Posterior Urethral Valve and Other Urethral Obstruction

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1. Introduction

Posterior Urethral Valves (PUV) is a congenital form of bladder outlet obstruction that occurs only in males. Leaflets of tissue or “valves” cause anatomical obstruction to the flow of urine, resulting in a variety of pathology. PUV has been traditionally categorized into three types based upon the classification scheme originally described by Hugh Hamptom Young in 1919.[1] Type I PUV are the most frequently encountered and the valves appear to emanate from the verumontanum and travel anteriorly to just proximal to the prostatomembranous junction. (**Figure 1)**

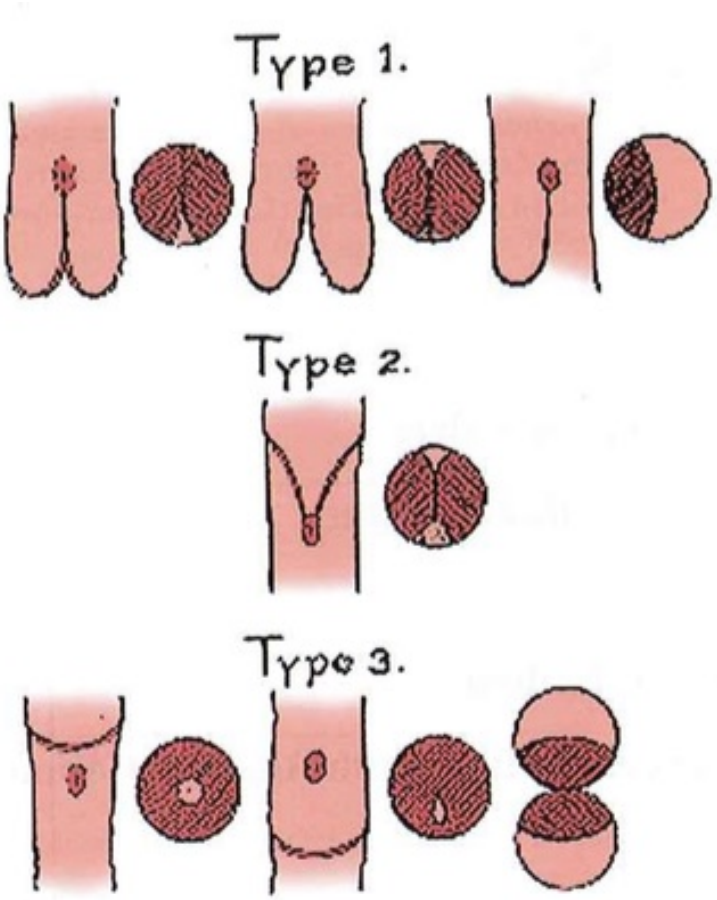

Figure 1: Three types of posterior urethral valves (PUV) originally described by Hugh Hampton Young in 1919.[1] Type I valves are the most frequently encountered and the valves appear to emanate from the verumontanum and travel anteriorly to just proximal to the prostatomembranous junction. Type 2 valves arise from the verumontanum and extend posteriorly and superiorly to the bladder neck, are not obstructing and have not been reported definitively since early reports. Type 3 valves are

an annular ring similar to that seen with a congenital urethral stricture, found at variable locations in the posterior urethra. [1]

