MECONIUM PERITONITIS: A RARE TREATABLE CAUSE OF NON-IMMUNE HYDROPS

Dr.Loukya Kodumuri, Dr.Arpana Verma, Dr.Sarita Rajbhar, Dr.Sarita Agrawal
Department of Obstetrics and Gynaecology
All India Institute of Medical Sciences, Raipur, C.G

INTRODUCTION:

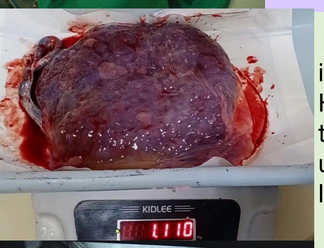
Meconium Peritonitis is defined as an aseptic localised or generalised chemical peritonitis which results from perforation of the gut in utero with reported incidence of 1 in 35,000 live births. Although, no definitive reason is found in half of the cases, possible causes include bowel perforation as a result of obstruction such as intestinal atresia, meconium plugs, volvulus, internal hernia, mesenteric ischemia, Hirschsprung's disease, torsion of a fallopian tube cyst, and cystic fibrosis. Meconium is clearly a strong pro-inflammatory mediator. Secondary inflammatory response results in production of fluid(ascites), fibrosis, calcification, and sometimes cyst formation. It can be classified into 3 types as follows: generalised, cystic, and fibro adhesive types. Successful outcome with conservative management has been seen in limited number of cases, however, surgery is imperative when signs and symptoms of intestinal obstruction are present. With the evolution of neonatal care, the prognosis of meconium peritonitis has improved much. Favourable results have been seen when the condition was detected in utero rather than a neonatal diagnosis.

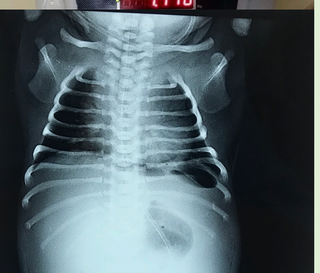
CASE DESCRIPTION:

A 32 year, G5P2L1D1(IUD)A2, non consanguineous marriage, was referred to our hospital at 33 weeks 2 days of gestation in view of isolated fetal ascites, diagnosed on antenatal scan at 32 weeks. Mother's blood group was "A" positive, ruling out the possibility of incompatibility. First trimester aneuploidy screen was indicative of low risk. Second trimester serum screening (quad test) and targeted imaging for fetal anomalies were normal. Immuno hematological work up including indirect coombs test, irregular antibody screening by 3 cell panel was negative. There was no history of congenital anomalies in family or in her previous babies. Screening done for syphilis, cytomegalovirus (CMV), parvovirus B19 and toxoplasmosis, were normal. Fetal echo was done, which showed structurally normal heart with mild pericardial effusion and echogenic foci in both ventricles. One week later, Repeat ultrasound was done which showed moderate fetal ascites, fluid collection in infra diaphragmatic space with echogenic bowel loops floating within it, also few areas of calcification in the bowel loops were noted with prominent Inferior vena cava(IVC), there was also associated polyhydramnios. Doppler middle cerebral artery peak systolic velocity was <1.5 multiples of median which rules out the probability of fetal anaemia. There was no evidence of hydrocephalus, hydrothorax or skin edema. The probable diagnosis of MP was made. Neonatologist and pediatric surgeon's opinion were taken regarding fetal prognosis and further management. She was planned for conservative management with an aim to prolong the pregnancy till 37 weeks. Dexamethasone was given for fetal lung maturation. Subsequently, she went into spontaneous labour and a preterm hydropic female baby of birth weight 3.05 kg was delivered at 35 weeks, cried immediately after birth, the baby had ascites, and vulval edema.

