Objective: The objective of the study was to share our experience of management of posterior urethral valve (PUV) and to suggest a paradigm to impede upstaging of chronic kidney disease (CKD) and prevent end-stage renal failure (ESRF).

Patients and Methods: We have treated 332 patient of PUV from March 2005 to April 2016, Of which 272 case records had adequate data to be analyzed. The mean age was 2.48 years (range: 1 day–18 years). We did primary fulguration in 231 patients, of which five patients needed bilateral ureterostomy for obstinate high creatinine level. The remaining 36 patients had primary fulguration done elsewhere.

Results: The mean duration of follow-up was 7.8 years (range 3–14 years). In the end of this study, 10 patients had down staging in CKD, 36 patients had up staging in CKD, and 9 patients ended in ESRF (3.8%).

Conclusions: Detection of deterioration of renal function with creatinine clearance along with identifying the causes of deterioration and necessary interventions would help to arrest upstaging of CKD otherwise that might end in ESRF. From this study and reviewing the literature, we presume that the rhabdosphincter spasm underneath actually renders bladder outlet obstruction, and cusps of PUV, particularly in neonates, amplify the obstruction, following that bladder outlet obstruction cascades detrusor hypertrophy, bladder neck hypertrophy/obstructions, and ureterovesical junction obstruction/reflux, causing gradual damage to the bladder and upper tract and deterioration of renal function as a consequence.

Keywords: Albumin creatinine ratio, bladder neck incision, chronic kidney disease, creatinine clearance, detrusor pressure, end stage renal failure, posterior urethral valve, rhabdosphincter spasm, ureterovesical junction obstruction, urodynamic study, vesicoureteral reflux, voiding cystourethrogram

INTRODUCTION

In recent years, the knowledge of posterior urethral valve (PUV) has been considerably upgraded through urodynamics studies, adaptation of specific pharmaceutical measures, sensitive imaging, and updated definition of chronic kidney disease (CKD). Nevertheless, published literature indicates that PUV ends in end-stage renal failure (ESRF) in around 24%–50%.[1-3] of patients treated due to upstaging of CKD. This disheartening fact led us to carry out this retrospective scrutiny to find the hidden facts and factors aggravating the deterioration of renal functions.

Our retrospective study was supplemented by the information acquired from the changed perspectives mentioned above. Our aim was to detect the basis of deterioration of renal function which would enable us to prevent upstaging of CKD. Primary endpoint of this study was reserved for the detection of upstaging...
of CKD and as a logical extension; the subsequent secondary endpoint was ESRF. We monitored stages of CKD with creatinine clearance (CCr) during follow-up.

This study derived insight from cumulative experiences of three authors. All of them managed patients of PUV according to the conventional and recent evidences available in literature. We compared the outcome of three cohorts managed by three different surgeons.

**Patients and Methods**

A total of 332 patients with PUV were treated at our center from March 2005 to April 2016. The age ranged from 1st day of life to 18 years. Of 332 patients, 272 had adequate data those could be analyzed [Table 1] including the analysis of our recorded videos. Patients with a minimum 3 years of follow-up with adequate data were included in this study. The mean age was 2.48 years (range: 1 day–18 years); 36 of 272 patients had primary management and fulguration done (“lateral entry”) elsewhere. Three neonates died of infection due to catheter and voiding cystourethrogram (VCUG) done before referral to our center were also excluded. Institute Ethical Committee approved this study.

The diagnosis of PUV was made either by antenatal ultrasound (USG) or postnatal USG along with lower urinary tract signs such as straining, dribbling, and palpable bladder. One-third of patients were diagnosed at the antenatal period. The second and third authors preferred VCUG in addition.

The first author skips VCUG following availability of endo camera and he relies on endovission. Prior to cystoscopy, he preferred repeat USG with a catheter in situ following 48 h of continuous catheter drainage of urine from bladder and ureters to confirm/exclude obstruction at ureterovesical junction. He also assessed rhabdo sphincter spasm (RSS) [Videos 1 and 2] during cystoscopy and used to do valve fulguration at 5 and 7 O’ clock only using Bugbee electrode in 91 patients under general anesthesia. He assessed the nature of the bladder neck, trabeculations, sacculations, diverticulations; and ‘trabeculations in posterior urethra’ (TIPU) and opted for bladder neck incision (BNI) in selected patient as per the practice mentioned in Table 2. The first author performed cystograms, not VCUG, after 48 h of fulguration of PUV, before removal of urethral catheter to confirm or exclude vesicoureteric reflux (VUR), uretero-vesical junction obstruction, VUR: Vesicoureteric reflux, DLPP: Detrusor leak point pressure, UDS: Urodynamic study

