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Case Report

Posterior urethral valves and recurrent vesicoureteral reflux: Surgical approach and postoperative challenges in a pediatric case

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Abstract

Posterior urethral valves (PUV) and Vesicoureteral reflux (VUR) are congenital urological anomalies that contribute to recurrent urinary tract infections (UTIs) and progressive renal damage. VUR, the retrograde flow of urine due to Vesicoureteral junction dysfunction, is classified as low-to-moderate (Grades I–III) or high-grade (Grades IV–V), with the latter increasing the risk of renal scarring. PUV, a posterior urethral obstruction, often coexists with VUR, exacerbating bladder dysfunction. Early detection via antenatal ultrasound and postnatal voiding cystourethrography (VCUG) is critical. Management varies from conservative therapy for mild cases to surgical correction for severe reflux, with long-term follow-up essential for renal preservation.

Keywords: Vesicoureteral reflux (VUR), Voiding cysto-urethrogram (VCUG), PUV fulguration, B/L Laparoscopic Ureteric Re-implantation, Posterior urethral valves (PUV), B/L DJ stent

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1. Introduction

Vesicoureteral reflux (VUR) is the most prevalent congenital urologic disorder, defined by the retrograde flow of urine from the bladder into the ureters and renal pelvis due to dysfunction of the Vesicoureteral junction. Epidemiological studies estimate the prevalence of VUR to be between 0.4% and 1.8% in the general paediatric population, with approximately 30% of children presenting with urinary tract infections subsequently diagnosed with VUR upon further evaluation.[1,2] VUR is the most common urological abnormality observed in neonates, with an estimated prevalence of approximately 1% among all new-born's.[3] However, this prevalence increases significantly, reaching up to 15%, in neonates diagnosed with prenatal hyderonephrosis.[4] VUR is typically categorized as either primary or secondary, with the secondary form often stemming from an improperly developed Vesicoureteral junction, neurogenic bladder dysfunction, increased intravesicle pressure, or the presence of posterior urethral valves.[5] VUR refers to the retrograde flow of urine from the bladder into the ureters and kidneys, with grades 1–3 classified as low-to-moderate VUR, involving reflux without significant ureteral dilation, and grades 4–5 as high-grade VUR, characterized by substantial ureteral dilation and tortuosity (**Figure 1**).[6] The management of primary VUR, occurring without secondary causes such as obstruction or neurogenic bladder, remains debated, as high-grade VUR poses a significant risk for recurrent urinary tract infections (UTIs) and potential kidney damage. Surgery has historically been the standard approach for high-grade VUR to prevent UTIs and renal damage, though conservative management is increasingly considered for lower grades.[7] The most common causes of VUR include congenital conditions such as neural tube defects like spina bifida and associated urinary tract abnormalities, including posterior urethral valves, ureterocele, or ureteral duplication. Treatment for Vesicoureteral reflux (VUR) depends on the child's symptoms, age, and overall health. VUR can range from mild to severe, with higher grades indicating more significant reflux. Most children with grade 1 to grade 3 VUR typically do not require intensive treatment, as the condition often resolves spontaneously within five years. However, children

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with grade 4 or grade 5 VUR may require surgical intervention. During surgery, a flap-valve mechanism is created for the affected ureter to prevent the reverse flow of urine into the kidney. In severe cases, removal of the scarred kidney and ureter may be necessary.[8]

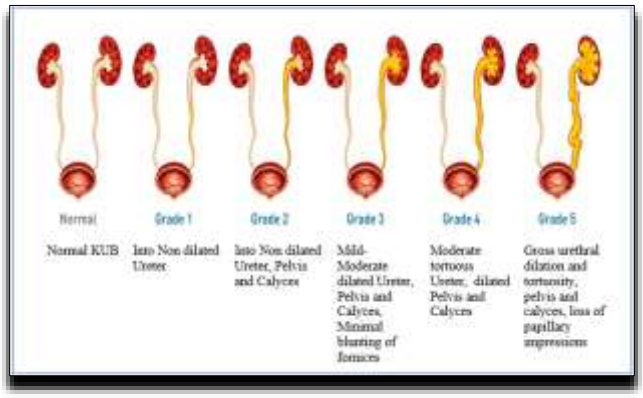

Figure 1: Vesicoureteral reflux (VUR)

