Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 13, (2024)
https://www.jneonatalsurg.com

Spectrum Of Urodynamic Findings In Posterior Urethral Valve Patients Post Fulguration

Dr Tariq Ahmad Mir [1], Dr Shoaib Nissar [1], Dr Aejaz A baba [1], Dr Gowhar nazir mufti [1], Dr Raashid Hameed [1], Dr Waseem Jan[1], Dr Nissar A Bhat [1].

[1]Sheri Kashmir institution of medical science soura (SKIMS), Jammu and Kashmir.

Crossponding-Author:

Dr Tariq Ahmad Mir,

M.Ch Paediatric surgery SKIMS Soura,

Email ID : tariqmir78@gmail.com

Cite this paper as Dr Tariq Ahmad Mir , Dr Shoaib Nissar , Dr Aejaz A baba , Dr Gowhar nazir mufti , Dr Raashid Hameed , Dr Waseem Jan, Dr Nissar A Bhat (2024) Spectrum Of Urodynamic Findings In Posterior Urethral Valve Patients Post Fulguration .Journal of Neonatal Surgery, 13. 1957-1971

ABSTRACT

Background:Posterior urethral valve (PUV) is a common cause of urethral obstruction in children that can lead to end-stage renal disease (ESRD). Bladder-directed management (BDM) is a recent approach used to treat these patients. Our objective was to investigate the urodynamic abnormalities in PUV patients, analyse the changing patterns of urodynamic parameters in different age groups, establish a BDM strategy based on urodynamic study (UDS), and evaluate its impact on short-term functional outcomes.

Materials and Methods: Patients of PUV, aged 3 – 18 years at time of inclusion, who underwent adequate fulguration at any age were enrolled in the study and a baseline UDS was performed. BDM was started based on the UDS findings and patients were followed regularly. A repeat UDS was performed after 6-12 months of treatment and the findings compared using standard statistical tools.

Results: A total of 39 patients were included in the study. Initial UDS revealed hyperreflexia in 18 patients (46.15%), myogenic failure in 13 patients (33.33%), and small capacity bladder in 4 patients (10%). After BDM, improved bladder dynamics were observed in 22 patients (64.7%), worsening in 8 patients (23.5%), and no change in 4 patients (11.8%).

Conclusion: This study supports the hypothesis that there is a natural progression of valve bladder dysfunction towards detrusor failure. Additionally, early detection of bladder abnormalities through UDS and the implementation of BDM may help prevent progression to myogenic failure after puberty...

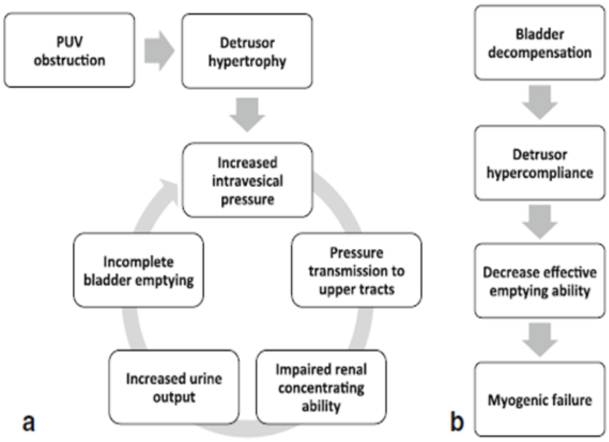
1. INTRODUCTION

Posterior urethral valve (PUV) is the most common form of congenital urethral obstruction with an incidence ranging from 1/3000 to 1/8000 male births [1,2]. Worldwide, a range of figures is quoted for the incidence of posterior urethral valves, with approximately 1:5000 live male births commonly quoted, with 50% progressing to ESRD within ten years.[3]

With the widespread use of prenatal ultrasonography, early diagnosis has allowed for timely intervention and monitoring of patients with PUV. The initial radiographic test to confirm the diagnosis of PUV is the voiding cystourethrogram (VCUG). Management of new-born babies with posterior urethral valves requires treatment plan as per the presentation of the patient. some patients present immediately in postnatal period with bilateral hydroureteronephrosis, they require immediate estab¬lishment of urinary catheter drainage from the bladder. After successful initial bladder drainage and when the patient's medical condition has stabilized, the next step is to fulgurate the valves. In few children urinary diversions are done prior to fulguration such as vesicos¬tomy, ureterostomy and pyelostomy. Preschool children who have underlying posterior urethral valve present with lower urinary tract symptoms such as urge incontinence and a poor urinary stream if they have not presented earlier. Following surgical treatment of the PUV, patients require close follow-up to detect and monitor for bladder dysfunction that may lead to renal injury.

However, after the obstruction is relieved, some of these patients continue to suffer because of bladder dysfunction associated with PUV. which contributes to CKD and potential progression to end-stage renal disease (ESRD) [4]. Management of the patient with PUV and bladder dysfunction needs urodynamic studies (UDS) to direct additional treatment. At present time, the term valve bladder (VB) encompasses all dysfunctional bladders in patients with PUV [5]. However, the term "valve bladder syndrome" was first used by Mitchell [6] to describe the pathophysiology were the underlying obstruction initiates cyclical events ultimately leading to bladder decompensation (Fig. 1). The intravesical obstruction leads to smooth muscle proliferation, detrusor hypertrophy, and compensatory increased detrusor voiding pressures. Transmission of the increased, detrusor pressures into the upper tracts subsequently causes renal tubular damage, a renal concentrating defect, and polyuria. As the bladder encounters increasing urine volumes, voiding results in a post void residual and the incomplete bladder emptying propagates the cycle. Progression of Valve bladder results in a large hypercompliant bladder, often losing the ability to generate effective emptying detrusor contractions, resulting in myogenic failure [7]. The bladder dysfunction is one of the important predictors which is seen in about 75 % of boys with PUV after valve fulguration with 50% progressing to ESRD within ten years..

