Filariform larvae of Strongyloides stercoralis-An unusual finding in ascitic fluid in an immunocompetent patient

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ABSTRACT:

Background

Strongyloides stercoralis, a nematode parasite in humans with free living and autoinfective cycles, is an asymptomatic infection of small intestine. Disseminated infection usually results due to increased worm burden in immune-compromised hosts. The adult female larvae remains embedded in the intestinal mucosa for years, producing eggs that develop into infective filariform larvae which on transmural intestinal penetration can reach the peritoneal cavity and produce ascites. Ascites with Strongyloides stercoralis infection in an immune-competent patient is extremely rare.

Case Report: *A 60 year old female presented with chief complaints of weakness, loss of appetite, alternating diarrhoea and constipation and frequent pain abdomen since 6 months with severe abdominal distension and marked pedal edema for 1 month. Diagnostic paracentesis showed numerous filariform larvae of Strongyloides stercoralis. Stool examination confirmed the presence of both rhabditiform and filariform larvae. The patient was found to be immunocompetent with HIV negative and HBsAg negative.*

Conclusion: Early detection of Strongyloides stercoralis may alter the often fatal course of infection.

Key Words: Ascites, Immunocompetent, Strongyloides stercoralis.

INTRODUCTION

Strongyloides stercoralis is a nematode common in tropical and subtropical areas infecting approximately 100 million people each year, worldwide. The prevalence of infection varies widely geographically and is commonly associated with rural areas and inadequate sanitation.(1)

The adult form can survive and reproduce either in the soil or in the human small intestine. The life cycle of S. stercoralis in human begins when the infective filariform larvae penetrate the skin and migrate to the lungs. Once the larvae reach the pulmonary capillary vessels, they migrate through the capillary walls into the pulmonary alveoli. The larvae are eliminated to the larynx and then are swallowed, gaining access to the small bowel. The larvae develop into adult females, which lay eggs that hatch non migratory (rhabditiform) larvae. They are passed in the stool and may also penetrate the mucosa, leading to internal auto-infection.(2)

A patient infected with S. stercoralis may present various clinical syndromes.(3) severe forms (hyperinfection) are reported, usually in immunosuppresed patients with potential severe outcome.(2) Dissemination may involve the gut, stomach, lung, the CSF and may develop ascites.

After an extensive literature search, we identified only few cases in which S. stercoralis was found in ascitic fluid. This case of Strongyloides hyperinfection highlights the protean picture of Strongyloides infection. Second, the patient´s immunocompetent profile is uncommon and did not reveal any of the identified risk factors that predispose to Strongyloides hyperinfection. Finally, the case depicts the underreported burden of disease and allows us to revisit Strongyloidiasis, a relevant and endemic parasitic infection.

CASE REPORT

A 60 year old female presented with chief complaints of weakness, loss of appetite, alternating diarrhoea and constipation and frequent pain abdomen for 6 months. For last one month she complained of weight loss, breathlessness, pedal oedema and abdominal distension. On investigation haemoglobin level was 5.4gms/dl. Total leukocyte count was 10,800/cumm of blood. Differential count showed N-57%, E-7%, B-0%, L-34%, M-2%. ESR was 87mm in 1ST hr. Chest X-Ray was clear. Ultrasonography of

abdomen showed mild hepatomegaly with moderate ascites. Electrocardiogram and Echocardiogram were normal. Diagnostic paracentesis obtained >1200ml of xanthochromic ascitic fluid. The fluid had a high parasitic load and the diagnosis of Strongyloides stercoralis was readily made by microscopic examination of direct smears and one thin prep slide.

Microscopic examination of a routine cytological preparation of the ascitic fluid revealed around 60 filariform larvae of Strongyloides stercoralis in a background of mixed inflammatory exudates and reactive mesothelial cells. (Fig-1A)

The filariform larva of S. stercoralis showed a characteristics blunt and notched end of the tail. (Fig-1B) The stool examination also showed numerous filariform and rhabditiform larvae. The patient was treated with thiabendazole for 2 weeks. Post- treatment paracentesis was performed twice and showed marked diminution in the number of larvae. Further investigations showed the patient to be HIV and HBs Ag seronegative with normal CD4 count. The patient showed a great improvement in the symptoms and signs of acute abdominal distress and discharged with follow up scheduled for management of S.stercoralis infection.