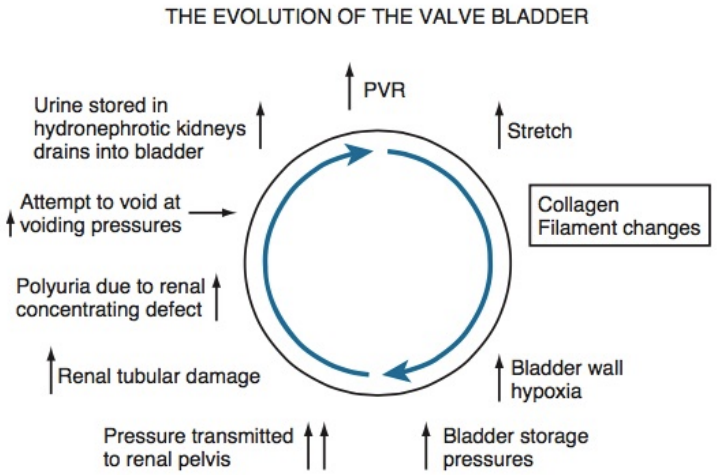

Figure 2: The valve bladder "vicious cycle." An affected bladder will lead to increasing postvoid residuals (PVR). The sustained stretch will cause increasing collagen deposition and other changes that cause increased pressures to be transmitted to the renal pelvis/upper tracts. Subsequent renal tubular injury causes polyuria and increased volume into a bladder that already empties poorly, and the cycle continues, leading to additive damage. [2]

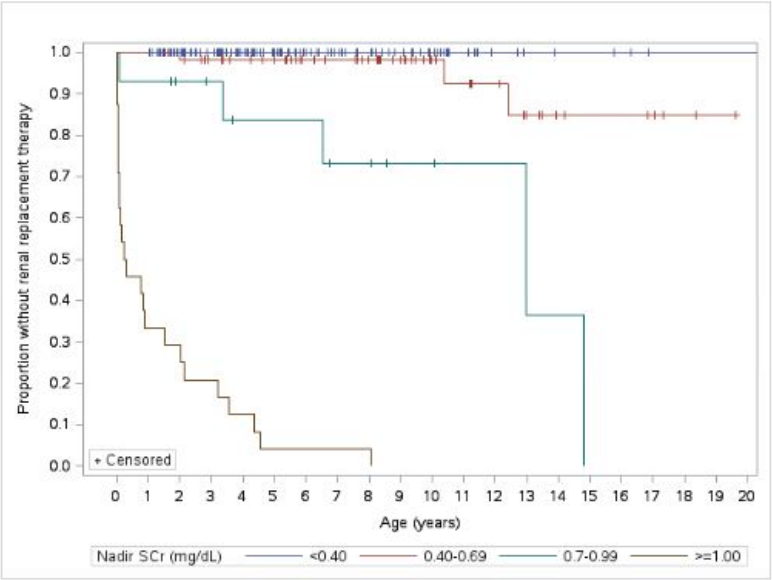

Figure 3: Nadir serum creatinine in the first year of life has proven to be a powerful predictor for need for renal replacement therapy (RRT) in the PUV patient. This is Kaplan-Meier estimates of age at RRT, stratified by nadir serum creatinine in the first year of life. In their series by 10 years of follow-up, a nadir serum creatinine <0.4 mg/dL was associated with 0% risk of RRT whereas 100% of patients with a nadir serum creatinine >1.0 mg/dL experienced outcome of requiring RRT. Additional ranges showed intermediate risk illustrating the variable renal

outcomes of the PUV patient and need for extended follow up[3].

2. Epidemiology

PUV can be diagnosed prenatally and postnatally and the phenotype expressed can vary within PUV. PUV has been estimated to occur in 1-2 per 10,000 live births.[2] A significant number of PUV patients will present with dilated upper and lower urinary tracts on prenatal imaging.[4] Postnatal diagnosis of PUV patients in certain studies comprises between 40-60% of all PUV patients.[5,6]

Between 20% - 50% of PUV patients will progress to end stage renal disease (ESRD) resulting in renal replacement therapy (RRT), either in the form of peritoneal dialysis, hemodialysis, or renal transplantation, throughout their lifetime.[3,6,7,8] In a multi-center study of 274 PUV patients diagnosed in the neonatal period, 16% had undergone RRT by 10 years of age, when accounting for differing lengths of follow-up.[3]

