



Original Article

## Comparative Profile of Pediatric Patients with Lower Urinary Tract Symptoms (LUTS) with and without Posterior Urethral Valve

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### ABSTRACT

**Background:** Posterior urethral valves (PUV) are the most common cause of congenital lower urinary tract obstruction in boys and are an important cause of bladder dysfunction and chronic kidney disease. Children with lower urinary tract symptoms (LUTS) may have PUV or non-obstructive functional and neurogenic etiologies, but comparative data between these groups are limited, particularly from developing countries.

**Objectives:** To compare the clinical, urodynamic, radiological, and renal profiles of pediatric patients with LUTS with and without posterior urethral valves.

**Methods:** This retrospective observational study was conducted at multiple tertiary care centers in North India between July 2022 and June 2025. Male children aged  $\leq 18$  years presenting with LUTS and undergoing ultrasonography, voiding cystourethrogram, and urodynamic evaluation were included. Patients were grouped as PUV ( $n=20$ ) or non-PUV LUTS ( $n=53$ ). Renal function was assessed using serum creatinine and estimated glomerular filtration rate (eGFR), and chronic kidney disease (CKD) was staged according to KDIGO guidelines. Bladder morphology, post-void residual urine, vesicoureteral reflux (VUR), and urodynamic parameters were compared. Statistical analysis was performed using appropriate parametric and non-parametric tests, with odds ratios calculated for major outcomes.

**Results:** Children with PUV had significantly higher prevalence of recurrent urinary tract infections (65% vs 39.6%,  $p=0.048$ ), neurogenic bladder (35% vs 11.3%,  $p=0.019$ ), and bowel-bladder dysfunction (40% vs 18.9%,  $p=0.041$ ). PUV patients had significantly lower eGFR ( $73.8 \pm 28.4$  vs  $91.2 \pm 24.9$  ml/min/1.73 m<sup>2</sup>,  $p=0.014$ ) and higher CKD stage  $\geq 3$  (35% vs 9.6%,  $p=0.008$ ). Bladder wall thickening, diverticula, trabeculations, and post-void residual urine were significantly more frequent in PUV. VUR was present in 60% of PUV compared to 26.4% of non-PUV patients ( $p=0.007$ ), with bilateral reflux in 40% vs 11.3% ( $p=0.004$ ). Urodynamic studies showed markedly lower bladder capacity, poorer compliance, higher detrusor pressures, lower flow rates, and greater outlet obstruction in PUV (all  $p<0.001$ ). PUV was associated with 4–5-fold higher odds of CKD, VUR, bladder trabeculation, and neurogenic bladder.

**Conclusion:** Posterior urethral valves are associated with severe bladder and renal morbidity in children with LUTS, underscoring the need for early diagnosis, comprehensive urodynamic evaluation, and long-term multidisciplinary care.

**Keywords:** Posterior urethral valves; Pediatric lower urinary tract symptoms; Bladder dysfunction; Vesicoureteral reflux; Urodynamic study; Chronic kidney disease; Obstructive uropathy; Pediatric urology.

## INTRODUCTION

Lower urinary tract symptoms (LUTS) in the pediatric population represent a varied group of clinical manifestations that may indicate underlying structural, functional, or neurological abnormalities of the urinary tract. LUTS commonly include urgency, frequency, incontinence, weak urinary stream, and a sense of incomplete voiding; when persistent, these symptoms can lead to recurrent urinary tract infections, vesicoureteral reflux (VUR), and progressive renal damage if left untreated [1]. Among structural causes of LUTS in male children, posterior urethral valves (PUV) constitute a significant congenital disorder characterized by obstructive membranous tissue in the posterior urethra that interferes with urinary outflow [2,3]. PUV is recognized as the most frequent cause of lower urinary tract obstruction in male infants and accounts for a large proportion of pediatric obstructive uropathy worldwide [2,4].

Epidemiological estimates indicate that PUV occurs in approximately 1 in 5,000 to 1 in 8,000 live male births, with exclusive occurrence in males due to its developmental origin within the posterior urethral lumen [2,3]. The malformation arises from abnormal embryological development of the urogenital sinus, resulting in varying degrees of urinary obstruction, which leads to increased bladder pressures and altered urinary dynamics that may impact both bladder function and renal development. Affected infants often present early with symptoms such as poor urinary stream, urinary retention, and palpable bladder distension, while milder cases may not be diagnosed until later in childhood through recurrent LUTS or infection [1,3].

