

Meconium Peritonitis

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ABSTRACT

In the second and third trimesters of pregnancy, hyperechogenic regions in the fetal abdomen can be used to identify meconium peritonitis (MP). According to studies on neonates, MP affects 1 in every 35,000 live births. The presence of an intrauterine ileal rupture causes a sterile chemical reaction that causes meconium peritonitis. The creation of fluid results in ascites, fibrosis, calcification, and cyst formation as a secondary inflammatory reaction. This process has four different outcomes, culminating in four types of meconium peritonitis: fibre adhesive, cystic, diffuse (diffuse), and healing. Below, we describe one case of fetal meconium peritonitis.

Keywords: Ileal perforation, inflammatory, meconium peritonitis.

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I. INTRODUCTION

An intrauterine intestinal perforation causes meconium peritonitis (MP), defined as an aseptic chemical inflammation. Intestinal atresia, volvulus, intussusceptions, meconium plug syndrome, inguinal hernia, Hirschsprung's disease, and meconium ileus are related to cystic fibrosis, the latter of which is uncommon in Asian populations [1], [2]. Abdominal calcifications, ascites, polyhydramnios, meconium pseudocysts, echogenic masses, and a dilated bowel or intestinal obstruction are all diagnostic characteristics of MP. A growing number of prenatal

ultrasonography diagnoses have been recorded recently—the unique characteristics of MP influence clinical outcomes and therapy.

II. CASE REPORT

A 35-year-old woman, her second pregnancy, 24-25 weeks, was referred by the SpOG physician with fetal hydrops for further examination and management. Previous history of delivery by cesarean section due to antepartum bleeding due to placenta previa. LMP: 09-09-2006. When referred, the patient also complained that her stomach was

larger than her gestational age and felt tense. No medical abnormalities were found.

Blood type B, Rh-positive. Ultrasound examination found a single male fetus, alive, breech presentation, with fetometry: BPD: 6.62 cm (25 weeks + 3 days); AC: 25.93 cm (30 weeks+1 day); FL: 3.74 cm (21 weeks+6 days) with mean: 26 weeks+3 days; EFW: 1224 grams. Ascites are seen in fetuses with echogenic bowel and polyhydramnios. No other anatomic abnormalities were seen. No fluid accumulation was seen in other body cavities. Placenta posteriorly with a thickness of 3.99 cm in the middle. Doppler examination of the umbilical artery S/D ratio: 3.24 with a pulsatile index: 1.16. Moreover, PSV (peak systolic velocity) on the MCA (middle cerebral artery) is 27.9 cm/second (<1.5 MoM), which means normal (no anaemia in the fetus). TORCH serological examination (Toxoplasmosis IgG(+), IgM(-); Rubella IgG(+), IgM(-); Cytomegalovirus IgG(+), IgM (-); Herpesvirus 1 IgG(-), IgM (-).

Amniocentesis was used in this case to check the karyotype, which revealed 46 XY chromosomes. Serial amnioreduction was also done because of polyhydramnios, and paracentesis was done because the fetus had significant ascites, which could have put too much pressure on the diaphragm. The fluid from the paracentesis is tested for bacterial growth. The results were completely sterile. When the gestational age exceeds 34 weeks, the amount of ascitic fluid decreases and the amniotic volume returns to normal.

Subsequently, weekly serial monitoring was performed by ultrasound. Spontaneous labor at 40-41 weeks of gestation, labor was performed by vacuum extraction because the patient was ineffective in straining. The baby boy was born, 3300 grams, birth length: 52 cm, head circumference: 35 cm, chest circumference: 34 cm, belly circumference: 33 cm, fit, APGAR score: 1': 8 and 5:10. Hemoglobin: 16,9 gram %, albumin: 4 gram %. Normal urination, normal bowel movements, good breastfeeding. An abdominal x-ray is done, showing air filling the upper intestine into the lower intestine. A CT scan is performed to look for signs of calcification in the brain associated with cytomegalovirus infection. No intracranial calcification was found, and hyperdense mass was found in the occipital, sulcus and cerebral gyrus with normal impression. A peer evaluation from the Department of Neurosurgery ended with an intracranial occipital haemorrhage with conservative management. The fetus's condition at the time of writing the report was in good condition.

III. DISCUSSION

According to animal research, calculations are only apparent after 8 days after meconium is discharged into the peritoneal cavity. Ileal ischemic lesions coupled with mechanical obstruction (atresia, volvulus, intussusception, congenital bands, Meckel's diverticulum, and internal hernia) are the most prevalent cause of meconium peritonitis. In 50% of cases with peritonitis meconium, this is the reason. A viral infection can also induce meconium peritonitis (cytomegalovirus or parvovirus B19). Meconium ileus is also the cause of meconium peritonitis in less than 25% of instances [3]-[5].

Intra-abdominal calcification, ascites, polyhydramnios,

and intestinal dilatation were identified on prenatal sonography to support the diagnosis of meconium peritonitis. The research documented intra-abdominal calcification in 86 percent, ascites in 64 percent, polyhydramnios in 71 percent, and indications of bowel obstruction in 46 percent. On prenatal ultrasound, three forms of meconium peritonitis can be identified: cystic (meconium pseudocyst), diffuse, and fibrous adhesive [6], [7].

The most common sonographic manifestation of meconium peritonitis is a hyperechoic mass called a meconium pseudocyst. Ascites are another prevalent condition. Polyhydramnios, fetal ascites, and certain intra-abdominal calcifications are common symptoms of diffuse meconium peritonitis. Due to oedema, the fetal abdominal wall may seem thicker. The presence of substantial calcium deposits in the peritoneum can seal off lesions in the intestines, causing fibroadhesive meconium peritonitis. The most common sonographic manifestation of meconium peritonitis is a hyperechoic mass called a meconium pseudocyst. Ascites are another prevalent condition. Polyhydramnios, fetal ascites, and certain intra-abdominal calcifications are common symptoms of diffuse meconium peritonitis. Due to oedema, the fetal abdominal wall may seem thicker. The presence of substantial calcium deposits in the peritoneum can seal off lesions in the intestines, causing fibroadhesive meconium peritonitis [8].

At 34 weeks of pregnancy, the ascites began to decrease independently. Is this related to the digestive tract's maturity, where intestinal leaks are repaired? The volume of amniotic fluid returns to normal as ascites reduce. Is fetal paracentesis (fetal abdominocentesis) beneficial to improving the prognosis in this circumstance? Furthermore, ascites in the fetus will go away on their own without treatment? This is an issue that needs to be addressed.

In this case, the examination for the detection of parvovirus B19 is not possible because it is not permitted in Indonesia. At the age of 20 days, a healthy infant appears.

IV. CONCLUSION

We do not know whether our case's MP was caused by a genetic defect or by chance. To discover the specific cause of MP, more investigations and case reports with genetic tests are needed. To better understand the genesis and clinical course of MP, we give a case report.

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