3. Risk Factors and Pathophysiology

PUV can have dramatic effects on bladder, ureteral and renal function as a sequela of the bladder outlet obstruction. The increase in bladder outlet resistance is initially compensated by an increase in voiding pressure and detrusor musculature. This change in detrusor architecture can also be accompanied by increases in collagen deposition. This detrusor to collagen ratio will determine the bladder's storage characteristics that, in severe instances, can result in reduced compliance, impaired bladder emptying and elevated storage pressures. The elevated storage pressures can be transmitted to upper tracts, impairing drainage of the renal pelvis and ureters. This increased renal pelvic pressure can alter renal tubular function, reducing urine concentrating ability and result in polyuria. The increased urine volume into an abnormal bladder, which empties incompletely, can continue a cycle where impaired bladder function perpetuates ongoing upper tract injury.[2] (**Figure 2**) This cycle has been referred to as "valve bladder" as originally coined by Dr. Michael Mitchell.[9]

The renal dysfunction and outlet obstruction, secondary to PUV, occurring *in utero* may result in oligohydramnios or anhydramnios. This reduction in amniotic fluid can have profound effects on pulmonary development. Uropathy-associated pulmonary hypoplasia appears to be predominantly the result of oligohydramnios during the canalicular phase of lung development, rather than renal dysfunction itself.

Risk factors for development of ESRD have been described in the PUV patient. Serum creatinine nadir in the first year of life has repeatedly been demonstrated as a predictor for renal outcome. A nadir serum creatinine of greater than1.0 mg/dL has been reported to be significantly associated with future development of ESRD; whereas a nadir of lower than 1.0 mg/dL is associated with better prognosis of renal function.[8,3,10] McLeod et al demonstrated that this risk of RRT in a cohort of PUV patients diagnosed in the neonatal period could be risk stratified based upon nadir serum creatinine in the first year of life[3] (**Figure 3**) Age at diagnosis is an unclear risk factor for development of ESRD. There was assumption that later diagnosis of PUV is a consequence of a milder phenotype of the condition and thus lower risk of ESRD. Conversely, prenatal detection of PUV would identify more severe phenotypes and thus an increased risk for development of ESRD. However, this is not universal based upon studies that report on renal outcomes in PUV patients presenting as neonates and those presenting later in life.[5,6,7,8,3]

PUV patients with high-grade vesicoureteral unilateral reflux into a poorly functioning kidney, whereas the contralateral renal unit appeared to have preserved renal function, has been described as *vesicoureteral reflux and dysplasia* (VURD) syndrome.[11] It has been hypothesized that the reflux served as a pop-off mechanism for bladder pressure, in which the dysplastic, poorly functioning kidney with reflux served as a pressure reservoir mitigating damage to the contralateral kidney. Originally it was theorized that these children would have better long-term renal function as a result of the pop-off phenomenon. However longer-term studies have demonstrated that the VURD syndrome does not improve renal prognosis. Cuckow et al found that, whereas 67% of patients affected by VURD during year 2 of life had a normal serum creatinine, only 30% of these children had normal values between ages 8 and 10 years.[12]

4. Diagnosis and Evaluation

PUV is most accurately diagnosed on cystoscopy; however, radiographic studies are the usual manner in which the bladder outlet obstruction is suspected and PUV identified. Ultrasound is the universal part of the diagnostic evaluation of most congenital urologic diseases, including PUV. Prenatal radiographic findings of a distended, thick-walled bladder with dilated posterior urethra, “keyhole sign”, is highly suggestive of PUV. (**Figure 4**) Postnatal diagnosis also occurs as a consequence of a clinical presentation of urinary tract infections or lower urinary tract symptoms in a significant number of patients. Perinephric urinoma is another presentation, detected by ultrasound in patients with PUV, and is characterized with a hypoechoic region along the renal capsule.

Voiding cystourethrogram (VCUG) plays a fundamental role in postnatal diagnosis. The bladder can have a trabeculated appearance, diverticula as well as a dilated posterior urethra, proximal to the PUV. (**Figure 5**) Vesicoureteral reflux (VUR) is frequently observed at time of diagnosis. A patent urachus can also be seen in patients with PUV as a consequence of the bladder outlet obstruction.

