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In Utero Urinary Bladder Rupture Causing Urinary Ascites due to Posterior Urethral valve: A case Report

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Abstract

Urinary ascites in a newborn infant is unusual and most commonly indicates a disruption to the integrity of urinary tract, the most common cause being posterior urethral valve. We report a case of fetal urinary bladder rupture due to posterior urethral valve. The case was diagnosed antenatally by ultrasonography and after birth the diagnosis is confirmed by ascitic fluid study and imaging technique and managed successfully by participation of gynecologist, neonatologist, radiologist and urologist. Urinary ascites should be considered in a male neonate with ascites.

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Introduction:

Posterior urethral valve (PUV) is a common congenital anomaly of the lower urinary tract in boys that impairs urinary flow. It is a prevalent cause of morbidity, mortality and end-stage kidney disease in infants and children[1]. The incidence of PUV is estimated to be 1 in 5000 to 8000 male births, but it may be more common for some fetal demise[2]. In the most severe cases, obstruction leads to urinary retention, hydronephrosis and renal insufficiency. Leakage of urine from the urinary tract can occur on rare occasions. In very few cases, rupture of the bladder has been described[3-5]. If it happens, the leakage is a protective event as it reduces the urinary pressure and prevents further kidney damage[6]. Predisposing factors for perforation of the neonatal bladder apart from posterior urethral valves include neurogenic bladder, congenital bladder diverticulum, and detrusor areflexia, profound hypoxia, abdominal trauma, difficult obstetric delivery, and iatrogenic injuries during urethral catheterization and umbilical catheterization[6-8].

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Here, we discuss a neonatal case of urinary ascites due to urinary bladder rupture due to posterior urethral valve.

Case report:

Antenatal course

The mother of our patient, 29 years old primigravida, nondiabetic, normotensive having blood group A negative was under regular antenatal checkup. Her pregnancy period was uneventful except antenatal fetal ultrasonography at 32 weeks of gestation revealed marked bilateral hydro-ureteronephrosis with distention of the urinary bladder and proximal part of the urethra giving a key whole appearance suggestive of posterior urethral valve, in particular there was no oligohydramnios (Fig 1a, b). A conservative approach was chosen, as the situation seemed to be well tolerated by the fetus, and mother was scheduled for follow-up and advised for repeat ultrasonography after 2 weeks. At 34 weeks, ultrasonography revealed that the fetus have bilateral hydro-ureteronephrosis but the measurements reduced than previous sonography, urinary bladder is normal in size, wall is thickened and irregular, there is a gap along the superior wall of the urinary bladder may be due to rupture of the urinary bladder, proximal part of the urethra is dilated giving a key whole appearance suggestive of posterior urethral valve and marked fetal ascites (Fig 2). Mother was under meticulous follow up of gynecologist and consultation was taken from a neonatologist. At 36 weeks of gestation elective cesarean section was done.

Neonatal Course

A male neonate weighing 3.1 kg was delivered by elective cesarean section at 36 weeks of gestation with APGAR score of 7 at 1 minute and 9 at 5 minutes. On physical

examination, the baby had signs of respiratory distress and tense distended abdomen with signs of ascites with no organomegaly and abdominal girth was 46 cm (Fig 3). Regarding Vital signs HR 152b/min, RR 68/min, Temp 98.6^0 F, SpO_2 92% with 6 liter/min oxygen therapy. Spine and back was normal. There were no other congenital malformations. The baby was admitted soon after birth and managed with oxygen therapy, intravenous fluid, antibiotics and other supportive treatment. Catheterization was done as the baby had urinary retention. Oxygen therapy was reduced gradually. Feeding started by nasogastric tube and as feed tolerated amount was increased gradually. In the absence of postnatal evidence of hydrops, evaluation for isolated neonatal ascites was initiated. Initial post-natal radiograph did not show any evidence of pleural or pericardial effusion. The neonate's blood group was A positive and Coombs test was negative. In view of suspicion of urinary ascites, diagnostic abdominal paracentesis was done and around 150ml of fluid was drained, which confirmed urinary etiology as there was presence of high urea and creatinine. Blood investigations were sent which showed raised blood urea and serum creatinine levels (Table 1). The electrolyte abnormalities were corrected with appropriate fluid management. As per advice of urologist continuous bladder drainage was continued, and renal parameters gradually normalized. The abdominal distension gradually decreased and resolved. After the clinical status of the neonate improved, voiding cystourethrogram was done which confirmed PUV (Fig 4). The neonate was discharged home with urology and neonatology follow-up. The defect of urinary bladder repaired with time without surgical exploration, but the patient need fulguration for posterior urethral valve at 2 months of age.