| Table 1: Patient demography and outcome |
|----------------------------------------|
| Total patients with adequate data       | 272 |
| Primary                                | 236 |
| Lateral entry                          | 36  |
| Patients with mini valve, Dhani Lanka valve or insignificant valve | |
| With moderate clinical and radiological features | 11  |
| With moderate-to-severe clinical and radiological features | 26  |
| Primary fulguration                    | 231 |
| Number of patients had BNI              | 120 |
| Primary                                | 86  |
| Secondary                              | 34  |
| Re BNI                                 | 6   |
| Number of patients had upstaging in CKD| 36  |
| First author                           | 2/91|
| Second author                          | 22/68|
| Third author                           | 12/77|
| Number of patients had down staging in CKD | 10 |
| First author                           | 4/91|
| Second author                          | 2/68|
| Third author                           | 4/77|
| Number of patients had ESRF            | 9   |
| First author                           | 0/91|
| Second author                          | 6/68|
| Third author                           | 3/77|
| DJ stenting in 18 patients              | 7   |
| Re-implantation of the ureter in UVJO   | 6   |
| VUR                                    | 1   |
| High Pdet Qₘₐₓ (60-130 cm of H₂O)       | 40/91|
| Decreased compliance                   | 2/91|
| No patient showed DLPP in UDS at Pdet max |  |

BNI: Bladder neck incision, CKD: Chronic kidney disease, ESRF: End-stage renal failure, UVJO: Ureterovesical junction obstruction, VUR: Vesicoureteric reflux, DLPP: Detrusor leak point pressure, UDS: Urodynamic study

| Table 2: Indications of bladder neck incision |
|---------------------------------------------|
| Indications of primary BNI                 |
| Whitis/glistening neck and or high neck, i.e., posturethra not flushed with trigone |
| Sacculcation, diverticulation and severe trabeculation (appreciable in half filled bladder) |
| Trabeculations in the posterior urethra    |
| Persistence of dilated ureter following catheter drainage for 48 h Noncompliant parents |
| Indications of secondary BNI               |
| Symptomatic following fulguration of valve |
| Recurrent UTI                             |
| Increased ACR                            |
| Deterioration in USG renometry            |
| High Pdet Qₘₐₓ in UDS                     |

BNI: Bladder neck incision, UTI: Urinary tract infection, ACR: Albumin-to-creatinine ratio, USG: Ultrasound, UDS: Urodynamic study
monitored by repeated USG renometry. Besides that, all patients were followed up with albumin-to-creatinine ratio (ACR), CCr, DTPA renal scan, and uroflowmetry. All patients of the first author were advised urodynamic study (UDS), particularly for study of detrusor pressure and pressure flow (Pdet and Pdet Q\text{max}), compliance, and detrusor leak point pressure. Repeat cystoscopy was done for secondary BNI and repeat BNI if bladder pressure found elevated on UDS [Table 2].

The second author did VCUG in all cases. He used Collins knife in 68 patients for cutting of PUV under general anesthesia. He preferred incision also at 12 O’clock if satisfactory flow was not achieved on Crede’s manoeuvre following ablation at 5 and 7 O’clock and he skipped BNI. During follow-up, he advised for ACR, CCr, DTPA renal scan, and uroflowmetry and preferred alpha blockers.

The third author did VCUG in all cases and used Collins knife in 77 patients for cutting of PUV under general anesthesia and he also assessed the nature of bladder neck, trabeculations, sacculations, diverticulations, and TIPU and opted for BNI in selected patient as per the practice mentioned in Table 2. During follow-up he advised for ACR, CCr, DTPA renal scan, and uroflowmetry.

Bladder neck muscle was incised of its full thickness either at 5 or 7 O’clock position. Our attempt to approximate the cusps of PUV by suprapubic pressure failed in all cases except in few neonates. Five neonates needed bilateral ureterostomy prior to fulguration of PUV owing to increasing creatinine level which refused to abate with continuous bladder drainage for 48 h.

UDS-guided management was skipped both by the second and third authors due to the lack of “convincing guidelines” regarding UDS, secondary BNI, and re-BNI from international literature and text books. Patients with suspected UVJO were kept on anticholinergics for few months and monitored with USG renometry, ACR, and CCr; following BNI. We had to take recourse to double-J (DJ) stenting/re-implantations on deterioration of renal function in the selected patients.

