Case Report

Meconium Peritonitis Connecting the Dots between Pathophysiology, Prenatal Diagnosis and Postnatal Outcome

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Abstract

Objective: To describe the antenatal findings, clinical course, and perinatal outcome of a case of meconium peritonitis.

Methods: This is a case report.

Case: A 28-year-old primigravida at 34–35 weeks' gestation was referred for evaluation of hyperechoic fetal bowel. Serial ultrasonography revealed echogenic bowel and fetal ascites without associated major structural anomalies. Maternal serology showed reactive IgG for toxoplasma and cytomegalovirus with high avidity, suggesting past infection. Based on the Zangheri classification, the condition was categorized as grade I meconium peritonitis. The patient delivered at term (38–39 weeks). The female neonate weighed 2,640 g, with a length of 50 cm and APGAR scores of 8 and 9 at 1 and 5 minutes, respectively. Postnatal evaluation revealed no signs of acute abdomen. Babygram findings were unremarkable. The neonate was diagnosed with meconium peritonitis and early-onset sepsis and received empirical antibiotic therapy for 6 days, resulting in clinical improvement.

Conclusion: Meconium peritonitis presents with a spectrum of antenatal findings, including intra-abdominal calcifications, ascites, pseudocyst formation, and bowel dilatation. The Zangheri classification is useful for predicting disease severity and the likelihood of postnatal surgical intervention. Mild cases may resolve spontaneously in utero, allowing for favorable outcomes with conservative management and term delivery.

Keywords: Meconium peritonitis, prenatal diagnosis, ultrasonography.

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INTRODUCTION

Meconium peritonitis is a rare clinical and radiological condition, occurring in approximately 1 in 30,000 to 35,000 live births.[1,2] It is characterized by sterile inflammation of the peritoneum resulting from the leakage of meconium into the peritoneal cavity due to intrauterine intestinal perforation.[3] Although uncommon, this condition can be diagnosed prenatally using ultrasonography, and its sonographic findings are strongly correlated with neonatal outcomes.[2,4]

Typical antenatal ultrasound features include intra-abdominal calcifications, with or without ascites, bowel dilatation, and meconium pseudocyst formation. Postnatal clinical manifestations vary widely, ranging from asymptomatic cases to abdominal distension and signs of intestinal obstruction. Therefore, affected pregnancies require close monitoring by obstetricians, multidisciplinary management, and preparation for possible postnatal surgical intervention, particularly in moderate to severe cases.[3,4]

Despite its rarity, meconium peritonitis poses diagnostic and therapeutic challenges. Fetal Meconium Peritonitis (FMP) has been associated with a mortality rate ranging from 43.7% to 59.6%.[5] In Indonesia, two cases of prenatally diagnosed meconium peritonitis have been reported in Jakarta, both requiring postnatal

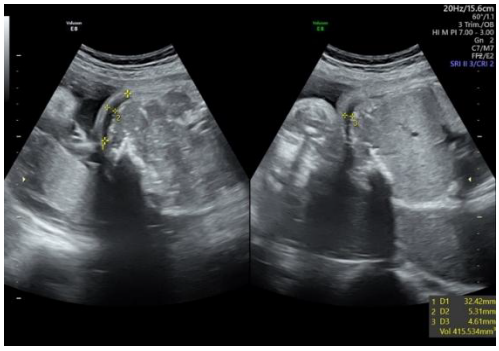
surgical management.[6] To our knowledge, this is the first reported case from Surabaya and the second case in Indonesia presenting with a mild manifestation that was successfully managed conservatively.

CASE

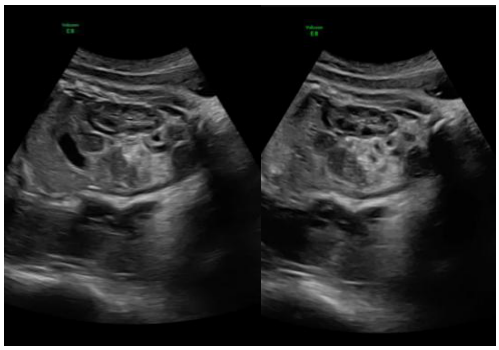
A 28-year-old primigravida at 34–35 weeks' gestation was referred to the High-Risk Pregnancy Clinic at Dr. Soetomo General Academic Hospital, Surabaya, due to a hyperechoic fetal bowel identified by a private obstetrician. The patient was asymptomatic and had no significant history of chronic, hereditary, familial, or infectious diseases.

