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Case Report

Early detection of meconium peritonitis in term neonate: a case report

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Meconium peritonitis (MP) is a very rare, sterile peritonitis that results from fetal gut perforation in utero. Late diagnosis and treatment may cause severe morbidity and mortality related to sepsis. Here we report a term, appropriate for gestational age (AGA) neonate with meconium peritonitis. A one-day-old baby was brought to our hospital with lethargy and progressive abdominal distention for the last 12 hours and hypovolemic shock on admission. The diagnosis of meconium peritonitis was established based on physical examination, supported by abdominal X-ray and ultrasound. A sigmoid colon perforation was found during emergency surgery and a sigmoid colostomy was done. The infant was discharged on the 20th post-operative day with good enteral feeding tolerance. **[Paediatr Indones. 2025;65:443-6; DOI: https://doi.org/10.14238/pi65.5.2025.443-6**].

Keywords: *case report; meconium peritonitis; term baby; appropriate for gestational age; emergency surgery; outcome*

Meconium peritonitis is a rare condition with an estimated incidence of 1 in 35,000 births. Its pathology was first reported by Morgagni in 1761.[1] Meconium peritonitis is a chemical peritonitis due to antenatal intestinal perforation that leads to meconium leakage into the abdominal cavity.[2] Although it is a sterile chemical peritonitis, it causes an exudative inflammatory reaction. A bacterial infection will occur if the process persists after birth.[3] Therefore, it is important to prevent morbidity and mortality related to sepsis.

Meconium peritonitis predominantly affects preterm babies due to intestinal hypoperfusion and hypoxia. It may also result from meconium ileus, intestinal atresia, intestinal stenosis, intrauterine intussusception, volvulus, gastroschisis, or other intestinal obstructive diseases.[3,4] It is also caused by intestinal necrosis due to vascular insufficiency or intrauterine ischemia.[5]

The clinical manifestations of meconium peritonitis include abdominal distention, bilious vomiting, or respiratory distress.[6] The objective of this case presentation is to emphasize the importance of early detection and treatment of meconium peritonitis in a term, appropriate for gestational age (AGA) neonate.

The case

A one-day-old nenonate was brought to Cipto Mangunkusumo Hospital with complaints of lethargy and progressive abdominal distention for the past 12

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hours. The parents also noted that he had diminished responsiveness to stimuli and had no urine output for the past 8 hours. On admission, we found a decreased level of consciousness, hypotension, tachycardia, and prolonged capillary refill time. We assessed the condition as hypovolemic shock and administered normal saline bolus infusion until the clinical signs of hypovolemic shock had resolved.

Antenatal history was negative for risk of intrauterine infection or maternal cardio-metabolic disease. The patient's mother received routine antenatal care; the last ultrasound examination was conducted at seven months of pregnancy. During pregnancy, the fetus was active and there was no history of fetal distress.

The baby was delivered via vaginal birth with an APGAR score of 9/10. The volume of amniotic fluid was normal and there was no sign of premature rupture of the membrane. Birth weight was 2,800 grams, AGA, and normal body length. After birth, the baby received standard formula milk due to low breastmilk supply. Meconium was passed within 24 hours, and stool was passed regularly. However, the infant refused to feed for 12 hours. The patient was brought to a district hospital, where he was immediately referred to Cipto Mangunkusumo Hospital for comprehensive treatment.

On physical examination, we found abdominal distention with normal peristaltic sounds. Laboratory examination revealed a significant increase in C-reactive protein (CRP). Abdominal X-ray showed multiple calcifications, air-fluid level, and pneumoperitoneum, consistent with findings of meconium peritonitis (**Figure 1**). We found loculated complex ascites and peritonitis from ultrasound examination (**Figure 2**). The infant was given empirical antibiotics within one hour from admission. During emergency exploratory laparotomy, surgeons found 10 mL of fecal material, fibrin, dense adhesion, and perforation in the sigmoid colon (**Figure 3**). Adhesiolysis and sigmoid colostomy were performed. The infant had a rapid clinical recovery and was able to be extubated on the third day of postoperative care. The patient did not fall into a septic state after the procedure. Intake via orogastric tube had been given since the third day of postoperative care. The patient was discharged on the 20th post-surgical day, when full enteral feeding was well-tolerated and the parents were confident to perform daily care at home, including the ability to perform stoma care.

