Sonographic diagnosis of meconium peritonitis: a case report

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European Journal of Medical Case Reports
Volume 2(2):87-90

https://doi.org/10.24911/ejmcr/2/21
Discover Publishing Group

ABSTRACT

Background: In certain situations, the radiologist may be the first physician to suggest the presence of a pediatric abnormality. The possibility of early diagnosis of potential life-threatening emergency conditions is one of the important contributions of ultrasound to the referring clinician. Our case demonstrates the value sonographic diagnosis of meconium peritonitis (MP).

Case Presentation: We present a case of a newborn infant who was diagnosed to have echogenic bowel with a few specks of peritoneal calcification and loculated ascites. Early detection with ultrasound led to immediate surgical intervention and patient recovered without any immediate or long-term complications.

Conclusion: MP is a chemical peritonitis caused by fetal intestinal perforation which occurs mostly in utero. Its incidence is extremely rare, but serious neonatal morbidity or even mortality can occur if the diagnosis is delayed. Prenatal diagnosis is essential in prompting early postnatal surgical intervention, and also, improving neonatal outcome.

Keywords: Ultrasound, ascites, meconium peritonitis, case report.

Received: 21 December 2017 **Accepted:** 2 May 2018

Type of Article: CASE REPORT

Funding: None

Declaration of conflicting interests: None

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Background

Meconium peritonitis (MP) is a sterile inflammatory reaction in the fetal abdomen resulting from in utero bowel perforation that nearly always involves the small bowel. Its prevalence is 0.29 in 10,000 live births [l–3]. In the neonatal period, this may lead to intestinal obstruction and requires surgical exploration. This form was first described by Morgagni in 1761 [3].

On rare occasions, this chemical peritonitis heals spontaneously without clinical manifestation. Attention to this type of pathology was drawn by the finding of intraperitoneal, inguinal or scrotal masses, or calcifications [4]. However, differential diagnosis with other intraperitoneal tumors or testicular neoplasm caused some unnecessary surgeries.

Recently, the natural history of MP and, even more exceptional, the extrusion of the meconium plug have been depicted [2].

The prenatal diagnosis process should include: testing for cystic fibrosis, chromosomal abnormalities, and congenital anatomical or structural anomalies as well as the chemical and histologic analysis of the peritoneal fluid obtained by fetal paracentesis [5,6]. The resulting diagnosis is of paramount importance in formulating a management plan for the obstetrical and perinatal management of the patient.

We present a patient with spontaneous MP. Neonatal surgery was required to treat ileal atresia.

Case Presentation

Our case is of new born baby boy born via selective cesarean section at 36 weeks of gestation. Birth history was insignificant. But on fifth day of life, the baby started to develop abdominal distention which was gradually increasing. The baby was initially managed in a small hospital which lacked tertiary care facilities.

The patient was then shifted to a tertiary care hospital where he was immediately admitted to intensive care unit (ICU) and initial workup was done which showed increased bilirubin levels up, for which phototherapy was started. Echo showed a small sized ductus arteriosus 2 mm in diameter, Patent Ductus Arteriosus (PDA)/ Lysophosphatidic acid (LPA) ratio 0.28 with left-to-right flow. Patent foramen ovale and moderate to severe right ventricle and pulmonary arterial hypertension was noted.

The patient was hemodynamically stable; however, on the second day of ICU admission, baby had issues of desaturation, tachycardia, and poor perfusion. The patient was intubated and troponin support was given. Blood transfusion and IV antibiotics were started. The patient was later shifted to isolation, and his blood culture growth showed gram-negative bacilli.

The pediatric team noted constant increase in abdominal girth. Supine X-ray abdomen was performed which showed the possibility of pneumoperitoneum. There was generalized haze in the abdomen secondary to ascites.

Later, portable ultrasound was performed in Neonatal intensive care unit (NICU) to look for collection. Moderate ascites with thick septations was identified (Figure 1). There was evidence of echogenic material within the bowel and echogenic mesenteric fat along with few specks of peritoneal calcification, which represented MP (Figure 2).

Under ultrasound guidance, using a 22-gauge needle, the radiology team performed diagnostic aspiration of fluid in the perihepatic locule and approximately 10 cc of clear yellow fluid was aspirated. Specimens were collected and sent for requested laboratory investigations. No complications were encountered. The ascitic culture was positive for enterococcus faecalis and morganella morganii.

With the diagnosis of MP, the pediatric surgical team was consulted. Considering the deteriorating status of the patient, the patient was moved to the operating room where ileal perforation was identified and ileostomy was done, and hepatic and pelvic drains were placed.

Following the surgery, the patient remained stable hemodynamically, and direct breast feeding trial was given which was tolerated well. The patient maintained temperature and was discharged alive as planned.

