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Radiological Manifestations of *Strongyloides stercoralis*

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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 conniventes. 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.

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Table 1. Clinical findings in 9 patients with strongyloidiasis

Patient	Age	Sex	Country of origin	Duration of symptoms	Weight loss (lbs)	Course	X-ray findings
C.M.	36	F	Trinidad	6 months	75	Died	Small bowel ileus
C.M.	70	F	Guyana	2 months	90	Relapsed, improved and gained 30 lbs	Stricture of duodenum and jejunum; free reflux of barium in biliary tree and pancreatic duct; tubular, featureless, colon
R.T.	39	M	Guyana	6 weeks	34	Improved	Tubular duodenum and jejunum; absent mucosal folds; terminal small bowel edematous and dilated
J.B.	30	M	USA	6 weeks	30	Improved	Edematous jejunum and ileum
J.F.	51	M	Trinidad	8 months	50	Improved	Prominent, thickened small bowel mucosa
C.Y.	30	M	Brazil/ China	3 months	30	Improved	Coarse mucosal folds; edematous dilated small bowel
O.T.	79	M	Trinidad	6 weeks	15	Died	Dilated tubular duodenum; paralytic ileus and edema of small bowel
M.M.	65	F	Trinidad	2 years	60	Improved after treatment; gained 30 lbs; relapsed 18 months later	Edema of duodenum and jejunum; stricture of third part of duodenum
S.D.	45	M	Haiti	8 months	14	Improved after treatment	Dilated small bowel; coarse mucosa; excess secretions

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 strictured 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 jejunum (Fig. 2 A). The normal mucosal folds were lost, and multiple superficial ulcers were present in the strictured 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, ahaustral 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.

