Case Report

Meconium peritonitis in a preterm infant: a surgical emergency

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ABSTRACT

Meconium peritonitis is sterile chemical peritonitis that occurs after intestinal perforation resulting in meconium leakage and subsequent inflammatory cascade within the peritoneal cavity. The clinical presentations after birth can range from completely sealed-off peritonitis without any symptoms, to severe peritonitis requiring emergency surgical intervention. We describe a case of meconium peritonitis in a premature infant following intestinal perforation. In the immediate postnatal period, the patient was intubated and a peritoneal drain was placed. Laparotomy with bowel resection was performed the following day. The postoperative course was uneventful and the patient was discharged home in good clinical condition.

Keywords: Meconium peritonitis, Intestinal obstruction, Intestinal perforation

INTRODUCTION

Meconium represents the initial substance present in the intestines of the developing fetus resulting from the swallowing of amniotic fluid along with mucus, epithelial cells, and lanugo. Meconium peritonitis (MP) is aseptic chemical peritonitis that results from intrauterine perforation of the bowel and exudation of meconium into the peritoneal cavity. The estimated incidence rate is 1 in 30-35,000 births, with a slight male predominance. Any cause leading to small bowel ischemia or mechanical obstruction may result in MP. The secondary inflammatory response triggered by the leakage of meconium into the peritoneal cavity results in the production of ascites, fibrosis, calcification, or cyst formation. The survival rate for MP has increased from 10-40 to 80-92% due to advances in perinatal care and surgical management. We present a case of MP which presented as abdominal distension with acute decompression, followed by bedside stabilization and peritoneal drainage. Surgical exploration was performed with good clinical outcome.

CASE REPORT

A 1,700-gm female infant was born at 29 weeks of gestation to a 24-year-old primigravida mother by vaginal delivery. The presentation was cephalic and the amniotic fluid was clear. An ultrasound (US) scan at 20 weeks of gestation was normal. Follow up scans were not done. The mother’s serum HBsAg and HIV were negative. The APGAR scores were 5 and 7 at 1 and 5 minutes respectively. Her length, head circumference, body temperature, pulse, and arterial blood pressure were determined to be 45 cm, 30 cm, 36.3°C (axillary), 148/min, and 50/32 mmHg, respectively with mean arterial pressure of 27 mmHg. On physical examination, she had normal facies with no gross congenital anomaly. Her muscle tone was poor, respiration was slow and irregular, the abdomen was firm and grossly distended with visible dilated veins. She was intubated and a dose of surfactant was given in the delivery room before transferring to the NICU. The patient was started on mechanical ventilation. An orogastric tube was placed. X-ray abdomen showed pneumoperitoneum (Figure 1).
From laboratory examinations, hemoglobin and white cell count were determined to be 14.4 g/dL and 18,300/mm³. Renal and liver function tests were normal. 2-D echocardiography did not show any congenital cardiac abnormality. Newborn screening was negative for cystic fibrosis. She was kept NPO (nothing by mouth) and total parenteral nutrition was started. Piperacillin/Tazobactam was administered and dopamine infusion was started for hypotension. Pediatric surgery was consulted and it was decided to perform the abdominal drainage procedure, enterostomy, and elective closure of the stoma. Approximately 150 ml of meconium-stained fluid was drained by the abdominal drain (Figure 2).

After 24 hours of abdominal drainage, an exploratory laparotomy was performed, and the perforation was found to be at the ileum. Enterostomy was performed and orogastric feed was started after five days of the surgery. The feeds were gradually advanced to the full feeds. The patient tolerated the feeds and was discharged at 35 weeks postmenstrual age.

DISCUSSION

MP represents the result of intrauterine bowel perforation with extravasation of meconium into the peritoneal cavity leading to sterile chemical peritonitis. The causes of meconium peritonitis include bowel obstruction due to stricture, atresia, volvulus, intussusception, Hirschsprung’s disease, meconium plug syndrome from cystic fibrosis, and others. Meconium is a complex mixture of bile salts, water, cell debris, mucus, amniotic fluid, and proteins. It has been shown that the extravasation of these constituents into the peritoneum activates immune cells including macrophages. Macrophages participate in a range of cellular functions, including phagocytosis, release of chemical mediators, and antibody-dependent cell-mediated cytotoxicity. Production of chemical mediator augments fibrin deposition and severe intra-abdominal adhesions. Massive abdominal cyst formation with ascites collection may cause fetal cardiac insufficiency, preterm labor, and a poor health condition after birth.

The clinical presentation after birth varies from severe peritonitis requiring emergency surgical intervention to sealed-off peritonitis without any symptoms. Clinical manifestations of the patients with MP include abdominal distension, ascites, respiratory distress, intra-abdominal calcification, polyhydramnios, bilious vomiting, hydrocele, inguinal hernia, and abdominal mass. Prenatal US demonstrating polyhydramnios, ascites, dilatation of loops, and abdominal calcifications should raise the suspicion for MP. The findings of intestinal pseudocyst and isolated polyhydramnios are specific and almost pathognomonic. MP is classified into three groups as per antenatal US findings, Type I: large meconium ascites, Type II: large pseudocyst, Type III: Intra-abdominal calcifications, small meconium ascites, and/or a shrinking pseudocyst. The diagnosis can be made by abdominal X-ray of neonates, with varied images, altering according to the etiology of the obstruction or perforation. Meconium undergoes dystrophic calcification resulting in abdominal calcifications on radiographs. The differential diagnosis of MP, mainly in relation to the imaging findings of intra-abdominal calcifications should include meconium pseudocysts, mesenteric cyst, hepatic calcifications, adrenal calcification, adrenal hemorrhage, teratoma, and hepatic granulomas.

Surgery remains the definitive treatment and surgical intervention is advisable within the first 24 hours of life with procedures including drainage, stoma creation, and final ostomy closure.

CONCLUSION

Prenatal diagnosis is the essential first step for the management of MP. Timing of the delivery and intervention required according to the fetal condition should be discussed with the pediatric surgeon.
and neonatologist. As a result of better antenatal diagnosis and with advances in pediatric surgery and perinatal care, neonatal outcomes of MP have improved over time. By selecting proper surgical procedures based on the information of prenatal diagnosis, the outcome of severe MP may be improved.

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**REFERENCES**


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