Case Report

Meconium Peritonitis – Two case reports

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Meconium peritonitis is a chemical peritonitis usually resulting from antenatal bowel rupture. We present two cases of meconium peritonitis diagnosed ultrasonographically.

Case 1

Mrs. S, a 23 year old primigravida married for one year had 5 months amenorrhea. Family history was negative for congenital anomalies. Her blood group was O+ve and serum antibody screen for TORCH group was negative.

Routine ultrasonography (USG) done in the second trimester, showed a single live fetus of 25 weeks gestational age with a fundal and anterior placenta. There was evidence of a dense focal area of calcification in the anterior aspect of right lower abdomen measuring 19 x 16 mm suggestive of a meconium pseudocyst. Few scattered calcified densities were seen in the abdomen. There was no suggestion of cerebral or hepatic calcification and bowel loops were not dilated. Subsequent USG done in the third trimester showed persistence of the focal area of dense intra abdominal calcification with few scattered calcific densities as seen in the previous scan.

Labor started spontaneously at 37 weeks. She delivered a baby girl weighing 2.3 kg with normal Apgar score. After the baby passed meconium spontaneously she was given feed, which was well tolerated. USG of the abdomen revealed a normal scan. X-ray of the abdomen confirmed intra-abdominal calcification consistent with meconium peritonitis. The baby was discharged on day 7 after delivery. She was fine at 6 weeks follow up and was not seen thereafter.

Case 2

Mrs. C, a 28 year old primigravida, came with history of 6 months amenorrhea.

Routine USG done in the 2nd trimester revealed a single live fetus corresponding to 26 weeks gestational age with posterior placenta. Fetal abdomen revealed peritoneal calcifications mostly in right upper quadrant seen as linear specks along the surface of the liver and colon. However, there was no fetal ascitis or focal space occupying lesion in the fetal abdomen. Fetal bowel loops were not dilated. Follow up USG revealed similar findings.

Pregnancy was supervised and cesarean section was done for fetal distress. Post-delivery x-ray and USG
confirmed the presence of intra-abdominal calcifications. The baby girl accepted feeds well and was asymptomatic. The mother and the baby were discharged on the 7th postnatal day. The baby was fine till 1½ years of age and is fine till date.

Discussion

Meconium peritonitis is a sterile chemical peritonitis, resulting from intra-uterine bowel perforation. The most common bowel disorder which lead to meconium peritonitis in utero are those resulting in bowel obstruction and perforation such as small bowel atresia, volvulus and meconium ileus. There is usually an intense inflammation from meconium peritonitis, which may incite calcification along the surface of bowel or peritoneum. In time the inflammatory response may seal the perforation or alternatively may form a pseudocyst or wall of meconium. This pseudocyst has a thin and often calcified wall.

Prenatal ultrasound findings include ascites, intra-abdominal mass, bowel dilatation and development of intra-abdominal calcification. In utero identification of meconium peritonitis by USG has important implications for obstetric and neonatal care. It is associated with a 20-30% incidence of prematurity and a 10-20% incidence of polyhydramnios. Dystocia secondary to massive ascites or abdominal distension with meconium although a rare occurrence has been reported. Postnatal outcome for such infants depend on the etiology for bowel rupture and the underlying disease.

She et al studied 115 cases of meconium peritonitis over 20 years. They found that in all the cases there was intra-abdominal calcification. There was no case of fibrocystic disease. Forty-one cases had neonatal obstruction. Mortality rate in their study was 42.6%.

Kamata et al evaluated 20 fetuses with meconium peritonitis. Of these 25% had massive ascites, 20% had giant pseudocyst and the remaining had abdominal calcifications or a small pseudocyst. Dilatation of the bowel was seen in 50% of the fetuses. Eighteen fetuses had intestinal atresia and two had fecal obstruction or ileus. Mortality rate was 20%, which is lower than that reported by She et al.

Dirkes et al reviewed their experience of nine cases with meconium peritonitis diagnosed in utero. They had five cases of simple meconium peritonitis that had an uneventful postnatal period. The other four cases had complex peritonitis with dilated bowel loops of which two cases also had meconium cyst. The two cases (22%, 2/9) required postnatal surgery.

Review of literature shows that if findings consistent with meconium peritonitis are observed in utero, the pediatrician should be alerted to look for early signs of bowel obstruction in the neonate. Surgery performed within 24 hours in such newborns with bowel obstruction may improve their outcome. However, asymptomatic infants may develop bowel obstruction secondary to adhesions later in childhood.

In our study both the infants were asymptomatic and had an uneventful postnatal period. Probably the site of perforation got sealed without any sequelae, indicating self limiting process. Such spontaneous and favorable outcome has been described by some authors.

Meconium peritonitis may occur without any underlying cause. The underlying cause may be innocuous and intervention may not be required. However, such cases should be closely monitored for any need for an urgent surgical intervention even later in childhood.

References