The physiological consequences of PUV extend well beyond mechanical blockage. Chronic obstruction elevates intravesical pressure, thereby impairing bladder compliance and detrusor contractility; such dysfunction may persist even after surgical correction of the obstructive segment [1]. Bladder dysfunction in children with PUV varies from detrusor overactivity to poor compliance and underactivity, contributing to ongoing LUTS, urinary incontinence, and an increased risk of upper tract deterioration [1]. Diagnostic urodynamic studies in this population often reveal significant abnormalities in bladder compliance, detrusor pressure, and bladder capacity, which have been linked to worse clinical outcomes [1].

One of the most serious long-term complications of PUV is chronic kidney disease (CKD). Despite early diagnosis and intervention, studies report that up to one-third of children with PUV may develop progressive renal dysfunction over time, reflecting a complex interplay between obstructive uropathy, primary renal dysplasia, and secondary effects such as VUR [4,5]. VUR—characterized by the retrograde flow of urine from the bladder to the upper tract—frequently coexists with both PUV and other forms of pediatric bladder dysfunction. The presence of VUR has been associated with an increased risk of recurrent pyelonephritis and renal scarring, thereby elevating the likelihood of long-term renal impairment [5,6].

In contrast to PUV, the broader category of non-PUV LUTS encompasses a wide range of functional and neurogenic etiologies. For instance, neurogenic bladder resulting from neurological conditions such as spina bifida or spinal cord injury disrupts normal synchronized bladder contraction and relaxation, leading to LUTS and potential upper tract involvement [7]. Another contributor to non-obstructive LUTS in children is bowel-bladder dysfunction, often seen in the context of chronic constipation; pelvic floor dyssynergia and altered voiding patterns in this setting may mimic or exacerbate urinary symptoms [8]. Functional disorders frequently require multidisciplinary management, including behavioral therapy, pharmacologic intervention, and careful monitoring through urodynamic evaluation to improve symptom control.

Despite the clinical importance of differentiating between PUV and non-PUV causes of pediatric LUTS, there remains a relative paucity of studies directly comparing these groups using comprehensive clinical, urodynamic, and radiological assessments. A clearer understanding of differences in bladder dynamics, renal outcomes, prevalence of VUR, and severity of symptoms in these populations would enable clinicians to tailor diagnostic and management pathways more precisely. Such comparative analyses can help distinguish reversible functional conditions from more serious congenital anomalies that require early intervention and long-term surveillance.

Currently, there is limited data from tertiary care centers in North India that contrasts pediatric LUTS due to PUV with LUTS of non-valve origin, particularly using a combination of clinical and urodynamic parameters. Given the high burden of pediatric urological disorders presenting at tertiary centers and the potential for delayed diagnosis in resource-limited settings, characterizing these subgroups in an Indian clinical population is both timely and relevant. Accordingly, this study—conducted at a tertiary care centre in North India aims to compare the demographic, clinical, urodynamic, radiological, and renal functional profiles of pediatric patients presenting with LUTS with and without PUV. Through such a comparative investigation, we intend to elucidate the distinguishing features and outcomes associated with PUV-related LUTS in the Indian pediatric population.

## MATERIALS AND METHODS

**Study Design and Setting:** This was a retrospective observational comparative study conducted at the Department of Urology and Pediatric Surgery of multiple tertiary care teaching hospitals in North India. The hospital serves as a major referral center for pediatric urological disorders from surrounding districts and neighboring states. The study included

pediatric patients evaluated for lower urinary tract symptoms (LUTS) over a three-year period from July 2022 to June 2025.

**Study Population:** All male children aged  $\leq 18$  years presenting with LUTS and undergoing comprehensive evaluation including ultrasonography, voiding cystourethrogram (VCUG), and urodynamic studies during the study period were screened for inclusion.

#### **Inclusion Criteria**

- Male children aged  $\leq 18$  years
- Presence of LUTS as per International Children's Continence Society (ICCS)
- Recurrent urinary tract infections (defined as  $\geq 2$  febrile UTIs or  $\geq 3$  symptomatic UTIs within the preceding 12 months)
- Availability of complete clinical, radiological, and urodynamic data
- Patients diagnosed (with cystoscopy) with posterior urethral valves (PUV) or non-PUV LUTS

#### **Exclusion Criteria**

- Female patients
- Children with prior lower urinary tract surgery (other than valve ablation in PUV patients)
- Children with congenital urinary tract anomalies other than PUV
- Incomplete or missing data on key study variables

**Sample Size Calculation:** The sample size was calculated based on the expected difference in the prevalence of chronic kidney disease (CKD stage  $\geq 3$ ) between children with PUV and those with non-PUV LUTS. Previous literature has reported CKD stage  $\geq 3$  in approximately 30–40% of children with PUV compared to 10–15% in non-PUV LUTS populations. Assuming an expected prevalence of 35% in PUV and 10% in non-PUV, with a two-sided alpha error of 5% and 80% power, the minimum sample size required was calculated to be 18 patients in the PUV group and 45 patients in the non-PUV group, giving a total minimum sample size of 63 patients. The final study included 73 patients, thus adequately meeting the calculated sample size requirement.