DISCUSSION :

Strongyloides stercoralis is a nematode with its life cycle initiated when infective filariform larvae penetrate the human skin or mucous membrane. The parasites enter the circulation, pass through the lungs, migrate up the larynx and are then swallowed. Larvae develop into adults in the duodenum. Female deposits their eggs in the intestinal mucosa, and these hatch into rhabditiform larvae that migrate into the intestinal lumen and pass into the feces. [4] What is unique in case Strongyloides strecoralis is its ability for autoinfection? This occurs when rhabditiform larvae develop into filariform larvae in the large intestine. The filariform larvae then invade the intestinal mucosa and enter the blood to start another cycle without leaving the body or host. Strongyloidiasis presents in wide variety of clinical syndromes, however almost 50% of chronically infected individuals are asymptomatic. [5].Hyper-infection syndrome refers to an increase in parasite burden due to acceleration of the autoinfective cycle, without an accompanying spread of larvae outside the usual migration pattern. In hyperinfection and disseminated state, secondary infection frequently occurs in the form of gram negative or polymicrobial baetormi. [6] Although the exact pathogenesis is unknown, it is believed that persons with impaired host immunity are at risk for hyper-infection. The literature identifies the following as major risk factots- immuno-suppressive therapy particularly corticosteroids, transplantation, haematological malignancies, HTLV-I and HIV infections. Additional risk factors include malnutrition, diabetes mellitus, chronic renal failure and chronic alcohol consumption. The most common antecedent factor is steroid use both endogenous and exogenous. Steroids have been shown to affect immunity by increasing T2 helper cell apoptosis, reducing the eosinophil count and mast cell response. Further more, it is proposed that it increases the production, mainly in the intestinal wall of ecdysteroid-like substance that act as molting signals lead to increased production of autoinfective larvae. [3]

S.stercoralis is unique in that the adult worms are parthenogenetic females that become localized (embedded) in the upper small bowel mucosa, where the worms lay eggs that develop into rhabditiform larvae. Rhabditiform larvae may metamorphose into filariform larvae and penetrate the host's intestinal wall or perianal skin, producing an autoinfective cycle that perpetuates the infection. [7]Gastrointestinal symptoms are most common but non-specific. Some case reports do not mention any gastrointestinal symptoms. Abdominal pain, often described as crampy or bloating in nature, watery diarrhoea , constipation, anorexia, weight loss, difficulty in swallowing, sore throat, nausea, vomiting, and gastrointestinal bleeding in any order or combination are frequently reported. Ileum and small bowel obstruction may result, with diffuse abdominal tenderness and hypoactive bowel sounds. Protein-losing enteropathy may give rise to acute or worsening hypoalbuminemia with peripheral edema or ascites. Hypokalemia and other electrolyte abnormalities may reflect these gastrointestinal abnormalities. [8] Above described patient presented with weakness and loss of apetite. Herein lies the unique profile of our patient. She did not have the most common risk factor of steroid use. There was no evidence of malignancy and therefore no exposure to any chemotherapeutic agent. She was HIV negative, HBSAg negative, and non-diabetic. Furthermore, weight loss was assessed to be more due to strongyloides hyperinfection. Eosinophilia is usually the only indication of strongyloides infection, but it is mild (5%-15%) and non-specific. [9] A limiting factor is the absence of this finding in a number of cases of hyperinfection or disseminated infection. In our patient with hyperinfection, the tools for arriving at a diagnosis were simple and straightforward with isolation of the larvae in both stool and ascitic fluid specimens. Strongyloidiasis is an important parasitic infection that has a propensity to disseminate and cause rare but treatable hyper-infection syndrome. It is

thus important to consider this entity in patients presenting with unexplained ascites in the background of chronic gastrointestinal symptoms and or acute onset pulmonary symptoms . The absence of risk factors for hyperinfection and /or dissemination should not mislead the physician as to lower the index of suspicion for this endemic infection, since early diagnosis and prompt treatment could be life saving. Finally we recommend doing research on the prevalence and epidemiology of strongyloidiasis in the country, including risk factor for hyperinfection and/or disseminated strongyloidiasis.

