CASE REPORT

GASTROINTESTINAL MANIFESTATIONS IN SEVERE STRONGYLOIDIASIS: REPORT OF 3 CASES AND LITERATURE REVIEW

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Abstract. We report here three cases of severe strongyloidiasis in normal and immunocompromised hosts. The first was a patient with a normal immune system who presented with acute severe abdominal pain. The second and third patients were immunocompromised due to steroid and chemotherapy treatment of underlying diseases. Both presented with obstructive symptoms. In all three cases, Strongyloides stercoralis larvae were detected in stool concentration samples, and in biopsied specimens from the duodenum in the first and second cases.

Key words: strongyloidiasis, S. stercoralis, GI manifestations

INTRODUCTION

Strongyloides stercoralis is an intestinal nematode in humans found in the tropics and sub tropics: Eastern Europe, Italy, Australia, Southern United States and Southeast Asia (Farthing, 1996). Many individuals infected with this parasite are asymptomatic. However, in patients with depressed delayed hypersensitivity (defects in cell-mediated immunity) as a result of malnutrition, corticosteroids or immunosuppressive drugs, an unrecognized asymptomatic infection may convert into severe infection (Burke, 1978).

We report 3 cases of severe strongyloidiasis during the years 2001-2008. The first case was immunocompetent while the other two were immunocompromised. All cases underwent esophagastroduodenoscopy. Clinical presentations, endoscopic findings, laboratory results, treatment and outcomes were described.

CASE REPORT

Case 1

A 5-year-old girl from northern Thailand presented with severe abdominal pain for 1 day. Two weeks prior to admission, she had a lower respiratory tract infection, the symptoms of which subsided within two days after treatment at a private clinic. About 5 days before coming to the hospital she developed generalized small pruritic vesicles. The skin lesions subsided in a few days, leaving the skin
dry. One day before coming to the hospital she vomited 5 times, each time approximately 20 minutes after eating. The vomitus contained no bile, she had generalized abdominal pain. The vomiting and abdominal pain continued, so she was admitted to the hospital.

Physical examination revealed no fever and the only positive finding was mild tenderness of the right upper and right lower quadrants. The CBC showed an Hct of 37.8%, WBC 30,900/mm³, and eosinophils 77%. Stool examination was negative for parasites. The patient had gastroscopy the following day which revealed duodenitis, duodenal fluid aspiration was negative for parasites. Stool concentration for parasites was done after gastroscopy because she had only one bowel movement since admission and the result was positive for *Strongyloides stercoralis* larvae. Biopsied tissues from the duodenum showed *S. stercoralis* larvae and eggs (Fig 1).

**Case 2**

A 12-year-old boy from northeastern Thailand with a known case of nephrotic syndrome for 4 months had been treated with prednisolone 60 mg daily without improvement and thus was referred to Queen Sirikit National Institute of Child Health (QSNICH) for renal biopsy. Three days before admission he had cough and a poor appetite. Epigastric pain, bilious vomiting and diarrhea occurred 1 day before hospitalization. Physical examination revealed mild to moderate dehydration, cushingoid facies, and mild tenderness of the epigastric region. After admission he had voluminous bilious vomiting which mimicked intestinal obstruction.

Management included fasting, placement of a nasogastric tube with intermittent suction, intravenous fluid and a stress dose of hydrocortisone. A complete blood count was within normal limits. Proteinuria and dysmorphic RBC with RBC casts were found on urine exam. *S. stercoralis* larvae were found on stool concentration exam and in the bile (900 larvae per slide each). Esophagogastroduodenoscopy with biopsy was performed and the duodenal mucosa was found to be inflamed and erythematous. *S. stercoralis* larvae were also found from duodenal fluid and biopsied tissues (Figs 2 and 3).

**Case 3**

A 9-year-old girl from central Thailand, a known case of acute lymphoblastic leukemia (L₁-L₂) for 5 years developed a low grade fever, a generalized erythematous maculopapular rash and vomiting for 2 weeks prior to coming to QSNICH for chemotherapy. Ten days after having chemotherapy she developed high fever and bilious vomiting. She was admitted to the provincial hospital and referred to QSNICH.

Physical examination revealed fever, moderate dehydration, a generalized erythematous maculopapular rash of the chest and abdominal wall with mild abdominal distention. Voluminous bilious vomiting was observed after admission. A complete blood count showed anemia and leukopenia (WBC 3,800/mm³ with eosinophils 3%). Urine analysis was normal. Stool examination yielded *Blastocystis hominis*. Stool concentration for parasites yielded hook worm ova and *S. stercoralis* larvae. Bile content from the nasogastric tube showed *S. stercoralis* filariform and rhabdiform larvae. A plain abdomen x-ray revealed abdominal distention and a bowel ileus. Blood chemistries showed hyponatremia (Na= 115 mEq/l) and hypoalbuminemia (alb = 2.98 g/dl). After admission she had voluminous bilious vomiting.
Fig 1–*S. stercoralis* eggs.

Fig 2–Cross section of *S. stercoralis* larva.

Fig 3–Cross section of *S. stercoralis* larva (high power).

Fig 4–An unidentifiable worm seen by gastroscopy.