Table 1:

Investigation	Results	Investigation	Results
CBC:Hb	17.3 gm/dl	**Ascitic fluid study**Color	straw
Total Count WBC	7000/cmm	Protein	30 mg/dl
Neutrophil	52%	Sugar	75 mg/dl
Lymphocyte	37%	Total cells	700/cmm
Monocyte	4%	Mononuclear cells	80%
Platelet	267000/cmm	Polymorph	20%
CRP	3 mg/dl	RBC	Few
S Electrolyte:	132 mmol/l	Amylase	45 U/L
Na+		Urea	56 mg/dl
K+	5.1 mmol/l	Creatinine	1.9 mg/dl
S. Creatinine	1.5 mg/dl	Gram stain	Negative
Blood urea	35 mg/dl	AFB Stain	Negative
S. Total Protein	4.7 gm/dl	Culture	No growth
S. Albumin	3.3 gm/dl		
S. Calcium	10.6 mg/dl		
Coombs Test	Negative		
Blood C/S	No Growth		
Urine R/E:			
Color:	Straw, Clear		
Sp. Gravity	1.004		
Albumin	Nil		
Sugar	Nil		
Pus Cells	0-2/HPF		
RBC	Nil		
Urine C/S	No Growth		

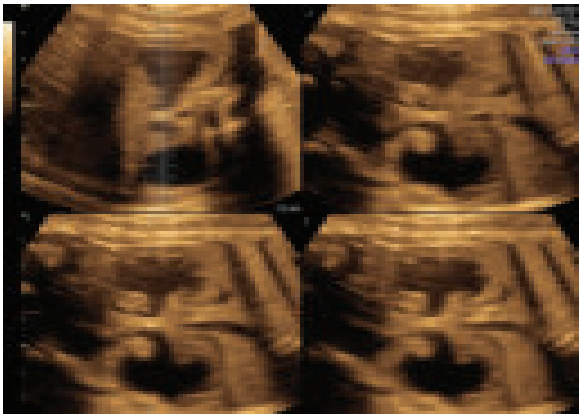

Figure 1 (a) *Gross bilateral hydro-ureteronephrosis with distention of the urinary bladder*