Fig. 1 a Depiction of the cyclical events occurring during infancy and early childhood in patients with posterior urethral valves. **b** Cascade of events occurring later in childhood and adolescence as the bladder begins to decompensate leading towards myogenic failure



In recent years, the overall prognosis of PUV patients has improved. Several factors such as maternal oligohydramnios with severe renal dysplasia, Vesicoureteric reflex, bladder dysfunctions, persistent hydronephrosis have been identified as predictors of long-term outcome [8-13]. In simplistic scenario, valve fulguration (VF) should cure the disease, but this does not necessarily happen, and children continue to have persistent problems with long-term morbidity including end-stage renal disease. While there is no control over developmental renal dysplasia, a better understanding of bladder behaviour and its appropriate management can go a long way in maximising long-term outcome in PUV patients, which centres on maintaining bladder function. A poor understanding and inappropriate management of bladder dysfunction can result in early onset renal damage and unnecessary morbidity. It is important to identify the bladder dysfunction, so that timely, adequate and appropriate management can be offered to the child. Follow up of patients is very crucial to recognize the risk factors and complications to improve the quality of life, it can be done with the help of somatic growth, renal function test, and various imaging investigations, most important is urodynamic studies (UDS). Urodynamic study provides a useful tool to identify the bladder dysfunction and to test the efficacy of treatment as well as determine any refinements that is necessary to improve the outcome [14,15]. The bladder dysfunction shows a changing pattern with the age of the child in PUV patients and urodynamic studies are necessary to track these changes in PUV patients throughout the first two decades of life [16].

We need to understand bladder directed managements for fulgurated PUV patients, for that necessary understanding of urodynamic studies in PUV patients need to be thoroughly studied and practiced. It is important to know these bladder changes by urodynamic studies to modify the treatment and thus arrest the progression of bladder dysfunction. As our departments enrolls approximately twenty new cases of PUV per year.so it was important to conduct this study knowing the background facts about the disease pathology and ongoing consequences related to disease progression. This study was conducted to provide a better knowledge and a basic idea about the bladder directed management in fulgurated PUV patients.

We sought to study the urodynamic abnormalities in boys with PUV following fulguration to identify the impact of bladder directed management and to formulate a bladder directed management strategy based as per urodynamic findings for improved bladder outcome. This study was first of its nature which was conducted in the department of paediatric and neonatal surgery. we intended to look for the change in the urodynamic parameters after the fulguration and subsequent medical management of bladder dysfunction in PUV patients.

2. MATERIAL AND METHODS:

The study was conducted in the Department of Pediatric Surgery at our tertiary care center from march 2021 up to April 2023. A total of thirty-nine (n=39) patients of posterior urethral valve (PUV) who have been fulgurated and are attending OPD of our department were enrolled in the study. This was a prospective observational study. Approval was taken from institutional Ethical committee.

Inclusion criteria:

Children with PUV who underwent fulguration at any age and are above the age of 3 years and below 18 years at the time of study.

Children with PUV who have been adequately fulgurated as examined by cystoscopy and VCUG.

Exclusion criteria:

Children with PUV who are still on urinary diversion of any type.

Children with PUV having high grade unilateral/bilateral Vesico-ureteric reflux.

Children with PUV, who have undergone multiple surgeries.

A detailed consent was taken from the parents before proceeding for urodynamic study (UDS) and procedure was explained in detail. All cases fulfilling the inclusion criteria were entered into the study. Detailed history of recurrent UTI, frequent hospital admission, anthropometry, age at valve fulguration was entered and initial presentation for posterior urethral valve was noted. It also included USG KUB (ultrasound kidney, ureters and bladder) with special focus on grade of hydronephrosis, cortical thickness at poles, Post void residual urine (PVRU), bladder wall thickness apart from routine renal parameters like kidney size, echo pattern of bilateral kidneys, corticomedullary differentiation. upper tracts were evaluated by functional studies Which included DTPA (Diethylenetriamine pentaacetate). Functional profile in fulgurated PUV patients was evaluated by RFT (renal function tests). As a standard care a check micturating-cystourethrogram (MCU) was also done in all patients. Any child with culture positive urine examination was deferred till urine became sterile.

Medications such as anticholinergic's (oxybutynin) and alpha blockers were stopped 48 hours before UDS. For conducting UDS we used MMS [medical measurement system]- urodynamics machine and followed conventional fill urodynamics which included an intravesical catheter, a rectal catheter and surface electrodes positioned at perineum of patient. We used a 6F (Technomed) intravesical catheter which was connected to a computer system through a pressure transducer. This was used to measure intravesical pressure. Another catheter was placed in the rectum (specialist rectal catheter, Technomed) to measure intra-abdominal pressure. Detrusor pressure was calculated by subtracting the abdominal pressure measured by rectal catheter from the intravesical pressure measured by intravesical catheter. Sphincteric activity was measured with simultaneous perineal EMG recordings by placing patch surface electrodes. All the measured data was directly fed into a computer for analysis and display of graphical measurements. Room temperature normal saline was used for filling the bladder at 5-10% of the patients expected bladder capacity per minute as per his age. Before the urodynamic study, uroflowmetry was done in all patients.