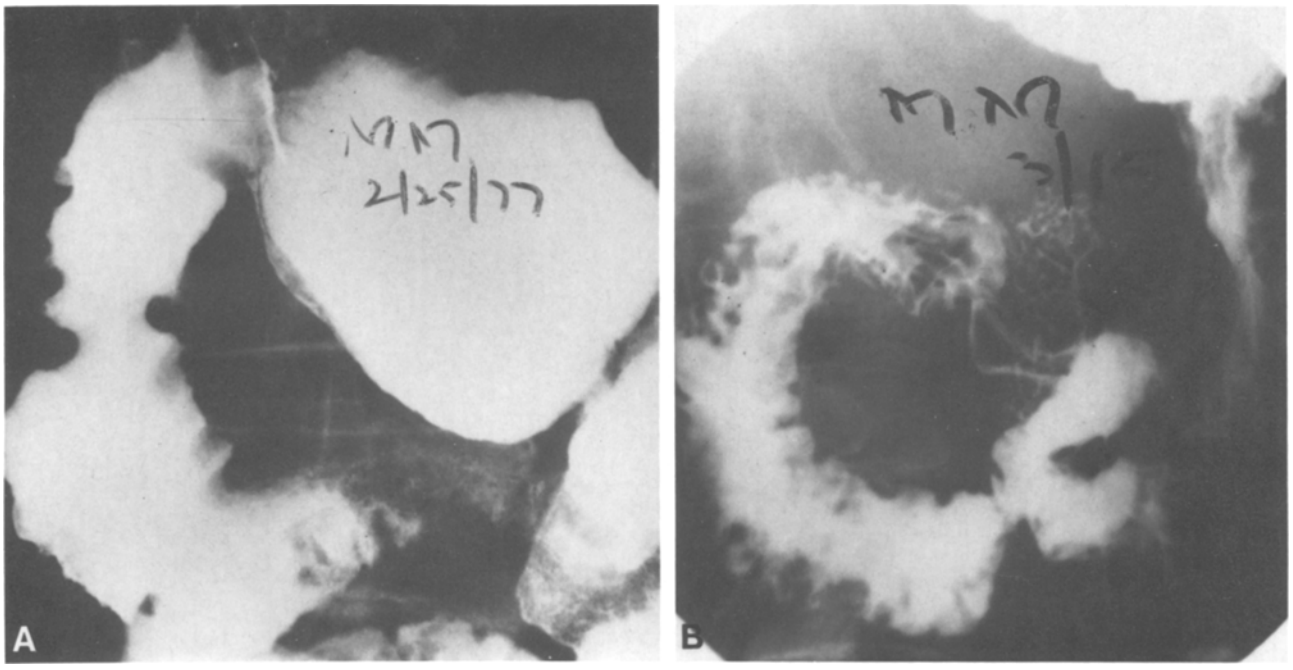

Fig. 1. *Case 1.* M.M., a 65-year-old woman. **A** UGI: duodenal sweep demonstrating dilatation, an edematous descending duodenum with loss of mucosal pattern. **B** Residual minor stricture in the 3rd portion, partially obscured. Duodenum otherwise normal

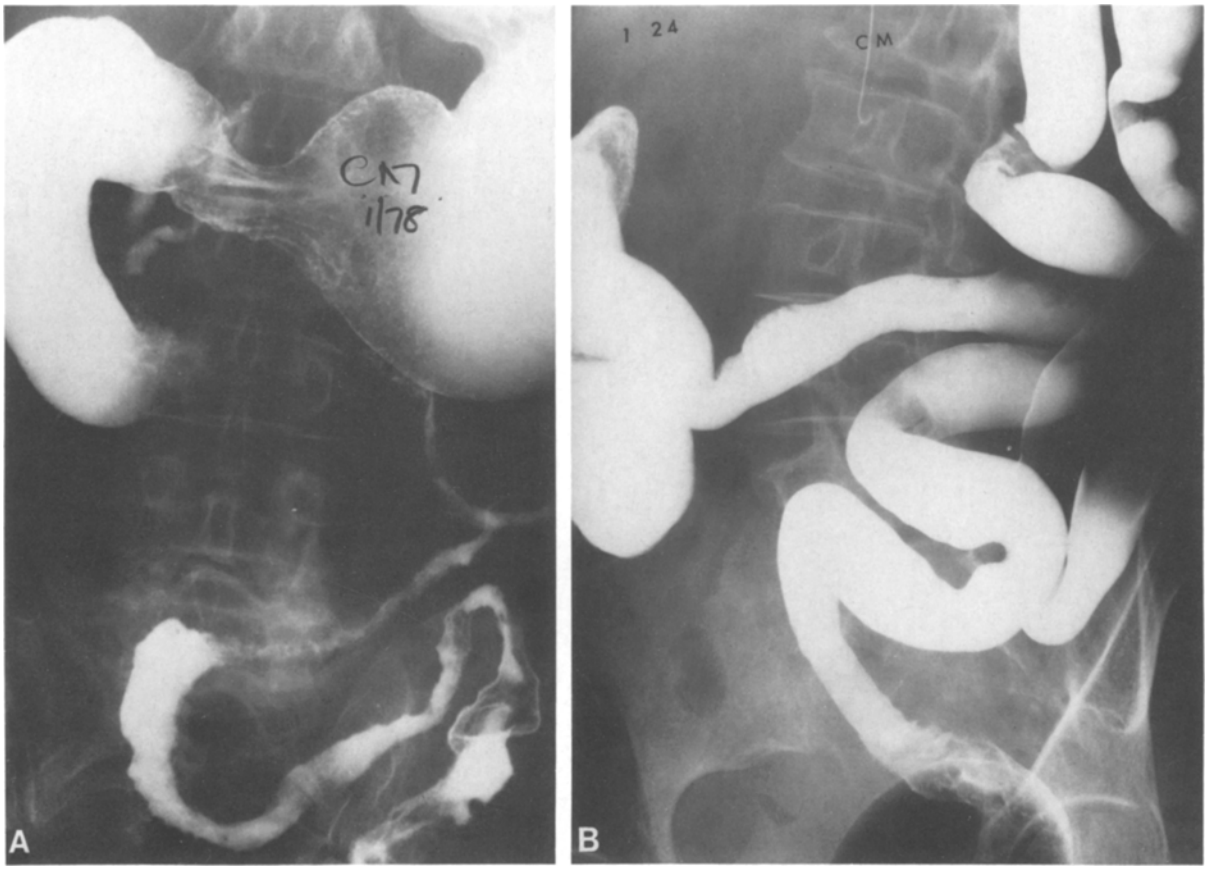

Fig. 2. *Case 2.* C.M., a 70-year-old woman. **A** UGI reveals a stricture of the 3rd portion of the duodenum with proximal dilatation and loss of mucosal pattern. Spontaneous reflux of barium through the ampulla into the pancreatic and common bile ducts has occurred. Marked narrowing of the duodenum and proximal small bowel is due to spasm and ulceration. **B** Tubular ahaustral colon without evidence of ulceration

