

Preterm with Abdominal Ascites

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Abstract

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Keywords: Preterm birth; Abdominal ascites

Introduction

A female baby of 29 week gestation preterm born to 24 year old Primi mother by spontaneous vaginal delivery. Mother Blood group A positive, HBsAg Negative, HIV negative. Ultrasound scan done at 20 weeks of gestation was normal. No further scan was done. Born by spontaneous vaginal delivery, cephalic presentation, no meconium stained amniotic uid present. Baby did not cried at birth and was oppy with APGAR score of 5 and 7 at 1 and 5 minute respectively. At birth abdomen was distended. Due to poor respiratory e ort she was intubated in labor room.

Vital signs

- Temp. 36.3 degree C
- HR 148/min, RR 56 breaths /min
- BP 50/32 (27) mmHg
- Birth weight 1.7 kg
- Length 45 cm
- HC 30 cm
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Discussion

Meconium peritonitis is aseptic chemical peritonitis which result due to intrauterine perforation of the gut in utero and exudation of meconium into the peritoneal cavity. The most common causes are intestinal atresia, meconium plugs, volvulus and cystic fibrosis. Meconium peritonitis leads to intense inflammation which results in calcification along the surface of bowel and peritoneum. The prevalence of meconium peritonitis is around 1 in 35000 [1] with a slight male predominance and a survival of 50%. The diagnosis of meconium peritonitis can be made with Ultrasound or by abdominal x-ray of neonates, with varied images, altering according to the etiology of the obstruction/perforation [2]. In general, at least four types of meconium peritonitis are recognized, to know: fibro-adhesive (dense mass with calcium deposits caused by chemical reaction), cystic, generalized and healed.

Meconium is a complex mixture of bile salts, cell debris and proteins. Spillage of these constituents has shown to activate immune cells including macrophages [3,4]. Macrophages infiltrate into the peritoneum and participate in a range of cellular functions, including phagocytosis, release of chemical mediators and antibody dependent cell mediated cytotoxicity [5].

Experimental animal studies have demonstrated that TNF production by macrophages is significantly increased in response to meconium stimulation [5]. Exaggerated production of chemical mediators including TNF enhances fibrin deposition and severe intraabdominal adhesion, resulting in short bowel syndrome due to massive resection or polysurgery. Moreover, if sealing of the perforation does not occur, huge abdominal cyst formation and progressive pro-inflammatory cytokine reaction with ascites collection may cause fetal cardiac insufficiency, preterm labor and a poor general condition at birth.

The poor prognosis can be due to some complications like: development of huge formations of abdominal cysts that may suffer

rupture; the formation of ascites that can cause fetal heart failure and the development of sepsis with rapid evolution due to frequent bacterial colonization after 72 hours of birth [6,7].

Conclusion

Meconium peritonitis is a rare condition with many complications and low survival, which requires specific therapy for each etiology.

Therefore, meconium peritonitis should always be suspected in neonates when the Ultrasound and x-rays present with abdominal ascites and perforation. All these babies should be screened to detect cystic fibrosis, because the association with meconium peritonitis is frequent.

References

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