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Case report

Chronic intestinal pseudo-obstruction due to *Strongyloides stercoralis*

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ABSTRACT

We report a case of chronic intestinal pseudo-obstruction due to a generalized visceral autonomic neuropathy in an immune-competent patient infected with *Strongyloides stercoralis*. The patient had immigrated to the United States from Sierra Leone in childhood but had not returned for decades. His symptoms resolved with ivermectin treatment. Clinicians should have a high index of suspicion for strongyloidiasis in any patient with abdominal complaints and a history of travel to endemic areas, even if the travel history is remote.

Introduction

Chronic intestinal pseudo-obstruction is a rare disorder caused by damage to the underlying neuromuscular apparatus of the gastrointestinal tract resulting in impaired gastrointestinal motility without an identifiable structural abnormality. There are multiple described pathogenic causes including inherited disorders, connective tissue disorders, paraneoplastic syndromes, endocrine disorders, neuromuscular syndromes, medications and infections [1]. We report a case of chronic intestinal pseudo-obstruction due to a generalized visceral autonomic neuropathy in an immune-competent patient infected with *Strongyloides stercoralis*.

Case report

A 59-year-old man was transferred to a tertiary care center for months of weight loss abdominal distention, constipation, nausea and vomiting. His medical history was significant for hypertension, past hepatitis B infection, and most interestingly, a sigmoid volvulus requiring a sigmoid colectomy with delayed reversal one year prior to admission. Of note, the patient immigrated to the United States from Sierra Leone in childhood but had not returned to West Africa in 20 years.

Physical exam was remarkable for abdominal distention with mild, diffuse tenderness and hyperactive bowel sounds. Table 1 shows his labs on admission which were most notable for eosinophilia. The patient's abdominal film was remarkable for diffuse gaseous distention of the small bowel greater than large bowel compatible with diffuse ileus (Fig. 1).

A small bowel follow-through was significant for globally diminished small bowel transit with retained contrast in the small bowel present 72 h post-administration, concerning for a global visceral neuropathy. Defecography and anal manometry were normal.

The patient underwent an upper GI endoscopy revealing a normal esophagus, an erythematous friable stomach with biopsies showing focal intestinal metaplasia, and normal examined duodenum. Antro-duodenal manometry was consistent with a generalized visceral neuropathy.

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Table 1
Lab results on admission.

Lab	Value	Reference Range
White blood cell count	5.7 K/uL	4.0–10.0 K/uL
Differential	31.7%	36.0–71.0%
Neutrophils	31.8%	20.0–50.0%
Lymphocytes	18.0%	6.0–13.0%
Monocytes	16%	0.0–6.0%
Eosinophils	0.9 K/uL	0.0–0.5 K/uL
Absolute Eosinophil Count		
Hemoglobin	11.8 g/dL	13.5–17.0 g/dL
Platelet	243 K/uL	150–400 K/uL
AST	45 IU/L	8–30 IU/L
ALT	33 IU/L	7–35 IU/L
Albumin	3.5 g/dL	3.5–4.9 g/dL
Creatinine	1.05 mg/dL	0.7–1.3 mg/dL
BUN	21 mg/dL	8–20 mg/dL

Parasite serologies were obtained and identified a highly positive *Strongyloides* IgG antibody of 4.96(NL < 1.49 IV.) Review of the patient's pathology from his sigmoid resection one-year prior showed enteric eosinophilia. Stool ova and parasites exams confirmed *Strongyloides stercoralis* larvae. Workup for primary immunodeficiencies, HIV, and HTLV-I/-II antibody screens were negative.

The patient was started on treatment for strongyloidiasis with ivermectin at 16,000 mcg/kg/day for 7 days. The patient was also started on pyridostigmine uptitrated to 90 mg three times daily to augment intestinal motility. With these interventions the patient was successfully weaned off total parenteral nutrition, able to tolerate an oral diet, and was discharged home. He was seen in clinic follow up 6 months later with complete resolution of symptoms.

Discussion

We present an unusual case of a 59-year-old immune-competent man from West Africa presenting with months of abdominal symptoms, weight loss and pseudo-obstruction with workup revealing a generalized visceral neuropathy and peripheral eosinophilia due to strongyloidiasis. Of note the patient had marked improvement in symptoms upon initiation of anti-parasitic therapy and a cholinesterase inhibitor.

Strongyloidiasis is the clinical syndrome caused by infection with the intestinal nematode *Strongyloides stercoralis*. Strongyloidiasis is endemic in tropical and subtropical regions and occurs sporadically in temperate areas. In tropical and subtropical regions, the overall disease prevalence may exceed 100 million people [2]. The highest rates of infection in the United States are among residents of the southeastern states and individuals who have been to endemic areas [3,6,7]. In addition, chronic infection has been documented in patients thirty years after leaving endemic areas with the majority of those infected being asymptomatic [4].