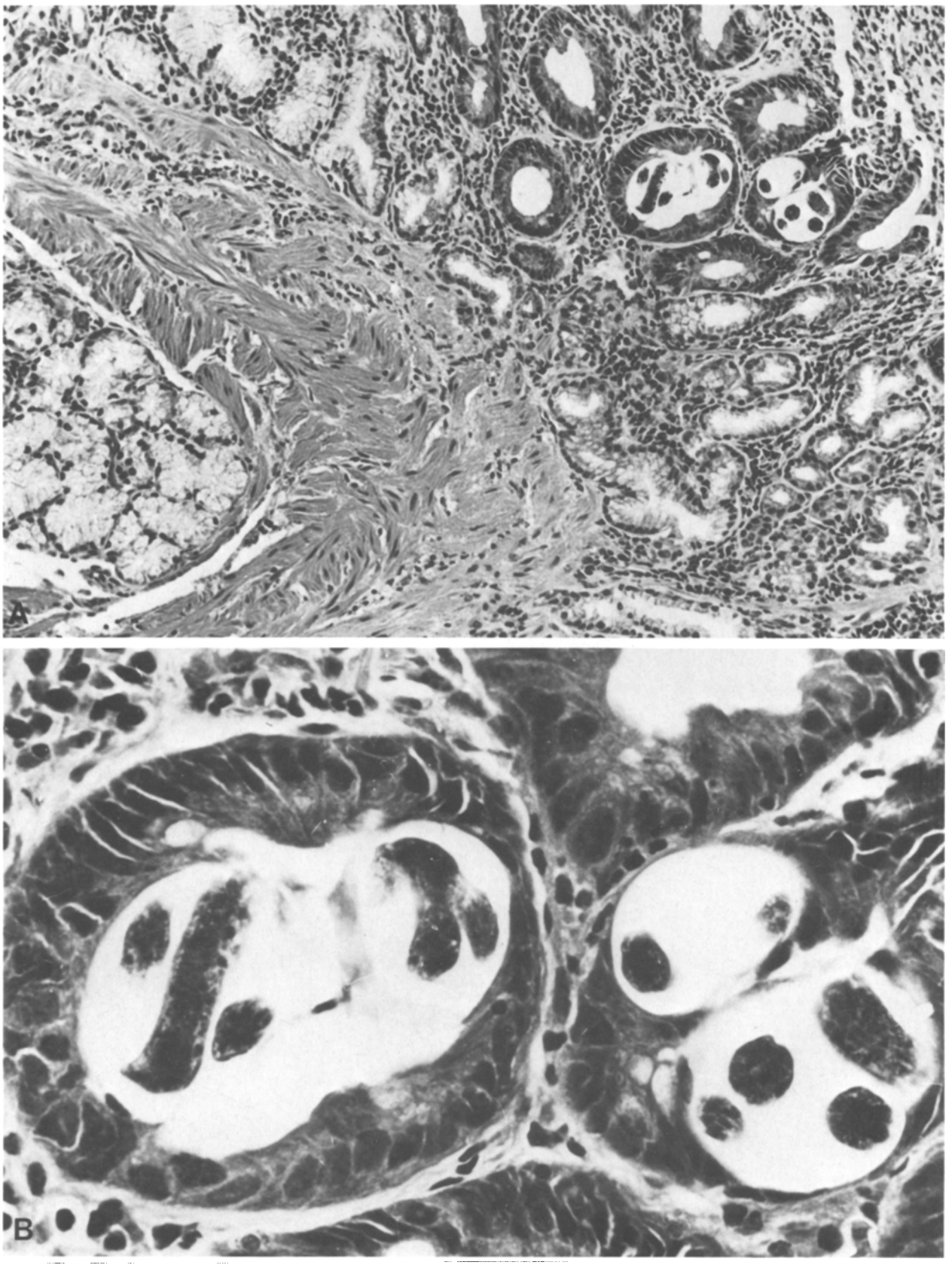

Fig. 3. *Case 2.* **A** Duodenal biopsy shows larvae of *Strongyloides stercoralis* within the mucosal crypts associated with an inflammatory exudate. The muscularis mucosae and submucosal Brunner's glands are on the left (H&E; ×100). **B** Higher power view of the larvae and duodenal crypts (H&E; ×400)