**RESULTS**

The mean duration of follow-up was 7.8 years (range 3–14 years). Outcome varied among the three authors [Table 1]. Patients of the second and third author were treated and followed up as per the current convention and have been included in this study as control data. Five of 36 patients from lateral entry (as mentioned earlier) were advised reimplantations for UVJO: two of them underwent surgery and are not in ESRF currently; of the other three, one is lost to follow up, one came back after 2 years with augmentation without reimplantation and he is in ESRF now, and the last patient who had no surgery was also in ESRF.

Another two of 36 patients aged 12 and 18 years had fulguration in their infancy at other center, 100 km away from their habitat, and had good flow of urine, so they shunned “needless” follow up and subsequently presented to us with bilateral hydroureretonephrosis (HUN) and ESRF. We tried the insertion of DJ stent in both which could not be negotiated, as ureteric orifices could not be identified.

The remaining 29 symptomatic patients of 36 (from lateral entry) had high Pdet Q\text{max} following primary fulguration and they needed secondary BNI and re-BNI [Table 1]. Six patients needed reimplantations for UVJO as insertion of DJ stent failed, in addition to BNI.

**DISCUSSION**

The first author additionally did UDS to monitor the detrusor pressure during follow-up and if necessary did secondary BNI and re-BNI as mentioned in Table 2. Possibly for that, the outcome differed among the three authors due to avoidance of UDS and necessary BNI by the second and third authors. Upstaging of CKD was more in the patients of the second and third authors [Table 1]. Similarly, ESRF in the patients of
second and third authors was also more in compared to the first author [Table 1].

The first author skips VCUG for the diagnosis of PUV as in his experience; VCUG may not show dilatation of posterior urethra because of “pop off” of contrast in dilated ureters by high-grade reflux or may be due to bladder neck hypertrophy/spasm in a good number of patients. The first author relies on USG and urethroscopy for diagnosis, confirmation, and fulguration of PUV, similar to other authors.[4,7]

Good flow of urine without dribbling and without strain following fulguration of PUV indicates “success.” However, this impression may masks elevated bladder pressure during voiding which may lead to deterioration renal function. This deterioration may occur from ongoing deleterious but reversible pathologies, i.e., from bladder neck obstruction (BNO), UVJO due to hypertrophy of detrusor, and deposition of matrix around.

The combination of VUR and elevated detrusor pressure is unsafe for the kidney. Elevated detrusor pressure more than 40 cm of H$_2$O$^8$ forces contrast through the obstructive matrix$^9$ of the UVJO, and normal ureteric pressure (around 15 cm of H$_2$O) is unsafe for the kidney. Elevation of contrast timely through the UVJO. We focused on three preventable and reversible factors, i.e. BNO, UVJO, and combination of VUR and high detrusor pressure$^{10}$ as the factors of upstaging of CKD that leads to ESRF. Possibly upstaging of CKD and higher incidence of ESRF are due to poor understanding and less awareness of BNO, UVJO, Pdet max and Pdet $Q_{max}$.

In earlier stages of CKD, gradual renal deterioration may not be appreciated merely with serum creatinine levels, as serum creatinine crosses normal boundary only following destruction of more than 50% of renal parenchymal function.$^{11}$ International classification of CKD is based on the glomerular filtration rate (GFR) $^{[2]}$ [Table 3], and as per the classification, “all patients of PUV are in CKD at different stages.” GFR varies patient to patient, depending on the body mass, although the serum creatinine is in the same level. That is why we monitor renal function with biochemical $CCr/GFR$ or GFR of DTPA renal scan to asss the upstaging of CKD. We do rely neither on serum creatinine alone nor with e-GFR to monitor upstaging of CKD in children, and prevention of upstaging of CKD remains our primary endpoint for management. Moreover, the deterioration in $CCr$ is most important determinant that harbingers to do necessary interventions to arrest or to slow down ESRF.

We prefer estimation of “markers of renal-strain,” i.e., ACR and plasma renin activity (PRA) to monitor$^{[12]}$ the presence or absence of ongoing “renal micro injury.” However, the estimation of ACR is less expensive than PRA. An elevation of ACR which results from increased permeation of albumin in urine from glomerular capillary is warming sign of “renal micro injury.” This injury occurs due to pressure, hypoxia, inflammations, and toxins, and these damage the slimy coating of glomerular endothelium called glyocalyx which prevents permeation of albumin from glomerular capillaries.