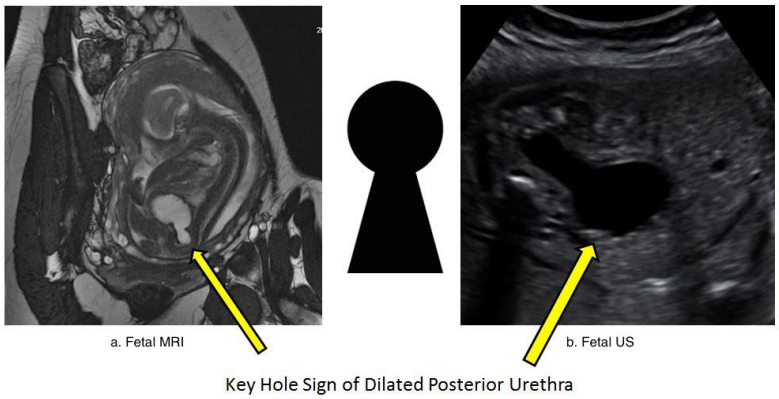

Figure 4: Radiographic images of fetus with a. magnetic resonance imaging and b. sonography demonstrating a dilated posterior urethra and distended bladder, which are highly suggestive of bladder outlet obstruction. Postnatally, the male neonate underwent voiding cystography and was found to have PUV.

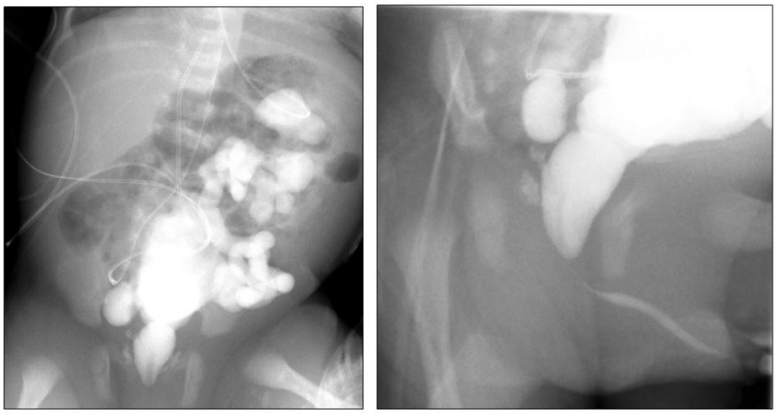

Figure 5: Anterior and oblique fluoroscopic images of a male patient with PUV on voiding cystourethrography, demonstrating a trabeculated bladder, vesicoureteral reflux and a dilated posterior urethra, diagnostic of PUV.

4.1 Other Urethral Obstruction

Fibroepithelial polyps (FEP) are rare, male predominant, benign tumors of mesodermal origin, occurring at all levels of the

urinary tract from renal calices to anterior urethra. Across all age groups FEP are most frequently identified in the upper ureter or renal pelvis, while in children, FEP are more likely to be located in the male posterior urethra and can result in bladder outlet obstruction.[13] Boys with urethral polyps (UP) typically present with hematuria, intermittent obstructive voiding complaints, and urinary retention. In girls, where reported cases are sparse, the most common presentation is an interlabial mass.[14] There is poor consensus as to whether UP are congenital or acquired with the majority presenting in the first decade of life.[15,16] Initial diagnosis is typically made by ultrasonography and voiding cystourethrogram, although confirmation by cystourethroscopy and histopathology is often necessary. Transurethral resection with either electrocautery or laser is preferred, however large UP may require cystotomy to remove the specimen. If resected at the base of the stalk, recurrence is rare.

