Case Report

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# Gastric Strongyloides with Ulceration and Klebsiella pneumonia Bacteraemia

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## Abstract

**Strongyloides infection is usually confined to small intestine in gastrointestinal tract. Gastric mucosal Strongyloides with ulceration is extremely uncommon. We describe a patient who presented with gastrointestinal bleeding with gastric Strongyloides diagnosed by biopsy from the base of the gastric ulcer. Patient was treated with ivermectin 9 mg once a day for 2 days. The hospital stay was complicated by severe Klebsiella pneumoniae bacteraemia which needed treatment with meropenem for 2 weeks. Patient was discharged after 40 days of hospital stay.**

**Key words:** Strongyloides, gastrointestinal bleeding, ulceration, Klebsiella pneumoniae bacteremia

## Introduction

Strongyloides is endemic in tropics and subtropics. The infection is usually confined to gastrointestinal (GI) tract. It usually resides in the mucosal glands of the small intestine [1]. Strongyloides in the gastric mucosa with acute GI bleeding from the gastric ulcer is uncommon [2,3,4]. We describe a case of gastric Strongyloides from the base of the gastric ulcer which caused acute GI bleeding and severe Klebsiella pneumoniae bacteremia.

## Case report

Mr. SM, 95 year-old gentleman, lives in private flat and has been homebound for the last 10 years. He was admitted on 21/6/06 with productive cough and breathlessness over 1 month. He had no hemoptysis or chest pain. He had past history of tuberculosis and chronic obstructive airway disease. He was treated for pulmonary tuberculosis for 9 months from August 2005 to April 2006. He was on inhalers and theophylline for chronic obstructive airway disease.

On admission, patient was tachypnoeic with respiratory rate of 22 per minute, blood pressure (BP) was 110/70 mmHg and pulse was 98/minute. There was no lymphadenopathy. Jugular venous pressure was not elevated and systemic examination was normal. On admission, blood investigations showed white blood cell $26.2x10^9$/L, neutrophils 93.2%, lymphocytes 1.3%, monocytes 5%, eosinophils 0.5%, basophils 0%, haemoglobin 15 g/dL, platelets $118x10^9$/L, urea 5.8 mmol/L, creatinine 84 µmol/L, glucose 4.9 mmol/L, electrolytes and liver function tests were normal. His chest X-ray showed scarring with bronchiectatic changes in bilateral apical and midzones.

He was treated with intravenous ceftriaxone and oral clarithromycin. In view of recent history of tuberculosis, he was isolated in a single room. His acid fast bacillus (AFB) smears (x2) were negative. The following day he appeared pale, but there was no haematemesis or melena. Investigations showed haemoglobin 9 g/dL, white blood cell $17.9x10^9$/L, neutrophils 93.3%, lymphocytes 1.3%, monocytes 4.1%, eosinophils 1.3%, basophils 0%, platelets $151x10^9$/L, reticulocytes 1.3% (normal 0.5-2.3%), reticulocytes $31.4x10^9$/L (normal 25-85), bilirubin 21

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μmol/L (normal 7-29), LDH 953 U/L (normal 250-580), iron 6 μmol/L (normal 10-30), TIBC 33 μmol/L (normal 40-70), iron saturation 18.2% (normal 15-45%), ferritin 516 μg/L (normal 24-336), folate 8 nmol/L (normal 8-30), vitamin $B_{12}$ 933 pmol/L (normal 133-675), stool for occult blood was positive (x2). In view of the sudden drop in haemoglobin and evidence of bleeding through GI tract, he was treated with intravenous omeprazole 40 mg per day. His haemoglobin was monitored and remained stable. His haemoglobin repeated on 27/6/06 was 8.5 g/dL. He underwent oesophagogastroduodenoscopy (OGD) on 28/6/06 which showed oesophagitis with linear oesophageal ulcers and multiple gastric ulcers (**Figure 1**). Biopsy taken from the gastric ulcer showed ulceration with chronic active gastritis with intestinal metaplasia. Larval forms of Strongyloides stercoralis were seen in the mucosa (**Figure 2**).

Patient was treated with ivermectin 9 mg per day for 2 days. His repeat investigations on 7/7/06 showed haemoglobin 10.3 g/dL, white blood cell $10x10^9$/L, neutrophils 51%, lymphocytes 6%, monocytes 11%, eosinophils 30%, basophils 0%, myelocytes 2%, platelets $270x10^9$/L. Patient responded well to the treatment but declined repeat OGD. He spiked a temperature 3 days after ivermectin treatment and blood culture showed Klebsiella pneumoniae bacteraemia. On examination patient was febrile, BP was 110/70 mmHg, pulse was 96/minute, chest was clear, abdomen was soft and nontender and there were no cardiac murmurs. He was treated with intravenous meropenem 1 gm twice a day for 2 weeks. Patient responded to the treatment. His repeat stool examinations (x2) was negative for Strongyloides. His anti-HIV screen was negative. Chest X-ray did not show any evidence of consolidation. CT scan thorax showed changes consistent with old tuberculosis with fibrosis and scarring in the right upper zone. Eye check up was done in view of Klebsiella pneumoniae bacteremia did not show any evidence of endopthalmitis. His repeat full blood count on 20/7/06 showed haemoglobin 10 g/dL, white blood cell $13.2x10^9$/L, neutrophils 88.5%, lymphocytes 6.9%, monocytes 2.1%, eosinophils 2.4 %, basophils 0%, platelets $215x10^9$/L . Patient did not agree for bronchoscopy and lavage in view of his chest symptoms. Patient was discharged well on 1/8/06.

