Radiological Manifestations of *Strongyloides stercoralis*

S. Dallemand, * M. Waxman, and J. Farman
Departments of Radiology and Pathology, SUNY – Downstate Medical Center, Brooklyn, New York, USA

**Abstract.** Nine patients with radiological changes due to *Strongyloides stercoralis* (SS) are described. A wide variation in appearance exists ranging from mild edema of the duodenal and small bowel mucosa to grossly enlarged, prominent valvulae coniventes. Small bowel dilatation is significant, and in overwhelming infestation toxic dilatation with paresis results. Spasm, ulceration, and stricture are encountered in addition. The appearances usually improve and reverse with treatment. Ampullary involvement is responsible for reflux of barium into the pancreatic duct and biliary tree through a patulous sphincter. In 1 patient the colonic changes resembled ulcerative colitis.

**Key words:** *Strongyloides stercoralis* – Edema – Dilatation – Patent ampulla – Stricture – Colitis.

*Strongyloidiasis stercoralis* (SS) is a parasitic infection of worldwide distribution. The reported incidence of human infection varies from 17% to 85% in endemic areas of Africa, Asia, and South America [1]. With current widespread travel and population migration, SS is being recognized with greater frequency in metropolitan areas of the United States [2]. Many of the infections are either trivial or asymptomatic and remain unnoticed for years while being sustained by a process of autoinfection [1, 3]. Changes in host-parasite relationship, especially in older patients, convert the chronic asymptomatic parasitosis into a debilitating and even lethal infection. In addition, patients who are on immunosuppressive therapy and steroids are likely to develop clinical evidence of the infestation [1, 4].

The varied radiological appearance of this disease needs to be stressed. Even comprehensive textbooks of gastroenterology omit mention of the various radiological patterns of this entity [5, 6].

**Life Cycle and Pathology**

SS is caused by infestation of the duodenum and jejunum with the nematode *Strongyloides stercoralis*. A filariform larva from the soil penetrates the bare skin, usually of the legs or feet, thus initiating the direct cycle within the human body. The larva subsequently migrates through the vessels or lymphatics to the right side of the heart to reach the lungs. Symptoms of cough or bronchopneumonia are usually mild when present at this stage. The larva settles finally in the duodenum and proximal small bowel, where the adult worms develop. In severe cases of infestation, the entire bowel including the colon and proximal part of the biliary tree and pancreatic duct may be affected. The female worms deposit ova that hatch into noninfective rhabditiform larvae, which are subsequently excreted in the feces. These are transformed into infective filariform larvae in the soil during the free life cycle of the parasite outside the human body. Within the intestinal wall, the rhabditiform larvae can be transformed into infective filariform larvae, thus producing a cycle of autoinfection. In SS with mucosal and submucosal invasion, edematous changes occur in the bowel wall. With severe infestation, mucosal ulceration, severe spasm, and even stricture are found. Involvement of the lymphatics results in absorptive deficiencies including steatorrhea.
Material and Methods

Our series consists of 9 patients, 6 men and 3 women. The patients ranged in age from 30 to 79 years, with a mean of 50 years. All our patients acquired the disease while in the Caribbean or South America (Table 1). One patient was U.S. born and had been a missionary for 13 years in Guyana. The clinical presentation in many cases was identical. Severe diarrhea and progressive and debilitating weight loss were the main presenting symptoms. Clinical findings were at times confusing, and several patients were suspected to have advanced malignancy (Table 1).

Case Reports

Case 1

M.M., a 65-year-old Trinidad-born woman, had lived in the United States for 14 years and was admitted to the hospital complaining of diffuse abdominal pain, bloating, vomiting, and a 60 pound weight loss. During her illness she developed peripheral edema; she had undergone 2 negative laparotomies for suspected pancreatic tumor.

Physical Examination. The patient was markedly cachectic with an obvious anemia. Hemoglobin was 4 g/100 ml, and serum albumin was 2 g/100 ml. An upper GI series revealed marked edema of the duodenal sweep with dilatation and poor peristalsis. The duodenal folds were coarse, thickened, and ulcerated. In addition, the third part of the duodenum was stricture for 3 cm (Fig. 1 A). A duodenal biopsy revealed an ulcerated, chronically inflamed mucosa with larvae of SS buried in the submucosa.

Medical Treatment. This consisted of thiabendazole, 15 mg/kg of body weight t.i.d., which was instituted for 3 days. A repeat upper GI series (Fig. 1 B) showed that there was a marked improvement in the appearance of the duodenal mucosa and proximal small bowel. The dilatation and edema had subsided. Clinically the patient did well and gained 30 pounds in 4 months.

Case 2

C.M., a 70-year-old woman born in Guyana, had been a resident of the United States for 10 years. She was admitted with a 2-month history of nausea, vomiting, and a 50 pound weight loss. She had severe diarrhea and denied any history of melena, jaundice, or alcohol abuse.

An upper GI series and small bowel follow-through examination demonstrated severe edema of the proximal duodenum and a long tubular stricture of the third part of the duodenum and the visualized jejunal (Fig. 2 A). The normal mucosal folds were lost, and multiple superficial ulcers were present in the stricture segment of the duodenum and small bowel. Spontaneous reflux of barium into the biliary tree and pancreatic duct occurred as well.

Barium enema revealed a tubular, ahastral colon without evidence of ulceration or polyp (Fig. 2 B). The diagnosis of SS was confirmed on duodenal biopsy (Fig. 3). In addition, multiple larvae were found in colonic washings. The patient received IV fluid, the electrolyte imbalance was corrected, and thiabendazole treatment was instituted. The patient improved rapidly and dramatically, as did the radiological appearance of the small bowel, although it did not return to normal (Fig. 4). Spontaneous reflux of barium through the patent ampulla did not occur after treatment. The patient was discharged 3 weeks later having gained 30 pounds.