

ILEAL PERFORATION CAUSED BY THE INNOCENT ASCARIS. A CASE REPORT
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ABSTRACT

Background: *Ascaris lumbricoides* is common cause of acute abdomen and is attributed to poor hygiene and low socio-economic background. About a quarter of world population is infected by this nematode. **Methods:** We report a case of a 10 year old female with symptoms of perforation peritonitis. Patient was subjected to exploratory laparotomy. **Results:** The patient had multiple ileal perforations due to a single ascaris worm with faecal peritonitis. A peritoneal wash and loop ileostomy was done and abdomen was closed back in layers. The patient recovered well post operatively. **Conclusion:** *Ascaris* induced perforation peritonitis is a very rare clinical presentation. Early diagnosis and surgical management is the only treatment modality.

KEYWORDS: *Ascaris*, Perforation peritonitis, Acute Abdomen.**INTRODUCTION**

Ascaris lumbricoides infection is one of the most common and largest nematodes infecting humans in the developing world.^[1] This nematode affects roughly a quarter of the world's population.^[2] In developing countries like ours, where public health, sanitation and personal hygiene are subpar, ascariasis infection is a significant medical and public health problem. Intestinal ascariasis manifests itself clinically in a variety of ways; from asymptomatic infections, colicky pain, growth retardation, and malnutrition to potentially fatal gastrointestinal sequelae such as obstruction, perforation, intussusception, and volvulus. Adult worms can migrate to the common bile duct, where they can cause cholangitis and pancreatitis.^[1] Although intestinal perforation caused by a worm is extremely rare, ileal perforation and gangrene secondary to worm bolus obstruction and pressure necrosis have been documented.^[3] We report an extremely rare case of worm induced ileal perforation and peritonitis.

CASE DETAILS

10 year old female child from rural area in India, presented to our emergency with pain abdomen and

distension for past 3 days. A detailed history including previous surgical, medical and personal history was taken which revealed important negative history of diarrhoea, fever or passing worms from any natural opening. On detailed general and systemic examination, the patient was conscious, co-operative and well oriented to time, place and person. Although she was dehydrated, pale and malnourished, her PR-130 bpm, BP-100/60 mmHg, RR-30/min, Temperature- 100^o F, GCS-15/15 and her Saturation-96% on room air. The abdomen was moderately distended and tender and had features of peritonitis like rebound tenderness. Digital rectal examination was unremarkable.

Baseline investigations- a complete blood count revealed a Hb of 10.2 gm% and TLC counts of 20,000/mm³ with predominant neutrophilia, serum electrolytes revealed- potassium 2.8 mmol/L, sodium-132 mmol/L, calcium-0.8 mmol/L. Renal/liver function tests, routine urine, radiography of chest were normal. Xray abdomen showed multiple air fluid levels and an Ultrasonography of the abdomen and pelvis was suggestive of free fluid with septations and echoes in it suggesting- bowel perforation (Figure.1)



Figure 1: Radiograph and Ultrasound imaging of the abdomen.

Due to lack of Computed Tomography facility in our emergency setup and high clinical suspicion of peritonitis, the patient was directly taken up for exploratory laparotomy under general anaesthesia. About 200ml of purulent foul smelling fluid was encountered from the peritoneal cavity. Multiple small and one large perforation was encountered in the region of distal ileum with a single ascaris worm visible through two of them (Figure 2). Rest of the bowel was healthy.

The ascaris worm was carefully pulled out through the most proximal ileal perforation (2 feet from the

ileoocaecal junction) (Figure 2). Peritoneal washes were done and of the three perforations encountered, the distal two were sutured primarily and most proximal ileal perforation was brought out as an ileostomy. An abdominal drain was put in the pelvis. Patient was extubated uneventfully and was then shifted to post operative ward for further management. The abdominal drain was removed on the post op day 3 and the patient was discharged on post day 6. Ileostomy closure was done at 3 months period. On the first follow up patient was given 400 mg Albendazole and was doing well on regular follow-up.

