



Gastroduodenal Strongyloidiasis Causing Protein Losing Enteropathy – A Case Report and Brief Review of Literature

S. Bharti ^a, A. Singh ^{a*}, R. Kirti ^{b#} and N. Kumar ^a

^a Department of Pathology, All India Institute of Medical Sciences, Patna, Bihar, India.

^b Department of General Medicine, All India Institute of Medical Sciences, Patna, Bihar, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Strongyloidiasis is a common disease worldwide, has a complex life cycle and infect humans percutaneously. We report case of 46-year-old woman, presented with complaints of abdominal pain, and distension. She was on PPI (Proton Pump Inhibitors) for recurrent gastritis. She also gives history of steroid intake for joint pain. USG abdomen showed moderate ascites. Upper GI endoscopy showed diffuse erythematous gastric mucosa with subtle mosaic pattern and edematous duodenal mucosa showing patchy erythema. Histopathological examination of gastric biopsy showed infestation by *Strongyloides stercoralis*, the gastric mucosal glands contained various parts of parasite. The patient recovered completely after course of antihelminthic drug.

Keywords: *Strongyloidiasis; gastritis; ascites; endoscopy; erythema; parasite.*

1. INTRODUCTION

The intestinal nematode *Strongyloides stercoralis* is a soil-transmitted parasite that infects humans.

Millions of people are thought to be infected over the world, while specific figures are unavailable [1]. It's a common disease all throughout the world, but it's more common in tropical and

[†]Assistant Professor;

[°]Senior Resident;

[#]Professor;

*Corresponding author: E-mail: avinashsingh02186@gmail.com;

temperate countries, such as India [2]. Although immunocompetent people infected with this worm are asymptomatic and merely have blood eosinophilia, *S. stercoralis* can cause a fulminant disseminated illness and septic shock in immunocompromised people [3]. Long-term corticosteroid medication, kidney transplant patients, the elderly, and cancers like lymphoma are also risk factors that make infection more severe [4]. Protein-losing enteropathy is a rare GI symptom that can cause immediate or progressive hypoalbuminemia, as well as peripheral edema or ascites [5]. In patients having gastrointestinal endoscopy or resection for neoplasia, the histologic diagnosis of strongyloidiasis from biopsies and surgical material is frequently an unexpected result. *S. stercoralis* is difficult to detect in tissues due to a lack of parasites, their small size, and pathologists' lack of familiarity with the topic, all of which contribute to an unanticipated risk of occurrence [6]. Before a diagnosis of strongyloidiasis is obtained, the majority of these individuals undergo numerous radiological and biochemical tests. Although *S. stercoralis* hyperinfection can affect a variety of organs, the involvement of the stomach and duodenum leading to protein losing enteropathy is rare [7]. Very few studies from India has reported localized involvement to disseminated strongyloidiasis [8]. Herein, we report a case of gastroduodenal strongyloidiasis with presenting symptom of abdominal pain and ascites and reviewed the literatures on strongyloidiasis.

2. CASE PRESENTATION

A 46-year-old woman, presented to medicine opd with complaints of abdominal pain, and distension since 1 month. There was abdominal tenderness on physical examination. Vital parameters were normal. About 3 months back, she had abdominal pain and was diagnosed as gastritis at a private clinic. Since then she was on PPI. There was no history of immunosuppressive disorders except history of steroid intake for joint pain. Laboratory tests revealed mild anemia, hypoalbuminemia, and hyponatremia. Absolute eosinophil count was normal. Her chest radiograph was normal. Patient was admitted and symptomatic treatment was started. A abdominal sonograph showed moderate ascites with grade 2 fatty liver. On upper gastrointestinal endoscopy, there was diffuse erythematous mucosa, subtle mosaic pattern and attenuated gastric folds. Duodenum shows edematous mucosa showing patchy erythema upto proximal D2. Biopsy was taken from multiple sites of stomach and was sent for histopathological examination. Diagnostic and therapeutic tapping was done. Biochemical examination of ascitic fluid showed high SAAG and normal ADA. Ascitic fluid microscopy showed total count of 100 cells/cumm with 100% mononuclear cells on differentials. After fixation there was four fragments of gray-white mucosal tissue. Microscopically, chronic gastritis with mild chronic inflammatory cell infiltrates was observed.