**Clinical, Radiological and Urodynamic Evaluation:** All patients underwent detailed clinical evaluation including documentation of presenting LUTS, history of urinary tract infections, bowel-bladder dysfunction, and neurogenic bladder. Bowel-bladder dysfunction was defined as the coexistence of lower urinary tract symptoms with constipation and/or fecal incontinence, as per International Children's Continence Society criteria. Neurogenic bladder was defined as bladder dysfunction secondary to an underlying neurological disorder or spinal pathology, confirmed clinically and/or radiologically. Renal-bladder ultrasonography was performed to assess kidney size, hydronephrosis, bladder wall thickness, trabeculations, diverticula, and post-void residual urine. VCUG was used to evaluate posterior urethral anatomy, bladder morphology, and presence and laterality of vesicoureteral reflux (VUR). Urodynamic studies were conducted according to standard pediatric urology protocols, recording maximum cystometric bladder capacity (MCBC), detrusor pressure at maximum flow (Pdet max), maximum flow rate (Qmax), bladder compliance, bladder outlet obstruction index (BOOI), bladder contractility index (BCI), presence of detrusor overactivity, and urinary leak.

**Renal Function Assessment:** Renal function was assessed using serum creatinine and estimated glomerular filtration rate (eGFR) calculated by the MDRD formula. Chronic kidney disease was staged according to KDIGO guidelines. Patients with an eGFR  $< 60$  ml/min/1.73 m<sup>2</sup> (corresponding to CKD stage  $\geq 3$ ) were classified as having clinically significant renal impairment for the purpose of this study. This cut-off was used to identify children with moderate to severe renal dysfunction and to compare renal outcomes between the PUV and non-PUV groups.

**Statistical Analysis:** Data were analyzed using SPSS version 26.0 (IBM Corp., USA). Continuous variables were expressed as mean  $\pm$  standard deviation, and categorical variables were expressed as frequency and percentage. Normality of data distribution was assessed using the Kolmogorov–Smirnov test. Continuous variables between the PUV and non-PUV groups were compared using the independent sample t-test or Mann–Whitney U test as appropriate, while categorical variables were compared using the chi-square test or Fisher's exact test. Odds ratios with 95% confidence intervals were calculated to assess the association between PUV and major outcomes such as CKD stage  $\geq 3$ , VUR, bladder trabeculation, and neurogenic bladder. A two-tailed p-value  $< 0.05$  was considered statistically significant.

**Ethical Considerations:** The study was conducted in accordance with the Declaration of Helsinki. As this was a retrospective record-based study, the requirement for informed consent was waived. Patient confidentiality was strictly maintained by anonymizing all data prior to analysis. No intervention beyond routine clinical care was performed.

## **RESULTS**

A total of 73 pediatric male patients with lower urinary tract symptoms (LUTS) were included in the analysis. Among them, 20 (27.4%) had posterior urethral valves (PUV) while 53 (72.6%) had non-PUV LUTS. Children with PUV had significantly higher prevalence of recurrent UTI, neurogenic bladder and bowel-bladder dysfunction than non-PUV LUTS

children. Table 1 shows comparative baseline and clinical profile of PUV versus Non-PUV patients with their statistical significance.

<b>Table 1. Baseline Demographic and Clinical Profile</b>			
<b>Variable</b>	<b>PUV (n=20)</b>	<b>Non-PUV (n=53)</b>	<b>p-value</b>
Age (years), mean $\pm$ SD	7.97 $\pm$ 6.47	7.72 $\pm$ 5.32	0.87
Weight (kg), mean $\pm$ SD	27.7 $\pm$ 20.1	24.2 $\pm$ 16.3	0.51
Recurrent UTI, n (%)	13 (65.0)	21 (39.6)	<b>0.048</b>
Neurogenic bladder, n (%)	7 (35.0)	6 (11.3)	<b>0.019</b>
Bowel-bladder dysfunction, n (%)	8 (40.0)	10 (18.9)	<b>0.041</b>

PUV patients had significantly lower eGFR and higher prevalence of CKD stage  $\geq 3$ , indicating worse renal impairment. Table 2 shows the comparative renal function profile of PUV versus Non-PUV patients with their statistical significance.

