Neonatal Posterior Urethral Valve associated with Acute Kidney Injury: A Case report

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ABSTRACT

Congenital obstructive uropathy is a major cause of kidney injury in children, with some cases requiring early neonatal intervention. Posterior urethral valve (PUV) is a common cause of congenital obstructive uropathy in male infants. We report the case of a 15 days old male, antenatally diagnosed with bilateral hydronephrosis, presented with vomiting and poor urinary flow. Serum creatinine was 5 mg/dL, indicating Stage 3 AKI (KDIGO criteria). After stabilization, PUV was confirmed and treated with valve ablation via electrofulguration in the neonatal period. As PUV can lead to early renal dysfunction, early diagnosis and timely intervention may preserve kidney and bladder function, but long-term follow-up is essential to assess outcomes.

Key words: Neonate, Obstructive Uropathy, Posterior urethral Valve

Introduction

Posterior urethral valve (PUV) is the most common lower urinary tract obstruction in male children with incidence varies from 1:5000 to 1:8000 live births.[1] PUV are obstructing membranous folds in the lumen of the posterior part of the urethra which can lead to hypertrophy of the bladder wall and detrusor muscle. Changes to the bladder can impact compliance and bladder filling as well as raise intravesical pressures. Elevated bladder pressures can be transmitted into the ureters causing vesicoureteral reflux. This, in turn, is associated with infection, incontinence, and progressively impaired renal function.[2] Around 24%–50% of patients with PUV evolve to chronic kidney disease.[3]

Presentation of PUV varies from dribbling of urine, poor flow, recurrent UTI, palpable bladder, or features of impaired kidney function like vomiting due to uremia, pedal oedema, decrease urine output etc.[3]

In patients with PUV, primary valve ablation is considered a standard treatment of choice.[4] However, temporary vesicostomy with delayed valve ablation may be considered if instruments are unavailable.[5]

Presentation of PUV in neonate associated with acute kidney injury is rare presentation with incidence of approximately 2-3% of total incidence of PUV.[6] We aim to describe this rare presentation of PUV in neonatal age group.

Case Report

A full-term, 15-day-old male baby presented with multiple episodes of vomiting for last 2 days. Vomiting was non-projectile, non-bilious with no abdominal distention or abdominal mass. Patient was passing stool normally. However, his voiding flow was inadequate with poor streaming. He was diagnosed with bilateral hydronephrosis antenatally at third trimester (right and left renal pelvic diameter 10mm and 10.5mm, respectively). Perinatal period was uneventful. On day 5 of life, an ultrasound abdomen scan showed mild hydroureteronephrosis with right and left renal pelvic diameter 11mm and 12mm, respectively. There was no renal parenchymal thinning.however, right and left kidney size were 4.3 and 4.2cm^2 respectively.

At the time of admission, patient had features of some dehydration. Vitals signs were stable. Abdominal examination revealed slightly palpable urinary bladder. Other findings were normal.

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Genitalia look normal. His biochemical parameters were S. Creatinine: 5 mg/dl; Urea: 82 mg%; Sodium 127 mmol/L; Potassium 5 mmol/L; Hb: 14.6 g/dl, WBC: 11500/cumm; Platelets: 250,000/cumm; CRP: positive; sterile urine. Arterial Blood Gas analysis showed metabolic acidosis with hyponatremia and low bicarbonate level.

Continuous urethral catheterization done with feeding tube and baby was resuscitated with iv fluids and associated electrolytes imbalance were corrected. Child was on post-obstructive diuresis state with urine output of average 700ml per day. Slowly, his creatinine fell to normal range in a week. Voiding cystourethrogram (VCUG) showed dilated and elongated posterior urethra with normal anterior urethra suggesting bladder outlet obstruction. His bladder was small in capacity with irregular margin and multiple diverticula was seen in VCUG (Figure. 1). DMSA/DTPA was not performed.

