Meconium Peritonitis and Periorchitis: Report of a Prenatal Case

Cem Y. SANHAL[1], İnanç MENDİLCİOĞLU[1], Adnan ASLAN[2], Murat ÖZEKİNCİ[1], Mehmet ŞİMŞEK[1]

Antalya, Turkey

Meconium peritonitis refers to rupture of the bowel prior to birth, resulting in fetal meconium escaping into peritoneum leading to inflammation (peritonitis). Meconium periorchitis is an extension of meconium peritonitis into the scrotum via a patent processus vaginalis. The most common causes of meconium peritonitis are ischemic lesions of the small bowel associated with mechanical obstruction (atresia, volvulus, intussusception, congenital bands, Meckel diverticulum and internal hernia). These likely account for 50% of the cases of meconium peritonitis. Meconium peritonitis may also be caused by viral infections (cytomegalovirus or parvovirus B19) and cystic fibrosis. Here, we report of a patient with fetal meconium peritonitis - periorchitis and perinatal management.

Keywords: Meconium, Peritonitis, Periorchitis, Etiology

Gynecol Obstet Reprod Med 2014;20:178-180

Introduction

Meconium peritonitis (MP) is the state of a bowel wall perforation during late fetal life or early postnatal that allows meconium to enter the peritoneal cavity.[1] Volvulus, intestinal atresia, vascular ischemic injury or meconium plug secondary to cystic fibrosis (CF) and infections are the common etiologic factors of MP.[2] Moreover, passage of the meconium through the patent processus vaginalis, may result in meconium periorchitis (MPO), which is a rare benign cause of a scrotal mass in the newborn.[3,4] In this case report, we present a patient with fetal MP and MPO, diagnosed at the third trimester by ultrasonography.

Case Report

A 23-year-old gravida 2 para 1 woman, at the 33th week of gestation, was referred to our perinatology unit for second opinion ultrasound (US). The couple's personal and family history was unremarkable and they had a non-consanguineous marriage. In the current pregnancy, she did not report any medication use, had no history of fever and exposure of radiation. First trimester screening for aneuploidy revealed a risk of <1:50000 for Down's syndrome, and serum immunoglobulins disclosed past infections of rubella, toxoplasma and cytomegalovirus. Her pregnancy follow-up until this referral was eventless.

On gray scale US; widespread intraperitoneal, liver capsular and scrotal echogenities (calcifications) were detected (Figure 1,2). Additionally, bowels were dilated and abdominal circumference of the fetus were increased. Other sonographic findings of the fetus were normal. Pathologic features were considered as fetal MP and MPO and proper counselling of parents, pointing out the etiologic factors of this situation and possible postnatal prognosis, were performed. We did not offer any invasive procedures because of the late gestational week.

A male infant (birth weight, 3325 gr) was delivered by an elective caesarean section for maternal anxiety at 38 weeks of gestation and Apgar scores were 6 and 8 at 1 and 5 minutes, respectively. All growth parameters including length and head circumference were appropriate for gestational age. The infant's first examination revealed ordinary findings except the soft, cystic and enlarged scrotal mass. At the 5th hour, bowel motions were normal and standart rectal meconium passage were seen. At the second day, feeding was commenced. Unfortunately, at the third day, general discomfort with abdominal distension were detected. Feeding was discontinued and an urgent laparotomy was performed after the X-ray showing infradiaphragmatic air. Abdominal observation revealed extensive meconium in the peritoneal cavity, fibrin plaques and caecal perforation (Figure 3). Resection and anastomosis of bowel and scrotal incisional meconium evacuation procedures were performed (Figure 4). The feeding was re-started at the sixth postoperative day. There were no complications and infant discharged in good condition two weeks later.

At the sixth month of his life, the infant was doing well.

[1] *Department of Obstetrics and Gynecology, Akdeniz University Faculty of Medicine, Antalya*

[2] *Department of Pediatric Surgery, Akdeniz University Faculty of Medicine, Antalya*

Address of Correspondence: *Cem Yaşar Sanhal*
Akdeniz University Medical Faculty
Obstetrics and Gynecology Department
Antalya, Turkey
cemsanhal@yahoo.com

Submitted for Publication: *18. 11. 2013*
Accepted for Publication: *11. 02. 2014*

On the other hand, mutation analysis for CF showed that the infant had a heterozygote V470 mutation on CFTR gene. He is under multidisciplinary follow up for CF and potential complications.