Discussion

In our case, meconium peritonitis occurred in a term, AGA baby. Previous studies revealed that meconium peritonitis more commonly affects preterm babies (59.5-60.5%).[6,7] Meconium peritonitis is classified

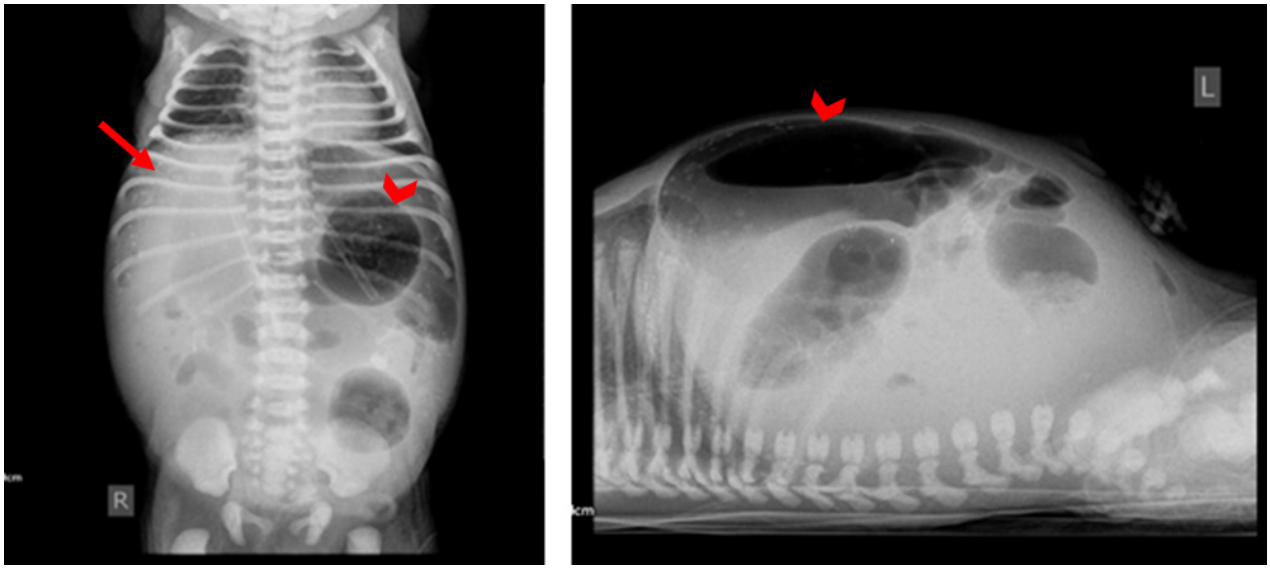

Figure 1. Abdominal X-Ray showed multiple calcification (arrow) and pneumoperitoneum (arrowhead)

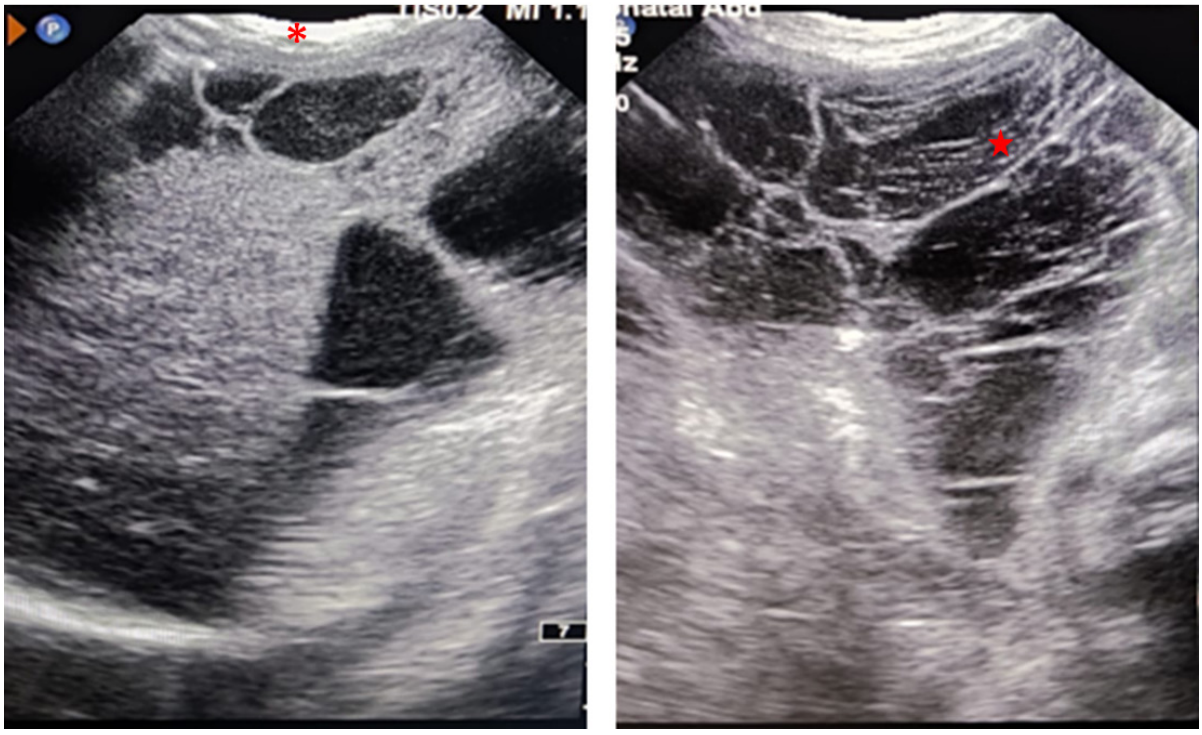

Figure 2. Abdominal ultrasound showed peritonitis (asterisk) and loculated ascites (star)