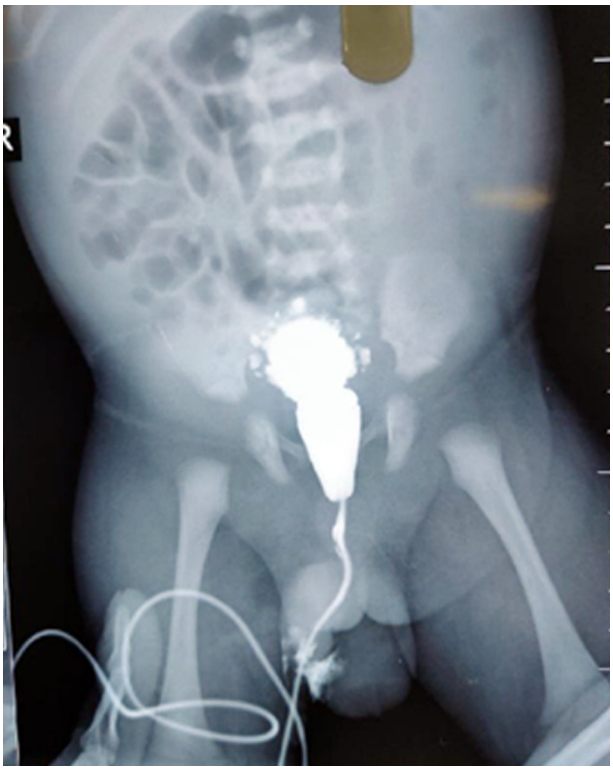

Figure 1: Small bladder with irregular margin and multiple diverticula seen in VCUG

After optimizing the general condition of the baby, Cystourethroscopy was performed which revealed Type I Posterior Urethral Valve at day 22 of life (Figure 2). Along with it, there was trabeculation of bladder and small multiple bladder diverticulum and golf hole right ureteric opening. However, left ureteric opening looks non-patulous slit like. Electro-fulguration of PUV at 5, 7 and 12 o'clock position done in same setting (Figure 3). The postoperative period was uneventful. Patient was under regular antibiotic prophylaxis and oxybutynin for bladder dysfunction.

Discussion

Approximately one third of children with chronic kidney disease have the illness because of urinary tract malformations and obstructive uropathy. Posterior urethral valve (PUV) is the leading cause

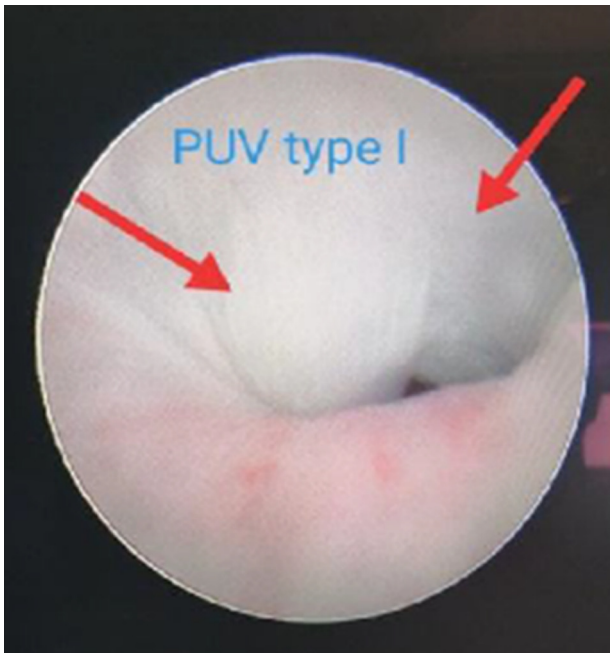

Figure 2: Cystourethroscopy revealing Type I PUV

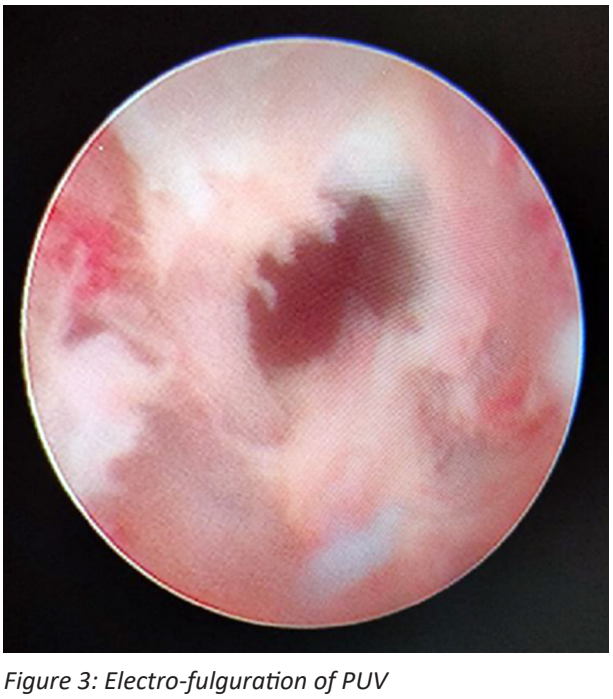

Figure 3: Electro-fulguration of PUV

of kidney failure in male child. However, there is significant drop in infant mortality and morbidity from it in recent decades. This is due to antenatal detection and early surgical interventiona and multidisciplinary management of PUV.[7]