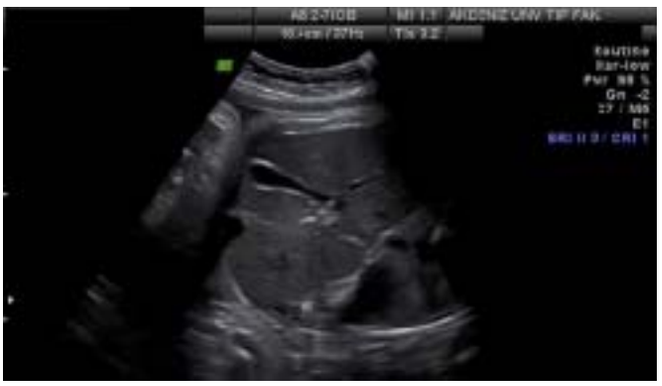

Figure 1: US view of abdominal calcifications

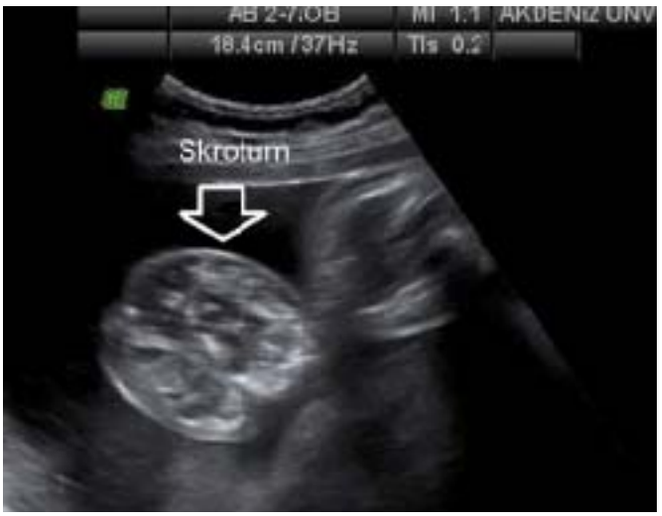

Figure 2: US view of scrotal calcifications.

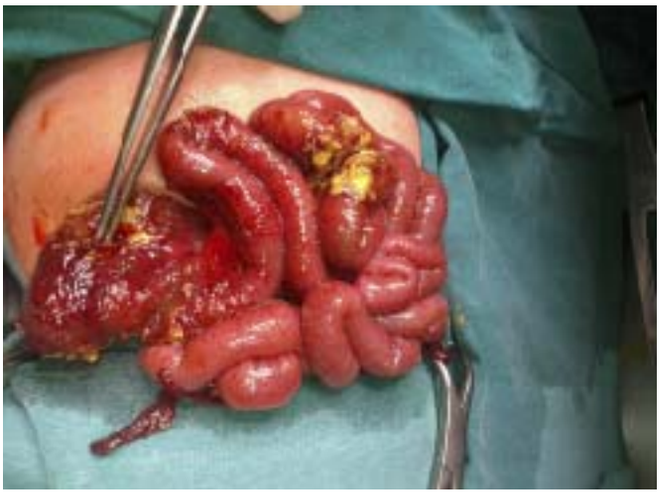

Figure 3: Surgical forceps pointing caecal perforation. Note bowel inflammation and free meconium

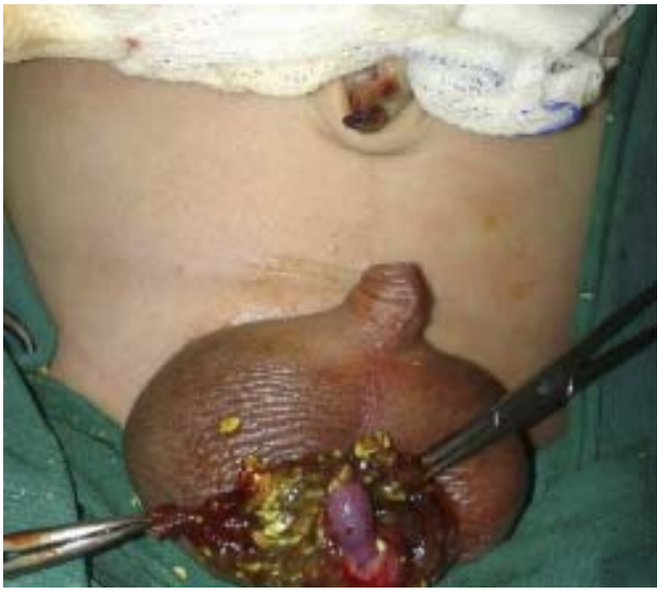

Figure 4: Meconium in scrotum

Discussion

Meconium peritonitis (MP) is a sterile chemical peritonitis resulting from intestinal perforation in utero.[1] Meconium periorchitis (MPO), first described in 1953, is an extension of MP into the scrotum via a patent processus vaginalis.[5] US is the certain instrument in prenatal diagnosis and abdominal, pelvic, scrotal calcifications, echogenic masses and bowel dilatation, which were also detected in our case, are the common findings.[6] Moreover fetal ascites; often the first sign of MP that is secondary to both spilled contents and inflammatory response, polyhydramnios; the result of obstruction and meconium pseudocysts; in the event of localized inflammatory response, are other sonographic markers.[7]