2. Case Report

A 7 year old male patient presented to Pediatric-Urology Department of Multispecialty center with chief complaints of Fever associated with Chills , irregular voiding of urine with No H/O DM, HTN, BA, TB, and Epilepsy. Upon arrival his vitals read PR-90 bpm, BP-120/84 mmHg, SPO2-97%@RA, and BSA 0.664 m^2. O/E no signs of pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema. Personal history revealed, he had mixed diet, regular bowel, normal appetite and sounded sleep with complete immunization record. Patient had long H/O Recurrent fever associated with chills, palpable bladder, dribbling of urine, foul smelling and lack of appetite since 9 months of age. ANC (Ante-Natal Care) scan at ninth month of pregnancy determining congenital obstruction of urethra. Blood investigations like CBC was found to be increased and Serum Creatinine 1.07, Blood Urea Nitrogen (BUN) 63.27, Urine routine pus cells 10-11/hpf and LFT/RFT was found to be normal. He was advised for voiding cysto-urethrogram (VCUG) for recurrent UTI revealing Posterior urethral valves (PUV) and Grade III VUR, henceforth he had underwent PUV fulguration procedure at the age of one. Post-surgery investigations revealed Sr.Creatine and BUN was gradually decreased compared to pre surgical value. Patient got better and discharged. Apparently patient was normal for few years, later came to OPD with above said complaints Immediate USG- KUB revealed findings suggestive of bilateral gross hydroureteronephrosis with significant parenchymal thinning on the right side and upon said history Re VCUG was performed revealing Grade V-VUR .Additionally, blood investigations like CBC reported TLC on higher side 9870cells/cumm, Neutrophils 39%, Lymphocytes 50%. Urine routine NAD, RFT- Serum chloride on the higher side 106mmol/L, Serum creatinine on normal side 0.4mg/dl, Serum uric acid in lower end 2.8mg/dl, RBS 98mg/dl. PT on the higher side 18.0, INR on the higher side 1.39, Serology was non-reactive to HIV 1&2, HBsAG, HCV. ECG- sinus arrhythmia, premature atrial complexes, high voltage (left ventricular), T abnormality indicating an abnormal ECG. Patient was taken up for admission under urology team were B/L Laparoscopic Ureteric Re-implantation (**Figure 2**). was performed under GA on 10/2024- Cystoscopy B/L RGP done, B/L Uretericdilatation noted with B/L HUN, patient shifted to supine position, lab ports placed, pneumoperitoneum created (A), left colon mobilized and left ureter dissected upto VUJ (B), excised and left ureteric re-implantation done over the DJ stent using V LOC 3-0 (C), Right ureter dissected distally (D), right ureteric re-implantation done over the DJ stent using V LOC 3-0 (E), B/L DJ stent insitu, B/L Detrusorapphy done, closure done, dressing done with drain insitu along with Foley's catheter. Patient withstood procedure well. OT finding camera port and two other conventional ports made under vision. Right and left gonadal vein, ureter traced till bladder and re-implanted with intravesicle length of 5:1 and sutured over a stent, drain put and patient catheterized. Post-surgical antibiotic INJ Piperacillin+ Tazobactam 1g Q8H IV and supportive medication such as antacid, antiemetic, antipyretic was administered for 7 days. All vitals, I/O was monitored and recorded. Repeated Blood investigations were done post OP day 2 were CBC- TLC was on higher side 16250cells/cumm, Neutrophils 80%, Lymphocytes 10%, and Monocytes 10%, Eosinophil 0% all indicating lower scale. Serum Sodium on lower side 133mmol/L. Patient was continued with same antibiotic for 5days and patient started feeling better and was advised for discharge with antibiotic Tab Cefuroxime axetil + potassium clavulanate 250mg Q12H for 5 days alongside antacid and antipyretic for 5 days.

