



Case Report

Neonatal meconium peritonitis presenting as non-immune hydrops fetalis

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Abstract

Background: Meconium peritonitis results from sterile intrauterine bowel perforation. While typically localized, it can manifest as NIHF — generalized fetal edema and fluid accumulation in multiple body cavities. Early identification is critical, as MP carries a significantly better prognosis than many other causes of NIHF when surgical intervention is timely.

Key words: Meconium peritonitis; Non-immune hydrops; Intestinal perforation

1. Introduction

Non-immune hydrops fetalis (NIHF) represents a final common pathway of diverse fetal pathologies, including cardiac, chromosomal, hematologic, metabolic, and infectious disorders. The etiology remains unidentified in nearly half of cases, and mortality is high. Gastrointestinal causes such as meconium peritonitis are exceedingly rare but potentially reversible. We describe a preterm infant with antenatal NIHF and polyhydramnios in whom stepwise postnatal evaluation revealed meconium peritonitis with ileal perforation—a distinctly uncommon yet treatable cause of hydrops.

2. Case Presentation

A male infant was delivered at 34 weeks' gestation, weighing 2.1 kg, by emergency cesarean section after the mother—diagnosed antenatally with non-immune hydrops and polyhydramnios—developed premature rupture of membranes in the third trimester. The antenatal course was otherwise uneventful; maternal screening for TORCH, hepatitis B/C, and HIV was negative, and there was no history of diabetes or drug intake. At birth, the baby had poor respiratory effort, generalized edema, and tense abdominal distension. He required positive-pressure ventilation, followed by endotracheal intubation and two doses of surfactant for respiratory distress syndrome. Mechanical ventilation was continued for 48 hours and subsequently transitioned to non-invasive support. Because of the antenatal diagnosis of hydrops, a comprehensive evaluation for non-immune causes was undertaken:

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- Cardiac evaluation: Echocardiography showed a closing ductus arteriosus with normal anatomy and function.
- Cranial and abdominal ultrasonography: Mild-to-moderate ascites with echogenic particulate content, suggestive of meconium peritonitis; no intracranial abnormalities.
- Infectious screen: Maternal and neonatal TORCH serologies negative.
- Hematologic workup: Hemoglobin 19.3 g/dL; DCT negative; reticulocyte 21.6%; normal ferritin.
- Metabolic evaluation: Urine succinyl acetone for tyrosinemia negative; galactosemia screen normal; blood glucose stable.
- Liver function: Within 24 hours, total bilirubin 14 mg/dL (direct 9.4 mg/dL); mild transaminase elevation; normal coagulation profile.

On day 2 of life, in view of progressive abdominal distension and tense ascites, a diagnostic paracentesis was performed, draining approximately 150 mL of dark-yellow ascitic fluid. Analysis showed high protein, low glucose, and sterile culture. Following the tap, abdominal girth reduced transiently, but the baby continued to have failure to pass meconium, even after rectal stimulation. Abdominal radiographs revealed a normal bowel-gas pattern without air–fluid levels or intra-abdominal calcifications, making intestinal obstruction less likely initially. However, when the baby continued to have persistent distension and bilious aspirates, distal ileal atresia or meconium ileus with perforation was considered. Meanwhile, the infant exhibited conjugated hyperbilirubinemia with markedly elevated α -fetoprotein, prompting evaluation for gestational alloimmune liver disease (GALD). Intravenous immunoglobulin (1 g/kg/day \times 2 days) was administered empirically. Minor-salivary-gland biopsy showed no hemosiderin deposition, thereby excluding GALD. A contrast study subsequently demonstrated poor passage of contrast beyond distal ileum, suggestive of obstruction. Exploratory laparotomy revealed extensive meconium staining, dense inter-loop adhesions, and multiple sealed perforations in a 10–12 cm segment of distal ileum with proximal dilatation. The diseased bowel was resected, and a proximal ileostomy with distal mucous fistula was fashioned; appendectomy was also done. Histopathology showed partial mucosal necrosis and ulceration with severe congestion, consistent with meconium peritonitis. Ganglion cells were present and no goblet-cell hyperplasia was observed. The postoperative course was stable. Feeds were initiated on postoperative day 3, briefly withheld for intolerance, and later advanced. The infant was discharged on full enteral feeds with a functioning ileostomy and was thriving at follow-up.

3. Discussion

Meconium peritonitis arises from antenatal intestinal perforation with sterile meconium leakage, leading to chemical peritonitis. Reported incidence is approximately 1 in 35 000 live births. Typical ultrasonographic features include echogenic ascites, pseudocyst formation, and peritoneal calcifications; however, atypical cases lacking calcifications, as in our patient, can obscure diagnosis. Hydrops fetalis secondary to meconium peritonitis is exceedingly uncommon. Chronic intrauterine inflammation and peritoneal irritation can result in hypoalbuminemia, capillary leak, and generalized fetal edema.

Several reports have documented this association:

- Kimura et al. described three infants with hydrops secondary to meconium peritonitis who survived following surgical correction (*Pediatr Surg Int* 2017; 33:1021–6).
- Bellini et al. noted gastrointestinal etiologies, including intestinal perforation, in <2% of NIHF cases (*Am J Med Genet A* 2021;185:2163–74).
- Ramachandran et al. reported improved outcomes in neonates where timely laparotomy identified meconium peritonitis as the cause of hydrops (*J Neonatal Surg* 2019; 8:42).

In the present case, prematurity, respiratory distress, and conjugated hyperbilirubinemia initially led to a broad differential including GALD and metabolic liver disease. The echogenic ascitic fluid suggested meconium peritonitis, but absence of calcifications delayed confirmation. Ultimately, surgical exploration established the diagnosis and allowed curative resection. Non-immune hydrops generally carries poor prognosis, yet when a correctable cause such as intestinal perforation is identified, survival may exceed 80%. This underscores the importance of systematic postnatal evaluation—cardiac, hepatic, metabolic, and gastrointestinal—whenever hydrops is detected antenatally.

4. Conclusion

Meconium peritonitis presenting as non-immune hydrops is rare but potentially reversible. In preterm infants with hydrops, ascites, and early cholestasis, gastrointestinal causes should be actively sought even when imaging lacks calcifications. Early recognition and surgical management can transform an otherwise fatal outcome into survival with full recovery.

Learning Points

- Non-immune hydrops fetalis often remains idiopathic; gastrointestinal causes such as meconium peritonitis, though rare, are potentially curable.
- Echogenic ascitic fluid without calcification should raise suspicion for meconium peritonitis, warranting close surveillance and paracentesis when appropriate.
- Systematic evaluation—including cardiac, hepatic, metabolic, and gastrointestinal investigations—can uncover reversible etiologies of hydrops.
- Timely surgical intervention following stabilization is lifesaving and markedly improves outcomes