

Meconium Periorchitis Diagnosed *In Utero* – Case Report Diagnóstico *In Utero* de Periorquite Meconial – Caso Clínico

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Abstract

Meconium periorchitis is a rare pathological entity arising from meconium leakage into the peritoneal cavity secondary to fetal bowel perforation. Extravasated meconium passes through a patent processus vaginalis, provoking a sterile inflammatory cascade and resulting in calcification within peritesticular tissues. Prenatal ultrasonography frequently reveals heterogeneous calcifications in the abdomen or scrotum, enabling timely therapeutic interventions and reducing neonatal morbidity and mortality. This report presents a fetus diagnosed with meconium periorchitis on routine ultrasound, confirmed postnatally. This case highlights the importance of considering both meconium periorchitis and meconium peritonitis in the differential diagnosis when prenatal ultrasonography raises suspicion.

Keywords: Meconium peritonitis; Meconium periorchitis; Intestinal perforation; Peritesticular calcification; Prenatal diagnosis.

Resumo

A periorquite meconial é uma entidade patológica rara, resultante do extravasamento de mecónio para a cavidade peritoneal, decorrente de uma perfuração intestinal fetal. Através do canal peritoneo-vaginal patente, o mecónio pode atingir a bolsa escrotal e iniciar um processo inflamatório, com consequente calcificação dos tecidos peritesticulares. A avaliação ecográfica poderá revelar calcificações heterogéneas na cavidade abdominal ou bolsa escrotal, permitindo uma intervenção precoce e reduzindo a morbilidade e mortalidade neonatais. Este caso evidencia a importância de considerar a peritonite e/ou periorquite meconial como diagnósticos diferenciais na presença de calcificações abdominais ou escrotais.

Palavras-chave: Peritonite meconial; Periorquite meconial; Perfuração intestinal; Calcificações escrotais; Diagnóstico pré-natal.

INTRODUCTION

Meconium peritonitis, an aseptic intrauterine inflammatory response, arises from the leakage of meconium into the fetal peritoneal cavity due to intestinal perforation. The estimated incidence is approximately 1 in 35,000 live births¹. Underlying causes in-

clude cystic fibrosis (owing to abnormally viscous meconium), congenital bowel obstruction (atresia, volvulus, congenital bands, or meconium ileus), and perforation unrelated to obstruction (resulting from vascular compromise, appendicitis, Meckel's diverticulum or ulcer rupture)². When meconium extravasates into the peritoneal cavity, it triggers a sterile chemical peritonitis. Subsequent translocation of meconium into the scrotum via a patent processus vaginalis leads to meconium periorchitis. Clinically, the condition may

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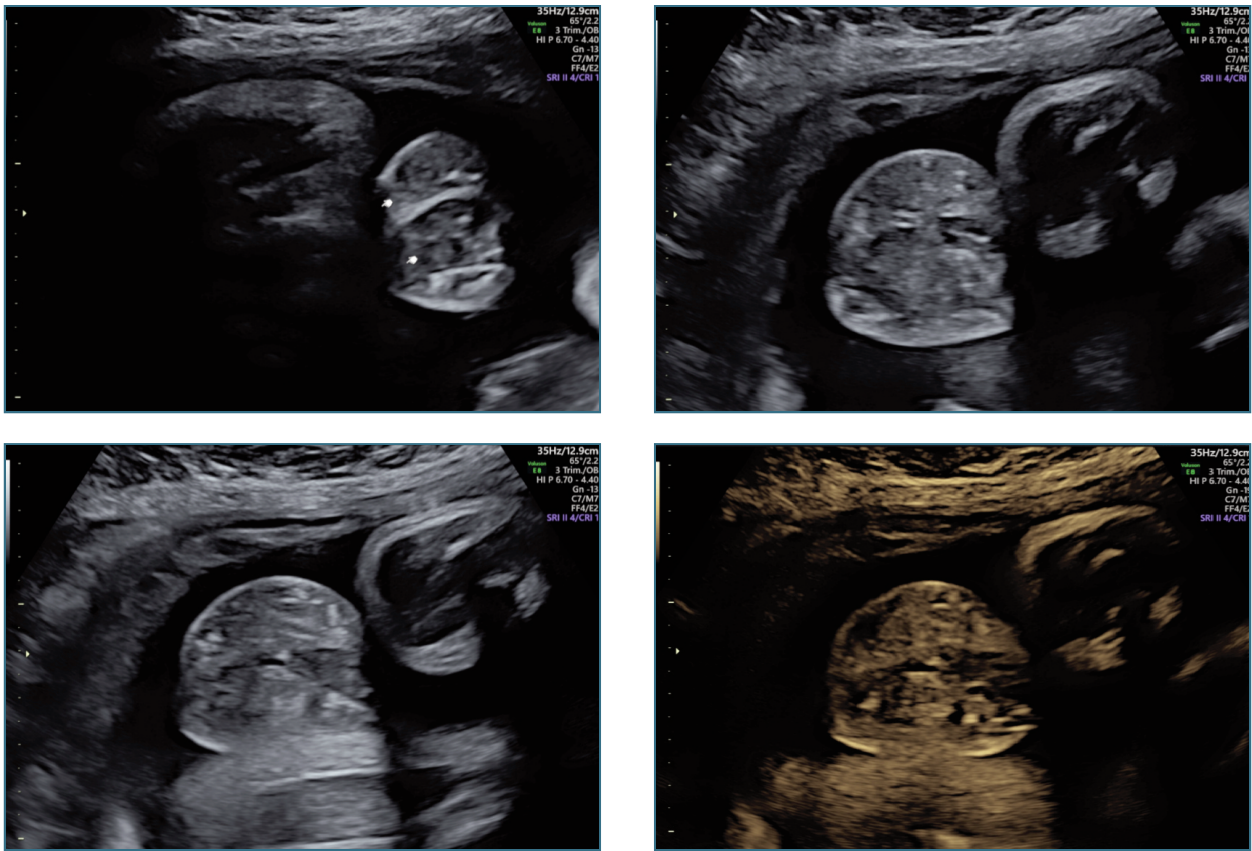