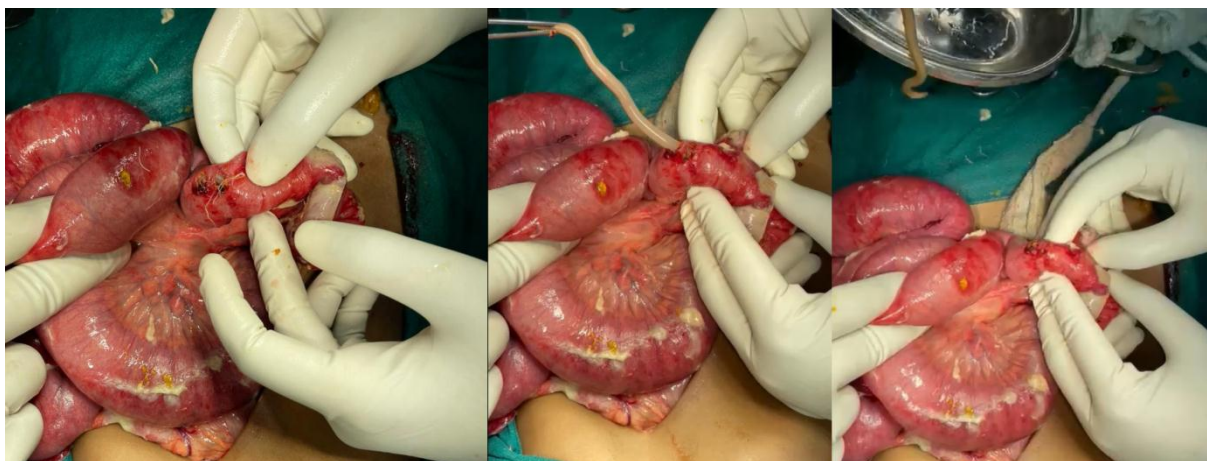


Figure 2: Intra-operative photograph showing ascaris with multiple worm induced perforations in the distal ileum.

DISCUSSION

The *Ascaris* species are very large (adult females:20-35cm; adult males:15 to 30cm) Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the faeces. Unfertilised eggs may be ingested but are not infective. Larvae develop to infectivity within fertile eggs after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm,

shaded soil). After infective eggs are swallowed, the larvae hatch, invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs. The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed. Upon reaching the small intestine, they develop into adult worms. Between 2 and 3 months are required from ingestion of the

infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years.^[4]

Most patients with *A.lumbricoides* or *A.suum* infection are asymptomatic. When symptoms do occur, during the early phase of infection (4 to 16 days following egg ingestion), migration of *Ascaris* larvae through the lungs maybe associated with transient respiratory symptoms and eosinophilic pneumonitis. During the late phase of infection (6 to 8 weeks after egg ingestion), non specific symptoms such as abdominal discomfort, anorexia, nausea, vomiting and diarrhoea. Pulmonary ascariasis should be suspected in individuals with no prior *Ascaris* exposure and potential egg ingestion within weeks prior to onset of respiratory symptoms (dry cough, dyspnea, fever, wheezing, substernal discomfort, and blood tinged sputum). Over half of patients have crackles and wheezing in the absence of focal consolidation. Urticaria occurs during the first five days of illness in about 15 percent of cases. Hepatomegaly may develop. Lymphadenopathy is generally not observed. Symptoms generally subside within 5 to 10 days; the syndrome is usually self-limited and very rarely fatal. This has characteristic radiographic findings (migratory bilateral round infiltrates), and peripheral eosinophilia. Symptomatic pulmonary involvement is rare among individuals in highly endemic areas with ongoing exposure. Pulmonary involvement associated with parasitic infection is known as Loeffler syndrome; the initial description of the syndrome consisted of eosinophilic pneumonitis later attributed by Loeffler to *A. lumbricoides* infection. Other parasitic infections associated with pulmonary syndromes include Strongyloides, hookworm (*Ancylostoma duodenale*, *Necator americanus*, and *Toxocara*), schistosomiasis, and lymphatic filariasis associated with tropical pulmonary eosinophilia.^[5, 6, 7]

Complication of ascariasis is intestinal obstruction due to a worm bolus. It can be complicated by intussusception, perforation and gangrene of the bowel. Intestinal obstruction and gangrene constitute the most common surgical complications of ascariasis. 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 cause perforations.^[8] In the study of Hassan, Y et al.; they reported 265 of 312 patients (84.94%) responded to conservative management of ascaridial intestinal obstruction using nasogastric decompression, hypertonic saline enema and supportive measures in absence of abdominal guarding or rigidity. One of their patients had worm-induced pancreatitis and 16 had biliary ascariasis out of which 8 were subjected to ERCP(Endoscopic Retrograde Cholangiopancreatography). One of their patients had developed features suggestive of septic shock and peritonitis and was found to have ileal perforation

secondary to bowel gangrene due to segmented volvulus.^[1] Although the worms causing perforation is very rare but intestinal perforation and gangrene secondary to worm bolus obstruction and pressure necrosis have been reported.^[9]

In our case, we for the first time report multiple isolated ileal perforation with the worm visible in the bowel lumen with no concurrent bowel disease. This is a very very rare circumstance which showed an otherwise healthy bowel with isolated ileal perforations. So in all patients of ascariasis who present with acute surgical conditions, an urgent laparotomy is indicated to deal with bowel perforation, to control the peritonitis and to decompress the worm bolus that may be causing the acute intestinal obstruction.^[10]

CONCLUSION

Children with acute abdominal pain in low-income and developing nations are frequently affected by ascariasis. *Ascaris* induced perforation peritonitis is a very rare clinical presentation. Early diagnosis and surgical management is the only treatment modality. To stop the occurrence of such devastating complications, mass deworming of children should be initiated in the endemic area.

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