Live *Ascaris lumbricoides* in the peritoneal cavity

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Infestation with *Ascaris lumbricoides* is a worldwide phenomenon with up to a quarter of the world population, mostly in the third-world countries, infected.^{1,2} *A. lumbricoides* resides mostly in the middle third of the jejunum, but perforations attributed to this parasite have been described mostly in the terminal ileum, cecum, Meckel's diverticulum and appendix. To date no case of duodenal perforation reported in the literature has been attributed to ascariasis. In previous reports there was also no evidence to support ascariasis as a cause of intestinal perforation.²⁻⁴ In China alone, some 500 million persons have been reported to harbor the parasite.¹ Although the exact number of *Ascaris*-infected persons in the United States is unknown, an estimated 4 million people are infected,⁵ mostly immigrants, travelers, and refugees.⁵

Complications of ascariasis are seen in both children and adults. They may present with bowel obstruction (due to worm bolus, small bowel volvulus and intussusception), appendicitis, cholecystitis, or pancreatitis.⁶⁻⁹ While most patients with *Ascaris* infection are asymptomatic, life-threatening gastrointestinal conditions may develop that require immediate medical and surgical intervention.¹⁰ Individuals living in poor sanitary conditions are infected when they ingest the fertilized eggs. The eggs migrate into the small intestine where the larvae are released into the duodenum and migrate to the lungs. The larvae are coughed up by the host and are swallowed. In the small intestine, the larvae mature into adult roundworms.¹¹ This report documents a case of migration of live *Ascaris* worms into the free peritoneal cavity through a perforation in the duodenum in a Southeast Asian migrant worker residing in Saudi Arabia.

CASE

A 35-year-old male expatriate from South Asia working in Saudi Arabia presented to the Aseer Central Hospital, Abha, with a 6-day history of epigastric pain associated with nausea, anorexia, constipation and vom-

iting. The pain became more severe and later involved the whole of the abdomen. The patient was not known to have any chronic medical illness and there was no history of previous surgery. On physical examination, the patient looked ill, in pain, toxic, and tachypneic. He was febrile (39°C). The blood pressure was 140/80 mm Hg, the pulse was 86 beats/minute and the respiratory rate was 24/minute. There was digital clubbing of the fingers. Examination of the chest and heart was non-contributory. Abdominal examination revealed a hugely distended abdomen and board-like rigidity on palpation. There was generalized tenderness on palpation and rebound tenderness on deep palpation. No bowel sounds were heard on auscultation of the abdomen. The hernia orifices and external genitalia were normal.

The white blood cell (WBC) count was $8.5 \times 10^9/L$, blood urea nitrogen was 145 mg/dL and serum creatinine was 1.5 mg/dL, serum sodium was 119 mmol/L, potassium was 4.5 mmol/L, and hemoglobin was 14.2 g/dL. The rest of the blood investigations were within normal limits. HIV serology was negative. An abdominal radiograph taken in the supine and erect positions showed air under the diaphragm with several air-fluid interfaces and free air in the abdomen (Figures 1A and B). A clinical diagnosis of acute abdomen due to hollow viscus perforation was made and active resuscitation was commenced in preparation for laparotomy. The inserted nasogastric tube drained copious amounts of feculent fluid and a lot of air.

Exploratory laparotomy under general anesthesia was carried out. Findings at laparotomy were 3 liters of pus and 2 viable *A. lumbricoides* worms were found in the peritoneal cavity (Figure 2A). There was a duodenal perforation (about 6 mm in diameter) just distal to the pre-pyloric vein of Mayo. The pus was drained and the live roundworms were removed. A third live roundworm, which was protruding through the duodenal perforation (Figure 2B), was manually extracted (Figure 2C) and the perforation was closed with a Graham's patch. The peritoneal cavity was washed

Figure 1. Plain abdominal radiographs: supine (A) showing air under the diaphragm (arrow) and erect (B) showing air-fluid levels (arrow).

Figure 2. Live *Ascaris lumbricoides* (roundworms) (A) lying free in the peritoneal cavity and (B) coming out through a perforation in the duodenum. (C) Manual removal of live *Ascaris lumbricoides* from the abdominal cavity.

thoroughly with a lot of warm saline. The abdomen was closed routinely over three suction drains placed in the left Morrison's pouch, pelvis and left sub-phrenic space, respectively. Postoperatively, the patient was admitted to the intensive care unit (ICU) for elective mechanical ventilation as he was in metabolic acidosis ($\text{pH}=7.284$; $\text{pCO}_2 = 31.6$ mm Hg; $\text{pO}_2 = 94.2$ mm Hg; oxygen saturation = 96.5%; $\text{HCO}_3^- = 14.5$ meq/L; standard base excess = +10.8 mmol/L).

The patient received intravenous antibiotics: ampicillin g IV every 6 hours, ceftriaxone (Rocheephine) 1 g IV every 12 hours, and metronidazole (Flagyl) 500 mg IV every 8 hours. He had temperature spikes for 5 days

postoperatively, but this eventually settled. He was taken off mechanical ventilation on the seventh day after the operation after which feeding was started through a nasogastric tube. He had the first bowel motion on the same day. The patient received anthelmintics (pantoprazol 40 mg IV every 12 hours). From the eighth day postoperatively it was changed to 100 mg of oral mebendazole twice a day and the patient was shifted to the general ward. The postoperative period was complicated by wound infection due to *Pseudomonas aeruginosa* and *Klebsiella* spp. This responded to daily wound dressings and high calorie/high protein diet. A gastrograffin study on the ninth day after operation showed

no gastrointestinal leak. On day 21 after the operation he had a left paracolic pus collection (20 mL) drained under CT guidance. The patient's condition improved and he was discharged home on day 40 after the operation.