FIGURE 1 to 4. Ultrasound at 33 weeks' gestation revealing bilateral hydroceles with echogenic nodular areas and occasional acoustic shadowing.

manifest initially as a soft hydrocele at birth, that gradually becomes hardened by meconium calcification over a period of weeks³.

Prenatal ultrasound is essential for early detection. Characteristic sonographic findings comprise dilated bowel loops, fetal ascites, intra-abdominal cystic masses, intraperitoneal or peritesticular calcifications and bilateral hydroceles⁴.

The present case report details the clinical course of a fetus with meconium periorchitis caused by meconium peritonitis, diagnosed before birth and subsequently managed in the neonatal period.

CASE REPORT

A 41-year-old gravida 2 para 1 underwent antenatal follow-up at our institution for chronic gestational

hypertension. Her previous pregnancy was uncomplicated and resulted in a term newborn delivered by spontaneous vaginal delivery. The couple reported no significant personal or familial medical history and there was no consanguinity.

Throughout pregnancy, clinical and ultrasound evaluations were normal until a fetal ultrasound at 33 weeks' gestation, which showed bilateral hydroceles with echogenic nodular areas and occasional acoustic shadowing, suggesting peritesticular calcifications (Figure 1-4). There was no ultrasonographic evidence of bowel perforation, ascites, or intra-abdominal calcifications. Serial weekly ultrasounds confirmed persistent bilateral hydroceles and peritesticular calcifications, without the presence of additional signs such as bowel dilation, intra-abdominal calcifications, or cystic masses.

A maternal TORCH serology test was negative.

At 38 weeks and 6 days, labor was induced with a dinoprostone vaginal insert due to increasing blood pressure levels in the context of chronic maternal hypertension, without diagnostic criteria for preeclampsia. A male infant (birth weight 3,030 g) was subsequently delivered via uncomplicated cesarean section due to fetal distress. Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. On physical examination, the neonate had bilateral hydroceles without overlying skin abnormalities or apparent congenital malformations. No abdominal distension was noted immediately postpartum; the neonate began breastfeeding appropriately and passed meconium within the normal period.

A scrotal ultrasound confirmed a heterogeneous or complex hydrocele with echogenic nodules, in keeping with calcified debris, and no Doppler evidence of vascular compromise to the testes. Serum alpha-feto-protein and beta-hCG levels were within normal ranges.

On the second day of life, the neonate was lethargic, with feeding intolerance and abdominal distension. Laboratory findings showed elevated inflammatory markers, hyperbilirubinemia (total bilirubin 4.47 mg/dL; direct bilirubin 3.06 mg/dL), and elevated liver enzymes (AST 112 U/L); coagulation parameters remained normal. Abdominal radiography revealed diffuse gas throughout the intestine. An abdominal ultrasound detected peritonitis, intra-abdominal calcifications, and perihepatic ascites, confirming meconium periorchitis. Liver, spleen, and bowel structures were anatomically unremarkable. Empirical antimicrobial therapy with gentamicin, ampicillin, and metronidazole was initiated.

On day four of life, the newborn's condition deteriorated, necessitating transfer to a tertiary center. Exploratory laparotomy revealed a large meconium cyst causing compression of the bile duct and multiple meconium concretions. No overt perforation was identified, and the intestinal tract remained patent.

Following surgical intervention, the neonate's clinical condition gradually improved. He was discharged on the 12th day of life. Cystic fibrosis screening was negative. At the one-month follow-up appointment, the infant remained asymptomatic, with normal growth and development.