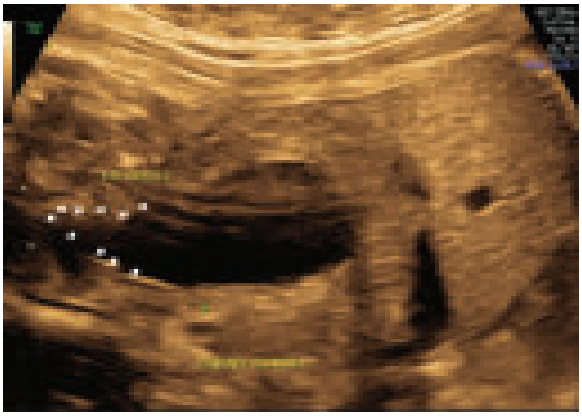

Figure 1(b) *Key whole appearance suggestive of posterior urethral valve*

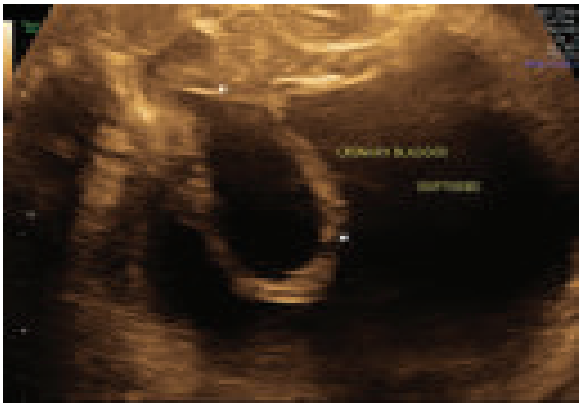

Fig 2: *A gap along the superior wall of urinary bladder due to rupture of the bladder*

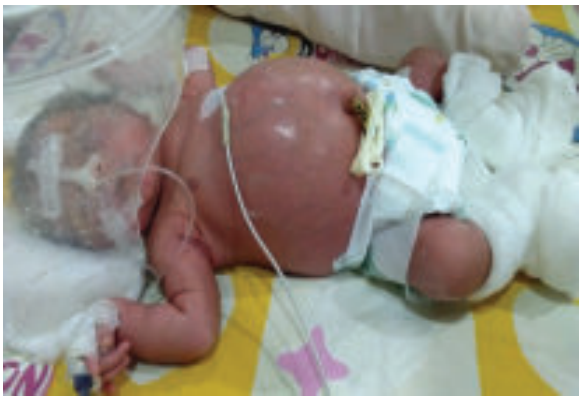

Figure 3: *Tense distended abdomen of the baby*

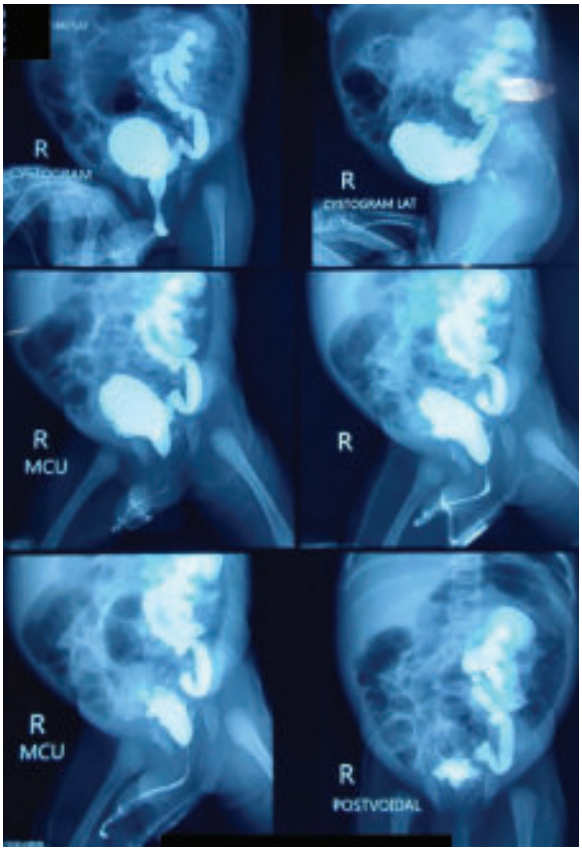

Figure 4: *Micturating cystourethrogram showing presence of posterior urethral valve.*

Discussion:

Urinary ascites in a newborn infant is rare. It indicates a disruption to the integrity of the urinary tract. Urinary tract obstruction leads to increased pressure which causes urine collection within the peri-renal spaces and subsequent urinary ascites either by calyceal or urinary bladder perforation or filtration through the walls of urinary tract. The cases of urinary ascitis reported in medical literature are generally related to bladder rupture secondary to the presence of a posterior urethral valve in boys[9-11].