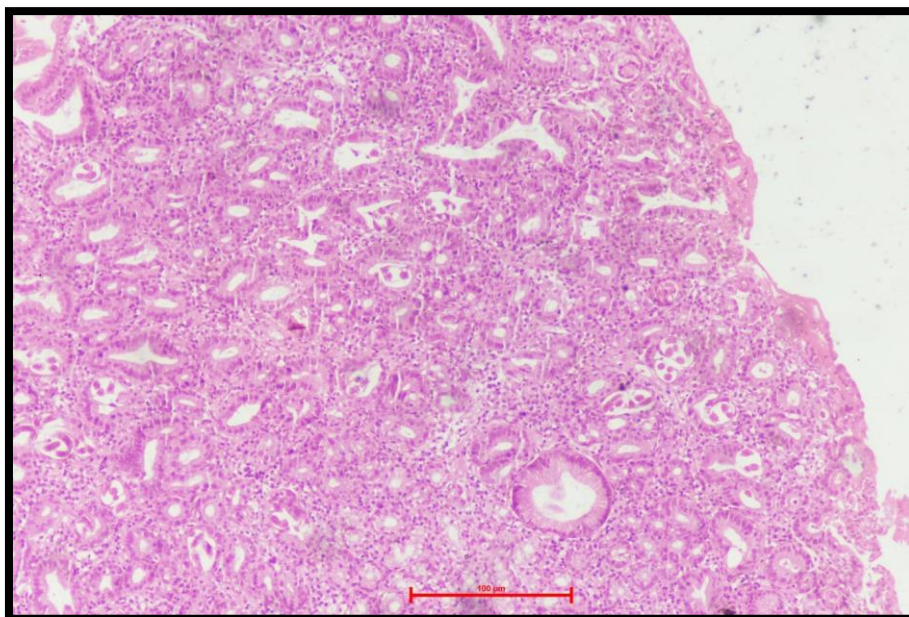


Fig. 1. Chronic gastritis with numerous sections of *S. stercoralis* adult worms. (H&E, 100x)

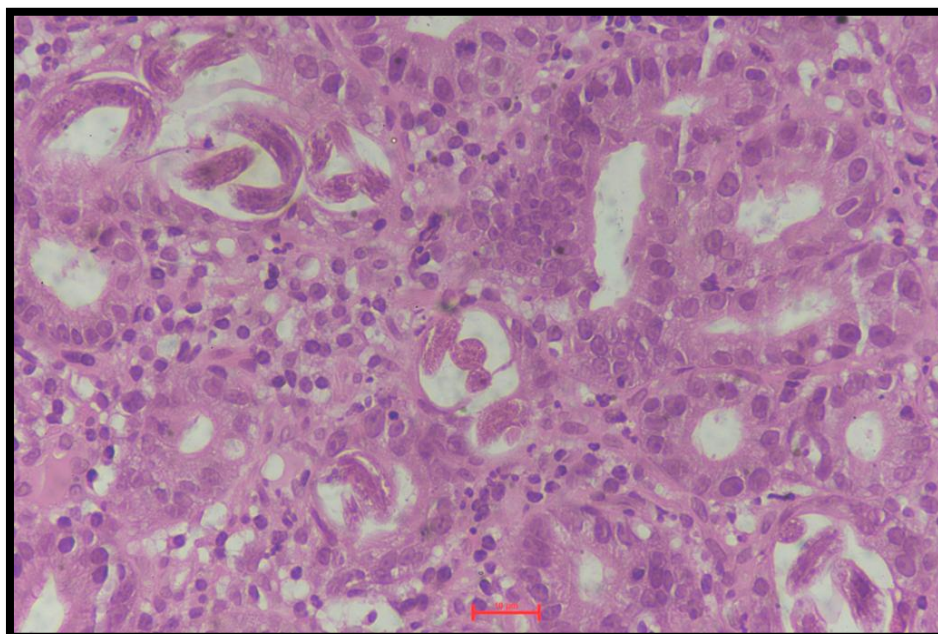


Fig. 2. Adult worm, larvae and eggs seen in the crypts (H&E, 400x)

Figs. 1 and 2 shows gastric crypts containing crosssections of adult worms, eggs, and rhabditiform larvae. Body wall of adult worm is composed of cuticle and weak muscle layer. Eggs in the crypts were basophilic granular mass. Histopathological diagnosis of chronic gastritis with infestation by *S. stercoralis* was made. After this, the patient was given 3 days course of tab ivermectin 10 mg and tab albendazole 400. After 3 days of therapy, patient improved clinically and was discharged.