Various urodynamic parameters which were noted in each patient included compliance, bladder stability, bladder capacity(observed/expected), detrusor pressure during voiding (sustained, waxing and waning or myogenic failure), detrusor sphincter dys-synergia and post void residue. Expected bladder capacity was calculated as [30×(age in years +2)] ml as per koffs formula. Stable bladder was defined, when the pressure during the filling of bladder does not rise more than 6-10 cm H_2O above baseline at the end of filling. Sustained detrusor pressure during voiding was defined when pdet was between 25 cm H_2O to 40 cm H_2O. small capacity bladder was defined when observed bladder capacity was less than 50% of expected bladder capacity and large capacity bladder was defined when observed bladder capacity was more than 150% of expected bladder capacity. Waxing and waning (unsustained) of detrusor pressure (impending myogenic failure) was defined when pdet during voiding does not sustain between 25 cm H_2O to 40 cm H_2O and drops down below the 25 cm H_2O in between. Detrusor overactivity (hyperreflexia)/Over active bladder (OAB) was defined as an involuntary detrusor contraction >15 cm of water from baseline during filling. Bladder underactivity (myogenic failure) was defined in patients who filled to >150% of their expected bladder capacity and have a poor (pdetmax <25 cm H_2O) or absent detrusor contraction during voiding. Poor compliance was defined when during filling; bladder pressure is equal to or more than 20 cm of water at expected bladder capacity. detrusor sphincter dys-synergia was labelled when detrusor muscle contraction occurred with concomitant involuntary urethral sphincter activation which was recorded on electromyography through surface electrodes applied on perineum.

After catheterization, any post void residue was noted. Post void residue was considered significant if the volume exceeds 20 % of the observed bladder capacity. After the procedure, oral antibiotics were given for 5 days. Patients were followed in pediatric surgery ward with ultrasonography (KUB) to see any post void residue (PVR) and upper tract dilatation. Urodynamic abnormalities were assessed after first UDS and Bladder directed management were followed for a period of six months.

During the follow up period detailed history of any recurrent UTI, urosepsis, frequent hospital admissions. blood pressure recordings were done; proteinuria was measured (spot P/C ratio) method was observed for analyzing proteinuria, anthropometry (weight/height percentile charting) and functional profile including RFT. Repeat upper tract functional studies including DTPA/DMSA was done. Detailed USG(KUB) was the integral part of repeat work up before check UDS, to see persistence of hydronephrosis, hydroureteronephrosis, diameters of distal ureter and PVRU. To check adequacy of valve fulguration a check MCU (micturating cysto-urethrogram) and a check cystoscopy was done. Check UDS was done after six to one year of follow up period and impact of bladder directed management on UDS was analysed. Pre and post bladder directed urodynamic studies were analysed and bladder dynamics were observed in terms of improving, worsened and status quo bladder dynamics respectively. The impact of urodynamic study after bladder directed management were studied and comparative results were obtained between two serial urodynamic studies

3. RESULTS AND OBSERVATIONS:

To evaluate urodynamic abnormalities in PUV patients of various age groups by studying urodynamic profile in PUV patients and to develop a bladder-directed management strategy based on UDS for better bladder outcomes and its effects on immediate functional outcomes as well, a study was conducted on Patients of posterior urethral valve (PUV) who had been fulgurated and were attending OPD at Department of Pediatric Surgery at our tertiary care center from March 2021 up to April 2023.Total of 39 patients of PUV who matched the inclusion and exclusion criteria were included in the study. Most of the patients had type 1 PUV (97.4%) as shown in table 1(a)

Table 1(a): Type of PUV

Type of PUV	**No. of patients (n=39)**	**Percentage%**
Type 1	38	97.4
Type 2	1	2.6
Type 3	0	0.0

Table 1(b): Age at Valve Fulguration (VF):

Twenty-three patients (59%) were fulgurated below the age of two years and remaining 16 patients (41%) were fulgurated after 2 years of age as depicted in table 1(b).

Age	**No. of patients (n=39)**	**Percentage%**
0-2yrs	23	59.0
>2yrs	16	41.0

Table (2): Baseline characteristics of patients studied

It shows the age group wise distribution of 39 patients. Over all 10 (25.6%), 23(58.9%), and 6(15.30%) patients belonged the age groups of 0-3 years, 3-6 years, and 6-12 years respectively at first UDS.

Age at first UDS	**No. of patients (n=39)**	**Percentage%**
0-3 YRS	10	25.6
3 YRS-6 YRS	23	58.9
6 YRS-12 YRS	6	15.3

3. Baseline UDS characteristics:

On initial urodynamic study we noted that thirty-one patients (79.5%) had normal observed bladder capacity, whereas 2 patients (5.1%) had increased bladder capacity and 6 patients (15.4%) had small capacity bladder. Detrusor overactivity was absent in twenty patients (51.3%) on their first UDS. Altered compliance was seen in twenty-four patients (61.5%). Poor detrusor contractions (PdetQmax<30 cm of H2O) were seen in eleven patients (28.2%). PVRU was noted in twenty patients (51.3%) as shown in table (3).

Table 3. Baseline UDS characteristics.

