

Hypoalbuminemia Secondary to Strongyloides hyper Infection - An Often Missed Entity

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Abstract: *Strongyloides stercoralis* is distinguished among helminths by its unique ability to replicate in the human host. Strongyloidiasis (infection by *S. stercoralis*) can be contracted by walking barefoot on contaminated soil or through autoinfection. A 53-year-old male farmer presented to our outpatient department for evaluation of a progressive increase in abdominal distension and pedal edema. History was positive for alcohol intake but there were no signs of liver failure. Examination showed bilateral grade 2 pedal edema till the knees and free fluid in the abdomen. Investigations revealed normal cell counts, hypoalbuminemia, mildly elevated ALT, AST and typical coagulation profile. Ultrasound abdomen showed mild hepatomegaly with moderate ascites. Urine microscopy was negative for proteinuria. Esophagogastroduodenoscopy which was done as a part of screening for signs of liver failure, revealed duodenitis with multiple small ulcers (1-2 mm) and focal edema, which were biopsied. Histopathological examination showed crypts containing multiple Strongyloides and larva containing eggs. So, the stool was sent for examination, which revealed the rhabditiform larva of Strongyloides. Hypoalbuminemia in this patient is concluded to be secondary to protein losing enteropathy due to strongyloides hyperinfection superimposed by alcoholism. He was commenced on a course of Ivermectin and Albendazole and protein rich diet. The patient's symptoms resolved on follow-up, and the albumin level gradually improved to 2.8g/dl.

Keywords: Strongyloides, Hyperinfection, Hypoalbuminemia, Protein-losing enteropathy, Duodenitis, Rhabditiform larva, Ivermectin, Albendazole

1. Introduction

The nematode, *Strongyloides stercoralis*, still spans almost 70 countries, affecting up to 100 million people, but most predominantly in tropical areas [1]. Humans are infected when the filariform larvae penetrate the skin and via the bloodstream travels to the lungs, from where they climb the trachea and larynx and are swallowed into the esophagus. The larvae then burrow into the duodenum and proximal jejunum and mature into female adults. These adults hatch eggs, which subsequently are shed in the stool as rhabditiform larvae. Albeit most larvae are shed, some re-enter the circulation by penetrating the intestinal wall, leading to auto-infection [2]. Most infections are asymptomatic. Sporadically, the only manifestation of the disease may be low-grade eosinophilia in a normal person. However, in those with immunocompromised status, presentations may range from abdominal pain, malabsorption, and intestinal obstruction to acute respiratory distress syndrome or sepsis.

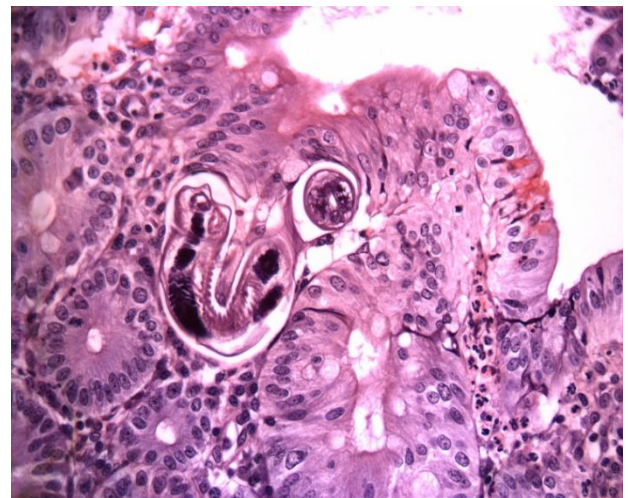
2. Case Report

Presentation

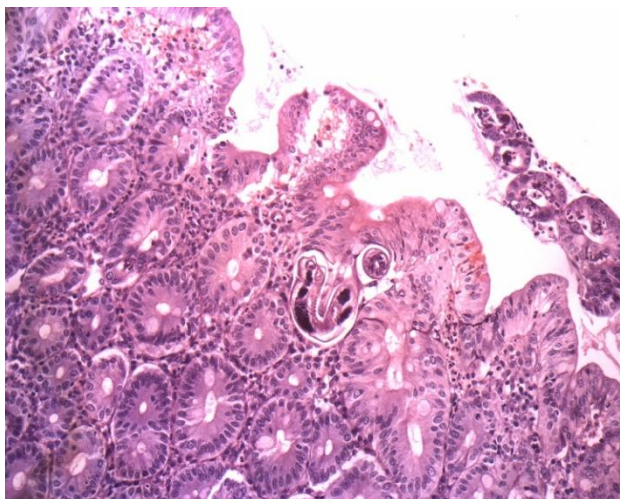
A 52-year-old male agriculturist had presented with complaints of generalized weakness for one month along with fever, abdominal distension, and pedal edema. He had no known comorbidities and was not on any medication but there was history of chronic alcohol abuse. General examination showed bilateral pedal edema till the knees and systemic examination was unremarkable except for ascites.