In utero presentations of posterior urethral valve vary from urinary retention with urinary tract dilatation, bladder wall thickening and vesico-ureteral reflux. In the most severe cases, this can lead to renal dysplasia, which may cause renal insufficiency. The renal parenchymal lesions are also due to urinary reflux in the renal cavities and parenchyma. Besides the urological and nephrologic manifestations of the PUV, the associated oligohydramnios can lead to pulmonary hypoplasia, facial dysmorphism and deformity of the extremities, in some cases, leading to fetal death[12]. Our patient didn't have such severe presentation.

Urinary bladder perforation and urinary ascites is a rare complication of PUV[13]. Other causes of urinary ascites in PUV include rupture of the pelvicalyceal fornices or transudation across the intact upper urinary tract. In obstructive uropathy, the upper tracts are in risk of high pressures in the intrauterine life, which may hampers the development of the kidneys and cystic renal dysplasia may occur. However, some protective mechanisms like-vesicoureteral reflux, bladder diverticuli, urinary extravasations do exist to prevent this irreversible damage to the developing kidneys. Urinary extravasations at the level of the fornices may result in urinoma formation around the kidneys, which may communicate freely with the peritoneal cavity, leading to urinary ascites[14].Fetal or neonatal urinary ascites is a life-threatening condition as the peritoneal membrane "autodialyzes" the urine, which may lead to gradual increase in the blood urea nitrogen (BUN) and derangement of the serum electrolytes. In these patients there may be elevation of serum urea and creatinine, hyponatremia and hyperkalemia. Thus, urinary ascites should be suspected in a neonate with ascites and abnormal blood biochemistry[15].

Posterior urethral valve (PUV) is the most common congenital cause of bladder outflow obstruction in male neonates and more than 50% of the PUVs are diagnosed prenatally[16]. Prenatal ultrasound findings include thick-walled bladder, the keyhole sign, unilateral or bilateral hydroureteronephrosis, echo bright kidneys, and oligohydramnios[17]. In severe cases Ultrasound helps to establish the presence of ascites, urinary bladder perforation and dilatation of the upper tracts with or without associated urinomas and cystic dysplasia of the kidneys. VCUG is the next choice of investigation to demonstrate the anatomy of the lower urinary tract for diagnosis of PUV, changes in the urinary bladder, vesicourethral reflux (VUR). Urinary bladder changes for longstanding PUV may include a large capacity or a contracted bladder with trabeculations, sacculations, diverticula formation or bladder neck hypertrophy. During voiding, fusiform dilatation of the posterior urethra with abrupt narrowing at the level of the valves may sometimes be noted as filling defects in the urinary stream[18].

The antenatal management of a patient with PUV with ascites is based on serial follow up of mother and fetus, with full parental consent. The decision of intervention is dependent on gestational age, decreasing amniotic volume and deteriorating renal function. The recommended procedures are vesicoamniotic shunt, vesicostomy and fetal endoscopic valve ablation. Interventions should not be tried in cases with the poorest prognosis[19,20]. After birth, management has to be prompt and the basic aim is to decompress the urinary tract. This may be achieved by abdominal paracentesis, catheter drainage or surgical exploration. Indications of abdominal paracentesis include significant respiratory distress, mechanical discomfort and sepsis[6,21]. Bladder catheterization by urethral route with or without vesicostomy achieves healing in most of the cases within 10-14 days, after that fulguration of the PUV is attempted. Catheter drainage fails to improve in ruptures with large rents and continued leak, loculated ascites and inadequate decompression of the upper tracts. In these patients surgical exploration and repair of the defect may require. Prognosis is variable depending on the age at diagnosis, severity of obstruction and the extent of changes in the urinary tract[22-23].

Conclusion:

Posterior urethral valve is a common neonatal problem but intrauterine urinary bladder rupture is a very rare condition which may be well tolerated by the fetus. Although it's a rare presentation, concerned physician need to keep in mind in practice if they find a fetus or neonate with ascites.

Conflict of interest: None.

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