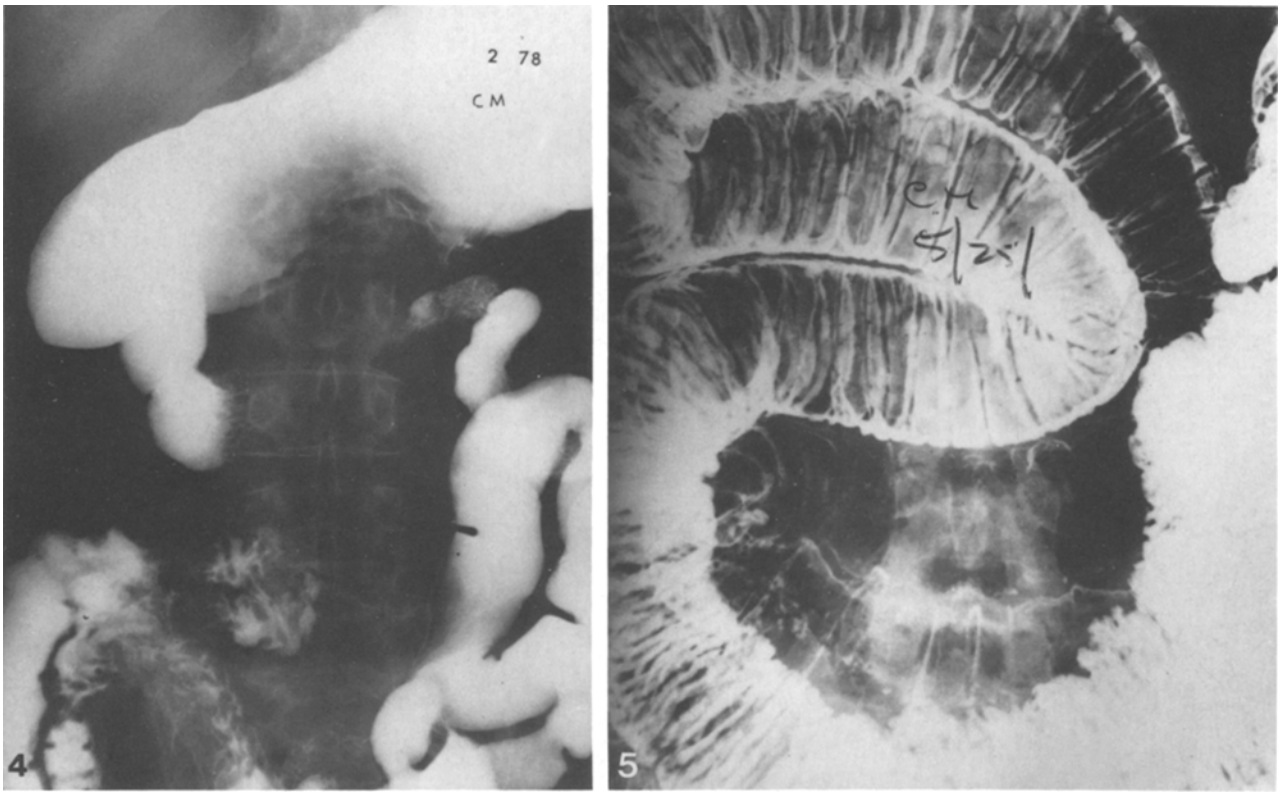

Fig. 4. *Case 2.* UGI and small bowel follow-through demonstrate marked improvement in the appearance of the small bowel. The mucosa has not returned to normal. The proximal jejunum is tubular. The duodenum has improved in appearance, but a stricture is still present. There is no reflux of barium through the ampulla