5. Treatment

5.1 Prenatal Evaluation and Intervention

As previously mentioned there can be fetuses diagnosed with urinary tract dilation (see Core Curriculum **Hydronephrosis** and **Hydroureteronephrosis**) and suspected lower urinary tract obstruction (LUTO). Unrelieved LUTO causes progressive damage to the bladder, renal and pulmonary dysfunction which impacts long-term health of fetus and child. Urine production begins around 10 weeks once the metanephros is formed but amniotic fluid is still a combination of maternal plasma ultrafiltrate and fetal plasma transudate. It is only at 16 weeks gestation low amniotic fluid levels secondary to LUTO are first able to be detected on prenatal ultrasound. The classic triad of PUV on prenatal sonography has been keyhole sign, megacystis and hydronephrosis (see Figure 4) however this historical teaching may not be as accurate as previously thought. In one retrospective series 29/42 suspected PUV fetuses confirmed to have PUV postnatally, the authors found that increased bladder wall thickness and bladder dilation were associated with diagnosis of PUV ($p<0.001$) while the keyhole sign did not predict diagnosis of PUV.[17]

The natural history of LUTO is variable and depends on disease severity and gestational age of onset. Severe LUTO causes massive bladder distention, hydronephrosis and renal dysplasia leading to oligohydramnios with secondary lung hypoplasia and soft tissue deformities (Potter's syndrome). Whereas mild forms of LUTO may have normal amniotic fluid volume throughout pregnancy and have favorable renal function after birth making prenatal intervention more risk than benefit. The correct selection of potential subjects for fetal therapy is important to avoid unnecessary intervention in those unlikely to survive as well as complications related to the procedure in those that are likely to survive without any intervention.[18] Most LUTO fetuses with normal amniotic fluid volumes at 24 weeks gestation will have stable postnatal renal function however upon follow up to 1/3 of patients will eventually require renal replacement therapy.[19] An assessment of fetal urine analysis parameters obtained by vesicocentesis has been promoted to better understand their prognostic significance on postnatal renal function. The primary function of the fetal kidney is to clear free water: therefore a more dilute fetal urine infers "better" urine and postnatal renal function. A review of fetal urine analysis has shown the following parameters to be associated with poor postnatal renal function: Urine osmolality >200 mOsm/L, Urine sodium >100 mEq/L, Urine chloride >90 mEq/L, Beta-2 microglobulin >13 mg/dL, Ca >95th percentile for gestational age, Na > 95th percentile for gestational age.[20]

Clinical studies examining patient outcomes with prenatal interventions aimed at alleviating the LUTO have been performed. The PLUTO study was a randomized trial in the UK, Ireland, and the Netherlands, women whose pregnancies with a male fetus were complicated by isolated LUTO were randomly assigned to receive either the intervention in the form of placement of vesicoamniotic shunt (VAS) or conservative management.[21] The primary outcome was survival of baby to 28 days postnatally. 31 fetuses enrolled with the median gestational age at shunt placement 19.7 weeks. The study was closed early due to poor recruitment. There were 24 total livebirths: 12 in each group. Twelve neonates survived to 28 days, with all 12 neonatal deaths secondary to pulmonary hypoplasia. 28 day survival greater in VAS group (8) vs. conservative management group (4), however this was not statistically significant in an intention to treat analysis. Complications with VAS were common: 7 complications in 6 patients [Spontaneous ruptured membranes (3); Shunt blockage (1) Shunt dislodgement (3)] along with 4/15 VAS pregnancies lost. An important observation of the study was that the probability of normal renal function was low whether VAS or conservative management performed. A

systematic review and meta-analysis of VAS vs. no intervention in LUTO encompassing 9 studies with 112 fetuses showed perinatal survival improved (OR 2.54, 95%CI 1.14-5.67) but no difference in 6-mo, 12-mo, or 2-yr survival and no difference with postnatal renal function[22] In general, the fetuses most likely to benefit from prenatal intervention are those with oligo/anhydramnios, severe bilateral hydronephrosis, absence of kidney cysts/dysplasia, favorable fetal urine paramters and intervention performed between 18-24 weeks gestation.

5.2 Medical

5.2.1 Neonatal Medical Management and Workup

Upon diagnosis of PUV, typically by combination of ultrasonography and VCUG, surgical intervention is imminent to address the anatomical bladder outlet obstruction. However, it is paramount for the urologist to make certain the patient is optimized prior to the procedure for the safety of the patient. This is most vital in the neonate with PUV. Assessment of respiratory status is critical to any neonate; but especially so in the PUV patient with a history of diminished amniotic fluid. Lung function may be diminished in this subset as a consequence of the oligo/anhydramnios. Ventilatory support may be needed for respiratory distress and the neonatologist will make this decision during the primary assessment.