<b>Table 2. Renal Function Profile</b>			
<b>Parameter</b>	<b>PUV (n=20)</b>	<b>Non-PUV (n=53)</b>	<b>p-value</b>
Serum creatinine (mg/dl)	0.87 $\pm$ 0.39	0.82 $\pm$ 0.34	0.61
eGFR (ml/min/1.73 m <sup>2</sup> )	73.8 $\pm$ 28.4	91.2 $\pm$ 24.9	<b>0.014</b>
CKD Stage $\geq 3$ , n (%)	7 (35.0)	5 (9.6)	<b>0.008</b>

PUV children showed significantly more severe structural bladder damage and higher residual urine. Table 3 shows comparative ultrasound and bladder morphology of PUV versus Non-PUV patients with their statistical significance.

<b>Table 3. Ultrasound and Bladder Morphology</b>			
<b>Parameter</b>	<b>PUV (n=20)</b>	<b>Non-PUV (n=53)</b>	<b>p-value</b>
Bladder wall thickening, n (%)	14 (70.0)	17 (32.1)	0.004
Bladder diverticula, n (%)	8 (40.0)	6 (11.3)	0.006
Trabeculations, n (%)	15 (75.0)	19 (35.8)	0.003
Post-void residual (ml)	86 $\pm$ 54	42 $\pm$ 29	<0.001

PUV patients had more than double the frequency of VUR, especially bilateral reflux, compared to non-PUV LUTS. Table 4 shows incidence and type of vesicoureteral reflux in PUV versus Non-PUV patients with their statistical significance.

<b>Table 4. Vesicoureteral Reflux (VUR)</b>			
<b>VUR</b>	<b>PUV (n=20)</b>	<b>Non-PUV (n=53)</b>	<b>p-value</b>
Any VUR	12 (60.0%)	14 (26.4%)	<b>0.007</b>
Bilateral VUR	8 (40.0%)	6 (11.3%)	<b>0.004</b>

PUV children had severely impaired bladder capacity, compliance and flow with marked outlet obstruction. Table 5 shows urodynamic parameters.

<b>Table 5. Urodynamic Parameters</b>			
<b>Parameter</b>	<b>PUV (n=20)</b>	<b>Non-PUV (n=53)</b>	<b>p-value</b>
MCBC (ml)	178 $\pm$ 69	252 $\pm$ 84	< <b>0.001</b>
Compliance (ml/cm H <sub>2</sub> O)	10.6 $\pm$ 4.1	17.4 $\pm$ 6.2	< <b>0.001</b>
Pdet max (cm H <sub>2</sub> O)	68.2 $\pm$ 18.4	41.6 $\pm$ 14.2	< <b>0.001</b>
Qmax (ml/sec)	7.8 $\pm$ 3.4	14.2 $\pm$ 5.1	< <b>0.001</b>
BOOI	46.1 $\pm$ 19.3	23.8 $\pm$ 12.6	< <b>0.001</b>

PUV was associated with a 4 to 5 fold increased risk of renal impairment and bladder damage. Table 6 shows the odds of major morbidities in PUV.

Table 6. Odds of Major Morbidities in PUV		
Outcome	Odds Ratio (95% CI)	p-value
CKD Stage $\geq 3$	5.1 (1.5–17.4)	<b>0.008</b>
VUR	4.1 (1.4–12.3)	<b>0.007</b>
Bladder trabeculation	5.4 (1.8–16.0)	<b>0.003</b>
Neurogenic bladder	4.2 (1.2–14.1)	<b>0.019</b>

## DISCUSSION

In the present study, children with posterior urethral valves (PUV) demonstrated significantly worse renal function, more severe bladder dysfunction, and a higher prevalence of vesicoureteral reflux (VUR) compared with non-PUV children presenting with lower urinary tract symptoms (LUTS). These findings reinforce the understanding that PUV represents a distinct pathological entity characterized by chronic bladder outlet obstruction leading to progressive bladder and renal damage.

The significantly higher proportion of chronic kidney disease (CKD stage  $\geq 3$ ) observed in the PUV group is consistent with earlier reports highlighting the long-term renal vulnerability of these children. Bain et al. (2022) reported that a substantial proportion of boys with PUV develop progressive renal dysfunction, with many requiring long-term nephrological follow-up and some progressing to end-stage renal disease [9]. Similarly, Klaus and Lange-Sperandio (2022) emphasized that renal dysplasia, hypoplasia, and fibrosis are frequently present in PUV, which makes these kidneys intrinsically vulnerable to further damage from postnatal obstruction and infection [10]. The significantly lower eGFR values in the PUV group in our study support these observations.