UDS BLADDER CAPACITY O/E	No. of patients (n=39)	Percentage%
Observed capacity>150%	2	5.1
Observed capacity <150 % & >50 %	31	79.5
Observed capacity<50 %	6	15.4
Detrusor overactivity		
Absent	20	51.3
Present	19	48.7
Compliance		
Decreased	16	41.0
Normal compliance	15	38.5
Increased	8	20.5
PDETQMAX		
PdetQmax<15 cm of H20	5	12.8
PdetQmax>15 cm of H20 &< 30cm OF H20	6	15.4
PdetQmax30 cm of H20 to 40 cm	8	20.5
Pdet Qmax>40cm of H20	20	51.3
PVRU		
Absent	19	48.7
Present	20	51.3
Sphinter activity		
Synergisim	6	15.4
Detrusor sphinter dysernergia	28	71.8
Sphincter no comments	5	12.8

4.UDS defined bladder abnormality:

The most common findings noted on initial UDS was detrusor hyperreflexia (overactive bladders). It was noted in eighteen patients (46.15%) followed by myogenic failure which was seen in thirteen patients (33.33%). Small capacity bladder was seen in four patients (10%). Four patients had sphincter dysernergia as shown in table (4).

Table 4. UDS defined bladder abnormality:

UDS detected abnormality	No. of patients (n=39)	Percentage%
Detrusor hyperreflexia (OAB)	8	20.51
Small capacity bladder	3	7.69
Myogenic failure	6	15.38
dyssynergia	4	10.25
Myogenic failure with dyssynergia	7	17.9
OAB with dyssynergia	10	25.64
small capacity bladder with dyssynergia	1	2.56

5. Bladder directed management:

About eleven patients (28.20%) patients received only anticholinergics as their bladder directed defined therapy as per initial urodynamic study. In addition to anticholinergic therapy one patient was prepared for augmentation cystoplasty. Six patients (15.4%) were put on clean intermittent catheterisation with addition of anticholinergics, night time drainage and biofeedback therapy. Twelve patients (30.76%) patients received clean intermittent catheterisation, alpha-blockers, night time drainage and biofeedback therapy. Eight patients (20.51%) received both anticholinergics and alpha-blockers apart from clean intermittent catheterisation with night time drainage and biofeedback therapy and one patient received (beta-3 adrenergic agonists) as per his initial Urodynamic abnormality as depicted in table 5.

Table 5 bladder directed management.

Bladder directed managment	No. of patients (n=39)	Percentage%
Anticholenergics	11	28.20
CIC, biofeed back therapy, night time drainage, anticholnergics	6	15.4
CIC, biofeed back therapy, night time drainage, alpha-blockers	12	30.76
Anticholenergics and prepartion for augmentation cystoplasty	1	2.6
CIC, biofeed back therapy, night time drainage, Mirabegron (beta-3 adrenergic agonists	1	2.6
CIC,biofeed back therapy,night time drainage,alpha-blockers/anticholinergics	8	20.51

6.Duration of follow up

Twenty-nine patients (74.4%) had completed one year of follow up post bladder directed therapy. Five patients (12.8%) had followed more than one year after their bladder directed therapy as shown in table 6.

Table 6: Duration of follow up;

Duration of Follow UP post BDM	**No.of patients (n=39)**	**Percentage%**
<6 months	5	12.8
6 months-1 year	29	74.4
1 year-2 year	5	12.8

7.serial urodynamic study post bladder directed therapy.

On serial UDS, normal bladder capacity was seen in thirty-one patients (91.2%) and three patients (8.8%) had decreased bladder capacity. DO (detrusor overactivity) was present in five patients (14.7%) after bladder directed therapy. Compliance was altered in eleven patients (32.10%). Detrusor hypercontractility was noted in fifteen patients (44.15%). PVRU was observed in fourteen patients (41.2%) as shown in table 7.

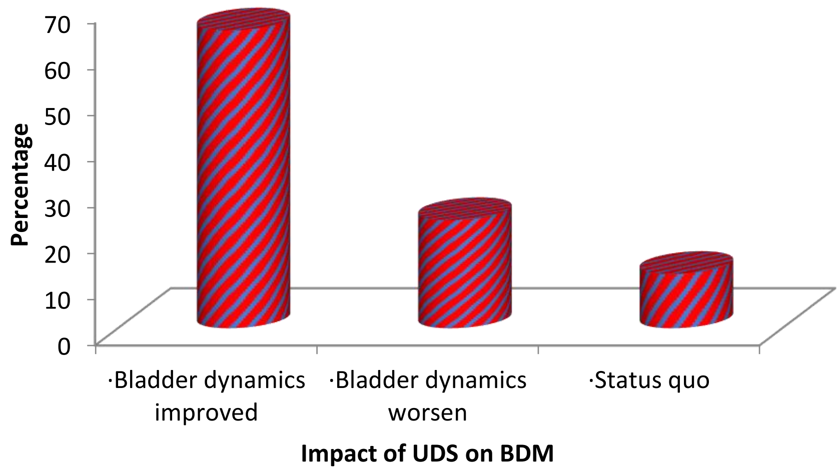
Table 7. Check UDS done after follow up period

bladder capacity	**N=34**	**Percentage%**
observed capacity>150%	0	0.0
observed capacity <150 % & >50 %	31	91.2
observed capacity<50 %	3	8.8
Detrusor overactivity		
Absent	29	85.3
Present	5	14.7
Compliance		
Decreased	5	14.7
Normal compliance	23	67.6
Increased	6	17.6
PDETQMAX		
Pdet Qmax<15 cm of H20	4	11.8
Pdet Qmax>15 cm of H20 &< 30cm OF H20	11	32.4
Pdet Qmax30 cm of H20 to 40 cm	4	11.8
Pdet Qmax>40cm of H20	15	44.1
PVRU		
Absent	20	58.8
Present	14	41.2
Sphincter activity		

SYNERGISIM	12	35.3
DYSSNERGIA	14	41.2
SPHINCTER no comments	8	23.5

8.Impact of Bladder directed management on serial UDS.