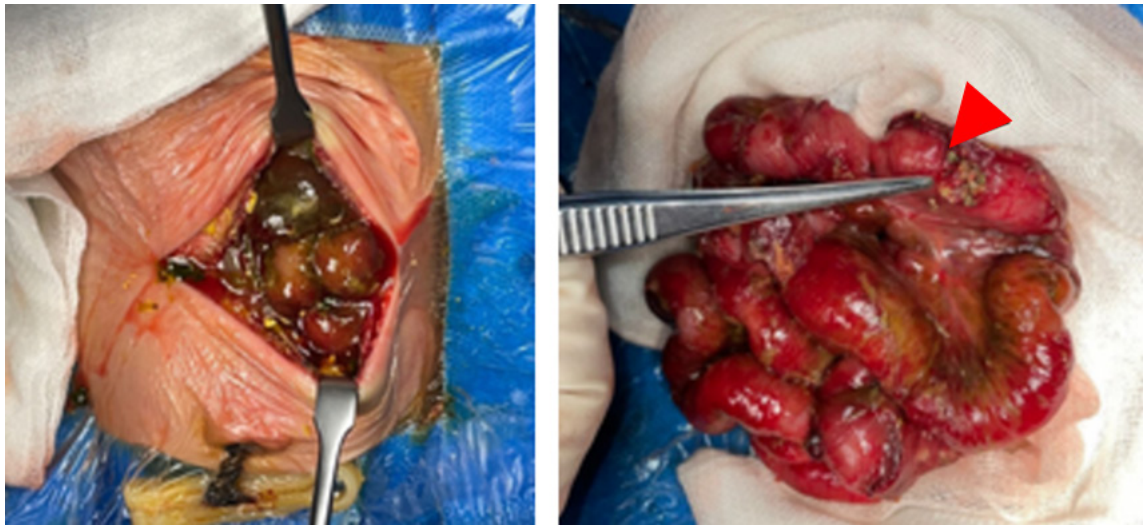

Figure 3. Intra-operative findings showed intestinal point perforation (triangle)

into three groups based on ultrasound findings: large meconium ascites (type 1), large pseudocyst (type 2), and intra-abdominal calcification with small meconium ascites and/or shrinking pseudocyst (type 3).[8] Our patient had type 3 meconium peritonitis.

Previous case reports showed that meconium peritonitis can be detected prenatally, with the earliest diagnosis possible from 20 weeks of gestation.[6] In our patient, the latest antenatal ultrasound examination was performed at seven months of gestational age. This implies that intestinal perforation occurred in

the last trimester of pregnancy. In fact, the baby had not shown any signs of fetal distress during pregnancy and did not require active resuscitation at birth, which suggests that the intestinal perforation occurred near the time of labor time.

In the present case, emergency surgery was performed after clinical stabilization of the patient's condition. Upon opening the peritoneum, the surgeon found 10 mL of meconium constituents mixed with fibrin in the abdominal cavity. Meconium is composed of bile salts, cell debris, and protein mixture. Although these constituents were sterile, the spillage will activate immune cells, especially macrophages. Macrophages infiltrate the peritoneum and activate phagocytosis, chemical mediators release, and antibody-dependent cell-mediated cytotoxicity. Tumor necrosis factor-α (TNF-α) that was produced by macrophages will aggravate fibrin deposition and severe intestinal adhesion.[7,9] These mechanisms explain the reason for fibrin deposition and severe adhesions during surgery, which required extensive adhesiolysis.

Our patient experienced a rapid clinical recovery, with extubation done on the third day of post-operative care. The infant did not fall into a septic state after the procedure. Intake via orogastric tube had been given since the third day of postoperative care. A previous study found that early surgical intervention leads to excellent outcomes with an overall survival rate of 95%.[7,10] Delaying surgery for more than 72 hours will cause intestinal bacterial colonization, which may cascade into severe sepsis with mortality rates of up to 75%.[11]

In conclusion, meconium peritonitis can occur in term, AGA babies. Early diagnosis can be achieved through comprehensive history taking, physical examination, X-ray, and ultrasound. Prompt surgical intervention is mandatory to prevent bacterial colonization and sepsis.

Conflict of interest

None declared.

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