Small Bowel Obstruction from Strongyloides Stercoralis: Case Report

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Abstract

Strongyloides Stercoralis is a nematode and infection from it is common in Sub-Saharan and South East Asia continent. It is mostly asymptomatic but if it presents, then is usually seen with vague gastrointestinal complaints. Small bowel obstruction is a poorly recognized and possibly underreported complication. We hereby present a case of partial small bowel obstruction from S. Stercoralis.

A 35 year old man from Africa presented with one year history of post prandial nausea, abdominal bloating and bilious vomiting along with significant weight loss. His abdomen X ray and CT scan were consistent with partial proximal bowel obstruction. He presented to different hospitals and was treated for his symptoms, but a definite diagnosis could not be made. We performed upper endoscopy and biopsy which was consistent with S. Stercoralis infection. He was treated aggressively with Ivermectin and Albendazole until his stool cultures became negative for infectious larvae. He was followed up after 3 and 6 months with complete resolution of symptoms. We conclude that, since S. Stercoralis is an uncommon etiology for proximal bowel obstruction, high index of suspicion should be exercised in patients presenting as above and recent travel to areas endemic for S. Stercoralis.

Introduction

Intestinal Obstruction is a poorly recognized and possibly under-reported complication of S. Stercoralis. We present a case of partial proximal small bowel obstruction secondary to S. Stercoralis infection. There are only a few cases described in literature of intestinal obstruction from Strongyloidiasis in last 40 years [1].

Case Presentation

A 45 year old African man who emigrated from Ghana a few years ago presented to our Emergency Department (ER) with complaints of progressively worsening nausea and vomiting after meals for over one year. He also had thirty pounds unintentional weight loss, intermittent dull diffuse abdominal pain. He had multiple ER visits for the same complaints in different local area hospitals. He denied any diarrhea, constipation, fever, recent travel or sick contacts. He last visited Ghana eight years back. He denied any hemoptysis or hematemesis. He was diagnosed with Diabetes Mellitus one year ago and was taking insulin and metformin. His symptoms were not relieved with cessation of metformin. On examination he was in moderate distress due to nausea and non-bloody recurrent bilious vomiting. His vital signs were stable. He had normal heart sounds and his chest was clear to auscultation. He had diffuse abdominal tenderness in the epigastric region.

Laboratory results revealed a white cell count of 5900/mm³ (Granulocytes 43%, Monocytes 13%, Eosinophils 6%), Hematocrit 36%, Platelet 162 k/mm³. His comprehensive panel was within normal limits (Granulocytes 43%, Monocytes 13%, Eosinophils 6%). Hematocrit 36%, Platelet 162 k/mm³. His comprehensive panel was within normal limits including liver function tests and lipase. HgBA1c was 10%. Abdominal X-ray was significant for multiple air-fluid levels and a dilated jejunum in the upper abdomen up to 3.5 cm. Chest X-ray was clear. CT scan of the abdomen 1 week ago at another hospital was remarkable for dilated loops of small bowel, but acute obstruction was ruled out. Surgery team was consulted and patient was given intravenous hydration and was made NPO. A nasogastric tube was inserted and set to continuous suction. It drained 900 cc of bilious fluid. Upper endoscopy was performed, which ruled out strictures and gastric outlet obstruction. It revealed inflammation of the antral and duodenal mucosa with some ulceration. The biopsy was sent and results were expected in 1 week.

The patient was given a diagnosis of partial small bowel obstruction with unclear etiology. Gastroparesis related to diabetes, viral or autoimmune causes were less likely in this setting. Meanwhile the patient improved symptomatically. He was discharged with close follow up when he started tolerating per oral diet. He did not follow up with his appointment and was again re-admitted for a similar presentation one month later.

On his second admission the gastric and duodenal biopsy were found to be positive for S. Stercoralis (Figures 1 and 2). Stool cultures

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Received December 30, 2013; Accepted February 22, 2014; Published February 24, 2014

Citation: Jain V, White SR, Sharma D, Iliev A (2014) Small Bowel Obstruction from Strongyloides Stercoralis: Case Report. J Clin Case Rep 4: 346. doi:10.4172/2165-7920.1000346

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Figure 1: H & E Stain. S. Stercoralis in Gastric Biopsy (arrow).
which were sent were also positive for *S. Stercoralis* showing numerous larvae. He was also HTLV-1 positive. The patient was diagnosed with *S. Stercoralis* Hyper infection Syndrome in the setting of HTLV-1, which lead to partial proximal bowel obstruction. Treatment was begun with Albendazole 400 mg twice daily for 3 days and Ivermectin 200 mcg/kg daily for total of 6 weeks until the infection was cleared and the stool cultures became negative. He was discharged with symptomatic improvement and serial stool cultures were followed in parasitology clinic until they became negative for parasitic larvae after 3 months. He reported no recurrence of symptoms and had started to gain weight.