Discussion

Any condition causing bowel obstruction may be responsible for bowel distension and perforation, leading to a sterile chemical peritonitis. Meconium ileus, which is related to cystic fibrosis in 90% of cases, accounts for less than 25%. More frequently, mechanical bowel obstruction is provoked by intestinal atresia, volvulus, intussusception, or herniation. Exceptionally colonic aganglionosis, resulting in an aperistalsis and a microcolon, is responsible for a meconium ileus and perforation of the intestinal wall [1,3,5].

Dilatation of the bowel loops leads to local vascular impairment of the intestinal wall, necrosis, and subsequent perforation. Some authors suggest that an intestinal hypoperfusion, as a result of fetal hypoxia, is the primary cause of bowel atresia and perforation. Bowel peristalsis forces meconium and digestive enzymes into the peritoneal cavity, resulting in an intense chemical inflammatory process. Within days, giant cells and histiocytes

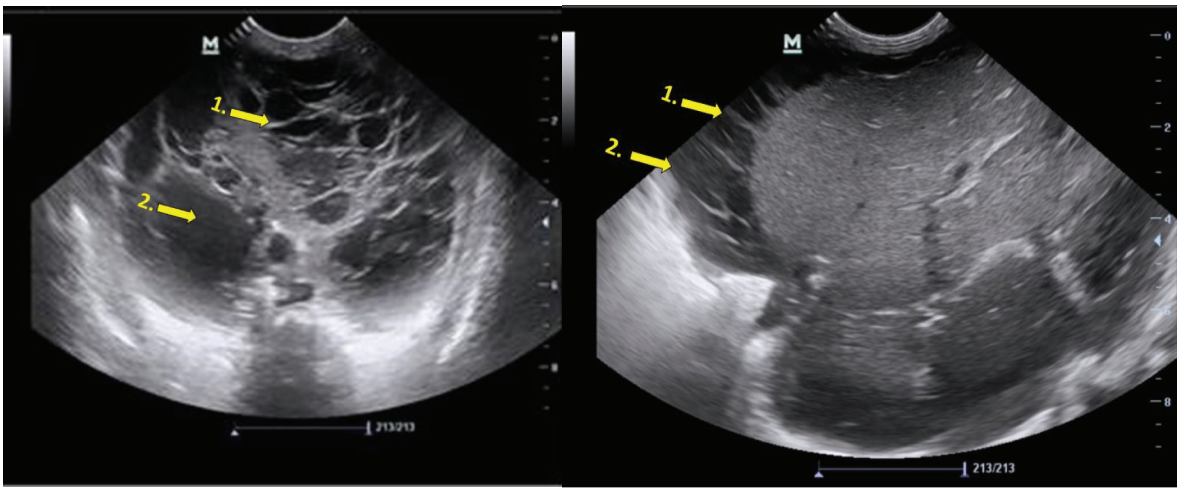

Figure 1. *Fetal ascities. (1) Thick septation (2) loculated fluid collection*

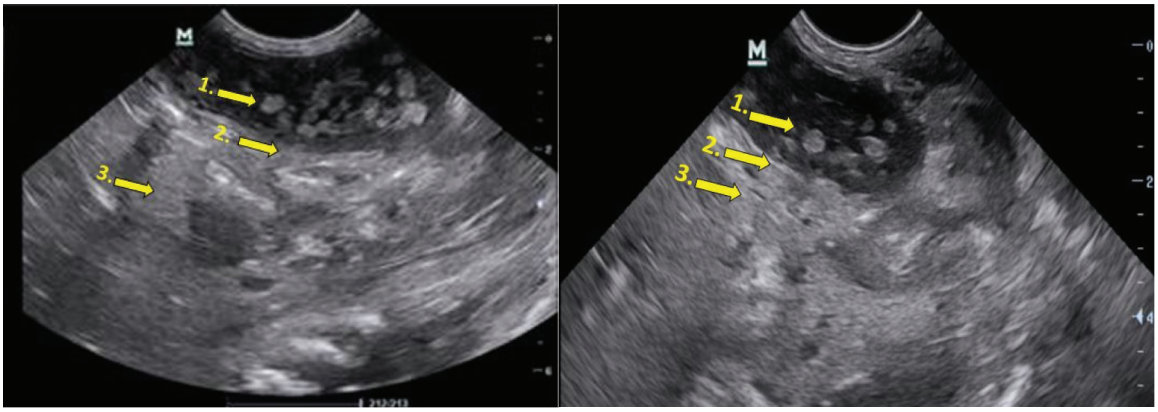

Figure 2. *Ecogenic material and calcified meconium plaques. (1) Mecomium plaque, (2) echogenic bowel, fetal bowel becomes progressively more visible by ultrasound and the lumen appears relatively "bright" if meconium accumulates within its lumen, (3) hyper echoic omentum, secondary to abdominal ascites and chemical peritonitis.*

surround the extruded meconium to form foreign body granulomas and calcifications. Depending on the spread of the inflammatory response, three pathological types are distinguished. In the generalized type, characterized by diffuse peritoneal fibrotic thickening and calcium deposits, the meconium spreads throughout the peritoneal cavity. The fibroadhesive variant, which is the most common, produces obstruction by adhesive bands sealing the perforated site. If the perforated site is not effectively sealed, a thick-walled cyst is formed by adhesion of the proximal bowel loops to the perforated site. So, the perforated intestinal area communicates only with the newly formed pseudocyst, which is lined by a calcified wall.

