Antenatal appendicular perforation


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Summary: Antenatal appendicular perforation leading to localized meconium peritonitis and intestinal obstruction is reported in a premature neonate. The baby was successfully treated by a limited ileocaecal resection.

Introduction

Appendicitis is rare in the neonatal period. We report a successfully treated case of localized meconium peritonitis secondary to an antenatal appendicular perforation in a premature low birth weight baby. This is believed to be the second case reported of meconium peritonitis due to in utero appendicular perforation.

Case report

A 12 day old female neonate, born at 32 weeks gestation, to a third gravida mother by normal vaginal delivery was admitted with constipation and abdominal distension since birth. The antenatal history was unremarkable except for prematurity. She had passed a small amount of meconium at 8 hours after birth. Later, she was able to pass only a few hard and dry pellets occasionally with rectal suppositories.

The baby weighed 1400 g and the abdomen was uniformly distended with visible intestinal peristalsis. A hard, nodular and fixed intra-abdominal mass 3 x 3 cm was palpable in the right iliac fossa. The anorectum was normal. The laboratory data were unremarkable. Plain radiographs of the abdomen showed the mass in the right iliac fossa to be calcified and gaseous distension of small bowel (Figure 1). A gastroconray study revealed a narrow calibre colon. Although repeated warm saline rectal washes relieved the obstruction temporarily, acute intestinal obstruction developed again, a week later. At laparotomy, the calcified mass was seen encasing the distal half of the appendix and the terminal ileum. There were dense adhesions around this mass. Lysis of adhesions and limited ileocaecal resection was performed with ileo-ascending colon anastomosis (Figure 2).

Histological sections from the terminal ileum and caecum showed normal mucosa and ganglion cells. Sections from the appendix showed ulceration of the mucosa with increased lymphomononuclear cells in the underlying tissue.

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lamina propria, oedema and hyperaemia in the submucosa, prominent lymphoid follicles, organizing serositis with perforation, and calcification. The postoperative recovery was uneventful. A rectal biopsy showed normal ganglion cells. The child was discharged home with normal bowel habits.

**Discussion**

Acute appendicitis with perforation causing bacterial peritonitis and presenting as an acute abdominal emergency in infants, neonates, and even premature babies has been well documented. However, only one case of prenatal appendicular perforation resulting in meconium peritonitis has been previously reported.

The usual obstructive appendicitis is rare in neonates as the base of the appendix has a conical configuration which makes obstruction of the lumen unlikely and before the differential growth of caecum occurs in the fetus, the lumen of appendix is larger at its junction with the caecum than at its tip. Perforation of the appendix in neonates may occur secondary to obstructive lesions like Hirschsprung's disease, colonic atresia, imperforate anus or ischaemic lesions like necrotizing enterocolitis. Idiopathic perforation of bowel in the absence of intestinal obstruction may be due to aplasia of the muscularis mucosa, primary vascular insufficiency or a localized vascular accident.

The presence of abdominal distension and abnormality of meconium evacuation since the day of birth and involvement of the appendix in meconium peritonitis was a strong pointer towards the probability of antenatal perforation as the cause of this child's illness. Ileocaecal resection was needed in our case because of the necessity for terminal ileal resection and extensive adhesions around this region caused by meconium peritonitis. Mucoviscidosis could be ruled out in view of normal bowel habits postoperatively in the present case.

**References**

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