## Discussion

Strongyloides stercoralis is a tiny nematode. Infection with this parasite usually occurs in the small intestines. The filiform larvae invade the human through the soil by break through the skin. Once in the body, they are carried by the lymphatic and venules to reach the lungs. They are coughed out and then reach small intestine where they reside and develop into adult worms. The adult worm lays eggs which are passed out in human faeces. In the soil this gives rise to rhabdiform larvae and invasive filiform larvae. In the human intestine, eggs mature into rhabdiform larvae in the mucosa. These do not penetrate intestinal mucosa in an otherwise healthy individual. In immunocompromised individual, rhabdiform larvae transform into invasive filiform larvae which penetrates the intestinal mucosa and causes disseminated infection [1,5]. This mucosal breach gives rise to Gram negative sepsis. This was seen in our patient who had evidence of Klebsiella pneumoniae sepsis. Systemic manifestations include pneumonia with eosinophilia and meningitis. Our patient also had eosinophilia which resolved with treatment.

Small intestine involvement is the usual presentation of Strongyloides. Gastric involvement can also occur but is extremely uncommon. Bleeding from gastric ulcer with Strongyloides infection is still uncommon. There have been very few case reports of Strongyloides invading stomach mucosa and causing intestinal bleeding [2,3,4]. In our patient, endoscopy showed evidence of gastric Strongyloides with ulcer. Other atypical presentations of Strongyloides are also known. The symptoms may mimic peptic ulcer disease, Crohn's disease or severe necrotising bowel disease. Kennedy et al has described Strongyloides with jejunal perforation [6]. Colonic Strongyloides mimicking ulcerative colitis has been described [5].

Besides directly demonstrating Strongyloides from biopsy specimen, larval forms in stool specimens can be seen. Repeated stool specimen with zinc floatation technique, Baermann technique or agarplate method increases chances of positive yield. Serological tests like immune haemagglutination tests may be useful [1]. The repeat stool specimens did not show evidence of Strongyloides. Recurrence of Strongyloides is common in immunocompromised hosts. Treatment

with ivermectin 200 µg/kg/day for 2 days has 97% cure rates. Thiabendazole 50 mg/kg/day is the alternative treatment but due to poor GI absorption, it is not a good choice for disseminated Strongyloides. Longer duration of treatment is recommended in such cases. Mortality is high with systemic dissemination. Death usually occurs due to Gram negative sepsis secondary to breach in the mucosal barrier created by Strongyloides infection [1,7].

## Conclusions

Strongyloides stercoralis infection of gastric mucosa with ulcer and Klebsiella bacteraemia is extremely uncommon. We need to investigate these patients for immunocompromised state and treat them aggressively as the mortality is high due to gram negative septicaemia.

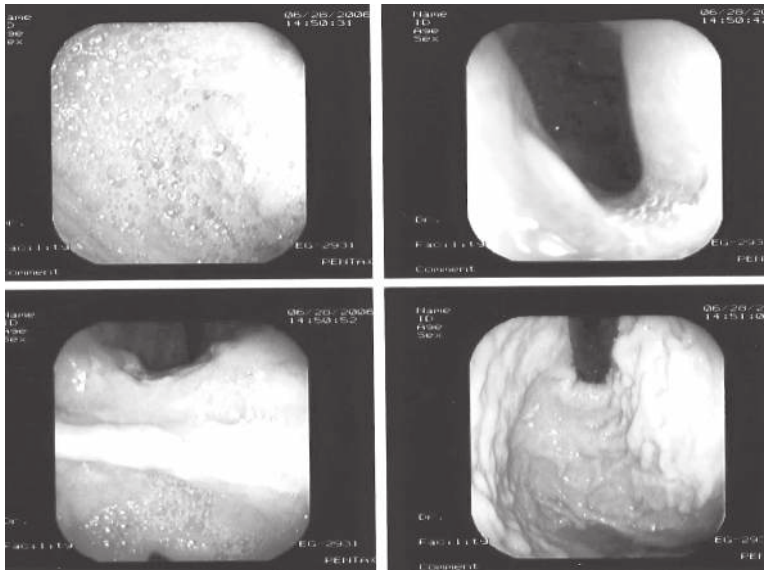

**Figure 1**. OESOPHAGOGASTRODUODENOSCOPY (OGD) SHOWS OESOPHAGITIS WITH LINEAR OESOPHAGEAL ULCERS AND MULTIPLE GASTRIC ULCERS.

