

Meconium Peritonitis: Prenatal Diagnosis and Postnatal Management - A Case Report

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Summary

The management of a case of antenatally diagnosed meconium peritonitis (MP) due to intrauterine intestinal perforation secondary to bowel atresia is reported. The literature is reviewed with reference to the significance and outcome of antenatally diagnosed MP.

Key Words: Meconium peritonitis, Fetal ascites, Bowel atresia, Intraabdominal calcification

Introduction

Intestinal perforation can occur in the fetus due to various causes. This results in meconium peritonitis (MP). Although, it is a rare condition with an incidence of about 1: 35,000 live births, the antenatal diagnosis of meconium peritonitis is well established in the developed countries. However, in Malaysia, the antenatal diagnosis of fetal anomalies, particularly, gut anomalies are made with increasing frequency only in recent times. The issues that need to be addressed in the antenatal counselling of the parents, especially in relation to the local context become important. In meconium peritonitis it includes the chances of the baby requiring postnatal surgical intervention, the possibility of meconium ileus due to cystic fibrosis as the etiological factor and the prognosis.

Case Report

A 43-year-old Indian woman, gravida 2, para 1, was referred at 36 weeks of gestation with polyhydramnios and suspected fetal ascites. A detailed antenatal ultrasound examination showed polyhydramnios, fetal

ascites with peritoneal calcification and bilateral hydrocele with scrotal calcification (Fig. 1). Dilated bowel loops were also noted. A diagnosis of meconium peritonitis was made. The parents were counseled jointly by the obstetrician and paediatric surgeon, regarding the prognosis and the possible need for neonatal surgical intervention. The pregnancy was allowed to proceed to term. The labour was uneventful and a male baby weighing 3,200 grams with an Apgar score of 9/10 was delivered vaginally. Examination of the neonate revealed a tense, distended abdomen and large bilateral hydroceles, which were not transilluminant. The infant did not pass meconium and rectal washouts failed to relieve the bowel obstruction. Plain abdominal radiograph showed ascites, dilated bowel loops and multiple areas of peritoneal and scrotal calcification (Fig. 2). Laparotomy was performed on the second postnatal day for unrelieved bowel obstruction. About 250ml of meconium stained ascitic fluid was drained. There was an obstructive lesion in the mid-ileum, but the exact cause of the obstruction could not be determined due to densely adherent bowel loops. An ileostomy was performed proximal to the obstructing lesion. Post-operative period was complicated by prolonged

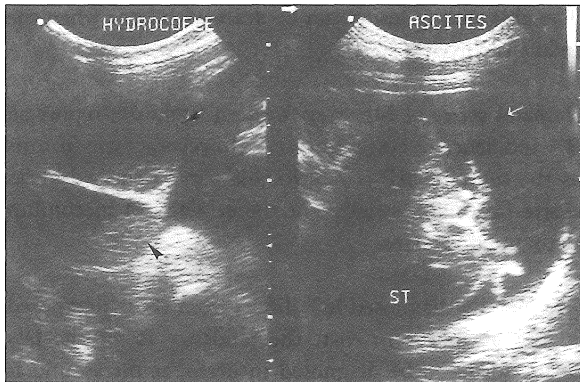


Fig. 1: Prenatal ultrasound showing ascites (arrow) and bilateral hydroceles (arrow heads).

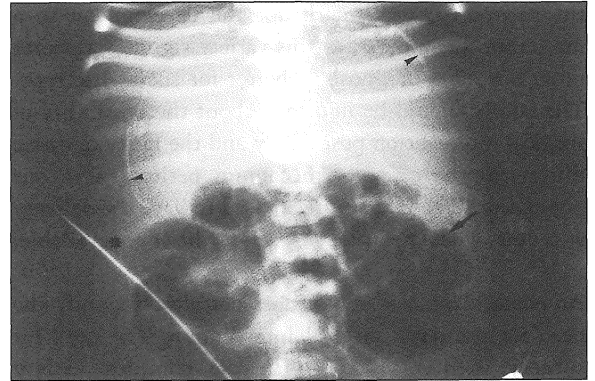


Fig. 2. Plain radiograph of the abdomen showing ascites (:), dilated bowel loops (arrow) and multiple areas of peritoneal calcification (arrow heads).

conjugated hyperbilirubinemia. The possibility of inspissated bile syndrome secondary to cystic fibrosis was suspected. Sweat test for cystic fibrosis could not be performed due to technical reasons. However, the jaundice resolved gradually. Barium enema showed the presence of a patent microcolon. Laparotomy was performed six weeks later and the obstructing lesion in the ileum was resected and bowel continuity was established by end to end anastomosis. The obstructing lesion was a septal atresia of the ileum. The baby recovered uneventfully and remained well. The final diagnosis was meconium peritonitis due to intrauterine ileal perforation secondary to an ileal atresia.