Initial infection with *Strongyloides stercoralis* begins when filariform larvae contact and penetrate the skin of a human host via contaminated material. The larvae then migrate through the skin, spread hematogenously to the lungs, penetrate the alveoli and migrate up the bronchi where they are ultimately swallowed. From there, the larvae mature and embed into the duodenum and

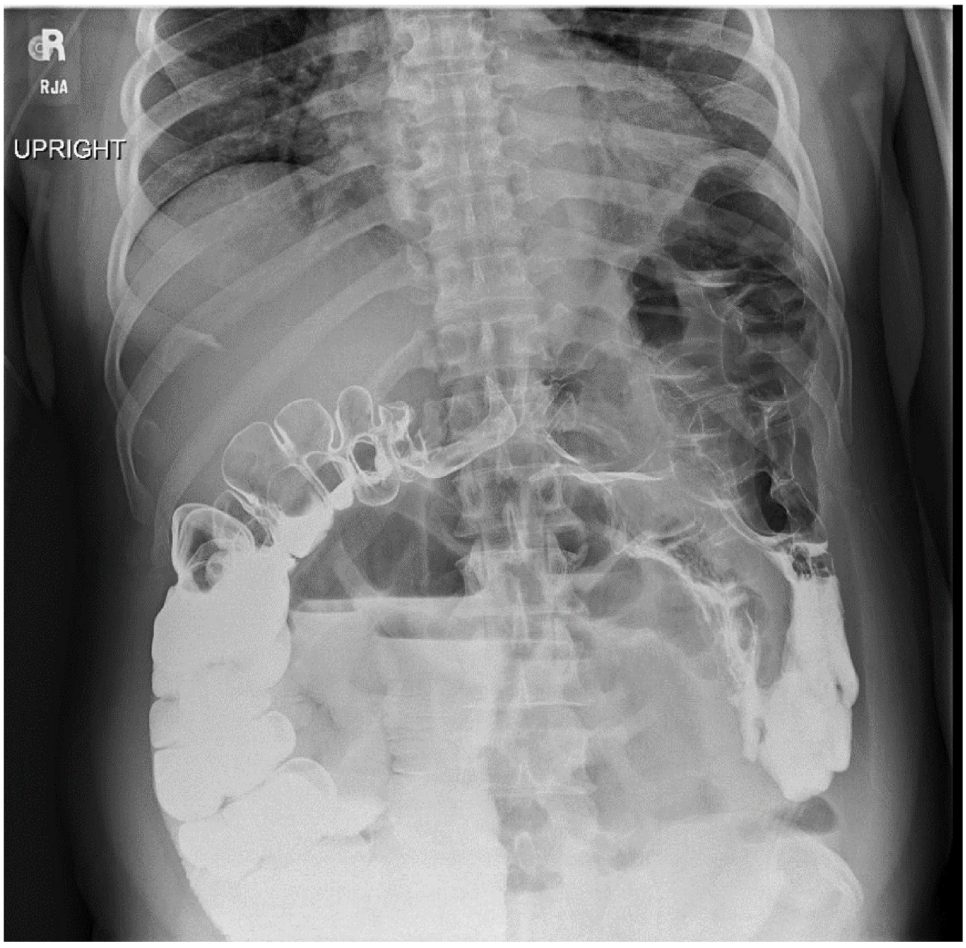

Fig. 1. Upright abdominal film performed six days after contrast administration.

jejunum where they produce eggs. The eggs mature into rhabdiform larvae that are excreted with the stool. Further, *Strongyloides stercoralis* possess the unusual ability to mature entirely into infective larvae within the host and re-enter the hematogenous circulation via the gastrointestinal tract (endoautoinfection) or perianal area (exoautoinfection) without an external cycle. Through repeated autoinfection, persistence of strongyloidiasis is possible long after the initial exposure for the lifespan of the host [5].

Manifestations of infection range from asymptomatic local gastrointestinal involvement in the immune-competent host to disseminated disease involving multiple organ systems in the immune-compromised host. Gastroenterological manifestations of infection are diverse and can include non-specific symptoms of nausea, vomiting, diarrhea, abdominal pain, weight loss, bloating, flatulence and less commonly duodenitis, enterocolitis, malabsorption, ulceration, hemorrhage, ileus, and obstruction [7,8]. The mechanisms responsible for visceral neuropathy from chronic *Strongyloides* infection are unclear. However, the resultant decreased bowel motility favors persistent auto-infection [8].

In conclusion, this case demonstrates the importance of considering global health conditions even in local practice. Given the rarity of symptomatic gastrointestinal strongyloidiasis and the diverse nature of its clinical presentations, clinicians should have a high index of suspicion in any patient with abdominal complaints and a history of travel to endemic areas especially in the setting of a peripheral eosinophilia.

Conflict of interest

None.

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Authorship

All authors had access to the data and a role in writing this manuscript.

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