3. DISCUSSION

Strongyloides is a genus of obligate gastrointestinal parasites that includes about 50 species. This parasite has both free-living and parasitic stages in its life cycle. Adult female worms infesting small intestine lay eggs in the mucosa of the gut, which hatch into rhabditiform larvae that are excreted in the faeces. Humans are typically infected transcutaneously, though infection has been experimentally caused by drinking filariform larvae-contaminated water [1]. After cutaneous penetration, filariform larvae move to the small intestine by unknown methods. A direct, auto-infective, and nonparasitic free-living developmental cycle are all part of *S. stercoralis*'s life cycle. The larvae penetrate the skin to enter the circulation, where they are coughed up and absorbed into the colon, where they mature into mature adult worms. This is a common way for the parasite to spread. In

healthy people, the parasite is asymptomatic and lives in the duodenum, seldom in the colon or stomach, letting the infection to go misdiagnosed and untreated for years. As a result, the parasite can survive in the host, raising the chance of infection throughout the population. Severe cramping, abdominal pain, watery diarrhoea, weight loss, nausea, vomiting, and gastrointestinal bleeding and small bowel blockage are also common symptoms. Acute or progressive hypoalbuminemia with peripheral edema or ascites can occur with protein-losing enteropathy [4]. Immunosuppressive therapy increases the risk of infection, and concurrent achlorhydria (often caused by histamine-2 blockers or proton pump inhibitors) can result in gastric strongyloidiasis [9]. In our case, the patient was given corticosteroid and antacid medication at the same time, which could have been a risk factor.

Eosinophilia is a commonly associated with parasitic infestation, but it is usually low in immunocompromised patients because eosinophils, like lymphocytes, are decreased in AIDS patients [10]. AEC was normal in our case. During hyperinfection, patients with increased peripheral eosinophilia appear to have a better prognosis [11]. Hyperinfection is a term used to describe a state of rapid autoinfection, which is often but not always caused by a change in immunological status. As a result, the presence of signs and symptoms related to enhanced

larval migration is required for the diagnosis of hyperinfection syndrome. Hyperinfection is characterised by presence of numerous larvae in stool and/or sputum, as well as worsening of gastrointestinal and pulmonary symptoms. Filariform larvae can affect any organ system by gaining systemic access through intestinal ulcers, but usually they are confined to the organs normally involved in the pulmonary autoinfective cycle (i.e., gastrointestinal tract, peritoneum, and lungs) in nondisseminated hyperinfection. The phrase "disseminated infection" refers to larvae migrating to organs outside of the pulmonary autoinfective cycle's spectrum. This does not always suggest that the disease is more severe [4].

Cough, wheezing, choking, hoarseness, chest pain (which may be pleuritic in origin), hemoptysis, palpitations, atrialfibrillation, dyspnea, and, in rare cases, respiratory collapse are all pulmonary symptoms. Only one case of pneumothorax has been reported. Filariform or rhabditiform larvae, as well as eggs, may be found in sputum [12].

The wet mount method is used to demonstrate larvae in stool in the laboratory to diagnose strongyloidiasis. Stool microscopy is a straightforward, quick, and low-cost examination. A single direct stool microscopic examination is said to have a sensitivity of less than 30%, increasing to 70% when three stool specimens are screened. The most common morphological type discovered is larva, but eggs have been found in sputum samples from patients with hyperinfection syndrome [8].

In some cases, parasite larvae are discovered in gastric or small intestinal biopsy specimens taken for causes other than strongyloidiasis; in these cases, the specificity of identification is questioned. To distinguish *S. stercoralis* infection from other intestinal nematode infections, more reliable special culture techniques such as the Harada-Mori filter paper strip culture method or the filter paper/slant culture approach are required [13].

In our patient, the microscopy showed chronic gastritis with a large number of parasites within the gastric crypt where eggs were deposited and larvae hatched. With involvement of duodenum there was development of protein losing enteropathy leading to hypoalbuminemia and later on causing transudative ascites and abdominal distension.

Recently, there are development of serological tests that will help in the detection and diagnosis of *S. stercoralis*. The test utilizes recombinant antigen, so follow-up of the titre is possible after treatment and eradication. Recently introduced serological test (NIE-ELISA) uses dried blood spots collected from finger prick on filter paper. The results were comparable to those obtained with blood collected through venipuncture [14].

Ivermectin is the treatment of choice for strongyloidiasis with higher efficacy than other drugs. Moreover, ivermectin is well tolerated with less adverse effects than benzimidazole group of drugs. However, other antihelminthic drugs like albendazole, mebendazole, and thiabendazole have also been prescribed in combination for disseminated and hyperinfection with successful cure [15].

4. CONCLUSION

Strongyloidiasis is a treatable disease. Thus it is necessary to diagnose it early and start appropriate therapy to reduce the mortality and morbidity. Unless severely infected, the clinical signs and symptoms are generally not severe and frequently nonspecific. Patients on steroid therapy or with HIV infection presenting with vague gastrointestinal symptoms should have their multiple stool examined for parasites.

We described a cases of gastroduodenal strongyloidiasis in which the diagnosis was made on identification of the parasite on histological examination. Hence, emphasis on the characteristic histological features was carried out to avoid missing the diagnosis.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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