Fig. 5. *Case 3.* C.M., a 36-year-old woman. UGI and small bowel follow-through reveal gross small bowel dilatation. The examination was performed in a phase of toxic dilatation shortly before the patient's death

Case 3

C.M., a 36-year-old woman, was admitted to the hospital with a 6-month history of progressive and severe weight loss, nausea, and abdominal pain. The patient had emigrated to the United States from Trinidad 18 months previously. An abdominal flat plate revealed gross small bowel distention, subsequently confirmed by a small bowel follow-through. There was, however, no evidence of obstruction (Fig. 5). SS ova and parasites were demonstrated on stool examination, and a course of thiabendazole was instituted for 3 days. However, the patient's condition deteriorated steadily and her abdominal distention worsened. She died 6 days after admission.

Discussion

A wide range of radiological findings occur in SS infestation. The stomach, duodenum, and small bowel may appear perfectly normal, and yet SS may be found in the stool examination or duodenal aspirate. Cases of this sort are especially well documented in immunosuppressed individuals and after renal transplantation or in lymphoma [1, 9]. Stomach changes are difficult to recognize radiologically and show evidence of mild gastritis with mucosal effacement. We were not able to document any gastric abnormality in our patients.

Edema of the duodenum and proximal small bowel with coarse, thickened folds and dilatation are commonly encountered. The radiological appearance at this stage mimics findings seen in hypoalbuminemia, ascites, and peritonitis [7]. With progression of infestation, the thickened folds assume a bizarre, coarse appearance. Excess secretions are evident in the intestinal lumen. Mucosal ulceration may or may not be associated with spasm. The appearances of the duodenal sweep are variable, ranging from massive, edematous, mucosal folds (Fig. 6) to mucosal effacement and stricture, especially involving the third part of the duodenum. These changes may reverse following treatment (Fig. 6C).

Reflux of barium through a patulous ampulla does occur, and barium may be seen outlining the biliary tree as well as the pancreatic duct [2]. Submucosal edema, ulceration, and fibrosis involving the ampullary musculature, result in loss of sphincteric control. Following treatment and improve-

ment the condition reverses, and ampullatory tone is restored. Loss of valvulae conniventes and a narrowed lumen lead to a "lead pipe" appearance indistinguishable from severe Crohn's disease. These changes are also reversible [8].

In its most severe form with massive infestation, continued autoinfection and poor host resistance, small bowel ileus may result [9]. This stage is often irreversible. Chronic malnutrition and electrolyte imbalance from relentless diarrhea add to

