

## Case Report

# Urinary ascites in a neonate with posterior urethral valve

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### ABSTRACT

Urinary ascites in a newborn is an extremely rare condition, most commonly due to posterior urethral valves, due to transmission of high intravesical pressure to calyceal fornices, and subsequent urinary ascites either by calyceal perforation or filtration through walls of urinary tract and their rupture. We describe a newborn male baby, who presented with huge abdominal distension at birth, and diagnosed as urinary ascites on paracentesis. Baby was asphyxiated and required resuscitation at birth, and ventilatory support for 4 days in v/o significant abdominal distension. Micturating cystourethrogram (MCUG) and magnetic resonance imaging (MRI) showed posterior urethral valves. Baby also had a left sided urinoma and grade 4 vesicoureteral reflux (VUR) on MCUG. Post paracentesis and drainage of 400ml of ascetic fluid, and urinary catheterization, baby had significant improvement of deranged renal parameters, and diuresis, and could be weaned from ventilation. In v/o inability to negotiate a urethroscope, a vesicostomy was done for urinary drainage and fulguration of valves planned on follow-up.

**Keywords:** Urinary ascites, Newborn, Male, Posterior Urethral valves

### INTRODUCTION

Urinary ascites is a rare clinical condition with different aetiologies. Posterior urethral valve is the most prevalent condition which is observed in 70% of the cases. The other risk factors include neurogenic bladder, congenital bladder diverticulum and detrusor areflexia. Bladder emptying at high intra-vesicle pressure can be transmitted to ureters and up to renal collecting system.<sup>1</sup>

Urinary ascites arises as a result of calyceal fornices rupture mediated by raised intra renal pressure. In some cases, perforation of posterior urethral valve also triggers urinary ascites.<sup>2,3</sup>

Ultrasound imaging is the mainstay in diagnosis of ascites with dysregulated renal function. Ultrasound detects the ascites and upper tract dilatation in the presence and absence of associated urinomas and kidney cysts.<sup>4</sup> Voiding cystourethrography (VCUG) reveals the urinary bladder leak by peritoneal cavity contrast extravasation and also

gives the information regarding the pathological changes in the urinary tracts. Urinary ascites in neonates is a life-threatening clinical event, since there exists an autodialyzes of urine in peritoneal membrane, which preludes to increased concentration of blood urea nitrogen (BUN) and alterations of serum electrolytes level. Treatment strategies encompass catheter drainage or surgery based on the neonate's condition with a primary goal of rerouting the urine from peritoneal cavity. The outcome relies on prompt diagnosis and efficient urinary drainage.<sup>4</sup>

Previous literatures show that the development of urinary ascites origin is due to the formation of posterior urethral valve which lead to rupture of bladder followed by ascites formation.<sup>5</sup> However, in one rare case urinary ascites secondary to renal calyx rupture has been reported in posterior urethral valves.<sup>6</sup> Thus, the site of occurrence is not important and the leakage is the best protective measure as it relieves the urinary pressure and prevents further kidney damage. Posterior urethral valve is one of the major causes of lower urinary tract obstruction in male

neonates. In majority of the cases, obstruction causes urinary retention, hydronephrosis and renal insufficiency. The present case describes a urinary ascites in male neonate with posterior urethral valves which was rare at our institution.

## CASE REPORT

A singleton, male baby with birth weight of 3.2 kg was delivered at 36 and 3 weeks period of gestation (POG) by emergency lower segment caesarean section (LSCS) at our hospital i/v/o severe oligohydramnios (AFI-3) and fetal distress. Mother was an unbooked pregnancy and was referred to our hospital at 36 weeks of POG in v/o intraabdominal cyst in the fetus. Baby was depressed at birth and required extensive resuscitation (bag and tube ventilation and chest compression). Baby intubated in delivery room. Apgar scores were-2/5/8 at 1 min/5 min/10 min. Injection vitamin K 1 mg stat was given in delivery room. Baby was immediately taken to neonatal intensive care unit (NICU) and ventilated in v/o significant respiratory distress.

On general physical examination, there was no pallor, cyanosis, dysmorphism or external congenital malformations. Baby had gross abdominal distension, uniform, and no palpable mass per abdomen. Baby had poor perfusion and hypotension which was corrected with fluid bolus and Inotropic support. A central line was secured for this purpose. Baby had one episode of seizure activity at 11 hours of life which was controlled with IV phenobarbitone.

A bedside postnatal ultrasound scan showed significant ascites (around 500 ml), with right hydronephrosis and left sided para-renal cyst, urinoma, with thickened trabeculated bladder. A diagnostic ascitic tap was done on day 2 which was consistent with urinary ascites (ascitic fluid creatinine 6.4 mg/dl and urea 94 mg/dl compared to serum creatinine of 1.6 mg/dl and blood urea of 54 mg/dl, significantly elevated in comparison with serum values). There was extreme difficulty in urinary catheterization, and urine output was documented during NICU stay which was normal. In v/o significant ventilatory requirement and deranged renal parameters, a therapeutic ascitic tap was done on day 4 and around 400 ml of ascitic fluid was aspirated. Subsequently, baby could be easily weaned from ventilation and was extubated in 24 hours. Renal parameters showed gradual improvement following paracentesis, from serum creatinine 1.6 mg/dl on day 1 improving to 0.3 mg/dl by day 6. Baby had polyuria following paracentesis (up to 6 ml/kg/hour) which normalized within 48 hours to 2-3 ml/kg/hour. There was no dyselectrolytemia during NICU stay. As part of work-up, sepsis screen was negative, serum calcium was normal, and chest X-ray and abdominal X-ray was normal. Blood culture was sterile.