Serial ultrasonography was performed and revealed findings suggestive of meconium peritonitis. At 35–36 and 36–37 weeks' gestation, intra-abdominal calcifications with minimal ascites were observed (Figure 1A and 1B). At 37–38 weeks' gestation, intra-abdominal calcifications and bowel dilatation were noted (Figure 1C). Fetal biometry showed an estimated fetal weight within the normal range. The placenta was located posteriorly, amniotic fluid volume was adequate, and fetal cardiac activity was normal. No major structural anomalies were detected in other organs.

Cardiotocography demonstrated a fetal heart rate of 150 bpm with normal variability and reactive tracing (Category I). Laboratory examination revealed a hemoglobin level of 10.4 g/dL (hematocrit 30.9%), leukocyte count of 5,730/µL, and platelet count of 213,000/µL. Serological testing showed reactive IgG for toxoplasma and cytomegalovirus with high avidity, indicating past infection. Based on Zangheri's classification, the diagnosis was grade I meconium peritonitis.



1A



1B

Figure 1. Ultrasonography at **(A)** 35/36 wga and **(B)** 37/38 wga showed intra-abdominal calcification and minimal ascites.

1C

Figure 1(C). Ultrasonography at 38/39 wga showed intra-abdominal calcification and bowel dilatation.

A multidisciplinary approach was initiated, involving consultations with the Neonatology and Pediatric Surgery departments. Both teams agreed to evaluate the neonate after delivery. The patient was subsequently hospitalized for induction of labor at 38–39 weeks' gestation. A female neonate was delivered vaginally with a birth weight of 2,640 g, length of 50 cm, and APGAR scores of 8 and 9 at 1 and 5 minutes, respectively. The amniotic fluid was clear. Initial neonatal assessment showed no signs of peritonitis, such as fever, abdominal distension, or abdominal wall erythema. No imperforate anus was identified. Babygram findings were normal, and blood culture results were sterile.

The neonate was diagnosed with mild meconium peritonitis and early-onset sepsis. Conservative management was initiated, including placement of an orogastric tube, enteral feeding, and administration of broad-spectrum antibiotics. The clinical condition

improved, and the neonate was discharged after six days of hospitalization. The maternal postpartum course was complicated by a grade IV perineal laceration, which required emergency surgical repair in the operating room. Follow-up evaluation showed good wound healing without urinary or fecal incontinence.

At two months of age, the infant was hospitalized due to acute diarrhea and severe underweight. Based on pediatric evaluation, the condition was attributed to lactose intolerance rather than a complication of meconium peritonitis.

DISCUSSION

Fetal meconium peritonitis (FMP) is a rare clinical and radiological entity, occurring in approximately 1 in 30,000–35,000 births.[1,2] A higher incidence has been reported in Taiwan, at 3.7 per 10,000 births.[7] Meconium peritonitis is defined as sterile inflammation of the peritoneum caused by the leakage of meconium from a perforated intestine into the peritoneal cavity in utero; perforations occurring postnatally are not included in this condition.[2]

Intestinal perforation may result from ischemia due to vascular insufficiency or from mechanical obstruction, such as meconium ileus, intestinal atresia, stenosis, volvulus, internal hernia, intussusception, or Hirschsprung's disease (Table 1).6,7 Meconium ileus associated with cystic fibrosis accounts for approximately 25%–40% of cases.[8]

Table 1. Etiology of Meconium Peritonitis[6]

Common etiology	Uncommon etiology
Meconium ileus (secondary to cystic fibrosis)	Meckel's diverticulum
Intestinal atresia	Colonic atresia
Intestinal stenosis	Torsion of a fallopian tube cyst
Intestinal hernia	Foetus in fetu
Hirschsprung's disease	Cytomegalovirus
Volvulus	Toxoplasma
Intussusception	Hepatitis
Extrinsic bands and adhesions	Parvovirus B19
Rectal strictures	Hydrometrocolpos
Imperforate ane	Appendicitis
Prenatal anoxic events causing bowel ischemia	
Vascular insufficiency including thrombosis	
Idiopathic	

Meconium contains bile salts, inorganic salts, proteins, and digestive enzymes. When meconium enters the peritoneal cavity, it induces an inflammatory response through increased production of proinflammatory cytokines. Calcium salts, a major component of meconium, react with inflammatory exudates, resulting in intra-abdominal calcifications, which are often the earliest and most prominent feature of this condition. The extravasation of plasma proteins and accumulation of extraluminal fluid lead to ascites, while localized calcium deposition may result in pseudocyst formation.[3] The natural history of FMP follows three possible pathways; spontaneous closure of the perforation leading to peritoneal calcifications; damage to the intestinal segment resulting in atresia; and persistence of perforation with thickened, calcified meconium surrounded by omentum, forming a pseudocyst.[9]

Ultrasonography plays a pivotal role in the prenatal diagnosis of FMP by identifying characteristic soft markers, including intra-abdominal calcifications, ascites, bowel dilatation, and pseudocyst formation. Calcifications typically appear as echogenic linear or clumped foci within the abdomen and pelvis, particularly outside the intestinal lumen or along the peritoneal surfaces.[4] They are visualized as hyperechoic areas with posterior acoustic shadowing.[10] Echogenic bowel is defined as bowel echogenicity comparable to that of bone. Bowel dilatation is defined as small bowel loops measuring >7 mm at any gestational age or colonic loops >18 mm in the third trimester.[1] Polyhydramnios is also frequently

observed.