Discussion

Meconium peritonitis is caused by antenatal perforation of the bowel, which may be secondary to bowel obstruction due to atresia (as in this case), meconium ileus, Hirschsprung's disease, intrauterine intussusception, strangulation of fetal hernia and volvulus¹. It can also occur in the absence of bowel obstruction due to intrauterine vascular accident, perforation of the appendix or Meckel's diverticulum¹. Parvovirus B 19 Infection has been reported to produce vascular inflammation leading to fetal bowel perforation². The meconium that leaks out induces a sterile chemical peritonitis and ascites. Organisation of

the meconium produces dense fibrous adhesions and calcification as seen in our case.

Meconium peritonitis can be of the ascitic type (as in our patient), generalized adhesive type or it can present as a meconium pseudocyst. MP can be of the simple or complex variety, depending on the absence or presence of dilated loops of bowel respectively³.

Antenatally various sonographic findings including bowel dilatation, ascites and polyhydramnios have been associated with MP¹. However, the presence of peritoneal calcification is the most important and consistent of all the findings for the diagnosis³. Abdominal calcification in the fetus can represent intraluminal, solid organ or tumor calcification, but abdominal calcification when present along with scrotal calcification is more specific for the antenatal diagnosis of meconium peritonitis³. The initial antenatal diagnosis in our patient was fetal ascites; however, the majority of cases of fetal ascites are due to other causes. The presence of ascites alone is not sufficient to diagnose MP³. The diagnosis in our case was clinched due to the presence of calcification and ascites. With reference to antenatal counselling, the parents should be informed that pregnancy could be allowed to go to term. The delivery can be vaginal except for obstetrical indications. There may be a need for repeated scans to assess the progress of

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the peritonitis, particularly for the development of a meconium pseudocyst, as this variety signifies a poorer prognosis. The prognosis is better for the ascitic type. The family should be informed about the possibility of surgical intervention postnatally and the rarity of cystic fibrosis as the cause of meconium peritonitis in our population. The parents are advised to have the delivery at centers where neonatal surgical facilities are available.

Antenatal diagnosis of MP does not always signify the need for postnatal surgical intervention. The need for surgery is more likely with the complex variety associated with dilated bowel loops³. Literature review suggests that the need for postnatal surgical intervention varies from 22% to 80%³. In a report of nine cases of MP detected antenatally, five were simple and four were complex. None of the 5 cases of the simple variety required surgical intervention, whilst only two patients (22%) of the complex type required surgery due to unrelieved bowel obstruction³. Sometimes, it may be necessary to modify the treatment in the presence of dense adhesions between the bowel loops by performing a temporary diverting ostomy proximal to the obstruction. This was done in the case reported here.

The obstructing lesion can be dealt with later when the infant is thriving well.

Prenatal series of MP from western literature report an 8% incidence of cystic fibrosis³. However, cystic fibrosis as the cause of MP is rare in this part of the world, as cystic fibrosis is usually not the etiology of meconium ileus among Asians.

Antenatally diagnosed MP differs from those diagnosed in the newborns in that, the tendency is for the less severe forms to be diagnosed in utero, as ultrasound is more sensitive in detecting calcifications. Many of the cases diagnosed in utero may remain asymptomatic postnatally. MP diagnosed in the newborns represents the more severe form of the disease with associated bowel obstruction and peritonitis. Therefore, the prognosis of MP diagnosed antenatally is obviously better than that diagnosed in the neonate (as seen in this report)³. Survival rate of 90% is reported for antenatally diagnosed MP whereas, overall, the reported mortality is about 55%³. However, with the advent of neonatal intensive care and total parenteral nutrition, improved survival is reported. This is illustrated in this case.

References

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