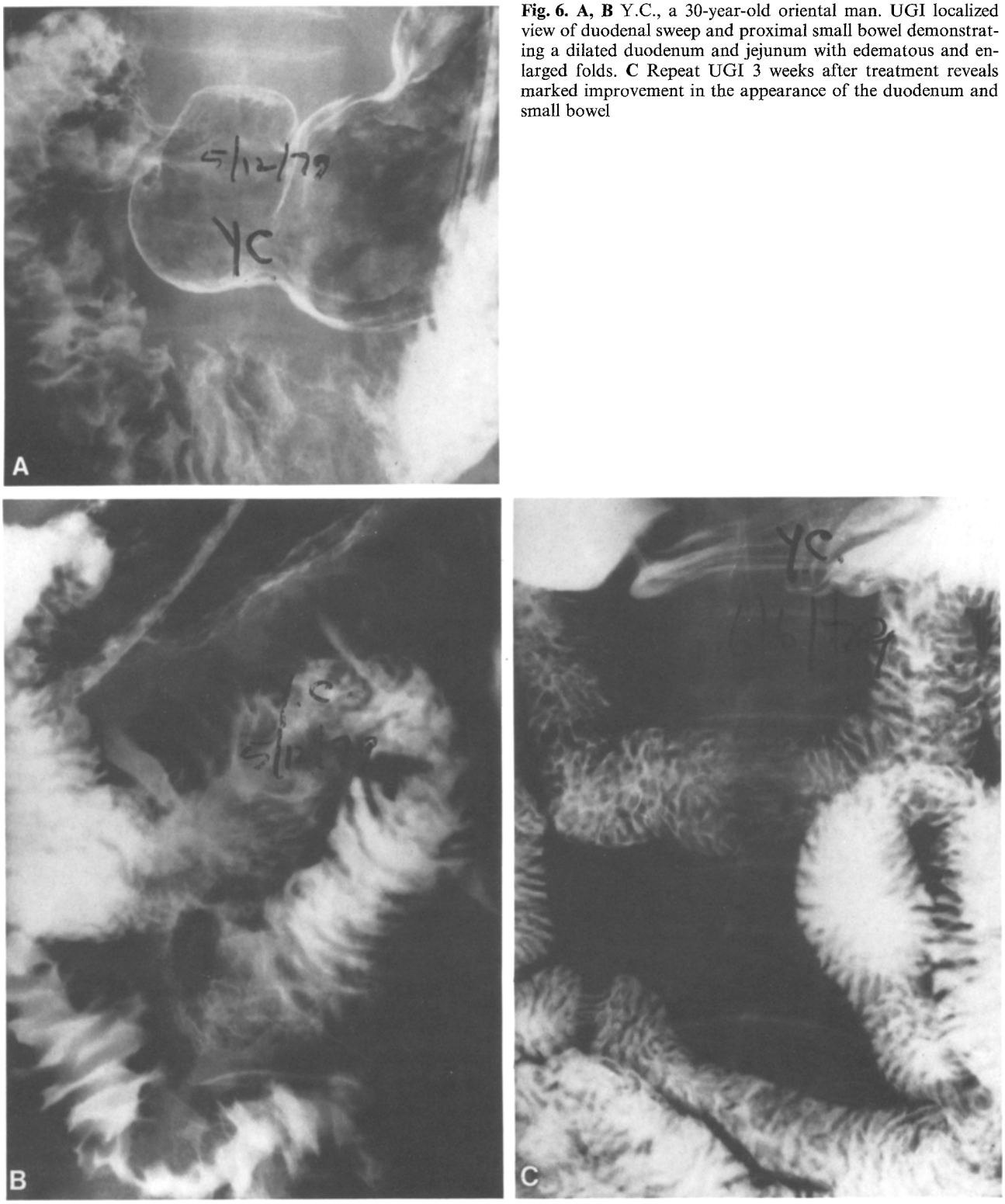

Fig. 6. A, B Y.C., a 30-year-old oriental man. UGI localized view of duodenal sweep and proximal small bowel demonstrating a dilated duodenum and jejunum with edematous and enlarged folds. **C** Repeat UGI 3 weeks after treatment reveals marked improvement in the appearance of the duodenum and small bowel

the difficulties of successful treatment. Death in this stage is not uncommon. Bras et al. [10] have commented on the high mortality rate. Two of the patients in our series died.

Colonic involvement has been well documented. In the acute phase, an ulcerative process resembling both the radiological and endoscopic features of ulcerative and amebic colitis has been described [11]. Our experience of colonic involvement was limited to 1 patient. The colon lost its normal haustral pattern, was narrowed, and had a tubular appearance indistinguishable from that of chronic ulcerative colitis. As in the small bowel, the radiological changes in the colon improved following treatment. The differential diagnosis of the appearance of small bowel should include sprue, lymphoma, Crohn's disease, secondary malignancy, radiation enteritis, and tuberculosis.

Toxic dilatation of the small bowel may be confused with an acute, severe bacterial infection, such as that caused by *Salmonella* or *Shigella*. Spontaneous ampullary reflux of barium must be differentiated from that caused by Crohn's disease of the duodenum, radiation duodenitis and scirrhous carcinoma of the ampulla.

There is no doubt in our minds that the radiological patterns of SS are readily recognizable in most patients. This together with a history of having been in the tropics should raise the physician's suspicions even more.

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