We observed that twenty-two patients (64.7%) had improved bladder dynamics after average follow up of nine months. whereas eight patients (23.5%) showed worsening of bladder dynamics after follow up period and in four patients (11.8%) a study-state was noticed as depicted in bar diagram below.



9.Correlation of serial UDS to see impact of BDM:

Fourteen patients who were fulgurated before 2 years of age out of total twenty-three patients showed improved bladder dynamics post BDM. Nineteen patients (86.36%) showed improved bladder dynamics with normal bladder capacity.DO (detrusor overactivity) was absent in twenty-one patients (95.45%) and in them bladder dynamics improved. We observed nineteen patients (86.36%) had normal compliance with improved bladder dynamics on check UDS. we also noted that the six patients (75%) out of total eight patients who had worsened bladder dynamics after BDM had hypo-contractile bladder on check UDS. In all eight patients, who showed worsened bladder dynamics after BDM, PVRU was significant in them as shown in table 9.

Table 9: correlation of serial UDS to see impact of BDM:

Variables	IMPACT OF UDS ON BDM			Total (n=34)	P value
	Bladder dynamics improved (n=22)	Bladder dynamics worsen (n=8)	Status quo (n=4)		
1.Age VF					
0-2yrs	14(63.6%)	4(50%)	3(75%)	21(61.8%)	0.671
>2yrs.	8(36.4%)	4(50%)	1(25%)	13(38.2%)	
2.Age at first UDS					

0-3 YRS	8(36.4%)	2(25%)	0(0%)	10(29.4%)	0.630
3 YRS-6 YRS	10(45.5%)	5(62.5%)	3(50%)	18(53%)	
6 YRS-12 YRS	4(18.2%)	1(12.5%)	1(50%)	6(19.3%)	
3. bladder capacity					
Observed capacity<50 %(small capacity bladders)	3(13.6%)	1(12.5%)	2(50%)	6(17.6%)	0.068+
Observed capacity <150 % & >50 %(normal capacity bladders)	19(86.4%)	6(75%)	1(25%)	26(76.5%)	
Observed capacity>150%(large capacity bladders)	0(0%)	1(12.5%)	1(25%)	2(5.9%)	
4.Detrusor overactivity					
Absent	21(95.45%)	5(62.5%)	3(75%)	29(85.3%)	0.065+
Present	1(4.5%)	3(37.5%)	1(25%)	5(14.70%)	

5.compliance					
Increased	2(9%)	3(37.5%)	1(25%)	6(17.645)	0.020*
Normal	19(86.36%)	3(37.5%)	1(25%)	23(67.64%)	
Decreased	1(4.5%)	2(25%)	2(50%)	5(14.70%)	
6.PdetQmax					
Hypocontractile	4(18.18%)	6(75%)	3(75%)	13(38.23%)	0.005**
Hypercontractile	18(81.81%)	2(25%)	1(25%)	21(61.76%)	
7.PVRU					
absent	19(86.36%)	0(0%)	1(25%)	20(58.82%)	<0.001**
present	3(13.63%)	8(100%)	3(75%)	14(41.18%)	
7.sphincter activity					
DSD	4(18.18%)	7(87.50%)	3(75%)	14(41.17%)	0.006**
Synergism	11(50%)	1(12.50%)	0(0%)	12(35.29%)	
no comments	7(31.82%)	0(0%)	1(25%)	8(23.52%)	

4. DISCUSSION

Morgagni first described obstruction by a posterior urethral valve in 1715. In the late 1970s, Mitchell (6) observed the relationship of the bladder to upper tract dilatation in such patients, and in 1980 he coined the term [a]valve bladder syndrome (VBS). VBS was described as persisting or progressive severe hydroureteronephrosis with no residual valve obstruction. Posterior urethral valve is primarily a disease of the bladder outlet, and the bladder possibly is a mere 'innocent victim' of the raised outflow pressures. Persistent detrusor dysfunction is a known entity which haunts patients of PUV long after treatment of the valves and hastens deterioration of renal function. Detrusor urothelium and smooth muscle cells act as mechano-receptors and mechanical stress induced by BOO gets mechano-transduced through gene expression and protein synthesis into detrusor hypertrophy. Also, such mechanical stress affects local neuronal pathways causing changes in threshold of micturition reflex and hence overactivity/ underactivity. Thus, the filling phase abnormalities observed in follow up patients of PUV may actually be related to persistently high outlet resistance.

Some degree of raised outflow pressures might persist in some children with PUV which may be caused due to bladder neck hypertrophy, urethral strictures or residual valve leaflets. Bladder-directed managements become important after serial urodynamic studies have recognized that management of bladder dysfunction are the only modifiable factors to avoid renal failure. The importance of bladder dysfunction in patients with PUV suggests the possible role of UDS evaluation yielding a higher incidence of abnormal bladder more so when there are clinical symptoms. It is also prudent to understand that normal urodynamic findings in the PUV patients do not rule out renal deterioration. Progressive renal tubular damage during childhood leads to inability to conserve sodium and free water and can lead to nephrogenic diabetes with large urine volumes. These exaggerated urine volumes can overwhelm the functional ability of the bladder, especially in one with limited functional potential.

