# Strongyloidiasis as a Hidden Cause of Protein Losing Enteropathy

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Parasitic infection still surprises us with amazing case presentation every now and then. Intestinal nematode infection is a worldwide health problem especially in humid and hot areas. *Strongyloides stercoralis (S. stercoralis)* is a common soil- transmitted nematode infection in tropics and subtropics. Its severity ranges from asymptomatic infection in immunocompetent patients to hyperinfection and even disseminated infection in immunocompromised patients. Here, we report a case of severe protein losing enteropathy caused by S. stercoralis.

## INTRODUCTION

The prevalence of *S. stercoralis* varies between countries worldwide. It was found to be 11.1% in Egypt **[1]**. In endemic areas, the parasite can persist and replicate in the host for years causing various clinical presentations including diarrhea, abdominal pain, vomiting, fever, GI hemorrhage, weight loss, adynamic ileus, skin manifestations and pneumonia. In hyperinfection , strongyloidiasis could be a strong etiological factor for chronic diarrhea and protein losing enteropathy **[2]**. Disseminated infection occurs when larvae invade organs other than GIT and lungs i.e, beyond the autoinfection range. Hyperinfection can be encountered in any host but disseminated infection is usually encountered in immunocmpromised patients **[3]**. The prognosis of Strongyloidiasis is usually benign except in hyperinfection and disseminated infection. Disseminated infection can be also complicated by secondary Gram- negative bacterial infection that adds to the bad prognosis of the condition **[4]**. Mortality rate can reach 71% in disseminated disease **[5]**.

## Case Presentation

Five years ago, we were astonished by an interesting case of chronic diarrhea that was discovered to be caused by strongyloidiasis. A female villager patient of 62 years' old, presented to Tropical Medicine Department, Zagazig University Hospitals, with severe abdominal pain, distention, anorexia, nausea, vomiting, watery diarrhea (3-5 times / day) and progressive loss of weight. Her complaint was of about 10 weeks duration and developed into bilateral pitting lower limp oedema. The patient denied any past history of chronic illness including diabetes mellitus and hypertension. Apart from the marked emaciation, abdominal distention and bilateral pitting lower limp oedema, the patient showed no clinical abnormality.

The patient was subjected to some investigations where CBC showed mild eosinophilia, stool analysis showed only mild *Entamoeba coli* (*E. coli*) infection, liver profile showed hypoproteinemia with hypoalbuminemia and serum electrolytes showed hyponatremia and hypokalemia. Abdominal ultrasonography showed minimal ascites that was confirmed by CT scanning of abdomen and pelvis.

Colonoscopy showed nonspecific findings while upper GIT endoscopy showed atrophic hyperemic inflamed mucosa of esophagus, stomach and duodenum. A biopsy was taken from the second part of the duodenum for histopathologic examination. The sample showed ulcerated, shortened and blunted villi. Lamina propria showed dense inflammatory infiltration with polymorphonuclear leucocytes, lymphocytes and eosinophils. The tips of villi showed multiple viable parasitic worms where acute parasitic duodenitis was diagnosed and Nematoda infection with *Strongyloid stercoralis* (*S. stercoralis*) was the most probably suggested (figure 1)

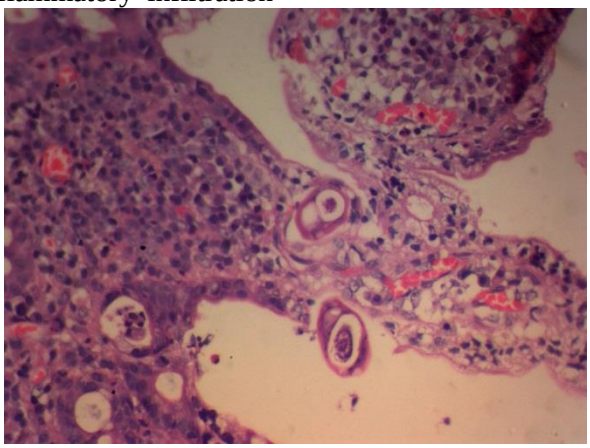

***Fig. (1):*** *Strongloids stercularis larva* is obvious in the tip of villus with dense inflammatory infiltration with PNL, lymphocytes and eosinophils ( H&E stain x40)

The patient received albendazole as a single daily dose of 400 mg / day for two weeks. The patient dramatically improved and reported complete recovery at her last visit - after 5 years of the first presentation - for another different complaint.