The Zangheri classification is based on prenatal ultrasound findings and is used to predict the likelihood of postnatal surgical intervention (Table 2). Isolated calcification (Grade 0) is associated with the best prognosis and no need for surgical intervention. However, when calcifications are accompanied by one or more additional findings, the likelihood of postnatal surgery increases, and delivery in a tertiary care center is recommended.[4]

Table 2. The Zangheri's Classification System for Fetal Meconium Peritonitis and Percentage of Postnatal Surgical Intervention[4]

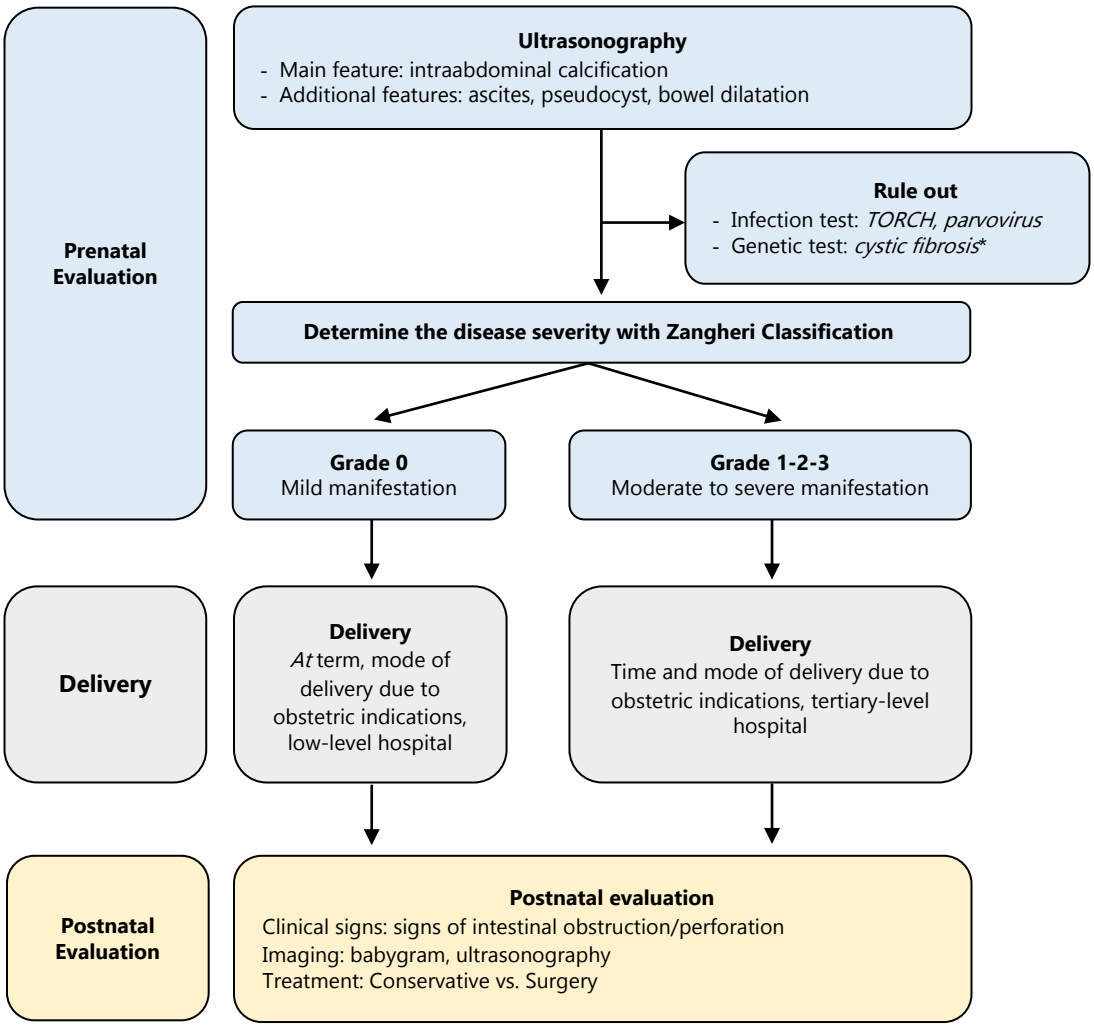
Grade		Prenatal Findings	Surgery (%)
0		Isolated calcification	0
1	A	Calcification associated with ascites	52
	B	Calcification associated with pseudocyst	52
	C	Calcification with bowel dilatation	52
2		Calcification with two associated findings	80
3		All features present	100

