other immunosuppressive therapy or infected by viruses such as Human T-Lymphotropic Virus (HTLV-1) and HIV infection, patients with haematological malignancies and those with diabetes and malnutrition [3,7,8].

## CASE PRESENTATION

A 69-year-old man was admitted in our institution with chief complaints of fever with pain and swelling of the right knee joint and right leg for 4 days. His past history revealed he had ankylosing spondylitis and had been under regular oral steroid therapy for the last 3 months. The patient was on chronic but irregular therapy of corticosteroids ever since he was diagnosed with ankylosing spondylitis 10 years back. On examination, he was found to be febrile with tenderness in right leg and knee joint. His blood pressure (100/60 mmHg) and arterial partial pressure of oxygen (PaO<sub>2</sub>) (97 mmHg) were noted. No oedema, cyanosis or clubbing was noted. He was given a preliminary diagnosis of right lower limb cellulitis with lower respiratory tract infections. Piperacillin/tazobactam was started as an empirical therapy.

On radiological assessment, CT study of thorax showed sub segmental consolidation in right middle lobe along with bilateral pleural effusion. Pleural fluid LDH level was high (996 mg/L). Correspondingly, serology was negative for Human Immunodeficient Virus (HIV), Hepatitis B Surface Antigen (HBsAg); C-reactive protein was high 222 mg (normal, <5 mg/L), white blood cell count was 14.6 ml (normal, 4.0=11.0), with neutrophil percentage of 90% (normal, 50%-70%). His eosinophilic count was normal probably due to steroid therapy.

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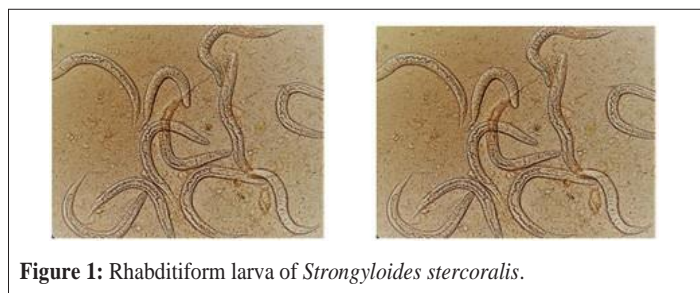
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His hemoglobin concentration, coagulation profile, liver function test and renal function test were within normal limits. Doppler study of right lower limb was normal with no impression of DVT or venous insufficiency.

Blood culture and synovial fluid culture of the patient revealed profuse growth of *Staphylococcus aureus*. He was continued on the same antibiotics.

However from day 2 of admission, the patient also complained of passing loose stool 4 to 5 times daily. The stool occult blood test was negative and culture did not show growth of any pathogenic bacteria. Direct microscopic examination of stool specimen revealed numerous rhabditiform larvae of *Strongyloides stercoralis* (Figure 1). Serial wet mount samples on the subsequent two days also showed the same rhabditiform larva. He was then treated with ivermectin in a single dose, 200 µg/kg orally for 2 days with repeated daily stool examination to verify eradication and to exclude the presence of other parasitic infections. Larval counts reduced significantly and were undetected in the stool sample from day 6. The patient was finally discharged with total resolution of infection after eleven days of admission here are 103 patients consisting of 68 males and 35 females aged 31-74 years. According to TNM classification of GC, there were 10 cases at stage I, 38 cases at stage II, 44 cases at stage III, and 11 cases at stage IV. According to TCM syndrome types of GC, there were 21 cases of liver qi invading the stomach type, 20 cases of qi stagnation and blood stasis type, 15 cases of Phlegm-dampness accumulation type, 7 cases of yin deficiency due to stomach heat type, 17 cases of qi and blood deficiency type, and 23 cases of deficiency-cold of the spleen and stomach type.



**Figure 1:** Rhabditiform larva of *Strongyloides stercoralis*.

## DISCUSSION

*Strongyloides stercoralis* remain asymptomatic in one third of infected cases [4]. Symptoms may vary from pruritic skin rashes, pulmonary manifestations to abdominal symptoms [1-2]. Hyper infection syndrome may occur with massive larval autoinfection in immune compromised patients. Gram negative bacteremia is one of the common manifestations of strongyloidiasis which occur as a result of mucosal injury and damage by filariform larva [5-8]. Filariform larva carry the bacteria to distal organs as it migrates through the body in the course of its life cycle leading to bacteremia. Secondary bacterial infection can result in pneumonia, meningitis, septicaemia and is frequently the immediate cause of death in hyper infection syndrome [5-8]. Corticosteroids have a particularly strong and specific association with the development of hyper infection syndrome [7]. Corticosteroids enhance the apoptosis of Th2 cells, and subsequently reduce natural immunity thereby leading to hyper-infection/disseminated infection.

Hyper infection syndrome has been described regardless of dose or route of steroid administration [7]. In the present case, the patient had a history of corticosteroid intake for several years and got admitted with septic arthritis and pneumonia with secondary bacteremia. Since the patient refused bronchoscopy, presence of larva in bronchoalveolar fluid could not be ascertained and hyper infection could not be confirmed. Peripheral eosinophilia may be seen in acute infections but may not be present in immune compromised patients as in our case though there was leucocytosis.

The gold standard for the diagnosis of strongyloidiasis is serial examination of the parasites with routine saline or wet mount preparations, concentration techniques (Baermann concentration, Horadi-Mori filter paper culture, quantitative acetate concentration technique), culturing the samples on agar plates and histopathological and cytological studies (duodenal biopsy, duodenal aspirate) [7]. If an infection is suspected and there is no specialised method of detection, several stool samples over consecutive days should be examined [6,7]. In our case, rhabditiform larva was detected in the first stool examination. The drug of choice for Strongyloidiasis is Ivermectin which is consistently more effective than Albendazole. The duration of therapy depends on the immune status and disease condition of the infected person [3-5]. The treatment should be aimed at complete eradication of the parasite [7]. Regular follow up is needed to check for hyper infection or dissemination in such patients.

## CONCLUSION

*Strongyloides* can cause asymptomatic infection which can lead to hyper infection and dissemination in chronically infected immune compromised patients. Hence treatment is needed for prevention and a regular follow up of such patients become essential to check the immune status and further re-infection or dissemination of infection.

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