CASE REPORT

Imaging findings of meconium peritonitis

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Introduction

Meconium peritonitis results from intrauterine gastrointestinal perforation, and can occur as early as the second trimester.[1] Meconium extrudes into the peritoneal cavity, inciting an intense fibroplastic reaction that results in intra-abdominal calcifications.[2] It is a rare condition occurring in 1 in 35 000 pregnant women.[3]

The clinical and radiological manifestations depend on whether the bowel perforation seals off *in utero* in the neonatal period or remains patent.[3] Accordingly, the radiological spectra range from the incidental demonstration of diffuse intra-abdominal calcifications to meconium ascites (free meconium in the peritoneal cavity), meconium pseudocysts (walled-off meconium concentrations), and meconium hydrocoeles. Meconium has also been reported in the thoracic cavity (via diaphragmatic hernias) and in the pelvic soft tissues.[4]

Antenatal ultrasound allows early detection of the condition, demonstrating free fluid, hydrocoeles and echogenic foci representing intraperitoneal calcifications.[5] In the newborn, plain abdominal radiographs demonstrating calcifications and/or ascites are sufficient for diagnosis.[1] Postnatal ultrasound is reserved for atypical presentations and can exclude intra-abdominal masses.[1] Although computed tomography (CT) was used as an ancillary tool in the case report described below, it is unnecessary, thus negating the need for radiation exposure and expenditure of time.

The imaging findings in a newborn with an ongoing bowel perforation, resulting in gross meconium ascites, is presented below. This case report serves also to describe the pathogenesis, radiological spectra, role of imaging and causes of meconium peritonitis.

Case report

A newborn presented with increased abdominal girth and a clinical suspicion of ascites. The mother had been a late booking at the antenatal clinic and was sent to the ultrasound department for an estimation of gestational dates. The fetus' measurements were estimated at 31 weeks and an incidental finding of a 'hydrocoele' was noted. Unfortunately, for reasons unknown to us, the hydrocoele was not investigated further and the next presentation was at delivery.

A 3 kg boy was born at 37 weeks' gestation following an uncomplicated delivery with good Apgar scores. On examination, the newborn had a markedly distended abdomen and scrotum with an abdominal girth of 40 cm. The abdomen was described as 'shiny' with poor bowel sounds. Diaphragmatic motion appeared restricted as a result of the increased abdominal pressure, resulting in mild respiratory distress. An abdominal radiograph, ultrasound and CT scan were performed by the on-call registrar and were reviewed by the consultant with the suggested diagnosis of meconium peritonitis.

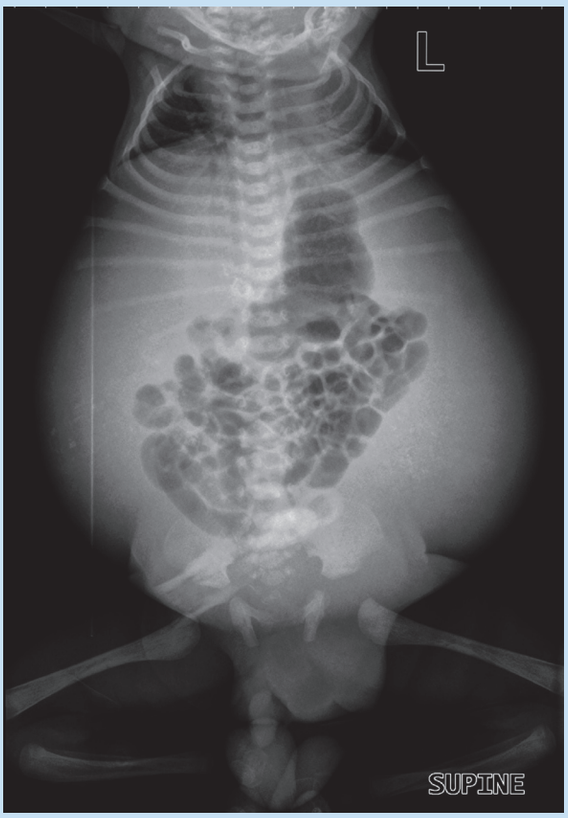

Fig. 1. Supine abdominal X-ray revealing a massively distended abdomen with poor lung capacities. The bulging flanks and central floating bowel loops indicate ascites. Note the intra-abdominal calcifications.

