CASE REPORT

GASTRIC STRONGYLOIDIASIS IN A MALAYSIAN PATIENT

Shekhar KC[1], Krishnan R[2], Pathmanathan R[3] and Fook CS[4]

[1]Department of Parasitology, Faculty of Medicine, University of Malaya, Kuala Lumpur 59100, Malaysia; [2]BP Clinical Laboratory Sdn Berhad, Penang, Malaysia; [3]Department of Pathology, Faculty of Medicine, University of Malaya, Kuala Lumpur 59100, Malaysia; [4]Consultant Surgeon, Penang Adventist Hospital, Penang, Malaysia

Abstract. *Strongyloides stercoralis* infection is of low prevalence in Malaysia. We report an unusual case presenting primarily with gastric symptoms. The patient was a 72 years old Chinese male admitted for progressive weight loss and abdominal bloating. Gastroscopic examination revealed mucosal prepyloric elevations in the gastric mucosa. Gastric strongyloidiasis was confirmed by the presence of adult forms, as well as ova and larval rhabditiform stages of the worm in the gastric mucosal crypts. We believe that this is the first histologically documented case of gastric strongyloidiasis in Malaysia.

Strongyloides stercoralis causing strongyloidiasis of worldwide distribution usually infects the mucosa of the small intestine. The disease is contracted by penetration of the filariform larvae into the skin by contact with infected soil, autoinfection or by fecal ingestion. The established adult worms locate in the sub-mucosa of the human small intestine and produce eggs that hatch to vegetative rhabditiform larvae. Most will leave the host after journeying through the entire small and large intestine while others develop to filariform larvae. These then penetrate the colonic mucosa and initiate the new parasitic cycle within the host.

Strongyloidiasis which is asymptomatic in otherwise healthy individuals, may suddenly become a serious and/or fatal infection in individuals whose immunologic defences have been altered by iatrogenic or natural means. Fatal overwhelming strongyloidiasis is a serious complication and is characterized by invasive filariform larvae that produces lesions in the colon, lungs, liver and other organs.

While several cross sectional surveys have been carried in different parts of Malaysia, *S. stercoralis* is believed to be of low prevalence *ie* between 0-3.8% (Sinniah, 1984). While infection status in these cross sectional surveys had been confirmed by stool examination, this case constitutes the first histologically diagnosed and documented case of gastric strongyloidiasis in Malaysia.

A 79 years old male Chinese patient, who had lived continuously in Penang, became bedridden after a fall. The patient could not give a good history because of his illness but it appeared that although his appetite remained good, he showed progressive loss of weight. He had been previously admitted to a private hospital for investigation of symptoms of recurrent epigastric pain and bloating accompanied by a generalised discomfort.

While in hospital, a stable wedge fracture of the first lumbar vertebra was diagnosed. The patient appeared acutely ill and dehydrated. His epigastrium was slightly tender but there was no guarding, rigidity, rebound tenderness, distension or masses on palpation. No abnormality was detected on rectal examination. The hemoglobin value was 10.0 g/dl, and the leukocyte count was 10.0×10^9/l with 3% eosinophils. The hematocrit was 0.29 Sl, with platelets 260×10^9/l and an ESR 40 mm/hour. The serum electrolytes were low. The total protein was 50 g/l, with markedly reduced albumin (17 g/l). There was trace proteinuria with 2 + glycosuria.

Ultrasound examination of the abdomen showed no significant abnormality. The chest x-ray was unremarkable. At gastroscopy, the esophagus, fundus and body wall of stomach was noted to be grossly normal. However, in the pre-pyloric region, there were isolated benign looking mucosal elevations which appeared mildly erythematous and edematous. These foci were biopsied. The duodenum was noted to have a grossly normal mucosal

surface up to the second part, and random biopsies of the duodenal mucosa were performed and tissue obtained for diagnosis.

Histopathological examination of the gastric mucosa showed numerous cross-sections of *Strongyloides* adult forms, eggs and rhabditiform larvae developing in the gastric crypts. Characteristic double lateral alae were identified in the walls of the adult worms (Figs 1-4). The gastric mucosa showed superficial ulceration, edema and increased cellularity of the lamina propria. The areas of ulceration were overlaid with fibrin, mucus and occasional necrotic polymorphonuclear leukocytes. Occasional scattered eosinophils were seen in the mucosa. In areas, a mild chronic gastritis was noted, with loss of specialized gastric parietal cells, and mild intestinal and Paneth cell metaplasia. In contrast, the biopsies of the duodenal mucosa did not show any significant pathology.

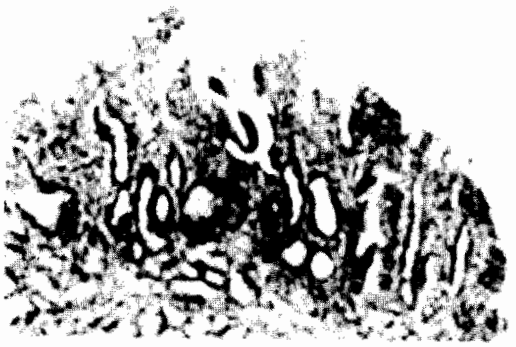

Fig 1–Cross-section of an adult *Strongyloides stercoralis* (arrow) from mucosal biopsy of the stomach. Although there is superficial ulceration and mild inflammation of the mucosa, there is no inflammatory infiltrate seen around the worm × 400.