In our study, a total of twenty-three patients (59%) were fulgurated before the age of 2 years as compared to sixteen patients (41%) who were fulgurated after 2 years of age. we have seen improved bladder dynamics after UDS based bladder directed management in terms of improved bladder capacity, normal compliance, good detrusor contractions and insignificant post void residual urine (PVRU). which makes us realize that early valve ablation would protect the bladder and allow normal cycling, which helps in bladder healing. Our study observed the same findings as noted by Ashwin mallya et al (20) about the valve fulguration (VF) which were done before 2 years of age. We found similar urodynamic outcomes in the group with early VF. Our study group is unique as sixteen patients (40%) underwent VF later as compared to other published series. This gives an insight into the lack of early detection and intervention and reflects the delayed referral and management as is commonly seen in developing countries. In addition, it also gives us a chance to study whether acceptable outcomes are achieved with later VF and to study the natural history of children with PUV on a longer follow up which has to be taken up in future. We observed that boys with VF at <2 years of age have better urodynamic profiles than those with fulguration over 2 years of age. Youssif et al (27), had compared the clinical and urodynamic outcomes of neonatal VF and compared them with those who underwent fulguration after the age of 1 year. They found that there was better resolution of hydroureteronephrosis and bladder function on UDS in the group with neonatal VF. These observations were consistent with our study group where early resolution of hydroureteronephrosis was noted on follow up period.

In our series detrusor overactivity was seen in nineteen patients (48.7%) before any bladder directed management. Significant improvement was observed after UDS based bladder directed therapy. where, only five patients (14.7%) continued with detrusor overactivity on their average follow up of nine months. Detrusor overactivity in our study was noted in young children less than five years of age and with age more than six years we noted over distended bladder with increased bladder capacity. Ziylan et al (28). noted detrusor overactivity in 50% of patients before 5 years of age however, it decreased to 20% after age of 10 years. As per De gennaro et al (26), they reported in a series of urodynamic studies in patients with PUV at different ages, 40% had instability and 37% had hypo contractility when assessed at 4 to 7 years of age. On the contrary, in patients aged 8 to 12 years, instability was present in 33% and hypo contractility in 45%. In a series of urodynamic studies in patients with PUV at different ages done by sajad et al (21), they had similar urodynamic findings. As 40% had bladder instability and 37% hypo contractility when assessed at 4-7 years of age. Lal et al (29) noted that small, hypo compliant, and unstable bladders were almost always seen in prepubertal boys and in the first 5 years following un-diversion, where, as large hypotonic bladders with impaired contractility were seen in older boys. These results seem to confirm that bladder dysfunction in boys with PUV changes from the unstable/hyper contractile bladder found in infants to hypo contractility in childhood, which may deteriorate with age, leading to a true myogenic detrusor failure after puberty.

In our series, decreased compliance, detrusor overactivity, normal bladder capacity, hyperreflexia was seen in young children and poor compliance, over distended bladder with increased capacity, waxing and waning (un-sustained) of detrusor pressure, myogenic failure was seen in older children. Serial urodynamic evaluation should be undertaken in all boys after valve ablation. In this way any impairment in bladder compliance, storage pressures and detrusor contractile power which can deteriorate the upper tracts, renal damage and early onset of renal failure can be detected early even if the boys do not complain of urinary incontinence or voiding problems. As in our series Altered compliance was seen in twenty-four patients (61%) on initial UDS, post BDM altered compliance was seen in eleven patients (32%). This observation was statistically significant (p valve <0.020). About two third of the patients had evidence of poor compliance on UDS. The reports from literature were variable ranging from 26 to 74.2% (22). So, observation of poor compliance in the present study falls in the mentioned range. The reason for the wide range of poor compliance reported in literature is multifactorial. There may be a factor of selection bias because UDS was done by most authors on those patients of PUV who had symptoms (30).

In our study, the main urodynamic pathology at the early stage was hypo compliance with decreased bladder capacity. This urodynamic abnormality was seen in twenty-two patients (56%), Decreased bladder capacity was observed in four patients (18%) and these children were younger than 4 years of age. Hypo compliance was present in eighteen patients (82%) of children below the age of 4 years. Among the four patients with small and hypo compliant bladders in the present series, two patients will require augmentation cystoplasty during the follow-up period. Although the literature reports that those presenting after 5 years of age represent the milder end of the spectrum of the disease with presenting features of voiding disturbance and well-preserved upper tracts (31). this was not the case in our series. All 4 patients in the small bladder capacity group were the ones treated after the age of 2.5 years. None of the patients treated before 2.5 years of age required bladder augmentation, even if they had low bladder capacity and hypo compliance, and all these patients were managed by conservative measures, such as antibiotic prophylaxis, anticholinergic drugs and clean intermittent catheterisation.in a study done by Emir et al (25). There main urodynamic pathology at the early stage was hypo compliance and decreased bladder capacity. Decreased capacity was observed in 64% of the children younger than 4 years and in 50% of the children older than 4 years, and hypo compliance was present in 57% of children below the age of 4 and in 41.6% of the children older than 4 years. Whereas, we noted that 82% of patients had hypo compliance and 18% had small capacity bladder. This contrasting urodynamic abnormality was noted because most of our patients were fulgurated before age of 2 years and some patients were even fulgurated in neonatal life. so, hypo compliance was our main urodynamic abnormality then the small capacity bladder. Among the 10 patients with small and hypo compliant bladders in their series, 5 patients required augmentation cystoplasty during the follow-up period. The small capacity bladder patients in our series are on follow up for requirement of augmentation cystoplasty as no one has undergone for the same procedure yet. we need to follow up these small capacity bladders with long duration of follow up with conservative managements including anticholinergic medications and to assess them on serial urodynamic study for an augmentation cystoplasty.