Relief of the bladder outlet obstruction should be pursued as soon as possible. The bladder should be drained with a urethral catheter of appropriate size as the first line option. Insuring proper placement of the catheter in the bladder is important in PUV patients as the dilated posterior urethra and high bladder neck can, at times, make positioning into the bladder challenging. Inability to place a urethral catheter, may necessitate a suprapubic tube placement.

Wu et al reported a clinical regimen of preoperative urethral indwelling catheter that was either gradually dilated up to an 8 French size or not dilated prior to transurethral resection of PUV (TURV) in 126 neonates.[23] They examined primary outcome of ability to perform TURV. Overall 97% of the cohort could have TURV completed. Only a larger catheter at time of TURV was associated with feasible procedure and not gestational age, infant's weight or urethral dilation. Progressive urethral dilation was associated with higher febrile UTI rate as a consequence of longer duration of indwelling catheter.

Initiation of a prophylactic antibiotic while the diagnostic workup occurs is frequently utilized under the logic that this may minimize the risk of catheter associated UTI.

Imaging studies of the urinary tract at bedside can be easily accomplished with ultrasound. Cystography to confirm the diagnosis of PUV is best performed with fluoroscopy in the radiology suite, but the stability of the patient will dictate the timing of this study. Measurement of the patient's renal function is key to the immediate and long-term treatment of patients with PUV. Neonatal creatinine reflects maternal creatinine in the first 24 hours of life and this should be kept in mind when interpreting initial result. After relief of bladder outlet obstruction, the serial trending of the serum creatinine until a plateau occurs may be helpful prior to proceeding with surgical intervention, assuming the child is stable.

5.2.2 Post Operative Medical Management

Whichever surgical technique is chosen to alleviate the bladder outlet obstruction, ongoing active management of the bladder is critical to minimizing the effects of PUV on the patient's health. The early bladder in PUV post-surgical relief of bladder outlet obstruction is characterized by detrusor hyperreflexia and bladder wall thickening, which is caused by detrusor wall thickening from increased collagen deposition. This bladder wall thickening can result in significant persistent upper tract dilation as a consequence of elevated detrusor pressure, impairing upper tract drainage. At times, bladder emptying can be impaired, making the upper tract dilation worse. In children who are toilet trained, timed voiding and double voiding may help with elevated post void residual. Abraham et al described the use of alpha-adrenergic blockade in a PUV cohort to relieve any counterproductive sphincteric hypertonicity and relax the bladder neck in children with high postvoid residuals, finding a significant reduction in residual volumes. [24] The thickened bladder can be modified with the use of anticholinergic medications in effort to improve bladder compliance and reduce intravesical detrusor storage pressures. Casey et al described the use of oral oxybutynin at 0.1 mg/kg twice daily in 18 consecutive infants undergoing urodynamic assessments at 3 months after transurethral valve ablation that demonstrated high voiding

pressures and/or small bladder capacity.[25] The authors reported that both of these parameters improved significantly with oxybutynin although therapy was stopped in 4 of 18 patients due to elevated bladder capacity on follow up studies and concern over urinary retention.

In patients where the aforementioned therapies of do not resolve the negative impact of abnormal bladder function, clean intermittent catheterization (CIC) is another therapeutic alternative. The utilization of CIC can be challenging in PUV patients due to the sensate urethra and bladder neck hypertrophy. Holmdahl reported their use of CIC in treatment of bladder dysfunction in 19 boys with PUV, who were started in the first year of life due to pronounced bladder dysfunction with poor bladder emptying, unsafe urodynamics, high grade reflux and decreased GFR.[26] Compliance with CIC was good with 17/19 patients continuing. GFR at 8 years follow up was similar between those who continued CIC and PUV patients who had never begun CIC. In contrast, in the 2 patients who stopped CIC when it was recommended, there was significant decline in GFR over the same study period. The authors concluded that treatment of bladder dysfunction in boys with PUV can counteract the deterioration in renal function seen during childhood, but the number of patients in their study was limited.