On endoscopy, we got classical (fin-like semi-transparent membrane united at 12'O clock) PUV in around 50% of neonates. The remainder had either small or thin incomplete cusps of PUV. We wondered whether these tiny structures could be the cause of impediment to urine flow: this reminds us the concept of Hendren who firmly postulated wide-spectrum effect on the bladder and kidney by “mini-valves$^{[13]}$ in the posterior urethra. This concept has been resurrected and highlighted by Pieretti.$^{[14]}$ Similarly, Imaji and Dewan$^{[15]}$ supported and showed that the seemingly insignificant valve may have considerable effect on the bladder and kidney. We also had encountered similar “seemingly insignificant valve” or almost absence of PUV in some patients presenting late with moderate-to-severe morphological changes in the bladder and kidney: We analogically call it Dhani Lanka (bird’s eye chilli)! In this scenario, we wonder how the use of a balloon catheter$^{[16]}$ still in practice in Nigeria for the treatment of PUV$^{[17]}$ could engage such tiny cusps of PUV and rupture it on pull to abate obstruction! It is also on record that some authors diagnosed PUV clinically and radiologically in neonates and infants, but at endoscopy, they found no valve. Hence, they attributed the clinical and

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**Table 3: Stages of chronic kidney disease, as per the international classification (2002)**

| Stage | Description | GFR |
|-------|-------------|-----|
| 1     | Structural or functional kidney damage for >3 months, with or without decrease in GFR | >90 |
| 2     | With or without kidney damage | 60-89 |
| 3     | Moderate decrease in GFR | 30-59 |
| 4     | Severe decrease in GFR | 15-29 |
| 5     | End-stage renal failure | <15 |

GFR: Glomerular filtration rate
radiological findings to “disharmony between detrusor and sphincter”\(^{[18,19]}\) or “spasm of sphincter,” i.e., RSS.

The obstruction due to RSS (external sphincter) is well appreciated in VCUG where the dilation of the posterior urethra extends up to the bulbar urethra with a “cut off” constriction. Physiologically, in children, posterior urethral diameter is 2.5 times more as compared to the anterior urethral diameter during voiding due to its compliance and resting tone of RS. Posterior urethral diameter balloons even up to 15–32 times when there is obstruction at its distal end.\(^{[20-22]}\) Anatomically, the main bulk of RS is present around the base of the verumontanum [Figure 2].

On endoscopy, in around 50% of the neonates, we found splayed out fin-like cusp abutting urethral wall. Patients who did not have classical PUV have enough gap between cusps even after discontinuation of irrigation pressure; in addition, we tried to bring those cusps in midline with manual suprapubic pressure to replicate obstruction but failed. This raises the question, “what is obstructing posterior urethra?”

On the other aspect of spectrum, we became aware of asymptomatic PUV on endoscopy done during DJ removal following pyeloplasty and during transurethral resection of prostate, i.e., TURP in adults. Similarly, Imaji and Dewan\(^{[15]}\) reported bigger cusps in the posterior urethra on endoscopy in 8% of patients who had no dilatation of the posterior urethra and had minimal changes in the bladder.

Smeulders et al.\(^{[23]}\) found the presence of residual PUV following fulguration in 40% of patients even after resolution of posterior urethral dilation in follow-up VCUG. Hence, we are baffled: Why there is no obstruction in posterior urethra even in the presence of bigger or residual cusps, and on other hand, why there is severe obstruction in patients of Dhani lanka PUV?

Discussions in the above paragraphs create confusion from “conflicting” evidences and observations, as we used to believe, from age-old hypothesis, that cusps of PUV are physical stumbling block. However, in the majority, we were unable to replicate the obstruction with manual suprapubic pressure during endoscopy. Hence, we had to rely on another “conflicting” hypothesis that advocates: it’s not the physical size of the cusp but the “response of the bladder.”\(^{[13-15]}\) This hypothesis clarifies that the severe clinical and radiological findings may be there in “mini valve”\(^{[13]}\) as well as in “no valve”\(^{[18,19]}\) on endoscopy. Contrasting with that, there are evidences of “asymptomatic PUV!” Hence, the mystery of urethral obstruction due to PUV continues. Is PUV really a stumbling block? Or an adjuvant to congenital “spasm of sphincter,”\(^{[18,19]}\) i.e., RSS, to intensify out flow obstruction!

