International Journal of Basic and Applied Medical Sciences ISSN: 2277-2103 (Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/jms.htm
2015 Vol. 5 (1) January-April, pp. 276-279/Medhi et al.

Case Report

ASCITES WITH *STRONGYLOIDES STERCORALIS* IN A PATIENT WITH TUBERCULOSIS AND HOOKWORM INFESTATION

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ABSTRACT

Strongyloides stercoralis is an intestinal nematode causing gastrointestinal (GI) infections that are often asymptomatic. Decreased host immunity may lead to hyper-infection, with the generation of a large number of flariform larvae that may disseminate throughout the body. We report a case of 40-year-old male presenting with tuberculosis that developed ascites with S. stercoralis and concomitant hookworm infection. A 40 yr old male presented with ascitis, dyspnoea and mesenteric lymphadenopathy with septatated ascitis on USG. Cytological examination of ascitic fluid revealed fair number of microparasites. A differential diagnosis of W. Bancrofti or S. Stercoralis was suspected. Patient received ATT based on USG findings and high ADA levels. Finger prick sample did not show microfilaria, *Wuchereria bancrofti* antigen specific test was negative. Morphology of the microparasite more consistent with S.stercoralis filariform larvae and a few eggs, which contain developing larvae were also observed. This confirmed the diagnosis of disseminated Strongyloides stercoralis. Patient also showed hookworm larva in stool sample. Strongyloides stercoralis in ascetic fluid is rare and need to be differentiated from filarial infestation.

Keywords: *Strongyloides Stercoralis, Ascitic Fluid*

INTRODUCTION

Strongyloides stercoralis is an intestinal nematode causing gastrointestinal (GI) infections that are often asymptomatic. It is endemic in tropical and subtropical countries. The prevalence of infection varies widely geographically and is commonly associated with rural areas and inadequate sanitation (RMG, 1989). In uncomplicated strongyloidiasis, most patients have a low worm burden and are asymptomatic or have only mild cutaneous and/or abdominal symptoms. When symptoms occur in these patients, they may do so irregularly and with asymptomatic periods (Liu *et al.*, 1993). Decreased host immunity may lead to hyper-infection, with the generation of a large number of flariform larvae that may disseminate throughout the body. This is associated with a high morbidity and mortality. In disseminated strongyloidiasis larvae may invade the gastro-intestinal tract, lungs, central nervous system, peritoneum, liver and kidney (Wurtz *et al.*, 1994; Liu *et al.*, 1998). We report a case of 40-year-old male presenting with tuberculosis that developed ascites with S. stercoralis and concomitant hookworm infection.

CASES

A 40-year old man, came with the chief complains of ascitis and dyspnoea for two weeks. Physical examination showed pale tongue and mucosa, poor nutrition, arterial blood pressure of 116/70 mmHg. Abdominal examination revealed ascitis with liver slightly palpable. Laboratory tests showed increased erythrocyte sedimentation rate (140 AEFH), leucocytosis (12,000/mm3), eosinophilia (AEC=2640/mm3), and hypochromic, microcytic anemia (Hb 8.5g/dl). Serum ADA level was high(40u/l) and serum albumin low(2.4g/l). Serum bilirubin and liver enzymes were within normal limits. PA view chest was normal and USG abdomen reported mesenteric lymphadenopathy with septatated ascitis. Cytological examination of ascitic fluid revealed fat globules, cholesterol crystal, and fair number of microparasites. A differential diagnosis of W. Bancrofti or S. Stercoralis was suspected. Patient was started on DOTS CAT-II regime based on contact history, high serum ADA and septated ascitis with lymphadenopathy

Patient was recovering well on DOTS and ascitis resolved. The absolute eosinophilia was much lower now (1280/ul). Finger prick thick blood smear (20 mm^3) during night at 2230 hour was taken, stained

with Giemsa did not show presence of microfilaria. *Wuchereria bancrofti* antigen specific test (BinaxNOW Filariasis) tested negative. Thick smears were prepared for atypical blood timings of the microfilaria at 1200 hr, 1600 hr and 2100 hr but these showed negative results. Subject's stool sample for helminth parasite's ova confirmed hookworm egg. Morphology of the micro parasite more consistent with S.stercoralis filariform larvae and a few eggs, which contain developing larvae were also observed. This confirmed the diagnosis of disseminated Strongyloides stercoralis. HIV status was negative.
The patient was prescribed albendazole 800 mg/day for 14 days and discharged on request. Control tests could not be done as the patient was lost on follow up.