DISCUSSION/CONCLUSION

Meconium periorchitis was originally described by Ol-nick and Hatcher in 1953 and remains a rare diagnosis that can mimic more common scrotal pathologies. It results from meconium leakage into the scrotum in utero or early postnatally through a patent processus vaginalis, often in the context of meconium peritonitis. Retained meconium in the scrotum can induce fibrosis and calcification, potentially mimicking paratesticular tumors such as teratomas, rhabdomyosarcomas, lymphomas, or metastatic lesions. Other differential diagnoses include scrotal hernia, hydrocele, hematoma, ascites, pseudocysts, and testicular torsion. Co-existing scrotal and abdominal calcifications on ultrasound and plain radiographs are strongly suggestive of meconium periorchitis³.

Prenatal ultrasound serves as a critical tool, aiding in the early identification of meconium peritonitis and its extraperitoneal complications. Wax et al. described a classic triad – scrotal calcifications, hydrocele, and reduced or absent Doppler flow surrounding the testis – that is highly indicative of meconium periorchitis⁴.

Therapeutic strategies range from conservative observation to surgical intervention. If malignancy cannot be excluded, surgical exploration is warranted; otherwise, conservative management is preferred, especially for asymptomatic neonates with stable intra-abdominal calcifications, which can resolve spontaneously³.

Because intrauterine meconium spillage is occasionally linked to cystic fibrosis, it is imperative to perform newborn screening for cystic fibrosis in these scenarios².

This case underscores the need to consider meconium periorchitis when encountering prenatal imaging findings of peritesticular masses with calcifications and hydroceles. An accurate prenatal diagnosis can critically influence neonatal management, permitting timely surgical intervention when needed or otherwise supporting a watchful, conservative approach. Current evidence indicates that there are no established criteria for routine intrauterine intervention in cases of meconium peritonitis or meconium periorchitis. Prenatal management is predominantly expectant, based on serial ultrasound assessment and multidisciplinary

planning. However, specific sonographic findings, including meconium pseudocyst, bowel dilatation, and ascites, have been identified as predictors of postnatal surgical intervention and should prompt delivery in a tertiary care center. In the absence of fetal compromise, delivery at term is recommended, and intrauterine intervention remains exceptional⁶.

REFERENCES

1. Konje JC, de Chazal R, MacFadyen U, Taylor DJ. Antenatal diagnosis and management of meconium peritonitis: A case report and review of the literature. *Ultrasound Obstet Gynecol.* 1995;6(1):66-69. doi: 10.1046/j.1469-0705.1995.06010066.x
2. Gilbert-Barnes E, Gunasekaran S. Male reproductive system. In: Gilbert-Barnes E, editor. *Potter's pathology of the fetus, infant and child.* 2nd ed. St. Louis: Mosby-Elsevier; 2007. p. 1414-1426.
3. Williams HJ, Abernethy LJ, Losty PD, Kotiloglu E. Meconium periorchitis: A rare cause of a paratesticular mass. *Pediatr Radiol.* 2004;34(5):421-423. doi: 10.1007/s00247-004-1171-6
4. Wax JR, Pinette MG, Cartin A, Blackstone J. Prenatal sonographic diagnosis of meconium periorchitis. *J Ultrasound Med.* 2007;26(3):415-417. doi: 10.7863/jum.2007.26.3.415
5. Alanbuki AH, Bandi A, Blackford N. Meconium periorchitis: A case report and literature review. *Can Urol Assoc J.* 2013;7(11-12):E495-E498. doi: 10.5489/cuaj.1667
6. Shinar S, Agrawal S, Yinon Y, et al. Fetal meconium peritonitis: prenatal predictors of postnatal surgery and clinical outcomes. *Ultrasound Obstet Gynecol.* 2022;59(1):87-94. doi:10.1002/uog.23650

AUTHORS' CONTRIBUTION

Daniela Rocha: Substantial contributions to the conception or design of the work; to the acquisition and interpretation of data for the work; manuscript writing; final approval of the version to be published. Soraia Cunha: Contributions to the acquisition and interpretation of data for the work; review of the manuscript; final approval of the version to be published. Vera Trocado: Contributions to the interpretation of data for the work; manuscript writing; review of the manuscript; final approval of the version to be published. Ana Paula Gama: Contributions to the interpretation of data for the work; review of the manuscript; final approval of the manuscript. Paula Pinheiro: Contributions to the interpretation of data for the work; review of the manuscript; final approval of the manuscript.

ETHICAL CONSIDERATIONS

The patient had provided written informed consent for publication of their data.

STATEMENT

This manuscript has not been published or presented elsewhere and is not under consideration by another journal.

CONFLICT OF INTERESTS

The authors have no conflicts of interest to declare.

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