Developmentally, the posterior urethra is a part of the bladder and rhabdosphincter (RS) is the partner. However, the bladder and posterior urethra are under control of the autonomic system, whereas RS is under somatic control. According to Shafik,\(^{[24]}\) synergy is essential for urination. Hence, it may be hypothesized that delay in the development of synergy between detrusor and RS leads to the obstruction to the bladder outlet, as is known to happen in nonneurogenic neurogenic bladder, and spectrum of damage would depend on the intensity of spasm and duration of delay in the maturation of synergy.

If so, how the obstruction gets relieved following fulguration of PUV? For elucidation, if we consider RSS is the cause of urethral obstruction due to delayed maturation of synergy between autonomic and somatic system, further line of reckoning would be as follows.

Actually, both cusps of PUV and RS are situated at the same location. RS encircles the urethra up to

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Figure 2: Diagram of rhabdosphincter, urethra, and bladder (Courtesy: BioMed Research International, http://dx.doi.org/10.1155/2014/906921, Edited Figure used in this article)
12 O’clock, mostly at the level of the base of the verumontanum and some fibers spread over into the posterior wall of the posterior urethra [Figure 2]. Adult urologists are concerned of this area as RS may get injury during transurethral resection of prostate around the verumontanum. In patients of PUV, valve cusps are easily visible on endoview, but RS is invisible at the verumontanum. However, the presence of RS is prominent at 12 O’clock position in the patients of PUV unlike in the normal urethra. Sometimes, jerky snap of RS might be appreciable in infants when cystoscopy is glided down from the prostatic urethra to just distal to the verumontanum [Video 1], and cystoscopy might get dislodged distally due to the same jerky snap of RS, particularly in neonates [Video 2]. Histologically, RS is close to the lower end of the prostatic urethral wall [Figure 3] by RS. Anatomically, the main bulk of RS is at the base of verumontanum [Figure 2] which is encircled, and on VCUG, the narrowing at the lower end of the dilated posterior urethra is caused by the spasm of RS.

Some authors are not satisfied with “adequacy of fulguration” unless further VCUG is done. They do not judge adequacy of fulguration with endoview; rather they prefer VCUG at follow-up. Plausibly, they do not rely on endoview in spite of adequate fulguration of PUV, as obstruction persists even following fulguration of PUV. Cystoscopically, visible cusps of PUV and invisible ring of RS are very close to base of the verumontanum anatomically. Contractions of RS might be perceptible from active Bugbee at cusp. The process of fulguration of cusp of PUV might dissipate thermal/electrical energy on RS to neutralize its spasm. Some authors claim that the outcome of fulguration done only at 12 O’clock, keeping cusps of PUV intact, is comparable with triple fulguration of cusps at 5, 7, and 12 O’clock. Possibly, dissipation of thermal/electrical energy on RS at 12 O’clock is more due to narrow cusps at commissure. Similarly, the second author of this article prefers to incise the commissure at 12 O’clock with Collin’s knife to get satisfactory flow on Crede’s manoeuvre done following fulguration. In addition, the placement of urethral catheter for few days in postfulguration period leads to continuous stretching of RS to alleviate spasm (simile... relief of anal spasm). Sometimes, the fulguration of PUV at the first attempt might not be able to neutralize RS spasm completely due to inadequate thermal/electrical dissipation.

Similarly, a pull of balloon catheter would easily engage RS in spasm and would do microrupture of RS. On the other hand, it is difficult for the balloon to engage mini cusps with wider gap in between to make it to rupture. Appreciated by the educated guess of Cain who suggested demonstration of laceration of cusps on endoview following balloon pull is necessary for proper understanding. This has not yet been demonstrated.

Five neonates were referred to us weeks after urethral catheterization, as they had radiological evidence of PUV. However, at endoscopy, we could not find PUV. Krishnan et al. have published a similar experience of relieved obstruction following catheterization for a long duration.

Thermal/electrical energy of fulguration, pressure of balloon pull, and long durational urethral catheterization lead to “benefit of spin-off-effect” or “side-benefit” to abolish/decrease the spasm of RS in small kids.

RSS cause dilatation and trabeculations in posterior urethra (TIPU) due to urethral obstruction and this TIPU appears similar to bladder trabeculations that we see following bladder outlet obstruction. Intensity of spasm and duration of obstruction by RS cause detrusor hypertrophy, bladder neck hypertrophy, BNO, and UVJO due to hypertrophy detrusor and deposition of matrix around intramural ureters and creates a vicious cycle [Figure 4]. This vicious cycle might persist as an aftermath even after the resolution of RS spasm and initiate upstaging and end staging of CKD due to high pressure in the bladder and upper tract with or without voiding dysfunction. Some authors are aware of UVJO following detrusor hypertrophy in PUV. Hence, they do not do vesicostomy but perform bilateral ureterostomies to evade UVJO.

