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Case Report

Antenatal diagnosis of spontaneous fetal bladder rupture presenting as progressive urinary ascites secondary to posterior urethral valves: a case report

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ABSTRACT

Fetal urinary ascites is a rare antenatal finding most commonly associated with lower urinary tract obstruction, particularly posterior urethral valves (PUV). Spontaneous fetal bladder rupture leading to urinary ascites represents a protective "pop-off" mechanism that may reduce intrarenal pressure and preserve renal function. Antenatal differentiation of urinary ascites from hydrops fetalis is crucial, as prognosis and management differ significantly. We report a case of progressive fetal urinary ascites detected at 29 weeks of gestation, characterized by increasing ascites, a persistently collapsed bladder, and evolving oligohydramnios on serial ultrasonography. Fetal MRI at 35 weeks demonstrated a 4-mm defect in the anterior bladder wall with associated hydroureteronephrosis, confirming antenatal bladder rupture. The neonate was diagnosed postnatally with urinary ascites and PUV on micturating cystourethrogram, and underwent successful valve ablation followed by vesicostomy, with favourable renal outcome. This case highlights the importance of meticulous serial sonographic surveillance, the complementary role of fetal MRI in confirming bladder wall defects, and the value of coordinated perinatal management in improving neonatal prognosis in foetuses with urinary ascites secondary to bladder rupture.

Keywords: Fetal urinary ascites, Bladder rupture, Posterior urethral valves, Fetal MRI, Pop-off mechanism

INTRODUCTION

Fetal ascites is an uncommon antenatal finding that may occur either as an isolated entity or as a component of hydrops fetalis. The etiological spectrum is broad and includes cardiac failure, fetal anaemia, infections, chromosomal abnormalities, gastrointestinal perforation, and urinary tract pathologies.[3] Accurate antenatal identification of the underlying cause is essential, as prognosis and perinatal management vary considerably depending on the etiology. Urinary ascites secondary to lower urinary tract obstruction (LUTO) is a rare but important cause of isolated fetal ascites, most commonly associated with posterior urethral valves (PUV).[1,2] In such cases, increased intravesical pressure may lead to spontaneous rupture of the fetal bladder with intraperitoneal leakage of urine. This phenomenon has been described as a protective "pop-off" mechanism that reduces back pressure on the upper urinary tract and may preserve renal function.[2-4]

Antenatal recognition of fetal bladder rupture is challenging and is frequently misinterpreted as hydrops fetalis unless careful attention is paid to bladder dynamics and associated urinary tract dilatation on serial ultrasonography.[7] While ultrasonography remains the

primary diagnostic modality, fetal MRI can provide additional anatomical detail in selected cases and may aid in confirming the diagnosis.[10] We report a case of antenatally detected progressive fetal urinary ascites due to spontaneous bladder rupture, subsequently confirmed postnatally to be secondary to PUV, with favourable neonatal renal outcome following timely surgical intervention.

CASE REPORT

A 27-year-old primigravida presented at 29 weeks of gestation with an ultrasound report showing moderate fetal ascites suspected to be urinary in origin, and an amniotic fluid index (AFI) of 14 cm. She was a known case of gestational hypertension on antihypertensive therapy. Routine antenatal investigations were unremarkable.