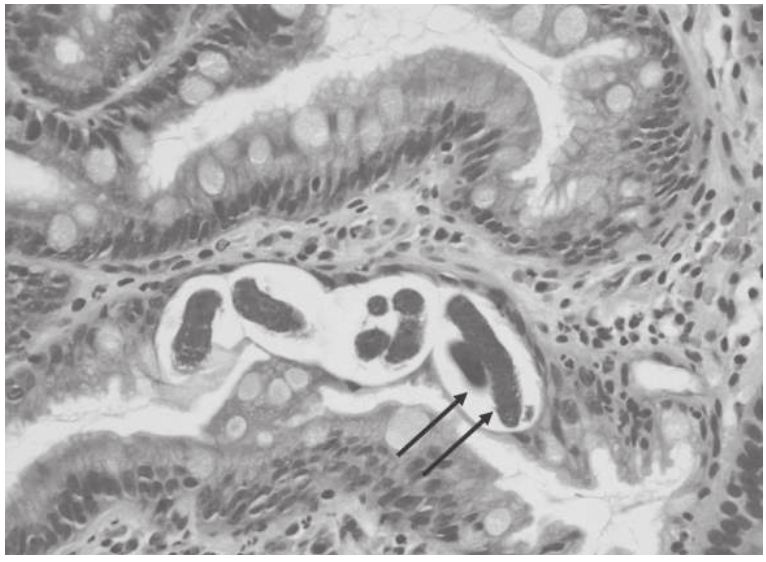

**Figure 2a.** HIGH POWER VIEW OF DEVELOPING LARVA IN THE GASTRIC EPITHELIUM (X40).

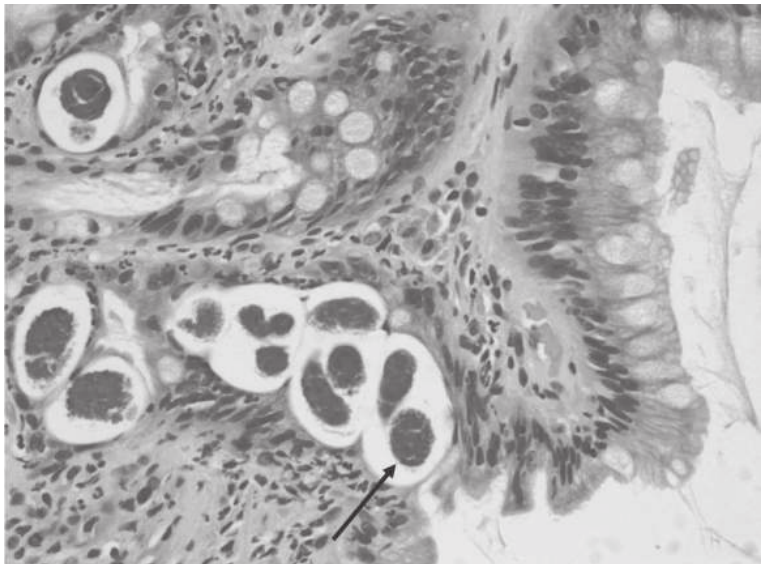

**Figure 2b**. HIGH POWER VIEW OF EGGS (X40).

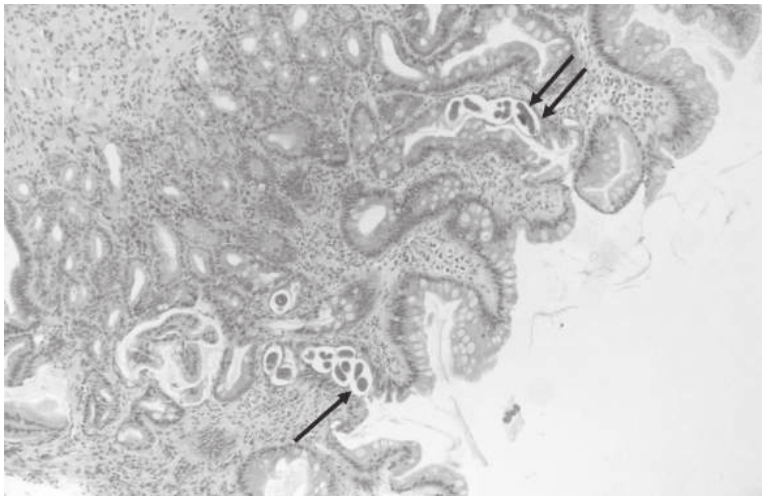

**Figure 2c**. DEVELOPING EGGS (SINGLE ARROW) AND LARVAE (DOUBLE ARROWS) ARE SEEN IN THE GASTRIC MUCOSA WITH INTESTINAL METAPLASIA (H & E, X10).

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