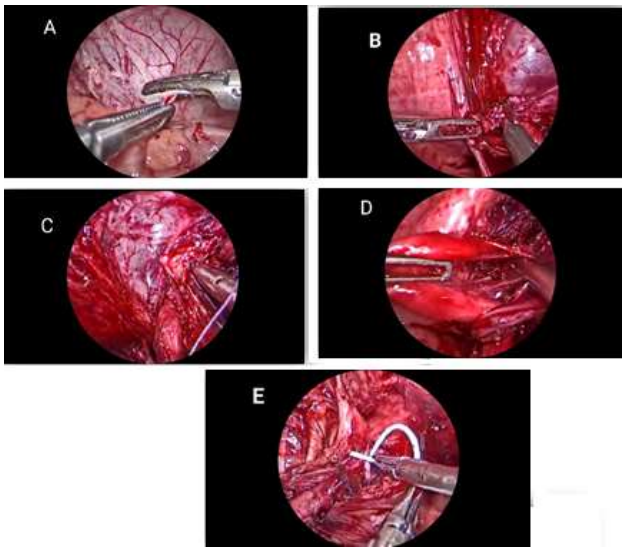

Figure 2: B/L Laparoscopic Ureteric Re-implantation

Patient was apparently well upto 3 days later started to experience fever, severe pain at post-surgical site and was taken to a secondary care hospital. Upon arrival to emergency OPD, his above chief complaints and past medical history

were noted and his vitals read temperature was 100.8̊ F, with Pulse rate of 150bpm, SPO2 98%@RA, BP 100/70mmHg and Foley's catheter in-situ. Blood investigations were advised and done CBC- TLC was on higher side 16540 cells/cumm, RBC count 5.30million/cumm, Neutrophils 77%, Lymphocytes 15%, and Eosinophil 0% all indicating lower scale. Serum creatinine on normal side 0.6mg/dl, Serum Sodium on lower side 133mmol/L. Patient was taken up for admission for medical management higher antibiotic Inj.Meropenem 250mg Q12H, antacid, antipyretic, nutrient powder was administered. All the vitals, I/O was monitored and recorded. Bladder wash was performed on fifth day of Foley's catheterization. Laparoscopic wound was clean, repeat blood test were done CBC – TLC 6000cells/cumm, Neutrophils 62%, Lymphocytes 43% on normal side, Serum creatinine 0.5mg/dl, Serum electrolytes- NAD, Foley's catheter was removed on day of discharge. Over the course of stay patient complaints came down and was symptomatically and clinically better. Patient was advised discharge with P/O antibiotic, antipyretic and nutrition powder. X RAY KUB was done which reported B/L DJ stent intact (**Figure 3**). Patient was advised for review after one week urology OPD, after six weeks for stent removal.

Figure 3: B/L DJ stent intact

3. Discussion

Posterior urethral valves (PUV) and recurrent Vesicoureteral reflux (VUR) represent significant challenges in paediatric urology due to their potential for recurrent infections, renal impairment, and the complexity of surgical management. This case presents a 7-year-old male with a complex history of congenital posterior urethral valves (PUV) and Vesicoureteral reflux (VUR) leading to recurrent urinary tract infections (UTIs) and progressive renal complications. The patient had a delayed initial diagnosis despite antenatal detection of urethral obstruction, which contributed to progressive upper urinary tract damage and eventual need for surgical intervention. The ante-natal scan at the ninth month of gestation detected congenital urethral obstruction; however, early postnatal intervention was not adequately pursued. The patient presented with persistent recurrent fevers, dribbling of urine, palpable bladder, and foul-smelling urine from infancy, suggestive of ongoing bladder dysfunction. The late diagnosis of Grade III VUR at one year, requiring PUV fulguration, indicates progressive renal deterioration.

Following initial surgical correction, the patient remained stable for a few years but later developed bilateral gross hydroureteronephrosis with significant parenchymal thinning, suggesting worsening VUR (progressing to Grade V) and chronic bladder dysfunction. The worsening reflux and structural kidney changes necessitated bilateral laparoscopic ureteric reimplantation. The bilateral laparoscopic ureteric re-implantation was performed successfully under general anaesthesia, with placement of Double J (DJ) stents to ensure urinary drainage. Postoperatively, the patient initially recovered well but developed fever and severe surgical site pain three days post-discharge, leading to readmission at a secondary care hospital. Investigations revealed elevated total leukocyte count (TLC), neutrophil, and hyponatremia, prompting initiation of higher intravenous antibiotics (Meropenem) and supportive management. A bladder wash was performed to address any potential catheter-related complications, and over the course of treatment, the patient's condition improved symptomatically and clinically**.**