DISCUSSION

Ascariasis is commonly a disease of poor countries. Increases in economic migrations, travel and refugee populations mean that the disease can manifest even in the most advanced and prosperous countries.¹⁰ The disease can exist for many years with the patient experiencing only vague abdominal symptoms. When *A. lumbricoides* infection is left untreated any of several complications may develop, some of which may be life-threatening and require prompt surgical intervention.⁷ In the case presented herein, although the patient was from a developing country, he presented in Saudi Arabia, a relatively prosperous country with high standards of hygiene.

The intestine has an immense capacity for dilatation. It has been claimed that it can accommodate >5000 worms without symptoms occurring.¹² It is thus unlikely that direct pressure by a few *Ascaris* can produce duodenal perforation. The commonest complication of ascariasis is intestinal obstruction due to a worm bolus.¹⁰ The obstruction may be acute or subacute. Acute upper airway obstruction due to roundworms has also been documented.¹³ It can be complicated by intussusception, perforation, and gangrene of the bowel. Acute appendicitis and appendicular perforation can occur as a result of worms entering the appendix. Hepatopancreatic ascariasis is a frequent cause of biliary and pancreatic disease in endemic areas.¹⁴ It occurs in adult women and can cause biliary colic, acute cholecystitis, acute cholangitis, acute pancreatitis, hepatic duct calculi and hepatic abscess.¹⁴ *Granulomatous peritonitis* in ascariasis can be due not only to the presence of dead adult worms in the peritoneal cavity,² but may be caused by reaction to the eggs¹⁵ as in the case reported from Benin by Kinde-Gazard, et al.¹⁵ In the case we have presented, peritonitis was due to the spillage of intestinal content into the peritoneal cavity due to duodenal perforation. In ascariasis, the cause of perforation of the small intestine remains controversial, with two main theories. In the tropics patients consistently have histories of diseases associated with ulceration of the intestines such as typhoid enteritis, tuberculosis, and amebiasis. During extreme conditions, such as inflammation, starvation, or worm bolus obstruction, some parasites are believed to migrate into the ulcers and to cause perforations.² Another possible explanation is that the large worm

bolus can lead to pressure necrosis and gangrene.¹⁶ The bowel, diseased in this way, becomes susceptible to rupture by the burrowing action of the worm.¹⁴

The hypothesis for small bowel perforation in cases of ascariasis highlights primary bowel diseases and intestinal obstruction and gangrene as the two pathogenetic factors involved. Primary bowel diseases include amebiasis, typhoid enteritis, tuberculosis, trauma, small bowel lymphoma, bowel ischemia, Crohn's disease, vasculitis-secondary to polyarteritis nodosa, radiation enteritis, strangulated hernia and non-specific ulceration secondary to drugs like the non-steroidal anti-inflammatory drugs (NSAID) causing intestinal ulceration. The *Ascaris* worm escapes into the peritoneal cavity through these perforations. Intestinal obstruction and gangrene constitute the most common surgical complications of ascariasis. The mechanism of obstruction is occlusion of the intestinal lumen by worms packed in the distal ileum as well as localized volvulus of a segment of terminal ileum owing to the weight of the roundworms inside. Localized gangrene, necrosis and perforation were noted in most reported cases. The mechanism of gangrene may be due to pressure necrosis caused by the roundworms and also the localized volvulus. Despite the fact that *Ascaris* is toothless and has soft lips, it is paradoxical that it can perforate normal healthy intestine.

Despite the large number of cases of infestation with *Ascaris lumbricoides* reported in the literature, no case of duodenal perforation by *Ascaris* per se was conclusively identified. In a recent case report by Louw,¹⁷ they found a bleeding duodenal ulcer during endoscopy with *Ascaris* worms adherent around the ulcer. Oozing blood from the duodenum might have attracted the parasite to migrate towards the duodenum, but whether the *Ascaris* was the cause of the ulcer could not be determined. In our patient the *Ascaris* worms probably migrated into the duodenum and exited into the peritoneal cavity through the perforated duodenal ulcer. In patients with ascariasis who present with acute surgical conditions, urgent laparotomy is indicated to deal with intestinal perforation, to control peritonitis and to decompress the worm bolus that may be causing acute intestinal obstruction.¹⁰

Ultrasonography can detect worms in the biliary tract and pancreas and is a useful noninvasive technique for diagnosis and follow-up of patients with hepatopancreatic ascariasis.¹⁴ Endoscopic retrograde cholangio-pancreatography (ERCP) can help diagnose biliary and pancreatic ascariasis, including *Ascaris* in the duodenum.¹⁴ Also, ERCP can be used to extract worms from the biliary and pancreatic ducts when indicated.¹⁴

Pyrantel pamoate, mebendazole, albendazole, and levamisole are effective drugs and can be used for mass therapy to control ascariasis in endemic areas. The possibility of

patients presenting with the sequel of ascariasis should be borne in mind when dealing with acute abdomen in migrants, travelers or refugee populations.

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