**Discussion**

Strongyloidiasis is a common parasitic infection of the intestine, which is caused by *S. Stercoralis*, a nematode prevalent in Southeast Asia and tropical sub-Saharan countries [1-3]. The parasite has a complex life cycle, which consists of two predominant larval forms—rhabditiform, a free-living, and filariform, which is the infective form [4]. The filariform larvae enter the skin and travel to the lungs either hematogenously or through the lymphatic system. The larvae proceed up the bronchial airways, are swallowed into the gastrointestinal tract and make their way to the duodenum and proximal jejunum. They settle in the intestinal mucosa and mature into adult females. Through asexual reproduction, the adult females lay eggs, which hatch and give rise to the rhabditiform larvae. These larvae may auto-infect the host by penetrating the intestinal mucosa or perianal skin, or they are freely excreted in feces [5,6]. The parasite thrives in the host and replicates for decades. Sometimes the larvae may travel to other organs outside the pulmonary and GI systems, which results in a disseminated infection and may lead to sepsis, if gram negative bacteria are translocated. This process is associated with a high mortality rate. Immune deficiency, hematologic infection, HTLV-1 infection, renal failure and transplant, steroid use and chronic alcoholism are predisposing factors for disseminated infection [1].

*S. Stercoralis* infection is normally asymptomatic, but may manifest with symptoms of nausea, vomiting, anorexia, weight loss, abdominal discomfort, flatulence and diarrhea [1,7-11]. Unusual presentation includes intestinal obstruction and GI bleed [12-14]. Loffler syndrome is descriptive for pulmonary symptoms like cough and wheezing. Heavy infestation of lungs may lead to dyspepsia, pleuritic pain and hemoptysis [12,13,15]. Larva currens (“racing larva”) is an itchy, cutaneous condition caused by infections with Strongyloides stercoralis. It is caused by the intradermal migration of Strongyloides [14,16,17].

Duodenal obstruction is an extremely rare complication of Strongyloidiasis, with only few cases reported in the medical literature up to 2010 [1,12,18]. Obstruction can be related to severe mucosal edema [15,17], inflammation or external obstruction by the superior mesenteric neurovascular bundle. Duodenal mucosa inflammation leading to incompetent Sphincter of Oddi may result in reflux of oral contrast in to the biliary tree on radiological exam [19]. Usually co-existing HTLV-1 immunodeficiency is present [3]. Another potential complication is paralytic ileus secondary to hyperinfection [19,20-23].

Eosinophilia is an inconsistent finding, present in up to 35% in acute phases. Eosinopenia and increased IgE levels have been associated with poor prognosis [14,24].

A high index of suspicion is essential for correct diagnosis of *S. Stercoralis* related duodenal obstruction. Travel history plays an important role. Diagnosis is confirmed with larvae detected in stool or duodenal aspirate/biopsy taken via EGD. White duodenal villi are a common endoscopic feature [25,26] though not significant in our case. Despite high sensitivity and specificity of the ELISA test, immunodiagnostic tests have certain limitations such as false negative results in immunocompromised hosts, presence of antibodies for prolonged periods even after treatment, and false positive results from cross reaction with other parasitic infections like Ascariasis [3,24]. Imaging studies are also very nonspecific. A unique radiographic feature of Strongyloidiasis is the reflux of oral contrast into the biliary trees, possibly due to an incompetent sphincter of Oddi [27]. In case of disseminated infection, the parasite can be detected in other specimens such as sputum, cerebro- spinal fluid and urine [21].

Stool studies have low sensitivity as the shedding of larvae in stools is only intermittently. Several specimens might be needed on consecutive days. ELISA tests have shown very good results [28].

Medical treatment is indicated even in the absence of symptoms to avoid further complications and hyperinfection syndrome. The drug of choice is Ivermectin 200 mcg/kg/day for at least 2 days, usually prolonged in disseminated or hyperinfection syndrome [28-30]. Combination therapy with Albendazole has shown very good results. Rectal administration has also been suggested where oral is not tolerated.

**Results and Conclusions**

Proximal partial small bowel obstruction is an unusual complication of *S. Stercoralis* infection. The large spectrum of clinical manifestation and lack of classical clinical syndrome (abdominal pain, bloating, heartburn, intermittent episodes of diarrhea and constipation) makes the final diagnosis of Strongyloidiasis difficult. Apart from high index of suspicion, it would be useful to get the basic stool studies, cultures and serological tests to evaluate for Strongyloides infection, if the cause for small bowel obstruction is not clear. In our case we would have send the stool studies and antigen tests for given parasite on prior admissions, the diagnosis could have been made earlier. On the other hand a biopsy of gastric and proximal small bowel by esophagogastroduodenoscopy proves very beneficial in such cases. We conclude that, in patients who have been suffering from recurrent symptoms of bowel obstruction it is prudent to rule out parasitic infections and work up should begin with stool and serological studies as soon as common causes are ruled out.

**Acknowledgement**

Special Thanks to Mladen Jecmenica, MD.
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