Like in this case, if the neonate is initially unwell, they may require correction of electrolytes, as well as support for respiratory distress or urosepsis. Along with it, the bladder may require drainage in the event of acute retention or difficulty to pass urine. This is usually performed with a soft feeding tube rather than a balloon catheter, which can cause spasm.[8]In this case, newborn presented with features of Acute kidney injury (AKI) stage 3. Regarding the fact that distal tubule, whose main functions are concentration and acidification of urine[9,10], is the first "front" facing the insult of retrograde urinary flow in the intrauterine period, it would be logical to state that this part would also be the first and most affected structure in a nephron in PUV disease.[11] all the blood parameters of thisnewborn are within normal limit, it is tough to comment on the precipitating factor of AKI in this newborn.

Many authors do believe that VCUG is the investigation of choice for PUV. However, VCUG showed classical sign of dilated posterior urethra in 51% and reflux in 53.9% only in case of PUV. Also, similar findings can also be observed in case of neurogenic bladder (Detrusor Sphincter Dyssynergia). Hence, cystoscopic examination should be preferred for all the cases to confirm PUVs regardless of VCUG results.[12]

Cystoscopic valve ablation is the current initial treatment of choice for posterior urethral valves. This will usually alleviate the obstruction, but vesicoureteral reflux will occur in a third.[13] In the case of extremely preterm infants, cystoscopic ablation is often not technically feasible and vesicostomy are preferred.[13] As in this case, continuous bladder drainage with feeding tube aids in optimizing serum creatinine, cystoscopic valve ablation was performed.

The usefulness of vesicostomy is diminishing because the bladders of patients with PUV can be drained efficiently and primary valve ablation can be performed. The first treatment of choice in patients with PUV is primary valve ablation.[4] In addition, initial urinary diversion in patients with PUV may lead to bladder dysfunction, by interfering with the physiological bladder cycle, leading to complications associated with stoma.[14] However, the usefulness of primary valve ablation and temporary vesicostomy with delayed valve ablation is controversial.

Prognosis of PUV is variable depending on the severity of obstruction and the in-utero sequelae. Most of the infants who survive the neonatal period go on to develop chronic kidney disease (CKD), and many have ongoing bladder dysfunction. Nevertheless, published literature indicates that PUV ends in ESRD in around 24%–50%of patients treated due to upstaging of CKD.[15] Roth et al. addressed that majority of PUV fulgurated patients may progressed to ESRD over a period of time and suggested that there will be slow and steady rise in serum creatinine (0.5 mg/dl per year) from serum creatinine of 1.5 to 5 mg/dl and rate of progression will be rapid after serum creatinine exceed 5 mg/dl.[7] Less than one third of cases may develop abnormal bladder function necessitating further intervention like medication, clean intermittent catheterization.[16] Sixteen percent of PUV cases required Renal replacement therapy within ten years who underwent initial treatment in first 90 days of life.[17] Similarly, around 60-70% of VUR secondary to PUV resolve after PUV fulguration spontaneously, however, remaining may require further interventions.[18] Hence, long term follow up is mandatory in every PUV fulgurated cases.

Oxybutynin enhances hydronephrosis improvement and vesicoureteral reflux resolution following primary endoscopic valve ablation in infants.[19] To enhance improvement in VUR and bladder function, antibiotic prophylaxis and oxybutynin have been regularly prescribed to patient.

Conclusion

PUV leads to a spectrum of problems, one of which is deterioration of renal function in early neonatal period. Early identification and intervention may improve bladder and renal function. However, long term follow up is necessary to know its actual impact. Standard therapy is valve ablation which should be carried out as early as possible if instruments, techniques and skill are available. Early intervention, check cystoscopy after ablation, close follow-up with appropriate laboratories and radiological investigation when necessary are recommended.

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