As advised by pediatric surgeon, a micturating cystourethrogram was done on day 2 which showed

bilateral mild hydronephrosis with right sided grade 4 vesicoureteral reflux (VUR) with posterior urethral valves. In v/o inconclusive micturating cystourethrogram (MCUG) (PUVs were obscured by the catheter), MRI of abdomen was done on day 3 of life which showed mild to moderate hydronephrosis, thickened urinary bladder, perinephric urinoma on left side with posterior urethral valve.

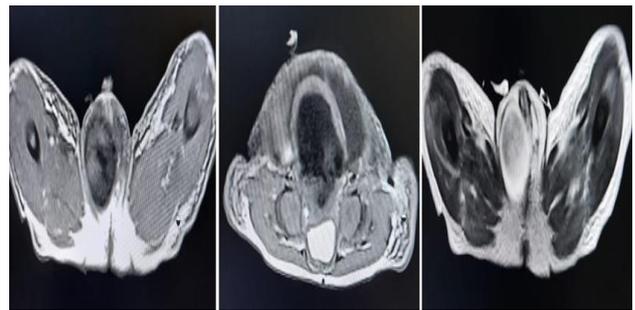
As baby's renal parameters improved after catheterization, a vesicostomy was done on day 7 of life, and baby was started on uroprophylaxis in v/o grade 4 VUR. Endoscopic fulguration could not be done as scope could not be negotiated through narrow urethra. Baby was discharged on day 11 of life. At discharge, baby was passing urine from both vesicostomy site and penile urethra. Fulguration of PUV has been planned at 4-5 months of age on follow-up. Follow-up of the baby 15 days after discharge showed baby accepting breast feeds well with appropriate weight gain. Vesicostomy site was healthy.

## Investigations

Serial serum creatinine level at 48 hours intervals, were as follows: 1.7, 1.5, 1, 0.7 and 0.3 mg/dl. Postnatal MCUG on day 2 of life after stabilization showed significant ascites, right sided grade IV VUR with hydro-nephrosis, left sided para-renal cyst, bladder marginally thickened, indirect inguinal hernia on right side, not consistent with posterior urethral valves.



**Figure 1: MCUG examination on day 2 shows right sided grade-III/ IV vesico-ureteric reflux.**



**Figure 2: MRI abdomen done on day 3 showing posterior urethral valves with perinephric urinoma on left side, thickened urinary bladder and gross ascites.**

Postnatal MRI on day 3 of life- B/L mild to moderate hydronephrosis, thick wall urinary bladder, gross ascites, posterior urethral valve with perinephric urinoma on left side, indirect inguinal hernia on right side.

## DISCUSSION

Previous published cases show that urinary ascites is as a result of rupture of bladder secondary to the existence of posterior urethral valves in males.<sup>1-3,7</sup> Posterior urethral valves are the major cause of bladder outlet obstruction in male neonates, with the incidence rate of 2 in 10,000 live births.<sup>8</sup> Progression of renal insufficiency in cases affected with posterior urethral valves is due to the increased pressure formed as a result of urethral obstruction which is transmitted to the upper urinary tract.

Accurate diagnosis is a challenging task in most of the cases using imaging modalities such as MCU and ultrasound. The initial management strategy of posterior urethral valves involves NICU support and placement of urinary catheter for drainage. Bladder catheterization must be done to prevent distension with simultaneous healing. In neonates affected with posterior urethral valves, catheter drainage by urethral route with or without vesicostomy elicits profound healing in 10-14 days. The valves can be ablated through endoscopic procedure, when the new-born is in stable condition. In neonates with small urethras, vesicostomy is recommended for the placement of endoscopy, meanwhile upper tract diversion will not offer any effective benefits with respect to drainage and improvement in renal functions.<sup>9</sup>

## CONCLUSION

The long-term outcome regarding renal functions is very poor, since around 20-60% of male neonates progresses to end-stage renal disease.

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## REFERENCES

1. Mauriceau F, Grosses T, Lauteur C. Paris, France, 3rd edition. 1681.
2. Cywes SJ, Wynne JM, Louw JH. Urinary ascites in the new-born with a report of two cases. J Pediatr Surg. 1968;3(3):350-6.
3. James U, Davies JA. Congenital urethral obstruction presenting in the newborn period. Proceedings of the Royal Soc Med. 1952;45:401.
4. Singh J, Khanna AC, Arora S. A rare case of urinary ascites in newborn. Int J Med Dent Sci. 2016;5(1):1098-100.
5. Ahmed S, Borghol M, Hugosson C. Urinoma and urinary ascites secondary to calyceal perforation in neonatal posterior urethral valves. Br J Urol. 1997;79:991-2.
6. Trulock TS, Finnerty DP, Woodard JR. Neonatal bladder rupture: case report and review of literature. J Urol. 1985;133(2):271-3.
7. Griscom NT, Colodny AH. Diagnostic aspects of neonatal ascites: report of 27cases. Am J Roentgenol. 1977;128:961-9.
8. Malin G, Tonks AM, Morris RK, Gardosi J, Kilby MD. Congenital lower urinary tract obstruction: a population-based epidemiological study. BJOG. 2012;119(12):1455-64.
9. Farhat W, McLorie G, Capolicchio G, Khoury A, Bägli D, Merguerian PA. Outcomes of primary valve ablation versus urinary tract diversion in patients with posterior urethral valves. Urol. 2000;56(4):653-7.

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