This case underscores the critical role of Early Diagnosis-Antenatal detection of congenital abnormalities, such as PUV and VUR, should be followed by immediate postnatal evaluation and intervention to prevent progressive renal deterioration, Delayed diagnosis, as seen in this case, can lead to chronic kidney disease (CKD) progression and recurrent infections. Recurrent UTIs as a Red Flag-Persistent recurrent fevers and voiding dysfunction in infants should prompt early urodynamic studies (VCUG, renal ultrasound) to identify structural abnormalities. Postoperative Monitoring and Secondary Complications-Infectious complications following urological surgeries, especially in cases with pre-existing renal dysfunction, should be anticipated, Close post-discharge follow-up is essential to detect early surgical site infections, electrolyte imbalances, or worsening renal function. Long-term Prognosis-The patient remains at risk for future renal insufficiency due to prior recurrent UTIs and structural kidney damage, Continuous monitoring of renal function, BP control, and urological follow-up are crucial for long-term renal preservation.

The intricate interplay between congenital abnormalities, recurrent infections, and surgical interventions, underscoring the importance of timely diagnosis, appropriate surgical management, and vigilant postoperative care. The patient's low socioeconomic background and limited awareness of congenital

abnormalities contributed to a delayed diagnosis. Consequently, the postponement of surgical intervention led to disease progression and complications.

4. Conclusion

This case emphasizes the critical need for early diagnosis and prompt intervention in congenital urological conditions such as posterior urethral valves (PUV) and Vesicoureteral reflux (VUR). Despite antenatal detection, delayed postnatal assessment resulted in progressive renal damage, recurrent infections, and the eventual need for bilateral ureteric reimplantation. Postoperative complications necessitated readmission and intensive management, highlighting the importance of close monitoring. Limited healthcare awareness contributed to delayed treatment, reinforcing the need for early intervention and parental education. Long-term renal function surveillance and multidisciplinary follow-up remain crucial to preserving kidney function and ensuring better patient outcomes.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

7. Acknowledgement

None.

Reference

1. Hoberman A, Charron M, Hickey RW, Baskin M, Kearney DH, Wald ER. Imaging studies after a first febrile urinary tract infection in young children. *N Engl J Med*. 2003;348(3):195–202.
2. Capozza N, Gulia C, Heidari Bateni Z, Zangari A, Gigli S, Briganti V et al. Vesicoureteral reflux in infants: what do we know about the gender prevalence by age. *Eur Rev Med Pharmacol Sci*. 2017;21(23):5321–9.
3. Hiraoka M, Hori C, Tsukahara H, Kasuga K, Ishihara Y, Kotsuji F et al. Vesicoureteral reflux in male and female neonates as detected by voiding ultrasonography. *Kidney Int*. 1999; 55(4):1486–90.
4. Van Eerde AM, Meutgeert MH, De Jong TP, Giltay JC. Vesico-ureteral reflux in children with prenatally detected hydronephrosis: a systematic review. *Ultrasound Obstet Gynecol*. 2007;29(4):463-9.
5. Sharifiaghdas F, Narouie B, Soltani MH, Bonakdar Hashemi M, Ahmadzade M, Rouientan H et al. Investigating the clinical outcomes of bulking agent injection in comparison with modified Gil-Vernet anti-vesicoureteral reflux surgery in children with high-grade reflux (4 or 5). *Afr J Uro*l. 2023;29(1):67. Doi: 10.1186/s12301-023-00403-0
6. González E, Papazyan JP, Girardin E. Impact of vesicoureteral reflux on the size of renal lesions after an episode of acute pyelonephritis. *J Urol*. 2005;173(2):571–4. DOI: 10.1097/01.ju.0000151263.36909.9
7. Deshpande PV. Vesicoureteral Reflux Fixed; Kidneys Not Fixed!–A Case Series. *Indian J Kidney Dis*. 2024;3(2):61–5.
8. Vesicoureteral reflux (VUR) in children Cedars-Sinai. Available from: https://www.cedars-sinai.org/health-library/diseases-and-conditions--- paediatrics/v/vesicoureteral-reflux-vur-in-children.html

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