DISCUSSION

There are very few reported cases of patients with ascites infected with S. stercoralis. In 2004, Hong et al reported the case of a man who came to the United States, four years earlier from Liberia and developed ascites and subsequently was found HIV positive. In this patient, the diagnostic paracentesis showed numerous filariform larvae of S. stercoralis and stool examination confirmed the presence of both rhabditiform and filariform larvae. The authors considered the case to be the second reported in the English-language literature, after the first one reported by Lambroza (1991) (Hong *et al.*, 2004, Lambroza *et al.*, 1991). Lawate and Singh (2005) reported a case of eosinophilic ascites in a patient from India (Lawate *et al.*, 2005) and Ramdial *et al.*, (2006) reported an autopsy case series of 5 HIV positive male patients from South Africa with mesenteric lymphadenopathy, intestinal pseudo-obstruction and ascites (Ramdial *et al.*, 2006).

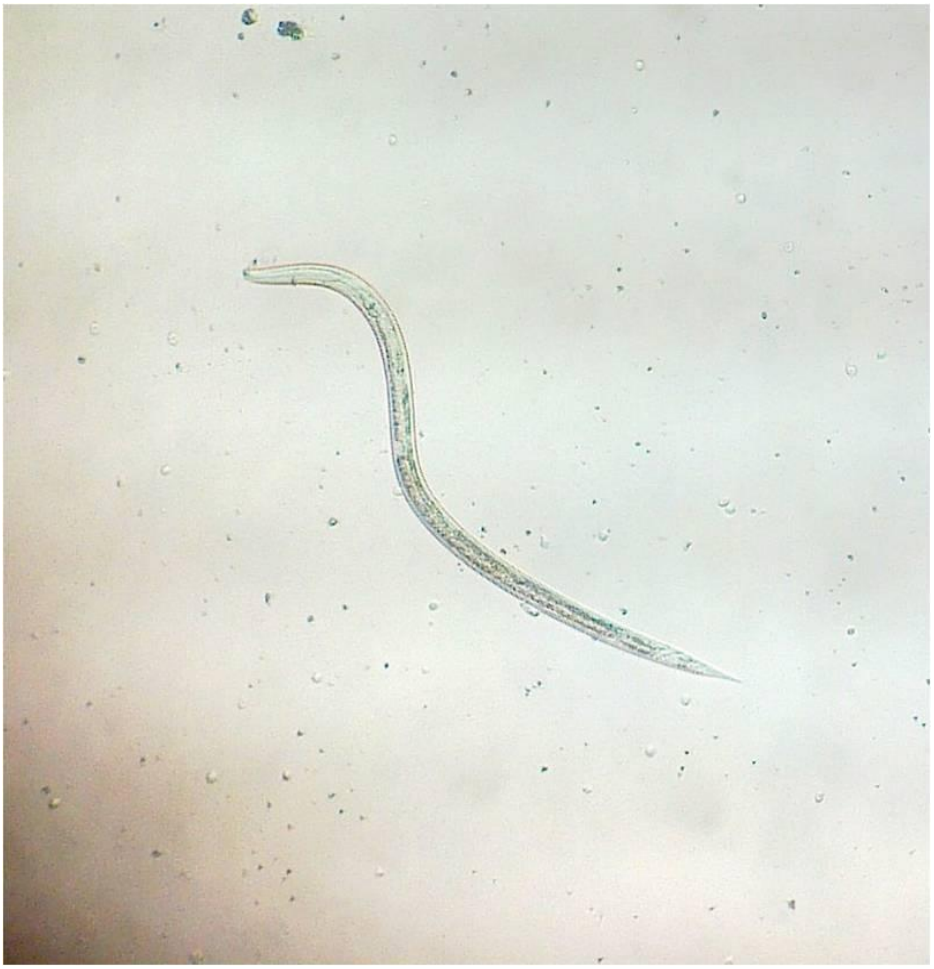

Figure 1: Showing ascitic fluid with mobile S.Stercoralis Larvae

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The adult female larvae can remain embedded in the mucosa of the small intestine for years, producing eggs that develop either in rhabditiform, noninfective larvae or filariform, infective larvae. Manifestations of dissemination occur when the filariform larvae penetrate the intestinal wall and migrate in the blood. Pulmonary involvement is common, and the central nervous system may be affected. Much less commonly described is invasion of the peritoneal cavity with peritoneal effusion (Hong *et al.*, 2004).

Disseminated Strongyloidiasis is commonly reported in immunocompromised patients; in our patient HIV status was negative with normal blood sugar level: however patient came from a very poor socioeconomic background with malnutrition, both high risk factors for disseminated strongyloidosis (Keiser *et al.*, 2004). The presence of abdominal adenopathy has been reported in infection with S. stercoralis (Ramdial *et al.*, 2006) and could also be a present in case of abdominal tuberculosis. However resolved lymphadenopathy on review ultrasound indicates the cause of adenopathy to be tuberculosis.

In conclusion, this is the first reported case form north-east india of infection of ascitic fluid with Strongyloides stercoralis in a patient with tuberculosis with co existence of hook worm infestation. Strongyloides stercoralis in ascetic fluid is rare and need to be differentiated from filarial infestation. Early detection of S stercoralis may alter the often-fatal course of infection.

Figure 2: Showing ova of S.Stercoralis in faeces

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