Cystic fibrosis is associated with meconium ileus and subsequent MP in about 15% of the cases [3]. Newer data that analyze the Cystic Fibrosis Transconductance Receptor gene suggest that the association is higher. Characteristic findings in the second and early third trimester include highly echogenic intra-abdominal masses. In the third trimester, often enlarged bowel loops are observed [7]. Parental carrier detection and prenatal diagnosis by DNA analysis is possible in about 70% of cases related to the mutation in DF508 allele on chromosome 7 [7,8]. Neonatal investigation should include repetitive sweat chloride tests.

Polyhydramnios is present in 10%–64% of cases and has been attributed to difficulty in swallowing as the result of deficient bowel peristalsis [2,9]. On rare occasions, fetal hydrops may be present.

Prenatal diagnosis is suspected when fetal intra-abdominal calcifications are observed, especially in association with fetal ascites and polyhydramnios [5]. Fetal bowel obstruction associated with fast developing fetal ascites or hydrops should alert the sonographer. On rare occasions, however, fetal ascites regresses, intestinal dilatation disappears, and peristalsis reappears. Only hyper echoic area remains [2,9]. Sometimes, intra-abdominal meconium pseudocysts are the sole remnants of the MP. Fetal abdominal hyper echoic masses or pseudocysts have recently been associated with congenital infections, chromosomal abnormalities, and cystic fibrosis [12,13]. Differential diagnosis further includes hematometrocolpos, ovarian, urachal, mesenteric and retroperitoneal cysts, and rare intra-abdominal tumors [8].

Prenatal investigation by fetal blood sampling should detect chromosomal abnormalities, rule out cystic fibrosis by DNA analysis, and exclude congenital infection through fetal hematological, immunological, and hepatic investigation. More precise information about fetal ascites and bowel perforation can be obtained by fetal paracentesis and histological analysis of the aspirated fluid.

Prenatally detected cases of MP most frequently have a fair prognosis, as proven by a rather low perinatal mortality rate. Exclusion of chromosomal or rare infectious etiologies results in a perinatal survival rate of more than 80%. The intense chemical peritonitis may seal the intestinal perforation permanently. Fifty percent of the newborns had a laparotomy and intestinal exploration, with resection of atretic or perforated segments in most cases.

Long-term prognosis is strongly affected by the presence of cystic fibrosis resulting in pancreatic insufficiency and digestive disturbances, multiple respiratory infections, and chronic lung disease. Therefore, prenatal investigation should include DNA analysis of the DF508 mutation on chromosome 7, or repetitive sweat chloride tests in the neonatal period.

Frequent sonographic observation permits the evaluation of the amount of fetal ascites, the evolution of intra-abdominal calcification, and the restoration of bowel peristalsis. Exclusion of chromosomal malformations, congenital infections, and cystic fibrosis is an essential element in the further management [5].

If MP resolves spontaneously, there is no need for induction of labor. Postnatal observation of bowel peristalsis and a plain radiography of abdomen should alert the pediatrician. Surgical exploration might be necessary.

In case of progressive deterioration of fetal condition with increasing amount of ascites, preterm delivery can be considered in a tertiary care center. Postnatal surgery will be required immediately. Cesarean section has not been proven to improve neonatal outcome [5].

Conclusion

MP is a chemical peritonitis caused by fetal intestinal perforation which occurs mostly in utero. Its incidence is extremely rare, but serious neonatal morbidity or even mortality can occur if the diagnosis is delayed. Prenatal diagnosis is essential in prompting early postnatal surgical intervention, and also, improving neonatal outcome.

Acknowledgement

None.

List of abbreviations

LDA	Left anterior descending (LAD, interventricular) artery
MP	Meconium peritonitis
NICU	Neonatal intensive care unit
PDA	Patent Ductus Arteriosus

Consent for publication

Informed consent was obtained from the parents of the patient to publish this case in a medical journal.

Ethical approval

Ethical approval is not required at our institution for publishing a case report in a medical journal.

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Summary of the case

Patient (gender, age)	1	5 days, male
Final diagnosis	2	Spontaneous MP
Symptoms	3	Abdominal distention, increased bilirubin
Medications	4	None
Clinical procedure	5	Neonatal surgery was required to treat ileal atresia.
Specialty	6	Radiology, Pediatric surgery, Emergency Medicine