Bladder contractions during peak flow of urine (PdetQmax) showed a significant improvement on bladder dynamics post BDM. In our study a cut off value of < than 25 cm of H20 was taken for hypo-contractile bladder, although bladder contractility index (BCI) was not taken into the consideration. An index for bladder contractility was derived from the contractility groups that Schaffer et al described (32), that is strong, normal, weak, and very weak. The slope of Schaffer's lines is given by the formula: PdetQmax +5Qmax and is called BCI. A strong bladder contractility is suggested by a BCI of >150, normal contractility by BCI of 100-150, and weak contractility by BCI of < 100.Patients with BCI < 100 have poor clinical outcome and continue to have persistent LUTS. In a previous study, a group of patients with BCI < 100 required prolonged catheterization or need of CIC after surgery [24]. But these parameters have been used only for adult populations. The paediatric population has varying voiding pressures across different ages. The voiding pressures in infancy tend to be higher than those in older children. Further, a Qmax of <12 mL/s is normal for a 7-year-old boy but low for a 15-year-old boy. It could be inferred that when BCI is calculated for children, it could be lower than the typical adult value as Qmax can be lower, but normal. As BCI has not yet been formulated for children, stratification of contractility based on BCI is difficult.

In our study, fifteen patients (38%) had poor detrusor contractions, (hypo-contractile bladders) which was comparable to that observed by Emir et al (25). Hypo contractile detrusor was also seen in 36% of patients in Lal et al (29). In our study, impact of UDS after bladder directed therapy seen on an average follow up of nine months, where Twenty-two patients showed improved bladder dynamics, these patients when described in terms of peak detrusor contractions, eighteen patients (81%) had hypercontractile bladder and only four patients (19%) had hypocontractile bladder. Out of total eight patients who had worsened bladder dynamics on follow-up six patients (75%) had hypocontractile bladder during peak flow and this was statistically significant (p value=0.005).Despite relief of anatomical obstruction resolving hypercontractility should be expected during the first year of life .The resolving instability and decreased high voiding detrusor pressure during infancy does not suggest that bladder dysfunction becomes normal in childhood, but that it changes with the progressive (`covert' hypocontractility) development of detrusor myogenic failure and functional obstruction**.** In study by De gennero et al (26), seven of the nine asymptomatic boys with a normal urodynamic pattern changed with time toward hypocontractility. The pdetmax decreased in these patients from the first urodynamic evaluation to the follow-up. This indicator of detrusor contractility was significantly different either in the follow-up of boys first evaluated as infants or during childhood, while the mean voiding detrusor pressure did not change significantly in older boys who already had unsustained detrusor voiding contractions. The development of the prostate gland may contribute to outlet obstruction, increasing urethral resistance with subsequent emptying difficulties and urinary overflow incontinence. In this situation the high urinary output of patients with chronic renal failure should be a determining factor in the development of overdistension in functionally obstructed bladders. In their series, all patients followed after puberty showed hypocontractility but only those with chronic renal failure had an overdistended bladder.so our observations are in coherence when discussed in terms of hypocontractile bladder seen with worsened urodynamics.

Classical teaching states that PUV bladder undergoes remodelling over time and passes through phase of small capacity hypo-compliant detrusor in young age to large capacity bladder before puberty and ultimately myogenic failure. As per these studies, three patterns of bladder dysfunction overlap considerably, hyper contractility, unsustained voiding contractions (hypo contractility) and myogenic failure with true bladder atony. Urodynamic patterns change with age and that hypo contractility was probably a step toward detrusor decompensation, which may lead to myogenic failure with time. Therefore, myogenic failure is the likely an end-stage of bladder dysfunction in boys with PUV. In our series sixteen patients (41%) had decreased compliance, and six patients (15.4%) had small bladder capacity. we found that this urodynamic abnormality was consistently seen in young children aged less than three years and fulgurated before 2 years of age. many of them were fulgurated in neonatal life. In our study group, thirteen patients (38%) had poor detrusor contractions, (hypo-contractile bladders) which was comparable to that observed by Emir et al (25). (38%). Hypo-contractile detrusor was seen in 36% by Lal et al (29) study.in our study group thirteen patients (38%) were hypo-contractile, had poor detrusor contractions and significant PVRU was noted in them, which brought our attention towards early myogenic failure. This pattern of urodynamics was profound in older children aged more than six to twelve years.

As we noted a changing urodynamic pattern from small capacity bladder with detrusor overactivity (overactive bladder) and decreased compliance to a state of increased bladder capacity with the increasing age and hypercontractility (hyperreflexia bladder). Finally reaching to over destinated bladder status with significant post void residual urine to a state of myogenic failure with poor detrusor contractions which are noted in older children. Similar changing urodynamic pattern were noted by Holmdahl et al (33). They correlated the patients' age with these three classical patterns to determine whether bladder dysfunction changed during infancy, childhood and adolescence. They reported that there is uniform pattern of initial hyper contractility and low bladder capacity which changes with resolving hyper contractility and increasing bladder capacity in first year of life. Subsequently, the same authors reported urodynamic studies in boys with PUV aged less than `15 years, comparing them with those in post pubertal patients. They found a decreasing prevalence of instability and more patients with un-sustained voiding, leading to an over distended bladder after puberty. Based on these observations, Holmdahl et al suggested that the three urodynamic patterns change with time, towards the detrusor decompensation and myogenic failure.