## DISCUSSION

The clinical presentation of the patient was not such classical picture of strongyloidiasis especially with the absence of criteria of immunocompromisation. The direct disappearance of the patient for 5 years after receiving albendazole therapy did not give us the chance to complete her investigations and exclude immunocompromising conditions including chronic illness, immunosuppressive therapy and HIV. However, a nearly similar clinical presentation was reported by Lakshmanan et al **[2]**. Moreover, the mere advanced age and malnutrition - manifested in this patient - can be considered as a cause of immunocompromisation that leads to enhancing the autoinfection, hyperinfection and even ultimately disseminated infection **[4]**.

The absence of some clinical manifestations such as dermatological and respiratory symptoms. However, these symptoms are usually suffered in acute stage. Even in chronic strongyloidiasis, these symptoms - if happen - take usually mild intermittent course in most instances. The combined effect of chronic diarrhea, malabsorption and protein losing enteropathy in this patient may augment the hypoproteinemia and hypoalbuminemia causing refractory oedema **[6]**.

The patient presented by hypoproteinemia and hypoalbuminemia that stressed the need to exclude the most suggested causes such as nutritional, hepatic and renal disorders to confirm the protein losing enteropathy syndrome. Some investigations were missed after disappearance of the patient, but the upper GI endoscopy with duodenal biopsy taking and histopathological examination saved the time and effort in the way of diagnosis.

*S. stercoralis* larvae can be detected in stool especially in hyperinfection and disseminated infection. However, stool analysis for this patient did not detect *S. stercoralis* larvae. This can be expected since single direct stool analysis is able to detect the infection in only 30 % of specimens in chronic disease **[7,8]**.

The endoscopic findings of duodenum - including inflammation and ulceration of the mucosa were reported also by Lakshmanan et al **[2]**. They may be not pathognomonic for

strongyloidiasis but they can be considered good diagnostic signs for strongyloidiasis in endemic areas **[9]**. Moreover, *S. stercoralis* colonizes the duodenum and the detection of parasitic larvae in duodenal biopsy is the most accurate method for the diagnosis **[10]**. Despite absence of a clue of immunocompromisation, the protein losing enteritis in this patient can be classified as the most severe form owing to the presence of ulceration and parasitic larvae in the mucosa of duodenum **[11]**.

Even though *E. coli* is a non- pathogenic organism, its presence in stool analysis of an old age patient could be a sign of the non-uncommon concomitant mixed parasitic infection especially in immunocompromised patients **[12- 14]**.

The association between strongyloidiasis and malabsorption was previously reported in a diarrheal case of uncontrolled celiac disease despite adopting a strict gluten- free diet by the patient a situation that provoke the etiologic role of *S. stercoralis* infection in the malabsorption **[15]**. Another case of malabsorption was reported presenting with severe weight loss (more than 20% of the total body mass) and the patient was salvaged by discovering *strongyloidiasis* as an etiologic cause of the illness **[16]**.

The patient was treated successfully with albendazole for two weeks. Ivermectin is the first line therapeutic medicine for strongyloidiasis. However, albendazole is a reliable alternative. The duration of treatment is usually modified according to the severity of the case and should be continued until symptomatic relief and repeated negative stool samples for 2 weeks. This management helps to guard against autoinfection and hyperinfection **[9, 17]**. Fortunately, this patient informed that she was improved dramatically after albendazole therapy despite her disappearance that time - 5 years ago - a situation that did not enable us to follow up her and confirm her recovery.

## CONCLUSION

Protein losing enteropathy can be caused by *strongyloidiasis* even in absence of overt immune suppression. In endemic areas, parasitic infections including *strongyloidiasis* should be put into consideration in every case of protein losing enteropathy especially in immunocompromised patients.

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