Meconium pseudocyst presenting as massive ascites in a new-born

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ABSTRACT

Meconium pseudocyst (MPC) is a rare but well-known surgical condition due to prenatal bowel perforation. A case of MPC secondary to prenatal bowel perforation is presented. Massive ascites requiring peritoneal drainage and disappearance of prenatal intraperitoneal calcifications have not been previously reported in MPC. MPC may present at birth with large ascites requiring peritoneal drainage to establish breathing and ventilation. Absence of prenatal intra-abdominal calcifications does not rule out MPC.

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Case presentation

Meconium pseudocyst (MPC) is a well-known condition that occurs secondary to bowel perforation taking place antenatally. Below, we describe a previously unpublished atypical presentation.

A 34-year-old, Caucasian woman was admitted at 32 + 5 weeks of gestation with a 2 day history of reduced fetal movement. She previously had a miscarriage at 6 weeks of gestation. She was otherwise in good health prior to and during this pregnancy. Antenatal scan at 32 + 1 weeks of gestation showed abnormal findings of polyhydramnios, foetal ascites along with hyper-echoic mass suggesting calcifications of unknown cause (Fig. 1). Estimated foetal weight on the scan was 2068 g (approximately 30th centile). She received 1 full course of steroids. Cardiotocographic examination showed pathologic tracing due to unprovoked decelerations and intermittent unprovoked bradycardia necessitating an emergency caesarian section.

A female infant weighing 1859 g (50th centile) was delivered. She was born in poor condition due to massive abdominal distension (Fig. 2) requiring therapeutic abdominal paracentesis (150-200 ml) to achieve effective lung volume and ventilation. She was initially given bag and mask ventilation and thereafter, intubated and mechanically ventilated. The Apgar scores were 4, 7, and 9 at 1, 5, and 10 minutes respectively.

The infant was then transferred to neonatal intensive care unit on synchronized intermittent mandatory ventilation for ongoing management.

General examination did not show any dysmorphic features. Notably, the abdomen showed generalized tense distension with extensive abdominal wall erythema (Fig. 2). Differential diagnosis included intra-abdominal tumor of vascular origin due to blanching abdominal wall erythema. Abdominal ultrasound confirmed ascites but excluded vascular malformation. Urgent X-ray abdomen (Fig. 3) showed ascites but absence of intra-abdominal calcifications noted antenatally. This led to delay in diagnosis of MPC. Repeat abdominal ultrasound raised the possibility of a walled intra-abdominal abscess necessitating surgical review and intervention.

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Fig. 1 – Antenatal scan at 32 weeks of gestation showing fetal ascites and hyper echoic mass suggesting calcifications.

Fig. 2 – Infant showing massive ascites and intense anterior abdominal wall erythema (photo taken after abdominal paracentesis).

Fig. 3 – Postnatal X-ray, A-P view of the abdominal cavity confirming ascites. There was no evidence of calcifications.
Laparotomy on day 2 of life revealed a MPC with extensive interloop adhesions (Fig. 4). There was an area of ileal perforation proximally, but this had sealed. There was no evidence of compression of blood or lymph flow. Ileostomy was created to facilitate repair and allow nutritional rehabilitation prior to stoma reversal. There was no reoccurrence of ascites postoperatively.

Extended genetic screen was negative for cystic fibrosis. Microarray was reported to be normal. Ascitic fluid analysis was normal.

The infant was discharged home after 32 days of neonatal care taking a combination of breast milk and formula milk. Her stoma was reversed successfully at 8 months of age. Recent outpatient review at 18 months of age showed that she was thriving at 25th centile with normal bowel habits and developmental milestones.

Discussion

Diagnosis of meconium peritonitis can be made based on antenatal scans, clinical findings, and postnatal imaging.

Even though the presence of ascites in meconium peritonitis is well described, our case is first to report that it can be to such an extent so as to warrant peritoneal drainage to establish effective lung volumes and ventilation at birth.

Imaging can reveal the presence of intraperitoneal calcifications in the newborn. The extruded meconium may or may not be calcified, but when present, is pathognomonic of meconium peritonitis. Twelve cases of meconium peritonitis were reviewed in a report. They found that intraperitoneal calcifications were present in 100% of patients without cystic fibrosis and in 60% of the patients with cystic fibrosis [1]. It is postulated that pancreatic enzymes, which are low in concentration in 80% of patients with cystic fibrosis, may be necessary for the calcifications to occur [2].

Our case is unusual as antenatal calcifications were not confirmed in postnatal imaging. In the absence of cystic fibrosis, as in our case, the exact pathophysiology of the disappearance of intra-abdominal calcifications remains unknown. We can only postulate that the severity of ascites masked the underlying hyper-echoic signals on postnatal ultrasound and X-ray.

Drainage of a large pseudocyst to enable adequate ventilation was reported in the presence of pulmonary hypoplasia [3]. Our case is unique due to the absence of pulmonary hypoplasia and drainage of ascites to establish ventilation.

Bowel ischemia and perforation may occur secondary to vasculitis by intrauterine infections such as Toxoplasmosis, Cytomegalovirus (CMV), or Herpes virus. However, the mother’s antenatal serology was negative for Toxoplasmosis and CMV. The infant did not show signs indicative of such infections and therefore a TORCH screen was not sent.

The presence of intense erythema of the anterior abdominal wall has also not been reported in MPC previously. We postulate that the presence of such erythema in this case was reflective of the intense inflammatory reaction of meconium peritonitis.

The case highlights the significance of recognition of these atypical presentations of MPC, namely, disappearance of antenatal intra-abdominal calcifications, massive ascites requiring peritoneal drainage, and intense abdominal wall erythema.

Author contributions

Suhail Ahmed Khan contributed to conception and design of article, acquisition of data, and drafting of article. Manjiri Khare, as consultant obstetrician and named foetal medicine specialist, provided antenatal ultrasound images and contributed to drafting of article. Haitham Dagash, as consultant paediatric surgeon, operated on the child, provided intraoperative pictures, and contributed to the drafting of the article. Venkatesh Kairamkonda as neonatal consultant, contributed to the write up.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2018.10.013.

REFERENCES

[1] Finkel LI, Slovis TL. Meconium peritonitis, intraperitoneal calcifications and cystic fibrosis. Pediatr Radiol 1982;12(2):92–3.
[2] Valladares E, Rodriguez D, Vela A, Cabré S, Lailla JM. Meconium pseudocyst secondary to ileum volvulus perforation without peritoneal calcification: a case report. J Med Case Rep 2010;4(1):292.
[3] Nam SH, Kim SC, Kim DY, Kim AR, Kim KS, Pi SY. Experience with meconium peritonitis. J Pediatr Surg 2007;42(11):1822–5.