The patient was warded for 2 days before relatives decided to take the patient home. The patient died at home a day later due to undetermined causes.

Strongyloidiasis has a low prevalence in Malaysia, has been reported occasionally in stool examination by several authors (Sinniah, 1984). The signs and symptoms of strongyloidiasis are non specific and include nausea, vomiting, epigastric pain and diarrhea. The commonest gastrointestinal sites are the duodenum and jejunum, where the parasite is reported to invade the mucosa, resulting in disruption of normal small bowel function and often, malabsorption. The severity of infection

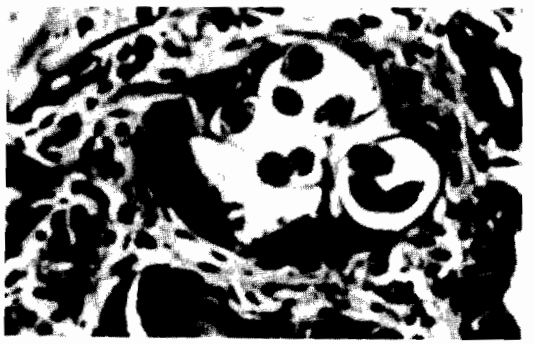

Fig 2–A low magnification showing location of coiled female, developing larvae and eggs deep within the crypts. Note the delicate cuticle and thin muscular layer × 200.

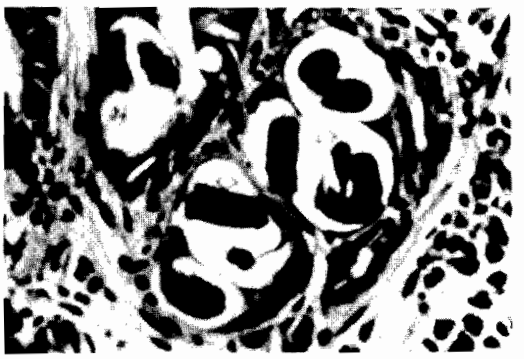

Fig 3–Developing larvae within the eggs and rhabditiform larvae (arrow) in the crypt × 400.

Fig 4–Higher magnification illustrating four cross-sections of adult worms within the crypt. Note double lateral alae (arrow) × 400.

influences the extent of the clinical disorder, and the clinical spectrum ranges from mild mucosal edema and diarrhea to a fulminant protein losing enteropathy. The majority of individuals have few or no complaints referable to this infection. The most frequent symptom, however, is midgastric pain which manifests as a burning sensation or a dull ache. The epigastrium is usually tender on deep palpation and foods which are spicy and alcoholic drinks tends to aggravate symptoms. Under these circumstances, the commonest clinical diagnosis is usually (mistakenly) that of peptic ulcer.

Eosinophilia found together with 'ulcer symptomatology' is suggestive of strongyloidiasis. In acute cases it varies from 25-35% while is chronic cases it falls between 6 to 8% (Berkmen and Rabinowitz, 1972). Based on the symptoms, and the low level of eosinophilia (3%), the patient probably suffered a chronic infection. The low level of hemoglobin and hematocrit is suggestive of chronic occult blood loss in the stool, usually seen in strongyloidiasis or in malnutrition and malabsorption. The appetite of the patient was maintained and it is likely that gut bleeding and malabsorption are the factors contributing to the anemia. Unfortunately, a stool examination was not carried out which would have further confirmed the diagnosis.

Although the patient did not give a good history, the marked serum albumin depletion suggest that the infection in this patient must have been severe. Gastric strongyloidiasis as a principal presentation is rare and the present case of gastric strongyloidiasis is the first report in Malaysia. Exceedingly unusual too in this case that we are currently reporting, the pathology is confined essentially to the gastric mucosa, since biopsies of the duodenum showed no evidence of parasitosis.

Strongyloidiasis is often overlooked because of the current scant attention it receives and because of the tendency of the disease to remain dormant for many years before giving rise to overt symptoms. Occasionally, the disease is discovered on biopsy or at autopsy. The present case demonstrates this fact rather nicely.

Based on the scant clinical data, lack of appropriate investigations and post-mortem findings, the cause of death of this patient is uncertain and may have resulted from complications of secondary infection, or cardiac failure precipitated by hypoproteinemia and hypokalemia.

It is established that *S. stercoralis* is worldwide in distribution and it is therefore important that physicians treating patients with diseases associated with immunodeficiency (such as lymphomas, leukemias, leprosy, systemic lupus erythematosus and other related diseases) or patients on immunosuppressive therapy be aware of the possibility of an infection with this parasite. Clinicians should be aware that although strongyloidiasis in Malaysia exists in low prevalence, this figure may be spuriously low, and could be attributed to misdiagnosis or under diagnosis of the disease.

REFERENCES

Berkmen YM, Rabinowitz J. Gastrointestinal manifestations of strongyloidiasis *Am J Roentgenol Radium Ther Nucl Med* 1972; 115 : 306-11.

Sinniah B. Intestinal protozoan and helminthic infections and control of soil-transmitted helminths in Malay school children. *Public Health* 1984; 98 : 152-6.