Established Facts:Strongyloides stercoralis infection is mostly asymptomatic .Strongyloides stercoralis induced ascites is seen in immunocomprised patients.

NOVEL INSIGHTS –strongyloides stercoralis has a propensity to disseminate and cause hyperacute infection with ascites even in immunocompetent patients

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Legends :

Fig 1 A –Characteristic coiled filariform larva of strongyloides stercoralis (MGG Stain x 100)

Fig 1 B- Larva showing blunt, notched end of tail in a background of

mesothelial cells and chronic inflammatory cells (MGG X 40)

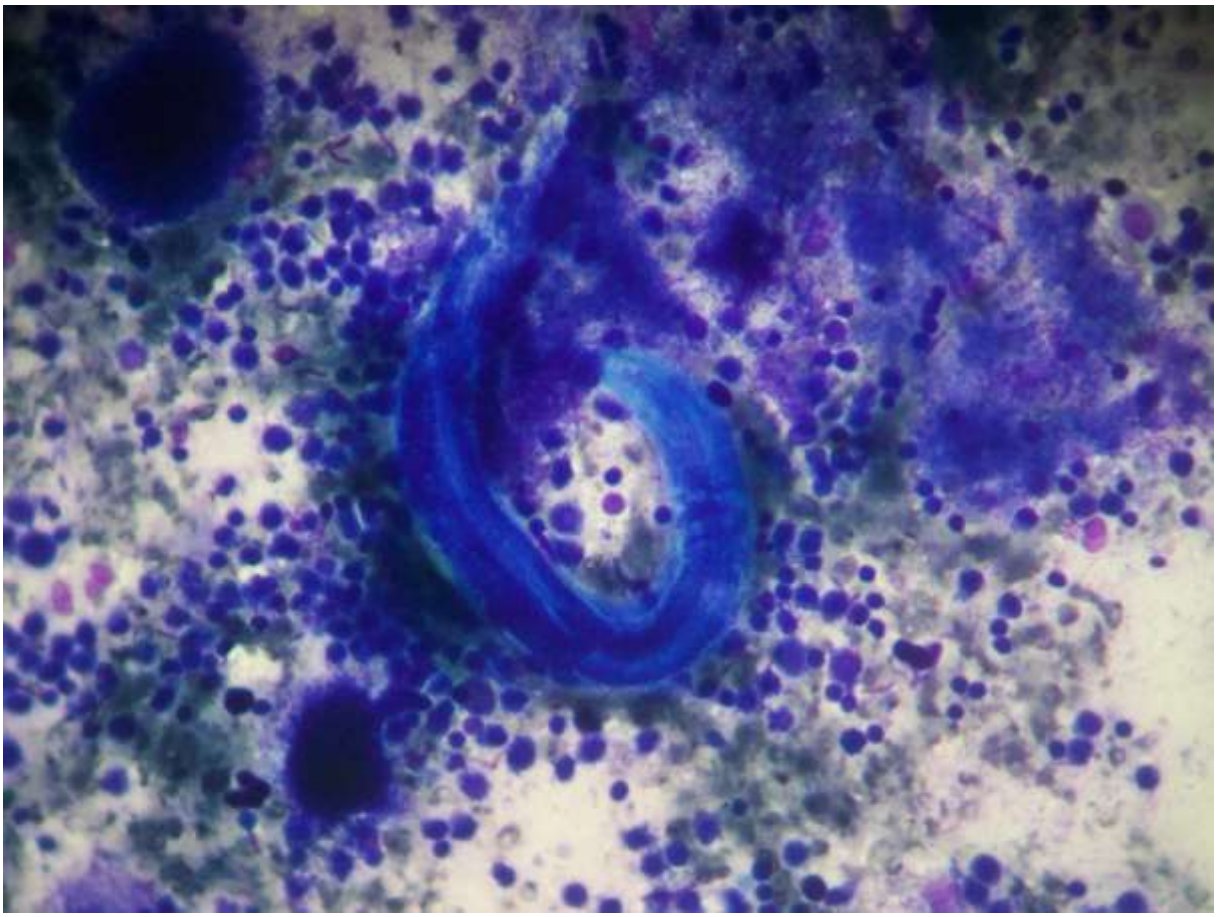

Fig1A

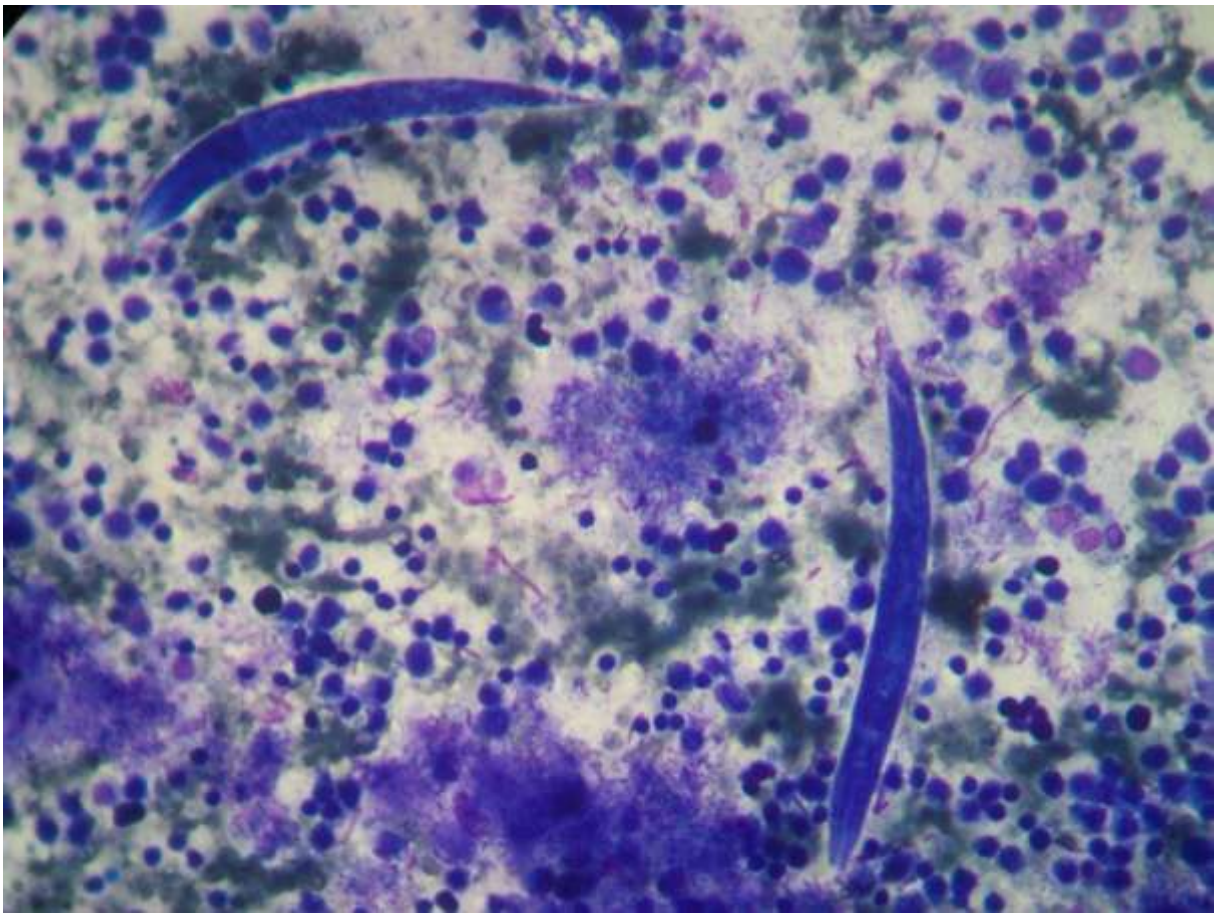

Fig1B