Antenatal Perforation of Meckel’s Diverticulum Presenting as Meconium Peritonitis: Case Report and Literature Review

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Abstract
The most common causes of meconium peritonitis (MP) are ischemic lesions of the small bowel associated with intestinal obstruction, such as intestinal atresia, volvulus, intussusception, and meconium ileus. There have been only 4 reports of MP due to perforation of Meckel’s diverticulum (MD) in the literature. In all cases, MP was diagnosed postnatally. We report a rare case of prenatally diagnosed MP, which was found to be due to a perforated MD at laparotomy. Our case suggests that perforation of MD should be included in the etiology of MP.

Key words: Meckel’s diverticulum, meconium peritonitis, prenatal diagnosis

I Introduction
Meconium peritonitis is a sterile peritonitis caused by bowel perforation during intrauterine life, occurring at a rate of 1 per 20,000-40,000 live births. Perforation during intrauterine life is usually secondary to congenital intestinal obstruction due to such condition as meconium ileus, intestinal atresia, intestinal stenosis, internal hernia, Hirschsprung’s disease, or volvulus.

Meckel’s diverticulum is the most common gastrointestinal congenital anomaly occurring in approximately 2% of the population, and most of them are asymptomatic. MD most commonly presents in children as gastrointestinal bleeding, intestinal obstruction, diverticulitis and umbilical discharge. Perforation of MD is less common and relatively rare in the neonatal period.

Here, we describe a case of MP, that was found to be due to perforated MD only after birth.

II Case Report
A 36-year-old woman was referred to our institute at 36 weeks gestation for further management of intrauterine growth retardation and fetal bradycardia. Antenatal ultrasound (US) showed ascites with tangled adhered bowel loops suggestive of MP (Fig. 1). A male infant, birth weight 1,776 grams, was born by cesarean section. Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. The infant had a considerably distended abdomen that contained ascites and lacked bowel gas on plain abdominal radiography. In view of the prenatal diagnosis of MP, laparotomy was performed on the first day of life. Operative findings showed that the peritoneal cavity was full of thick, yellow-green fluid and the loops of intestine were adhered. The diagnosis of generalized meconium peritonitis was confirmed. Adhesiolysis revealed MD, 1 cm in length, located 15 cm from the ileocecal valve, perforated at the tip (Fig. 2). The rest of the bowel was of normal caliber with neither perforation nor obstruction.
Fig. 1  Antenatal ultrasonography.
Sagittal view of the fetus is shown. Ascites (arrows) and high echoic bowels (arrowheads) are detected.

Fig. 2  Intraoperative findings.
Meckel’s diverticulum perforated at its distal end with stoma-like eversion of the mucosa.

Fig. 3  Histopathology of the resected Meckel’s diverticulum.
Ectopic gastric mucosa and pancreatic tissue are not present. The muscle layers are intact.

such as meconium ileus or intussusception identified. Meckel’s diverticulum was excised and a double barrel ileostomy was created at the excision site. Histopathology of the MD showed that there was neither ectopic gastric mucosa nor pancreatic tissue and the muscle layers were continuous in all sections (Fig. 3). Contrast enema and a rectal suction biopsy were performed to exclude atresia/obstruction of the distal bowel and Hirschsprung’s disease. He later underwent a stoma closure and remained well at 1-year follow-up.
III Discussion

Antenatal perforation of Meckel’s diverticulum causing meconium peritonitis is an extremely rare condition and its pathophysiology remains unknown. Rosza and Gross reported the first case of suspected intrauterine perforation of MD presenting with MP. They postulated that heterotopic gastric mucosa present in the MD caused perforation through an intense inflammatory reaction, although no gastric mucosa was identified on histopathology. However, it seems unlikely that antenatal perforation occurs as a complication of gastric acid secretion since gastric glands do not normally become functional before birth. Gilbert and Rainey suggested that proximal obstruction resulted in distention of the diverticulum and subsequent necrosis and perforation. In our case, however, neither gastric nor pancreatic tissue was identified and no obstructive structure or muscle defect was seen in the diverticulum. Thus, we cannot describe the exact mechanism for spontaneous perforation in our case based on intraoperative findings or histopathology.

As shown in Table 1, review of the literature for MP due to perforated MD identified 4 cases. All 4 presented after birth (range: birth to 3 months); 2 with abdominal distention, 1 with an abdominal mass, and 1 with an inflamed hydrocele containing intestinal material and were diagnosed as perforated MD only at laparotomy. No other anomaly was noted to be present. One died from dyspnea due to peritonitis 9 hours after delivery and another infant was in shock at delivery and died 6 hours later. The other two were alive, and both had MD resected with end-to-end anastomosis. Regarding the type of MP, the oldest case seemed to be fibroadhesive, two were generalized, and the other was cystic type. Suspected etiologies for perforated MD were obstruction of the proximal portion of MD in 2, and ectopic gastric mucosa in 2.

We believe this is the first case of prenatally diagnosed MP caused by perforation of MD. Although the exact etiology in our case cannot be identified, we suggest perforated MD be included in the etiology of MP.

References


Table 1 Cases of meconium peritonitis due to perforated Meckel’s diverticulum.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Year reported</th>
<th>Sex</th>
<th>Antenatal diagnosis</th>
<th>Presentation and onset</th>
<th>Timing of operation/ Procedure</th>
<th>Type of MP</th>
<th>Ectopic gastric mucosa</th>
<th>Possible mechanism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1953</td>
<td>F</td>
<td>Abdominal distention at birth</td>
<td>-</td>
<td>-</td>
<td>Fibroadhesive?</td>
<td>-***</td>
<td>Ectopic mucosa</td>
<td>Dead</td>
</tr>
<tr>
<td>2</td>
<td>1986</td>
<td>M</td>
<td>Abdominal distention at birth</td>
<td>-</td>
<td>-</td>
<td>Generalized</td>
<td>-</td>
<td>Obstruction</td>
<td>Dead</td>
</tr>
<tr>
<td>3</td>
<td>2004</td>
<td>M</td>
<td>Inflamed hydrocele at birth*</td>
<td>Unknown/MD resection</td>
<td>Generalized</td>
<td>Generalized</td>
<td>+</td>
<td>Ectopic mucosa</td>
<td>Alive</td>
</tr>
<tr>
<td>Present case</td>
<td>2008</td>
<td>M</td>
<td>Meconium peritonitis</td>
<td>Asces on prenatal US</td>
<td>Day 0/MD resection ileostomy</td>
<td>Generalized</td>
<td>-</td>
<td>Unknown</td>
<td>Alive</td>
</tr>
</tbody>
</table>

US: ultrasound. F: female. M: male. *: Antenatal torsion of spermatic cord was suspected, **: A choledochal cyst was suspected, ***: Although ectopic gastric mucosa was not detected microscopically, the authors suspected that it had been destroyed by perforation.


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