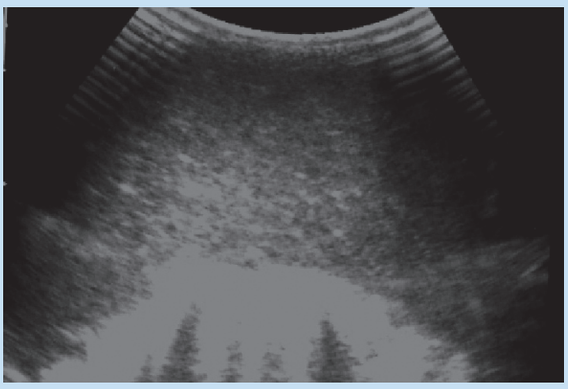

Fig. 2. Transverse section cut on abdominal ultrasound depicting echogenic material with a 'snowstorm' appearance.

The abdominal radiograph (Fig. 1) revealed a massively enlarged abdomen with elevated hemidiaphragms, resulting in small lung capacities. The central floating bowel loops, bulging flanks and loss of soft tis-

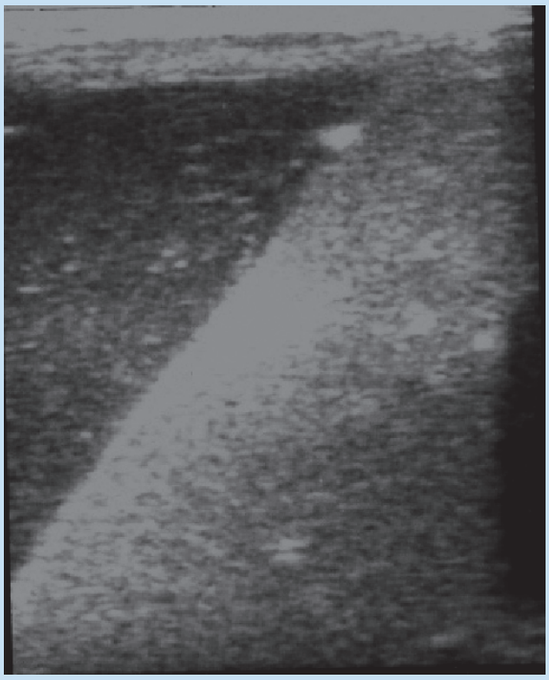

Fig. 3. High-resolution abdominal sonar showing echogenic foci in keeping with calcifications at the periphery of organs.

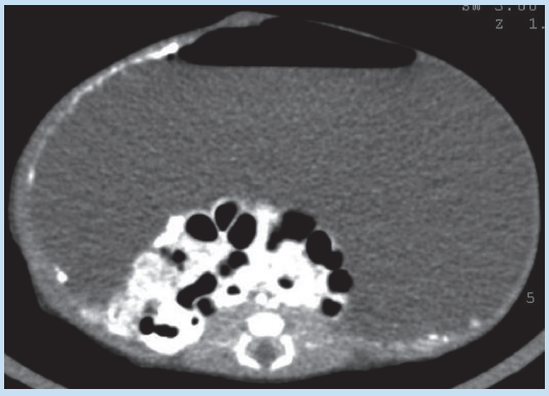

Fig. 4. Axial CT revealing anterior intraperitoneal free air. The meconium ascites causes posterior displacement of bowel loops. Note the peripheral calcifications.

sue planes and properitoneal lines were indicative of ascites. There were intraperitoneal calcifications that appeared to be irregular and scattered through the abdomen. No free air was visualised.

Ultrasound (Figs 2 and 3) demonstrated complex fluid. This highly echogenic material spread throughout the abdomen to produce a 'snowstorm' appearance. Clumped echogenic foci depicting the intraperitoneal calcifications were scattered around bowel loops. Dense calcifications with shadowing were demonstrated in the scrotum.

A CT scan (Fig. 4) confirmed the complex fluid extending into the scrotum. Calcifications were seen at the periphery of the fluid and interspersed among posteriorly displaced bowel loops. Bowel loops appeared dilated. Central free air (not visualised on plain films) was demonstrated on CT.

Air usually enters the small bowel within 3 hours of birth.[1] As CT was the last investigation performed, free air penetrated the ongoing perforation and was visible on CT but not on the plain abdominal radiograph which was done immediately after birth.