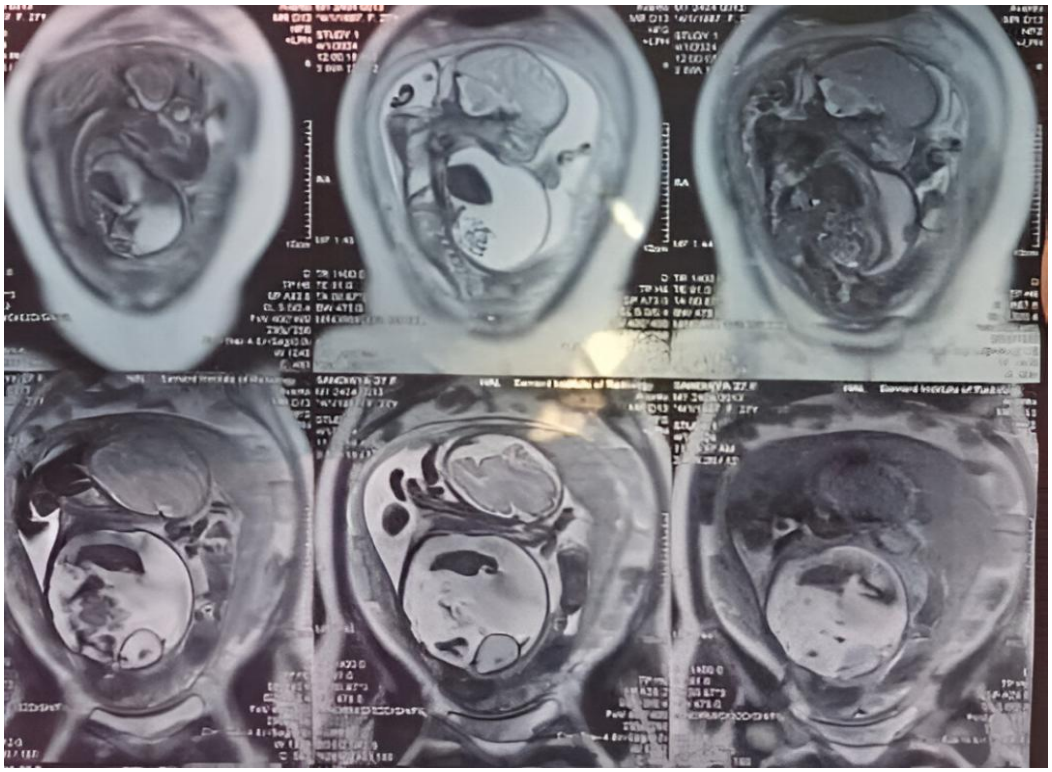

Figure 1: Fetal MRI (t2-weighted sections) showing massive t2 hyperintense urinary ascites with a collapsed urinary bladder and centrally displaced bowel loops, consistent with spontaneous fetal bladder rupture secondary to posterior urethral valves.

Detailed fetal evaluation revealed moderate ascites with suspicion of bladder rupture, normal fetal echocardiography, normal doppler studies, negative TORCH panel, and a normal male karyotype (46, XY).

The presence of isolated ascites with a persistently collapsed urinary bladder, associated hydroureteronephrosis, and absence of skin edema, placentomegaly, or cardiomegaly favoured a diagnosis of urinary ascites rather than hydrops fetalis.[3] Serial ultrasonographic surveillance was planned to monitor ascites, bladder filling, renal parenchyma, and amniotic fluid volume.

At 32 weeks, ultrasound showed persistent ascites, a collapsed urinary bladder, and mild oligohydramnios (AFI 6 cm). Conservative expectant management with maternal hydration was advised. Paediatric surgical opinion recommended close surveillance. Antenatal corticosteroids and magnesium sulphate were administered. Patient was advised to take an MRI but she was not willing. At 34 weeks, repeat ultrasound demonstrated massive ascites with minimal pleural and pericardial effusion without features of hydrops. AFI was 5.7 cm. She was admitted for observation.

At 35 weeks, fetal MRI revealed gross ascites, grade III hydroureteronephrosis, severe oligohydramnios, and a 4-mm defect in the anterior bladder wall suggestive of bladder rupture. In view of severe oligohydramnios, breech presentation, and gestational hypertension, emergency caesarean section was performed. A live preterm male neonate weighing 2.67 kg was delivered and admitted to NICU. Postnatal ultrasonography confirmed ascites, bilateral hydroureteronephrosis, and thick-walled urinary bladder. Ascitic fluid analysis showed elevated creatinine levels confirming urinary ascites.[4,5] Micturating cystourethrogram demonstrated posterior urethral valves. The neonate underwent endoscopic valve ablation followed by Blocksom vesicostomy. There was gradual resolution of ascites with improvement in renal

parameters. Both mother and baby were discharged in stable condition.

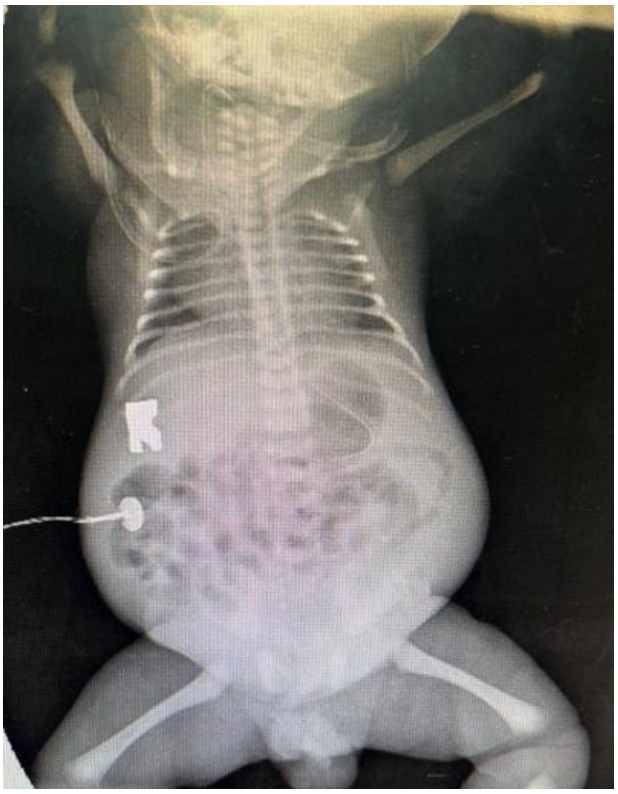

Figure 2: Postnatal abdominal radiograph.