Awareness of BNO was dawned in patients of PUV from the publication of Koff et al. who justly suspected the combination of elevated bladder pressure in the bladder and upper tract with or without voiding dysfunction. Some authors are aware of UVJO following detrusor hypertrophy in PUV. Hence, they do not do vesicostomy but perform bilateral ureterostomies to evade UVJO.

Figure 3: Longitudinal section of the urethral wall and surrounding muscles: rhabdosphincter and inferomedial edge of the levator ani. (Courtesy: BioMed Research International, http://dx.doi.org/10.1155/2014/906921)
pressure and VUR as a cause of renal deterioration. However, they never demonstrated BNO; as they conceptually focused themselves on residual valve and stricture which were also not demonstrable. Others hesitated to focus on BNO in patients of PUV. Rather they kept themselves contented with the conception of bladder dysfunctions (valve bladder) which is the end-product of permanent alteration in the bladder histology consequent to prenatal obstruction. [31-35] Afterward, Manning et al. [36] found that the relief of prenatal obstruction by interventions did not abate the deterioration of bladder histology as compared to control group. They concluded that prenatal decompression relieved bladder pressure temporarily but did not cure the long-lasting BNO and elevated pressure permanently. Hence, the ultimate prognosis concerning ESRF did not differ.

Gradually, bladder pressure and BNO came into focus. Jeffrey et al. emphasized on the persistence of HUN following valve ablation in patients of PUV that should not be considered as residual dilatation until an urodynamic evaluation demonstrates normal detrusor pressure and normal bladder compliance. One can expect significant lessening of HUN and long-term preservation of renal function following reduction of detrusor pressure [37] after successful treatment of PUV. Gradually, the concept of bladder neck hypertrophy which may occur in 50%-70% of patients [33] and BNO causing elevated detrusor pressure is getting legitimacy. That is why people started taking recourse to use of alpha blockers [38] or even clean intermittent catheterization (CIC) [39] to keep intravesical pressure under control after getting checked by UDS at regular interval. Androulakis et al. [40] suggested that secondary BNO was a cause of myogenic bladder decompensation in boys with a history of PUV. With the advancement of treatment of BNO, BNI is making its place in preference to pharmaceutical agent and CIC to reduce bladder pressure. [41,42] Apprehension of retrograde ejaculation after BNI has proved to be unfounded by the studies of Hennus et al. [43] and Keihani et al. [44]

BNI appears to be more effective than alpha blockers and CIC, in long term, particularly for the patients who reside far away from tertiary care centers and are financially not endowed.

In this communication, we are making an endeavor to draw attention toward BNO, UVJO, and importance of UDS for the prevention of upstaging of CKD as well as bladder dysfunction. It is true that some authors [3] start pondering on BNO, RSS, and UDS only after the onset of detrusor dysfunction (valve bladder).

**Conclusions**

From this study and reviewing the literature, it appears that the RSS plays a major role in bladder outlet obstruction, and cusps of PUV, particularly in neonates, amplify the obstruction. This RSS with or without the support of PUV cusp may set off detrusor hypertrophy, bladder neck hypertrophy, BNO, and UVJO. These are preventable and reversible factors which are treatable to prevent or delay the ESRF. These reversible as well as preventable factors have been sporadically discussed with minor emphasis in international literature and textbooks. Therefore, we emphasize on five points, added in paradigm, i.e., CCr, UVJO, BNO, UDS, and ACR (CUBUA) for the prevention of upstaging and end staging of CKD. We have kept renal dysplasia out of the purview of present communication as we have no control on such dysplasia which may or may not have bearing on upstaging of CKD. Hence, we conclude that, "In the End we need not Stage the Renal dysplasia on a pedestal to mask our Failure!" Nevertheless, we cannot assert the ascendancy of this retrospective study and of CUBUA unless prospective studies with randomized controlled trial are done for high level of evidence.

**Future direction**

Now, we are in the process of evaluating the RSS with the help of electromyography, and its effect on Pdet and Pdet Qmax in UDS both in pre and post fulguration period. This might also help us to manage overactive bladder, voiding dysfunction, and dysfunctional voiding.

**Acknowledgment**

We are grateful to Professor Kalyan Kumar Sarkar, MS, FRCS, Head of Department of Urology, Vivekananda Institute of Medical Science, Kolkata, for his consultation and guidance on Urodynamic study and his sincere help in correction and revision of this manuscript.
Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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