VUR spontaneously resolves in a large percentage of patients (66% in one series) following relief of bladder outlet obstruction.[27] Reflux can be associated with pyelonephritis and in cases of high grade reflux, where the risk of febrile UTI is highest, antibiotic prophylaxis can be helpful to minimize this risk. Distinguishing any persistent VUR as primary or secondary in etiology is crucial to its proper management. As previously described, the PUV bladder has variability in its characteristics such that lower voiding or storage pressures can be beneficial to increasing secondary VUR resolution rates.

Given the increased risk of ESRD, it is critical that longitudinal measurement of glomerular filtration rate (GFR) be performed to identify chronic kidney disease stage and appropriately treat the kidney dysfunction. Annual assessment is recommended and changes during adolescence may be reflective of metabolic demand of the associated growth spurt and decreased renal reserve. Delayed myogenic failure of the detrusor as a result of the “valve bladder cycle” is a concern and should always be investigated when urologic testing reveals increased post void residual or worsening upper tract dilation.

Table 1. Medications used in treatment of PUV

Therapeutic Class	Example Agents	Mechanism of Action	Outcomes	Adverse Events	References
Anticholinergic	Oxybutinin, Tolterodine	Inhibits acetylcholine signal at the neuromuscular junctions of the detrusor muscle, thus leading to inhibition of detrusor contractions	Decreased detrusor end filling pressures, Reduced Voiding pressures	Common: Dry mouth, constipation, flushing of skin. Rare: elevated post void residual, behavioral changes in	Casey et al[25]
Alpha-Adrenergic Antagonist	Tamsulosin, doxazosin	Relax smooth muscle at the bladder neck and prostate thereby helping to relieve bladder outlet obstruction	Reduced bladder neck resistance leading to decreased post void residual	Orthostatic hypotension	Abraham et al[24]

5.3 Surgical Management

The choice of surgical procedure can be dependent upon multiple factors but the size of the urethra usually is the most limiting factor. While transurethral resection of the urethral valve (TURV) is the most commonly performed procedure to address the PUV in the neonate, placement of a pediatric resectoscope to perform a transurethral resection of the urethral valve can be challenging in the premature or low weight PUV patient. Options in this circumstance include prolonged transurethral catheter drainage to dilate urethra,[23] suprapubic tube drainage, vesicostomy or use of a pediatric cystoscope with use of a small 3 French catheter and bent wire to allow for diathermy to ablate the valve.

TURV is the preferred initial surgical choice given studies comparing TURV vs. vesicostomy, which have not demonstrated superiority of vesicostomy.[28,29,30] The treatment goal of TURV is to restore flow of urine through the urethra and enable normal cyclic filling and emptying of the bladder, which is superior to urinary diversion and passive urine drainage. There have been different techniques described to ablate the valves with cold knife, Collins knife with diathermy or Holmium laser. The valves are incised at the ventral 5 o'clock and 7 o'clock positions with or without an incision at the dorsal 12 o'clock position. No energy modality, "hot" or "cold", for TURV has proven superior to the other and surgeon preference dictates which is utilized. Postoperatively, it is not uncommon to have an urethral catheter placed for brief time, but this is not universally practiced.

Circumcision is often performed at the time of TURV to decrease risk of UTI. A recent randomized trial of circumcision vs no circumcision in 91 male infants with PUV showed a 3% risk of febrile UTI in the circumcised group compared to 20% in the uncircumcised group over the first 2 years of life.[31]