Fetal ascites is the most frequently reported ultrasound finding, present in 67.7%–100% of cases, followed by other soft markers with variable frequencies (Table 3). In our case, intra-abdominal calcifications and minimal ascites were observed at 35–36 weeks' gestation, with bowel dilatation appearing at 37–38 weeks (Figure 1A–C). Based on Zangheri's classification, the case was categorized as Grade 1. However, further subclassification into Grade 1A or 1C was not possible due to the mild degree of ascites and bowel dilatation. In some cases, spontaneous resolution of intrauterine findings has been reported, reflecting in utero healing of the perforation.[10]

Table 3. Percentage of each Type of Prenatal Ultrasound Soft-Markers of Meconium Peritonitis

Prenatal findings (%, n)	Related studies			
	Shinar et al. (2022)	Fu et al. (2022)	Chen et al. (2019)	Wang et al. (2008)
Intraabdominal calcification	29.4 (12/34)	88.9 (8/9)	29.2 (7/24)	92.9 (13/14)
Ascites	67.6 (23/34)	55.6 (5/9)	70.8 (17/24)	100 (14/14)
Pseudocyst	14.7 (5/34)	55.6 (5/9)	8.3 (2/24)	29 (4/14)
Bowel dilatation	44.1 (15/34)	100 (9.9)	45.8 (11/24)	57 (8/14)
Polyhidramnios	32.4 (11/34)	66.7 (6/9)	25 (6/24)	50 (7/15)
Echogenic bowel	73.5 (25/34)	Not evaluated	29.2 (7/24)	Not evaluated

Following the identification of intra-abdominal calcifications, potential etiologies should be investigated through serological testing for cytomegalovirus, toxoplasma, hepatitis viruses, and parvovirus B19, as well as genetic testing for cystic fibrosis.[3,8] Viral infections are more commonly associated with calcifications within the liver or spleen, whereas cystic fibrosis is typically associated with intraluminal calcifications. Although approximately 10% of cases are related to cystic fibrosis, routine genetic testing is not always recommended in Asian populations due to its low prevalence.[4,9] Fetal Magnetic Resonance Imaging (MRI) may improve diagnostic accuracy, particularly in differentiating pseudocysts from other intra-abdominal cystic lesions such as mesenteric, duplication, or ovarian cysts.[9] In our case, toxoplasma and cytomegalovirus infections were excluded as etiologies. A clinical pathway was established to guide prenatal diagnosis and postnatal management (Figure 2).



*Only for countries with a high prevalence of cystic fibrosis

Figure 2. The continuing pathway from prenatal diagnosis to postnatal management for fetal meconium peritonitis.

Selective termination of pregnancy is generally not recommended unless there are additional indications.[9] Most studies report a mean gestational age at delivery of 35–36 weeks.[1,2,5,7] After birth, clinical manifestations typically appear within the first 1–2 days and range from asymptomatic cases to abdominal distension and signs of intestinal obstruction.3 Signs of obstruction include a distended abdomen with edematous, congested, and shiny skin, visible superficial vessels, and, in severe cases, a palpable abdominal mass.[9]

Neonates without signs of intestinal obstruction can be managed conservatively, whereas those with obstruction require surgical intervention. Conservative management includes gastrointestinal decompression, enteral or parenteral nutrition, and administration of broad-spectrum antibiotics. In cases requiring surgery, end-to-end anastomosis is the most commonly performed procedure, as intestinal obstruction is the underlying cause in most cases.

CONCLUSION

Meconium peritonitis remains a rare but clinically significant condition that presents diagnostic and therapeutic challenges. Prenatal ultrasound plays a crucial role in early detection through key findings such as intra-abdominal calcifications, ascites, pseudocyst formation, and bowel dilatation. The Zangheri classification is a valuable tool for stratifying disease severity and guiding perinatal management, particularly in anticipating the need for postnatal surgical intervention. This case highlights that mild forms of meconium peritonitis may resolve spontaneously in utero and can result in favorable outcomes with conservative management and term delivery. Nevertheless, accurate prenatal

diagnosis, appropriate risk stratification, and multidisciplinary planning remain essential. Early identification and tailored management strategies are critical to optimizing neonatal outcomes, especially in moderate to severe cases, which should be managed in tertiary care centers with access to neonatal intensive care and pediatric surgical services.

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