The final diagnosis was meconium ascites with an ongoing perforation. At exploratory laparotomy, an ileal perforation was demonstrated. Copious amounts of meconium were removed. A portion of necrotic bowel was removed at ileostomy. No identifiable causes were found.

Discussion

Meconium peritonitis results from prenatal intestinal perforation nearly always involving the small bowel.[5] Meconium and digestive enzymes are extruded into the peritoneal cavity, inciting an intense chemical peritonitis and secondary inflammatory response.[5] Within days, giant cells and histiocytes surround the meconium, resulting in foreign body granulomas.[5] These often calcify, resulting in the characteristic intraperitoneal deposits identified prior to birth via antenatal ultrasound and after birth with plain radiography.[5] The calcifications appear amorphous and irregular on plain radiographs.[1] Antenatal and postnatal ultrasound depicts these calcifications as highly echogenic linear or clumped foci exhibiting posterior acoustic shadowing.[5] Ancillary features on antenatal sonar include polyhydramnios, fetal ascites and bowel dilatation.[5]

Calcific plaques may be the only finding on incidental radiographs and antenatal ultrasound when perforations are small and seal off *in utero*.[3] Eventually, most of these disappear; it is rare to encounter an older child with residual calcifications.[1]

Hydrocoeles may also occur in cases of spontaneous perforation closure *in utero*. They result when meconium migrates into the fetal scrotum via a patent processus vaginalis. Meconium interacts with the tunica vaginalis to incite an intense inflammatory reaction resulting in local calcifications.[1] Meconium pseudocysts may also present in this category. They present as walled-off concentrations that display a mass effect mimicking abdominal tumours.[1]

Meconium ascites and pseudocysts result from perforations that seal off/remain patent after birth. Meconium peritonitis, as in our case, features bulging flanks and central bowel loops on plain radiographs. Ante/postnatal sonar demonstrates highly echogenic material spreading through the abdomen and around bowel loops.[2] This results in multiple speckled echoes and has been described as a 'snowstorm' appearance.[2] Pseudocysts may feature heterogenous collections as a result of debris and calcifications.[2] Ongoing perforations display free air or encysted air pockets.

The radiological categorisation mentioned above is essential for management. Surgical intervention, for example, is required to eliminate obstructing meconium, for respiratory compromise, and ongoing perforations. Prior to delivery, meconium is sterile; however, bacterial contamination can occur after delivery, and severe meconium ascites should therefore be cleared.

The causes of meconium peritonitis include bowel obstruction from strictures, small bowel atresia, volvulus, intussusception, and meconium ileus from cystic fibrosis (CF).[2] The cause is sometimes idiopathic, as in this case.[2]

Foster *et al*.[5] found that meconium peritonitis diagnosed *in utero* was associated with causes other than CF. Interestingly, this study also found that the presence of abdominal calcifications was associated with causes of meconium peritonitis other than CF. There are different proposals to explain this: some feel that pancreatic enzymes that are deficient in 80% of patients with CF may be necessary for calcifications to occur; others speculate that the abnormal, thick and tenacious meconium that is characteristic of CF cannot spill freely into the peritoneal cavity, resulting in a poor inflammatory response, and hence poor calcifications.

Most babies with meconium peritonitis do well. At one time, this condition carried a poor prognosis, but this has changed with advances in surgical techniques and postoperative care, with recent literature indicating a 100% survival.[7]

Conclusion

The division of meconium peritonitis into groups depending on whether the perforation occurred *in utero* or neonatally is necessary for determining the type of management. Surgical management is essential in conditions of respiratory compromise, meconium ascites, and ongoing perforation.

Improvements in antenatal clinics in South Africa are leading to earlier detection by antenatal ultrasound. Earlier detection will alert the neonatologist, obstetrician and paediatric surgeon to possible complications such as dystocia, respiratory distress, bacterial peritonitis, acid-base disturbances and septic shock.[6]

In the postnatal period, plain radiographs are usually sufficient for diagnosis.[1] Ultrasound is indicated for atypical presentations and to exclude other causes. The use of CT, while adhering to ALARA principles of paediatric radiology, is not indicated, though it proved useful in demonstrating free air in this case. It is also useful for the radiologist and sonographer to know that most antenatally diagnosed cases of meconium peritonitis are unrelated to CF, and that patients with CF rarely present with intraperitoneal calcifications.[5]

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