Investigations

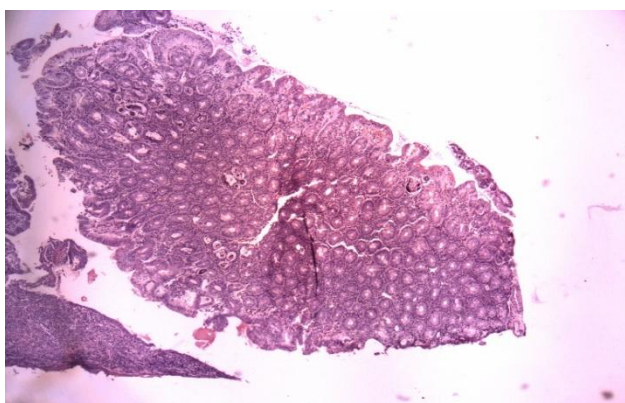
Labs showed hypoalbuminemia, and slightly elevated SGOT (98mg/dl) and SGPT (58mg/dl). Remaining tests were within normal limits. Abdominal ultrasound revealed mild hepatomegaly with moderate ascites, bilateral pleural effusion with collapse. Urine microscopy was negative for proteinuria. After that, echocardiography proved to be a regular study. As a part of screening for signs of liver failure, the patient underwent an upper gastrointestinal endoscopy, which showed evidence of duodenitis, for which the patient was started on a course of anti-H.pylori medications pending biopsy report. Biopsy came back positive for numerous Strongyloides and eggs with developing larvae.



1) Original Histopathology showing Larva in the crypts of Duodenum.



2) Original Histopathology showing multiple crypts and embedded larva of *Strongyloides* in the Duodenum.



3) Original Histopathology showing multiple larvae in the crypts.

Stool microscopy was undertaken, which revealed the larvae of *Strongyloides stercoralis* confirming our diagnosis. HIV-ELISA and CD4 counts were performed to rule out other superimposed condition. Both turned out to be negative.

Treatment and response

He was treated with Ivermectin and albendazole for seven days along with protein rich diet and supportive therapy and strict abstinence to alcohol. Repeat stool examination showed no larva, and there was a remarkable improvement in the symptoms and albumin levels improved over course of time.

3. Discussion

Strongyloides' incidence is usually underestimated because of its subclinical manifestations and low sensitivity tests for diagnosis. The incidence of *Strongyloides* hyperinfection depends on the demography of a particular area, for example as in locations where moist soil and improper disposal of human waste coexist, especially in West Africa, the Caribbean, and Southeast Asia, tropical sections of Brazil its incidence accounts to 40 % [1]. But the incidence in India is very low. In a study conducted in north-eastern India, the incidence was around 8.5% [2]. *Strongyloides* infection can be acute, chronic, disseminated, and hyperinfection. The latter two are most common in immunocompromised individuals.

In uncomplicated *Strongyloidiasis*, many patients are asymptomatic or have mild cutaneous symptoms like recurrent urticaria involving buttocks and wrists or may have abdominal symptoms. Migrating larva can lead to *larva currens* (running larva). While passing through small intestine, adult worms can burrow into the duodenojejunal mucosa leading to duodenitis, which can cause midepigastic pain resembling peptic ulcer pain. Nausea, diarrhoea, gastrointestinal bleed, mild chronic colitis, and weight loss can occur. But the above discussed patient did not manifest any of these typical findings.

Abrogation of the host immunity which confines the autoinfection cycle, leads to hyperinfection, with the generation of huge number of filariform larvae which can cause duodenitis, colitis, enteritis or malabsorption. The duodenitis in this patient lead to exudative protein loss and lead to decreased albumin levels and caused the pedal edema and ascites. Risk factors to contract infection in our patient were he being a farmer by occupation and working barefooted on damp soil. And his chronic alcohol abuse landed him in immunocompromised state which caused the parasite to flourish and cause hyperinfection. The mortality in hyperinfection is about 87% [5]. So prompt recognition and treatment determine the prognosis.

The peripheral eosinophilia, which is a common finding in various parasitic infections and which is noted in acute or chronic *Strongyloides* infection, is not common in hyperinfection, which was the case in our patient obscuring the possibility of parasitic infection at our first glance.

In uncomplicated *Strongyloidiasis* infection, stool examinations may be repeatedly negative. But luckily, the sensitivity of stool examination increases in case of hyperinfection due to a large number of larva and eggs which was the case in our patient and pointed us towards a definitive diagnosis. We were able to respond promptly and the patient's symptoms improved gradually.

After one month, a follow-up with a repeat stool microscopy confirmed the patient to be asymptomatic and no longer passing larvae.

4. Conclusion

Strongyloides hyper infection is a rare entity in cases of immunocompetent individuals. They present predominantly with lung and gastrointestinal manifestations. This case highlights an atypical presentation of *Strongyloides*, which presented as protein-losing enteropathy leading to ascites and pedal edema, which initially caused a delay in diagnosis. In any patient from endemic background, with significant risk factors, without any identifiable cause of protein loss, the possibility of any parasitic infection should be definitely ruled out.

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