**Hyperinfection: Sequential Changes of Gastrointestinal Radiology after Treatment with Thiabendazole**

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**Abstract.** Sequential changes of gastrointestinal radiology are described in a case of *Strongyloides stercoralis* hyperinfection. Duodenal dilatation, reflux of barium into the biliary and pancreatic duct, and mucosal thickening of the small intestine were the striking features before treatment. Although treatment with thiabendazole led to rapid and complete clearance of rhabditiform larvae, some abnormal radiologic changes in the small intestine persisted for approximately 4 months before the mucosal pattern reverted to normal.

**Key words:** *Strongyloides stercoralis*, hyperinfection

Most patients with *Strongyloides stercoralis* (SS) infestation are asymptomatic. Some may have nonspecific symptoms such as abdominal pain, diarrhea, and weight loss. The most serious syndrome of the disease, hyperinfection, results from the compromised host-parasite relationship and massive invasion of the tissue by SS [1]. Patients susceptible to hyperinfection include those receiving corticosteroids or immunosuppressive drugs for leukemia, lymphoma, renal transplant, systemic lupus erythematosus, or malnutrition-poor conditions. Malabsorption, protein-losing enteropathy, or even death may occur in advanced stages [1–3].

This case report illustrates the sequential changes of radiology in the gastrointestinal (GI) tract before and after treatment with thiabendazole.

**Case Report**

A 48-year-old woman was admitted to the First Department of Surgery of Miyazaki Medical College Hospital on May 8, 1979, complaining of abdominal pain, diarrhea, and weight loss. In September 1976, the patient developed sudden onset of abdominal pain with increasing abdominal fullness. Under a presumptive diagnosis of mechanical obstruction, a laparotomy was performed, but no obvious abnormality was found in the peritoneal cavity. Approximately 1 year before the present admission, the patient had an episode of colicky abdominal pain, which brought her to another hospital. An upper GI series showed that the lumen of the duodenum was 2–3 times larger than normal size. Five months before admission, abdominal pain, diarrhea, and weight loss increased.

On admission, the physical examination revealed a cachectic patient weighing 34 kg with a fever of 38°C. Abnormal physical findings included abdominal distention with visible peristalsis and hyperactive bowel sounds. There was no organomegaly, palpable mass, or ascites.

Abdominal scout films revealed increased gas in the small intestine and the presence of air in the biliary tract. A barium meal study showed dilatation of the duodenum with serrated margins. The bulb was markedly enlarged. Regurgitation of the contrast medium into the biliary and pancreatic duct was also observed. There was an interruption of barium flow at the third portion of the duodenum, which mimicked the superior mesenteric artery syndrome. Barium follow-through revealed thickening of folds and disappearance of the normal mucosal pattern from the duodenum to proximal ileum, producing a pipe stem appearance (Fig. 1).

An upper GI panendoscopy showed edematous gastric mucosa. The mucosa of the duodenum appeared injected and edematous, with patchy superficial erosions. Biopsy from edematous mucosa of the duodenum disclosed a large number of rhabditiform larvae (RL) in the pits with moderate inflammatory cell infiltration in the mucosal lamina propria. The RL were also found in the duodenal aspirates as well as in the stool specimens. In spite of the remarkable radiologic changes in the small intestine, a flat curve in the glucose tolerance test and slight increase in fecal excretion rate of $^{131}$I-Triolein were the only abnormal findings. SS hyperinfection was diagnosed on the basis of roentgenologic characteristics in the GI tract and the presence of abundant RL in the duodenal mucosa. Thiabendazole 1.5 g twice daily was given for 2 days. The RL were present for only 2 days in the stool specimens after treatment. Subsequently, complete disappearance of RL was noted in the duodenal mucosa and aspirates.

Barium follow-through 2 weeks later demonstrated that barium passed normally beyond the third portion of the duodenum. However, reflux of the contrast material into the biliary tract persisted (Fig. 2). The radiologic findings 1 month later showed slight improvement and recovery of the mucosal thickening. Exudation subsided. Not until 7 weeks later did the size of the bulb return to normal and the reflux of barium into the biliary tract disappear.
Fig. 1. Disappearance of the normal mucosal pattern showing pipe stem appearance with hypomotility of the segments of small intestine. However, minimal changes of mucosal edema were still present in the vicinity of the upper jejunum. The patient gained 6 kg during the hospitalization and was discharged free of abdominal symptoms.

Four months later she was readmitted for repeat radiologic check-ups and absorptive studies. The duodenal aspirates and biopsy again failed to demonstrate the presence of RL. Endoscopic examination showed normal gastric and duodenal mucosa. The patient remained asymptomatic. There was slight improvement in the glucose tolerance test. The $^{131}$I-Triolein fecal excretion rate was unchanged. Other laboratory studies were within normal ranges. Final barium study demonstrated complete disappearance of mucosal thickening of the small intestine (Fig. 3).

Discussion

Gastrointestinal changes in SS infestation are mainly localized to the duodenum and upper jejunum, although other areas of the small intestine and even the colon may be involved in patients with hyperinfection [2, 4]. Hyperinfection is usually characterized by malabsorption, diarrhea, electrolyte imbalance, and gram-negative or opportunistic fungal sepsis [3]. It may lead to coma or death.

Roentgenologic features vary depending on severity and chronicity of the infection. Involvement of the stomach with a mild antral gastritis may show effacement of the mucosal pattern and delay in gastric emptying [2, 5]. Radiologic findings of duodenal involvement consist of mucosal thickening, dilatation, and delay of the bolus in the third portion of duodenal loop with abnormal motility [2, 6, 7]. The underlying mechanism of arrest of the barium bolus at the level of the mesenteric crossing is thought to be a combination of (a) duodenitis with dilated duodenal loop and (b) the pressure from an edematous mesentery and dilated superior mesenteric artery [7].

Varying degrees of mucosal thickening, dilatation, and hypomotility are also prominent in the upper jejunum [7, 8]. Diffuse infective process, allergic edema, and hypoproteinemia or malnutrition are factors involved in these findings [2]. The jejunum sometimes shows a pipe stem appearance. This is described by Paterson [9] as the smooth, tubular transformation of the upper part of the small intestine from severe inflammation, edema, and fibrosis due to diffuse infiltration of the helminths. It is virtually specific for SS infections [10].

Long-standing or far-advanced cases may result in narrowing and stricture of the small intestine secondary to chronic inflammation and fibrosis [1, 2]. These advanced cases often need to be differentiated from granulomatous disease, tuberculosis, and lymphoma of the small intestine [2, 7]. Infiltration of RL into deeper layers of the bowel may give rise to subacute obstruction or segmental ileus [11, 12]. Reflux of barium into the biliary or pancreatic duct is a rare feature. An incompetent sphincter of Oddi...