POST DELIVERY:

Immediate intubation was done in view of respiratory distress and shifted to neonatal unit. Placental examination revealed large placenta weighing 1.1 kg, however no other placental abnormality seen. Placental tissue was sent for histopathological examination. Umbilical cord blood sent for cytogenetic analysis and revealed normal chromosomal complement. Post natal ultrasound of the neonate was done which showed gross thick particulated ascites, a giant cyst compressing the IVC, and minimal bilateral pleural effusion. X-ray whole abdomen revealed few spots of intra-abdominal calcification. Emergency exploratory laparotomy was performed on day three of life. Intra operative findings consisted of clumps of collapsed small bowel loop, a giant meconium cyst of 8x10 cm containing approximately 150 ml of thick meconium, which was drained out, there was also terminal ileal perforation of about 10-14 cm proximal to ileo cecal junction which was brought out as loop stoma. Postoperatively baby was extubated and was maintaining saturation with oxygen by nasal prongs, edema started resolving and over all neonatal condition improved. On postoperative day five, baby had one episode of febrile seizure and was started on injection levetiracetum. Ileostomy started functioning. Low volume enteral Feeds were initiated with gradual advancement by seven days of life. Ileostomy closure was planned at fifth month follow up. From Third week of life, baby's condition started deteriorating with recurrent episodes of fever and poor weight gain. Higher antibiotics were started, unfortunately the baby could not be saved and succumb to sepsis on fourth week of life.





DISCUSSION:The occurrence of immune hydrops has been reduced drastically due to universal use of immunoprophylaxis for red cell isoimmunisation.Consequently,NIHF accounts for almost 90% cases of HF.MP has been reported as one of the treatable etiologies of NIHF. Withe the evolution in the imaging technologies increasing number of foetuses with MP are being diagnosed prenatally. Most common ultrasound finding in case of MP is isolated foetal ascites, other presentations include dilated bowel loops, calcification, echogenic bowel and polyhydramnios.

Spillage of meconium constituents secondary to in utero bowel perforation has been shown to activate immune cells including macrophages. The intense inflammatory reaction leads to the formation of a dense, adherent membrane that practically seals off the intestine at the site of perforation. However, if the sealing is incomplete, a thick-walled cystic space is formed, and meconium will continuously keep collecting in this cystic pocket. Any cause of small bowel ischemia or associated mechanical obstruction such as intestinal atresia, volvulus, intussusception, congenital bands, and meconium plug syndrome, as in cystic fibrosis, may result in the genesis of meconium peritonitis

CONCLUSION : Prenatal diagnosis is crucial for the first step of perinatal therapy for MP. Management and the need for surgery depends on the clinical presentation and the overall condition of the newborn. Surgery is required when signs of intestinal obstruction are present. Early diagnosis and management of acid base imbalance, superimposed bacterial peritonitis, and septic shock can prevent mortality. Timing of delivery should rely on composite decision of gynaecologists, neonatologists, and neonatal pediatric surgeons in perinatal and maternal care centre's. Surgery performed within 24 hours in newborns with bowel obstruction may also improve their outcome.

REFERENCE:

1. Meng D, Li Q, Hu X. et al. Etiology and Outcome of non-immune Hydrops Fetalis in Southern China: report of 1004 cases. Sci Rep 9. 2019;10726.

2. Kosinski P, Krajewski P, Wielgos M, Jezela-Stanek A. Nonimmune Hydrops Fetalis—Prenatal Diagnosis, Genetic Investigation, Outcomes and Literature Review. *J. Clin. Med.* 2020; *9(6)*: 1789.

3. Gaddam S A, Tirunagari S. Meconium Peritonitis – A Rare Cause of Ascites. JCR 2018; 8:190-193.

4. Türkyilmaz G, Kurt D, Sarac Sivrikoz T, Kalelioğlu I, Has R and Yüksel, A. Prenatal diagnosis and conservative management of complex meconium peritonitis: a case report. J Clin Invest Surg. 2019; 4(1): 48-52.

5. Uchida K, Koike Y, Matsushita K, Nagano Y, Hashimoto K, Otake K, Kusunoki M. Meconium peritonitis: Prenatal diagnosis of a rare entity and postnatal management. Intractable Rare Dis Res. 2015 May; 4(2): 93–97.