Ileal Strongyloidiasis in a Malaysian Patient

K C Shekhar*, R Pathmanathan**, V S Loo***, K S Chan****, *Department of Parasitology, University of Malaya, **Subang Jaya Medical Centre Sdn Bhd, ***V S Loo Surgical Clinic Sdn Bhd, Ipoh, ****B P Clinical Lab Sdn Bhd, Ipoh

Introduction

Strongyloidiasis is a human intestinal nematode infection which is common among children and adults in the tropics and sub-tropics, especially in warm and humid regions. It causes chronic ill health in an incalculable number of people and occasionally produces explosive, overwhelming illness in immuno-suppressed persons treated for systemic diseases.

Strongyloides stercoralis, usually infects only the mucosa of the small intestine. Hyper-infection by S. stercoralis, however, is a serious complication and is characterised by invasive filariform larvae that produce lesions in the liver, lungs, colon, stomach and other organs. Individuals with intestinal strongyloidiasis are often asymptomatic but may have abdominal pain, weight loss, diarrhoea and other non-specific complaints. Occasionally, an illness occurs which is related to massive systemic invasion by a larval stage of the parasite. Death is frequent.

While S. stercoralis is a problem in many parts of the world, the prevalence in Malaysia is low. We reported a case of gastric strongyloidiasis recently in a Malaysian Chinese patient. This patient is a second clinical case of severe infection presenting with lower gastrointestinal bleeding.

Case Report

A 68-year-old Chinese female was admitted with a three-hour history of bleeding from the rectum. There was no associated abdominal pain or vomiting. She felt dizzy after the rectal bleeding. She gave a history of passing “dark stools” for 2 days allegedly after taking herbal remedy for “flu”. She was also treated one week before for diarrhoea by a physician, after which she became constipated for 3 days. No details of treatment are available. There was no recent change of bowel habits, history of ingestion of NSAID and no dyspepsia or haematuria. She was hyperglycaemic but not on any medication.

On examination, she was small built, poorly nourished, pale and dehydrated. The blood pressure was 100/60 and pulse was 100/min with decreased volume. The abdomen was distended and tympanic but soft. Bowel sounds were hyperactive. The liver and spleen were not palpable and no ascites was detected. Rectal examination showed a moderate amount of stale blood. Haemoglobin concentration was 5.7 mg/100mL. Blood urea nitrogen was 25 mg/dL but the serum electrolytes were normal. Liver function tests showed hypo-proteinemina and a normal alkaline phosphatase, bilirubin and transaminase levels. The prothrombin time was 20 seconds and the INR was 1.6. HBsAg and HIV antibodies were not detected.

Upper gastrointestinal endoscopy showed moderate atrophic gastritis. No ulcers, erosions, oesophageal varices or duodenal lesions were noted. Colonoscopy up to the hepatic flexure showed accumulation of a large amount of stale blood in the ascending colon. A total of 6 units of whole blood were cross-matched and two units transfused preoperatively. Resuscitation included i.v. crystalloids (Ringers lactate). She continued to pass unclear stool and stale blood per rectum after 16 hours. In the absence of facilities such as angiographic, or scintigraphic studies, operation was advised on a suspicion of bleeding from angiodysplastic lesion of the right colon.
At laparotomy, there was a moderate amount of stale blood in the upper and distal ileum and clotted blood in the colon. The stomach and duodenum was empty. A caecal diverticulum was present but no blood was detected in the ascending colon. Two patches of submucosal ecchymoses about 0.5cm on the antimesenteric border of the upper ileum was noted. On ileotomy, there were acute small ulcers with submucosal haemorrhages. The ulcers were excised for histopathology and the ileotomies closed. The other abdominal organs were found normal. The caecal diverticulum was invaginated and an appendectomy was performed for multiple appendiceal faecolitis. The post operative recovery was uncomplicated. She was transfused a total of four units of whole blood perioperatively. She was discharged well on the 5th postoperative day.

Histopathologically, there was goblet cell hyperplasia and ulcerative enteritis (Fig. 1 & 2). Numerous adult female worms in cross section were found within the crypts and rhabditiform larvae and eggs were present between the epithelium and the basal membrane. Glandular destruction was noted. There was marked increase in inflammatory cells especially eosinophils in the lamina propria. There was no increase in mitotic activity of the crypt epithelium. Central lacteals of many of the villi were moderately to markedly dilated while the submucosa appeared edematous. (Fig. 3)

Strongyloides is universal in distribution and infection is usually chronic and asymptomatic. The disease is contracted by penetration of the filariform larvae into the skin from infected soil, by auto-infection or by faecal ingestion. About 25% of patients excrete the parasite in faecal samples and in 90% of patients, the parasite is obtained by duodenal aspiration. Confirmatory diagnosis is by duodenal biopsy.

In Malaysia, Strongyloides has a low prevalence as it has been reported only occasionally on stool examination. Recently we described the first case of gastric strongyloidiasis diagnosed histopathologically suggesting that this disease may be more prevalent than suspected. The present case represents the first case in the intestine documented histologically. Presentation of the patient with bloody diarrhoea is unusual.

Eosinophilia, although not investigated in this case has been reported in approximately 25% of the patients with hyper-infection and its absence in severe infection has been thought to indicate a poor prognosis. Eosinophilia may be absent if systemic steroid therapy is instituted.
Strongyloidiasis may present in a variety of ways including paralytic ileus, an acute surgical abdomen and a protein losing enteropathy with malabsorption. It should be considered in patients presenting with abdominal symptoms. Severe gastrointestinal disease with bleeding should arouse suspicion that the disease can be strongyloidiasis. In this particular patient, diagnosis is confirmed by the presence of larvae, adults and eggs on biopsy. The presence of stale blood and clotted blood maybe due to profuse bleeding attributed to haemorrhages due to hyper-infection with the parasite.

Infection with *Strongyloides stercoralis* is under diagnosed because of the insidious clinical presentation and lack of clinical awareness. Unfortunately, most cases are diagnosed only at autopsy or at laparotomy.

Strongyloidiasis is world wide in distribution. Gastroenterologists and surgeons should be aware that this particular condition is often associated with immunodeficiency and in those on corticosteroids or immunosuppressive agents, fair placed clinical awareness is likely to lead to an improved detection of the disease.

---

**References**