Management of obstructive symptoms was the same as in the second case. Esophagogastroduodenoscopy was performed which revealed swelling and a mottling appearance of the antrum and the duodenum appeared edematous with enlarged folds. An unidentifiable worm was endoscopically seen in the second part of the duodenum (Fig 4). Duodenal fluid was negative for parasites and biopsied specimens showed only chronic duodenitis. Albendazole was started during the procedure via endoscopy.

**DISCUSSION**

*Strongyloides stercoralis* is a widespread soil transmitted intestinal nematode. Infected filariform larvae penetrate the intact skin by contact with infected soil and enter the venous microcirculation via the lymphatics (Grove, 1994). Larvae penetrate pulmonary alveoli, ascend the respiratory tree and then are swallowed. The larvae grow to adult females in the duodenum and jejunum, burrow into the submucosa, releasing eggs (Concha *et al.*, 2005).

The gastrointestinal manifestations of *S. stercoralis* infection include abdominal pain (crampy or bloating), watery diarrhea, constipation, anorexia, weight loss, nausea and vomiting (Keiser and Nutman, 2004). These symptoms begin about 2 weeks after infection with larvae detectable in the stool after 3 to 4 weeks. Chronic
Infestation is usually limited to the duodenum and jejunum. Massive overwhelming strongyloidiasis may cause intestinal obstruction (Suvarna et al, 2005) as in the 2nd and 3rd cases. Adult worms invade the intestinal mucosa and produce an inflammatory response involving mononuclear cells and eosinophils. There may be some degree of partial villous atrophy (Paola et al, 1962).

Endoautoinfection plays an important role in some chronic cases (Cruz et al, 1966; Berkmen and Rabinowitz, 1972). Malnutrition, lymphoma, treatment with immunosuppressive drugs and corticosteroids may alter host-parasite equilibrium leading to severe strongyloidiasis (Stemmermann, 1967; Smith et al, 1977; Burke, 1978). Suppression of the local inflammatory response by corticosteroid use, changes in the intestinal mucosa and an increase in the transformation rate from rhabditiform into the infective filariform larva may enhance the invasive potential of the parasite (Neefe et al, 1973).

All three reported cases presented with abdominal pain, which is the most common presenting symptom. For the second and third cases which were immunocompromised, there were accompanying symptoms mimicking intestinal obstruction. Upper gastrointestinal endoscopy was done in the first case because of severe abdominal pain. The other cases underwent upper endoscopy for direct observation, obtaining tissues and giving the antiparasitic drug directly into the duodenum. The findings of upper endoscopy in strongyloidiasis have been described as enlarged gastric folds (Milder et al, 1981), gastric ulcers, gross inflammation and hypertrophy of the second part of duodenal mucosa (Bone et al, 1982) and edematous duodenal mucosa (Kazuoki et al, 1996). The only significant finding in our cases was edematous duodenal mucosa in all cases. Small bowel obstruction is secondary to intense infestation and mucosal edema (Bras et al, 1964; Suvarna et al, 2005).

Investigations for detecting this type of infection should be done in all symptomatic patients who have ever resided in an endemic area. Analyzing three consecutive stool specimens has been considered an effective means of diagnosing the disease (Celedon et al, 1994). The sensitivity of this method ranges from 33 to 60% because of intermittent passing of the larvae. The diagnostic yield of duodenal aspiration and endoscopic duodenal biopsy is > 90% (Milder et al, 1981; Choudhry et al, 1995) due to the nature and life cycle of the parasites that grow and release eggs in the duodenum and jejunum (Concha et al, 2005).

Treatment of strongyloidiasis consists of three oral drugs: thiabendazole, albendazole and ivermectin. Thiabendazole has been used widely in the past and gives cure rates of 80 to 90% (Celedon et al, 1994). There are multiple side effects and it is not available on the market at the moment. Albendazole is effective (Archibald et al, 1993) but for hyperinfection or systemic illness in immunocompromised patients, prolonged treatment for 7 to 14 days or until clearance of parasite larvae from stool occurs is recommended. Our three reported cases used this drug for 21 days and follow-up stool specimens after completed treatment were negative in all. Ivermectin is highly effective as a single oral dose and gives cure rates of up to 88% in immunocompetent patients (Naquira et al, 1989). However, a repeated course is necessary in immunocompromised patients. Patients with disseminated strongyloidiasis and intestinal obstruction are a difficult
problem. Oral administration of drugs via nasogastric tube or rectal enema may be alternatives. Our second and third cases which were immunocompromised presenting with obstructive symptoms were started on the medication (albendazole) via upper endoscopy directly into the duodenum. Resolution of the obstructive symptoms was seen in just a few days.

In summary, we report here three cases of severe S. stercoralis infection: one normal host (case 1) and 2 immunocompromised hosts (cases 2 and 3). The presenting symptom in the first case was severe abdominal pain and in the second and third were obstructive symptoms. Stool concentration for parasites yielded S. stercoralis larvae in all three cases. Upper GI endoscopy benefited all patients not only by obtaining tissue but also for administering the medication via the endoscope in the second and third cases. Albendazole 800 mg per day was used for treatment in all 3 cases for 21 days. Stool concentration for parasites after treatment was negative. Prior to starting immunosuppressive drugs for patients in an area endemic for this parasite, evaluation and treatment should be done beforehand in view of the high mortality rate associated with hyperinfection with this nematode.

REFERENCES