Recurrent urinary tract infections and neurogenic bladder features were also significantly more common among PUV patients. Sarhan et al. (2021) demonstrated that even after early valve ablation, many children continue to have bladder dysfunction including detrusor overactivity, poor compliance, and incomplete emptying, all of which predispose to recurrent infections and renal scarring [1]. Similarly, Uwaezuoke (2022) highlighted that persistent bladder dysfunction in PUV is driven by detrusor hypertrophy, fibrosis, and altered neuromuscular signaling, contributing to long-term morbidity [11]. These mechanisms likely explain the higher rates of recurrent LUTS and neurogenic bladder observed in our PUV cohort.

Urodynamic abnormalities were strikingly more severe in children with PUV, as evidenced by significantly lower maximum cystometric bladder capacity, reduced compliance, higher detrusor pressures, and elevated bladder outlet obstruction index. These findings mirror those reported by Mallya et al. (2018), who showed that even after surgical relief of obstruction, many PUV patients continue to exhibit abnormal urodynamic patterns reflecting irreversible bladder remodeling [12]. This highlights that valve ablation, while necessary, is often insufficient to reverse established detrusor pathology, and long-term bladder management remains crucial.

The significantly higher prevalence of vesicoureteral reflux, particularly bilateral reflux, in PUV patients further explains their worse renal outcomes. Pellegrino et al. (2023) demonstrated that high intravesical pressures in PUV disrupt the anti-reflux mechanism of the ureterovesical junction, leading to secondary reflux and upper tract deterioration [13]. The presence of reflux has been shown to independently predict poorer renal survival in children with obstructive uropathy, which is consistent with our observation of a higher CKD burden in PUV patients.

Structural bladder abnormalities such as trabeculations and diverticula were also significantly more frequent in PUV. Hennis et al. (2012) systematically reviewed long-term outcomes of PUV and reported that chronic outlet obstruction leads to permanent bladder wall thickening, reduced elasticity, and formation of diverticula, which further impair voiding and promote urinary stasis [14]. These morphological changes explain the higher post-void residual volumes and reduced compliance observed in our cohort.

The odds ratio analysis in our study quantified the high-risk nature of PUV, showing a several-fold increased likelihood of CKD, VUR, bladder trabeculation, and neurogenic bladder compared to non-PUV LUTS children. These findings emphasize that PUV is not merely a cause of voiding difficulty but a progressive urological and nephrological disorder that requires lifelong surveillance and multidisciplinary management.

**Study Limitations:** This study has certain limitations. Being a retrospective, single-center analysis, it is subject to selection bias and may not fully represent the entire pediatric population with lower urinary tract symptoms. The reliance on existing medical records limited the ability to control for all potential confounding variables and precluded uniform follow-up for long-term renal and bladder outcomes. Additionally, the sample size, particularly in the PUV group, was relatively small, which may have reduced the power to detect some associations. Age-stratified renal outcomes were not analyzed, as this

was not a primary objective of the study, although age may influence the progression of renal dysfunction. Furthermore, the timing of renal function assessment in relation to valve fulguration was not standardized across patients; as the study spanned three years, renal function may have deteriorated over time or transiently improved after surgical intervention, potentially introducing temporal bias. Despite these limitations, the detailed urodynamic, radiological, and renal assessments provide robust comparative insight into the impact of posterior urethral valves in children.

## CONCLUSION

Children with posterior urethral valves represent a high-risk subgroup among pediatric patients presenting with lower urinary tract symptoms. In this tertiary-care cohort, PUV was associated with significantly worse renal function, higher prevalence of chronic kidney disease, more severe bladder structural damage, greater frequency of vesicoureteral reflux, and markedly impaired urodynamic parameters compared to non-PUV LUTS patients. Reduced bladder capacity, poor compliance, high detrusor pressures, and increased outlet obstruction highlight the irreversible bladder remodeling caused by chronic obstruction. The strong associations of PUV with CKD stage  $\geq 3$ , neurogenic bladder, VUR, and trabeculation emphasize the need for early diagnosis, comprehensive urodynamic evaluation, and long-term multidisciplinary follow-up. Differentiating PUV from functional and neurogenic causes of LUTS is crucial, as valve patients require more intensive surveillance and proactive management to prevent progressive renal and bladder deterioration.

## Declarations:

- The authors declare that there is no conflict of interest related to this study.
- No external funding was received for this research.
- The study was conducted in accordance with institutional ethical guidelines.
- All data used in this study were obtained from routine clinical records.
- The authors take full responsibility for the integrity and accuracy of the data and analysis.

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