Meconium ileus, intestinal atresia, stenosis, internal hernia, Hirschsprungs disase, intestinal volvulus, intrauterine intussusception, congenital extrinsic band, duplication and CF are the main underlying pathologies of MP and MPO.[8] The anatomical obstructive conditions were eliminated at the time of laparotomy in our case. Furthermore mutation analysis for CF, that is responsible for 7-40% of cases of MP, revealed a heterozygote mutation in CFTR gene and pointed out the possibility of the disease. On the other, the diagnosis of CF is challenging and based upon compatible findings with biochemical or genetic confirmation.[9] Until now, the criteria regarding the clear CF diagnosis have not been fulfilled yet but the infant is under close follow-up by pediatricians.

In conclusion we want to emphasize that in a pregnant with fetal MP and MPO, prenatal diagnosis and counselling is important for the labor in a tertiary hospital. Maternal rubella, toxoplasma and cytomegalovirus screening together with CFTR gene analysis of parents should be the first step at the

prenatal management. Furthermore invasive procedures should be offered for the certain diagnosis of these situations, particularly at early gestational weeks. Couples should be informed that the postnatal prognosis depends upon the etiology and perfect if MP and MPO is not associated with CF.[10]

Mekonyum Peritoniti ve Periorşiti: Prenatal Bir Olgunun Sunumu

Mekonyum peritoniti doğumdan önce bağırsağın bütünlüğünün bozulması ile fetal mekonyumun peritona serbestleşmesi ve inflamasyona (peritonit) yol açması durumunu ifade etmektedir. Mekonyum periorşiti ise, mekonyum peritonitinin patent prosesus vaginalis ile skrotuma yaygınlaşmasıdır. Mekonyum peritonitinin en sık sebepleri mekanik obstrüksiyonla ilişkili iskemik lezyonlardır (atrezi, volvulus, intususepsiyon, konjenital bantlar, Meckel divertikülü ve internal herni). Bunlar mekonyum peritonitinin %50'sinden sorumludur. Mekonyum peritoniti ayrıca enfeksiyonlar (sitomegalovirüs, toksoplazma, Rubella) ve kistik fibrozis nedeniyle de oluşabilir. Burada, fetal mekonyum peritoniti - periorşiti olan bir hastayı ve perinatal yönetimini bildirmekteyiz.

Anahtar Kelimeler: Mekonyum, Peritonit, Periorşit, Etyoloji

References

1. Patton WL, Lutz AM, Willmann JK, Callen P, Barkovich AJ, Gooding CA. Systemic spread of meconium peritonitis. Pediatr Radiol 1998;28(9):714-6.
2. Kenney PJ, Spirt BA, Ellis DA, Patil U. Scrotal masses caused by meconium peritonitis: prenatal sonographic diagnosis. Radiology 1985;154(2):362.
3. Algaba F, Mikuz G, Boccon-Gibod L, Trias I, Arce Y, Montironi R, et al. Pseudoneoplastic lesions of the testis and paratesticular structures. Virchows Arch 2007;451 (6):987-97.
4. Herman TE, Siegel MJ. Meconium periorchitis. J Perinatol 2004;24(1):53-5.
5. Olnick HM, Hatcher MB. Meconium peritonitis. J Am Med Assoc 1953; 152(7):582-584.
6. Eckoldt F, Heling KS, Woderich R, Kraft S, Bollmann R, Mau H. Meconium peritonitis and pseudo-cyst formation: prenatal diagnosis and post-natal course. Prenat Diagn 2003;23(11):904-8.
7. Foster MA, Nyberg DA,Mahony BS, Mack LA,Marks WM, Raabe RD. Meconium peritonitis: prenatal sonographic findings and their clinical significance. Radiology 1987;165(3):661-5.
8. Reynolds E, Douglass B, Bleacher J. Meconium peritonitis. J Perinatol 2000;20(3):193-5.
9. Yankaskas JR, Marshall BC, Sufian B, Simon RH, Rodman D. Cystic fibrosis adult care: consensus conference report. Chest 2004;125(1 Suppl):1S .
10. Chan KL, Tang MH, Tse HY, Tang RY, Tam PK. Meconium peritonitis: prenatal diagnosis, postnatal management and outcome. Prenat Diagn 2005;25(8):676-82.