After UDS based BDM, twenty-two patients (64%) showed improved bladder dynamics in terms of compliance, bladder capacity, detrusor contractions, PVRU and eight patients (23%) worsened after BDM. Whereas, four patients (11%) remained in a study state. All these eight patients who showed worsening on repeat UDS, had significant PVRU, this observation was statistically significant, (p value<0.001). The presence of significant PVRU was not alone responsible for their worsened urodynamics. It was associated with overdistended bladder, increased bladder capacity, hyper compliance with poor detrusor contractions ultimately progressing towards myogenic failure. In our study population the incidence of patients with myogenic failure with or without detrusor-sphincter dyssynergia was seen in thirteen patients (33%). This subset of patients had their first UDS done after the age of six years. Significant PVRU was seen in twenty patients (51.3%) in total and about half of them had myogenic failure on initial UDS. Five patients had significantly poor detrusor contraction (pDetQmax<15cm of H20). All of these five patients constituted about 12.8% of total study population with significant PVRU and they had myogenic failure.

Significant PVRU alone cannot predict the development of myogenic failure on ongoing serial urodynamic assessments but the presence of increased bladder capacity (over distended bladder) with poor detrusor contractions are also noteworthy for calling a bladder in a state of myogenic failure. Our study showed only six patients (15%) had myogenic failure at initial UDS, where as in many studies' incidence of myogenic failure ranges up to 30%. This conflict was seen because most of our patients were in the early pre-pubertal age group and only two of our patients were of pubertal age. As mentioned above they tend to develop myogenic failure at or after puberty. In our study, both increased bladder capacity and high residual urine were observed in 2 patients who were older than 4 years of age. As our patients mostly belonged to the prepubertal period, we observed less decompensation findings. It is known that voiding dysfunction may occur after valve ablation in 13 to 38% of patients and may or may not be reversible even after the relief of obstruction (34).

In our series, the urodynamic investigations were performed at an average of four years after fulguration and all the children had urodynamic pathologies. As boys approach puberty, hypertonicity and hyperreflexia decrease. Myogenic failure is more common after puberty and the cause seems to be nephrogenic diabetes insipidus with abnormal patterns of voiding with large residual urine. Koff et al (35). stated that polyuria, impaired bladder sensation and residual urine volume prevent the bladder from becoming normal after valve ablation, and progressively reduce functional bladder capacity, inducing bladder overdistension. The resulting functional obstruction leads to bladder decompensation, upper tract dilation and renal injury, which characterize valve bladder syndrome (VBS). The presence of significant PVRU in fourteen patients (41.2%) in their series after bladder directed managements, as demonstrated by the follow up urodynamic studies. Approximately 40% of these patients have developed chronic renal failure and this supports the statement that even after valve fulguration half of these patients will develop chronic kidney disease by the age of ten years.

Dyssynergia in our study was only observed on urodynamic tracings of patients, we couldn't follow this on video urodynamic, perineal needle electrodes and on urethral pressure flow studies. As, all of these modalities were not available with us. We used surface electrodes on the perineum of our patients for EMG recordings. Contraction of the pelvic floor or urethral sphincter during voiding in a neurologically intact patient should be characterized as "dysfunctional voiding" rather than DSD. Although needle electrodes placed into the anal sphincter are considered the gold standard for EMG recordings, perineal placement of needles can be challenging and painful for a patient. Perineal surface electrodes are easier to place and more comfortable, but the signal may be confounded by lack of adhesion to the skin or by the amount of fat between the muscle and the electrode.

Detrusor sphincter dyssynergia (DSD) is the urodynamic description of bladder outlet obstruction from detrusor muscle contraction with concomitant involuntary urethral sphincter activation. DSD is associated with neurologic conditions such as spinal cord injury, multiple sclerosis, and spina bifida and some of these neurogenic bladder patients with DSD may be at risk for autonomic dysreflexia, recurrent urinary tract infections, or upper tract compromise if the condition is not followed and treated appropriately. It is diagnosed most commonly during the voiding phase of urodynamic studies using EMG recordings and voiding cystourethrograms, although urethral pressure monitoring could also potentially be used. Yalla et al (36). described how concomitant urethral narrowing during VCUG correlated with increased sphincter activity on EMG in the setting of a detrusor contraction in DSD patients. Blavias et al (37). also published several reports on using fluoroscopy during urodynamics to assess sphincter function and defined DSD on fluoroscopy as a dilated posterior urethra obstructed by the external sphincter. However, a diagnosis of DSD can sometimes be challenging due to technical limitations of the study modalities. In our study population, as already mentioned we had used perineal surface electrodes for EMG recording during voiding phase and on these findings categorising patients into sphincter dyssynergia and to follow them post bladder directed managements as per initial UDS is crude in itself. For proper definition of DSD, we need a battery of modalities involved in detecting this particular urodynamic abnormality to allow a better understanding about the disease prognosis in future trails.

5. CONCLUSIONS:

PUV is one of the most common congenital obstructive uropathy of the posterior urethra in male having deleterious effects on upper urinary tract in long term follow up. Hence, regular follow up till adolescent is mandatory in all the cases. Amongst the various parameters of follow up both MCU and UDS followed by either cystoscopy for surgical management or medical management for bladder dysfunction are the important modulating factors for achieving better long-term outcome in such cases. Urodynamic pattern shows considerable overlap during childhood and through adolescence with hyper contractility generally seen in young children after the fulguration and hypo contractility and myogenic failure in older boys. The urodynamic follow-up of the present patients supports the hypothesis of a natural development of valve bladder dysfunction toward detrusor failure. The early detection of `covert' hypo contractility and the possibility of early bladder rehabilitation might be helpful in preventing emptying difficulties secondary to functional obstruction and the progression of hypo contractility to a state of detrusor myogenic failure and overdistension after puberty

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