Primary Ascaridal Perforations of Small Intestine and Meckel's Diverticulum

Y K HANGLOO, INDIRA KOUL, R SAFAYA, S KOUL, U DHAR, S KUMAR, R K CHRUNGGOO

Department of Surgery, Medical College, Srinagar

Abstract

The clinical presentation of six cases with primary ascaridal perforations of the terminal ileum (4 cases) and Meckel's diverticulum (2 cases), without any associated underlying intestinal disease, is described. All the cases presented clinically as peritonitis and had worm masses in the intestinal lumen with maximal impaction in the terminal ileum. Ileal perforations were surgically closed in two layers after removal of the worms. Diverticulectomy after removal of the worms was performed for Meckel's diverticular perforations. Peritonal toilet was done in all the cases. Pressure necrosis from the impacted worm masses probably resulted in these perforations.

Key words: Helminthes, Ascaris lumbricoides

Introduction

The commonest location of Ascaris lumbricoides is in the small intestinal lumen, where it can cause intestinal obstruction, volvulus, intussusception, strangulation and perforation.1,2 Primary ascaridal perforations (PAP) of the small bowel, if in the absence of predisposing intestinal disease, resulting in peritonitis are rare.1,2 PAP results from pressure necrosis from worm masses with or without production of lytic substance(s) by the parasite,1 or subsequent to patchy gangrene in the wall of the small intestine filled to a bursting point with worms.3,5 Six cases with PAP resulting in generalized peritonitis are reported.

Material and Methods

These six patients with PAP of the small bowel (4 Intestinal, 2 Meckel's) were found amongst 200 intestinal perforations treated during the period. All of them presented clinically as peritonitis. Their clinical symptoms and signs including findings on rectal examination were recorded. Hemogram, urine analysis and plain X-ray of the abdomen were done in every case. In one case paracentesis of the abdomen was done for the diagnosis of peritonitis. All these patients were subjected to laparotomy for peritonitis.

Results

Five patients were between 6 and 10 years of age, and one patient was 12 years old. Abdominal pain was present in all the cases, vomiting in 4, constipation in 2, and worms on rectal examination in 4; one patient had vomited worms. In one case anti-helminthic was given before hospitalisation. Abdominal tenderness, rigidity and rebound tenderness were found in five cases. One patient had guarding with minimal tenderness; abdominal paracentesis in him was positive for pus. Plain X-ray abdomen showed gas under the right dome of the diaphragm in two cases, and one case had multiple air-fluid levels.

Four patients had terminal ileal perforations within 60 cm of the ileocecal region on the antimesenteric border; the terminal ileum was packed with worms, with one to four free worms in the peritoneal cavity. One case had worm partly in the lumen and partly projecting into the peritoneal cavity via a perforation. The perforations were single, small sized, and non-indurated. The two cases with perforated Meckel's diverticulum had this structure packed with worms. The gut wall adjacent to the perforations was congested. The peritoneal cavity was full of pus in all the six cases. No other associated cause of bowel perforation was found. The lymph nodes and the rest of the gut and viscera were normal.

The two patients with PAP of Meckel's diverticulum underwent Meckel's diverticulectomy with removal of worms. The four cases with PAP of the terminal ileum had standard closure in two layers after removal of worms. In two cases milking of worms from the terminal ileum into the large gut was done before closure, but in the other two cases, the perforation opening was enlarged into an enterotomy through which worms were removed, as milking was impossible without traumatizing the gut wall because the worms were tightly impacted. This enterotomy was closed in two layers. In all cases peritonal toilet was done and temporary drainage of the peritoneal cavity via the right flank was provided. Two patients had mild wound infection which subsided under treatment.

Histopathology of biopsies from the wall of the perforations and resected Meckel's diverticula revealed non-specific inflammatory changes.

Discussion

Ascaris lumbricoides can invade the peritoneal cavity through operative suture lines or preexisting typhoid or tubercular perforations of the small intestine.2 Primary ascaridal perforations of the small intestine and Meckel's diverticulum occur in the absence of associated underlying intestinal disease and result in migration of the worm into the peritoneal cavity with
resulting peritonitis. Such PAP result due to irritation by impacted ascarial masses in the intestinal and Meckelian diverticular lumen, leading to necrosis, ulceration and perforation. Local hyperemia, edema and necrosis induced by pressure from the parasite together with production of lytic substance(s), also contribute. Localized gangrenous patches may occur in the wall of the small intestine and Meckel's diverticulum filled to a bursting point with ascaris.

In our series since the terminal ileum and Meckel's diverticulum were tightly impacted with worms, and the rest of the small intestine, lymph nodes and other visera were normal, and since histopathology revealed nonspecific inflammatory changes, it is suggested that pressure necrosis resulting from impaction resulted in the PAP. Lytic secretion(s) from the ascaris also probably contributed.

References