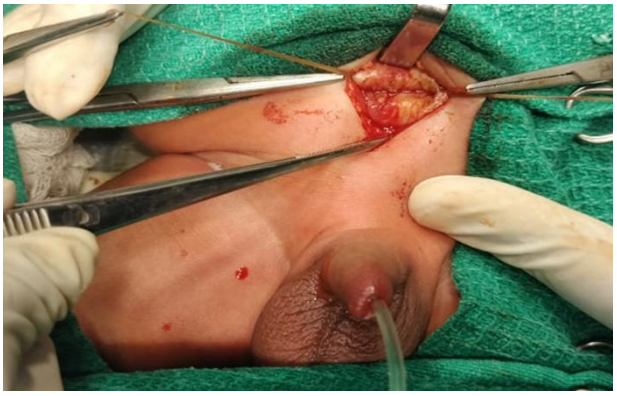

Figure 3: Intraoperative image during vesicostomy.

DISCUSSION

Fetal urinary ascites is an uncommon antenatal finding and represents a diagnostic challenge because it can mimic hydrops fetalis and other causes of fetal fluid accumulation. Among the etiologies, LUTO, particularly PUV, remains the most frequent underlying cause.[1,2] Increased intravesical pressure secondary to obstruction may lead to rupture of the bladder wall with extravasation of urine into the peritoneal cavity, resulting in urinary ascites. This phenomenon has been described as a protective "pop-off" mechanism, wherein spontaneous urinary diversion reduces intrarenal pressure and potentially preserves renal parenchyma.[2,4,8] Dinneen et al first emphasized this concept, demonstrating that foetuses with urinary extravasation such as urinoma, urinary ascites, or bladder rupture often have comparatively better renal outcomes than those without such decompressive mechanisms.[8] The favourable postnatal renal recovery observed in our neonate following valve ablation supports this theory.

A major clinical challenge lies in differentiating urinary ascites from hydrops fetalis. Fontanella et al noted that isolated fetal ascites without skin edema, placentomegaly, or cardiomegaly is more likely to be urinary in origin and carries a significantly better prognosis.[3] In our case, the presence of massive ascites with a persistently collapsed bladder, associated hydroureteronephrosis, and absence of generalized edema strongly favoured urinary ascites rather than hydrops, allowing expectant antenatal management rather than premature intervention.

Shukla et al described that fetal bladder rupture is frequently misdiagnosed antenatally unless careful attention is paid to bladder dynamics and urinary tract dilatation on serial ultrasonography.[7] The progressive nature of ascites with non-visualization of the bladder on serial scans in our case served as an important diagnostic clue. This highlights the importance of meticulous serial sonographic surveillance in suspected cases of fetal ascites.

While ultrasonography remains the primary diagnostic modality, the role of fetal MRI in urinary tract anomalies is increasingly recognized. Levine et al demonstrated that MRI provides superior delineation of fetal genitourinary anatomy when sonographic findings are equivocal.[10] Very few reports in literature document direct antenatal visualization of a bladder wall defect on MRI. In our case, MRI clearly demonstrated a 4-mm anterior bladder wall defect along with hydroureteronephrosis and oligohydramnios, providing definitive antenatal confirmation of bladder rupture. This radiological correlation adds significant value and rarity to the case.

Farrugia et al reported that foetuses presenting with urinary ascites in the absence of hydrops had better survival and renal outcomes compared to those presenting with hydrops fetalis.[9] Our case aligns with this observation, as despite massive ascites, the absence of generalized edema translated into a favourable neonatal course following timely surgical intervention.

Postnatally, ascitic fluid creatinine estimation remains the gold standard for confirming urinary ascites.[4,5] Early recognition allowed prompt urological evaluation in our neonate, and micturating cystourethrogram confirmed PUV, enabling definitive management by valve ablation and vesicostomy. Long-term studies by Smith et al have shown that early valve ablation in PUV significantly improves renal prognosis, particularly in cases where a prenatal pop-off mechanism has occurred.[5]

Detailed counselling of parents about the potential outcomes like fetal and neonatal mortality, preterm delivery, poor postnatal pulmonary function, prolonged ventilation and bladder dysfunction should also be given.

Compared to previously reported cases, the uniqueness of our report lies in the serial antenatal documentation of disease progression, clear differentiation from hydrops, MRI visualization of the bladder wall defect, and complete antenatal-to-postnatal clinic radiological and surgical correlation. Such comprehensive documentation is rarely illustrated in literature and provides important teaching value for obstetricians, radiologists, and paediatric surgeons.

CONCLUSION

Spontaneous fetal bladder rupture should be actively considered in the differential diagnosis of isolated or progressive fetal ascites, especially when accompanied by a persistently non-visualized bladder and urinary tract dilatation. Recognizing this entity antenatally is crucial, as urinary ascites represents a decompressive "pop-off" mechanism that may confer a renal protective effect and carries a far better prognosis than hydrops fetalis.

This case underscores three key clinical messages: the value of meticulous serial ultrasonography in tracking bladder dynamics and ascites progression; the complementary role of fetal MRI in confirming a bladder wall defect when sonographic findings are suggestive; and the importance of coordinated perinatal planning to enable prompt postnatal urological intervention.

Early identification, close surveillance, and timely neonatal management can translate a potentially ominous antenatal finding into a favourable renal and neonatal outcome.

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