The dilated appearance of upper tract can persist after relief of bladder outlet obstruction and this is important to recognize. Studies have examined the etiology of the persistent upper tract dilation and anatomical ureterovesical junction obstruction is very rare. [32,33,34] A functional ureterovesical junction obstruction due to combination of elevated bladder pressures, high urine output and impaired bladder compliance, with the bladder empty or full, is the more common cause of hydroureteronephrosis.[35] In these instances, time for the bladder to respond to relief of bladder outlet obstruction with or without aggressive bladder management may obviate the need for additional surgical procedures in form of upper tract diversion. However upper urinary tract diversion may be considered in an infant with complete decompression of the lower urinary tract with associated worsening renal function, increasing upper tract dilation, and possible clinical picture of an infected and obstructed upper tract. Upper tract diversion can be in the form of percutaneous nephrostomy or cutaneous ureterostomy or pyelostomy. Contemporary studies and long-term follow-up of these patients, however, failed to detect a long-term renal protective benefit of high diversion, which necessarily requires an often complicated undiversion procedure as the child matures. Whether upper urinary tract diversion or vesicostomy preserves renal function better than valve ablation alone cannot be definitively concluded because of the lack of controlled comparative studies or other available studies.

Instances where maximal medical management does not improve characteristics of the bladder in conjunction with signs or symptoms such as worsening renal function, recurrent UTIs or incontinence, makes surgical intervention a consideration. Maximal medical therapy may be defined as maximum tolerated anticholinergics, continuous overnight bladder drainage and use of daytime intermittent catheterization. Bladder augmentation is infrequently performed in the PUV patient as the natural history of the valve bladder is originally a hypertonic, low capacity reservoir and over time becomes a more compliant, larger than expected reservoir. [36] The attendant risks of bladder augmentation makes delaying performing the procedure, if an option, advantageous as bladder characteristics can evolve. The role of botulinum toxin injection into detrusor in the PUV patient has not been described but presumably may be a less morbid way to modify problematic elevated detrusor pressures and uninhibited detrusor contractions when maximal medical therapy fails.

Persistent VUR in the PUV patient may lead to antireflux surgery when medical therapy is not successful. Standard indications for proceeding with antireflux surgery (i.e. breakthrough UTI, new renal scarring) are applied in PUV patients. However surgical intervention should not be taken lightly as success rates of antireflux procedures in an abnormal bladder

are lower than in a normal bladder.[37] Therefore bladder function should be optimized in all PUV patients, but especially before unavoidable anti-reflux surgical procedures.

6. Treatment Complications

Complications of PUV from a medical standpoint have been mentioned already but the pulmonary hypoplasia from severe oligohydramnios is the most life threatening to the neonate. Orthopedic complications such as clubfeet may result from the decreased fluid in the amniotic space as well. ESRD can result from PUV due to congenital renal dysplasia however post-natal factors such as UTI may play a role in acquired insult to renal function. Cetio et al reported renal and sexual function outcomes in men >18 years with 46% having ESRD and with 35% undergoing renal transplantation. A subset of the 39 patients in their study had sexual function evaluation (n=32) and none reported erectile dysfunction. However 4 patients reported slow ejaculation.[38]

Bladder dysfunction leading to UTI and incontinence is common. A recent study by Jalkanen et al, showed that patients with PUV achieved daytime and nighttime urinary continence significantly later than their healthy peers. Prenatal or neonatal diagnosis and high serum creatinine were factors associated with later attainment of continence.

Surgical complications secondary to intervention, such as TURV, can occur. Urethral injury/stricture can easily occur if there is forceful placement of a rigid cystoscope or resectoscope in the delicate neonatal urethra with associated long term consequences.[39] Persistent obstructing valvular tissue following TURV may require repeat TURV when clinical or radiographic improvement is not observed. Postoperative VCUG may not be as accurate as cystoscopy; however routine cystoscopy post TURV is not universally practiced, but is recommended by some.[40,41]

The requisite undiversion for vesicostomy or supravesical diversion may be considered by some as a complication of the procedure itself. Cutaneous urinary stomas all suffer from the possibility of stomal stenosis. Stomal prolapse is more relevant complication of a vesicostomy than it is to pyelostomy or ureterostomy. A key operative step during creation of the vesicostomy to prevent prolapse of the back wall of the bladder through the incision is to bring the dome of the bladder to the skin, thus ensuring that the posterior wall of the bladder is taut.[2]

7. Post Operative Pathways

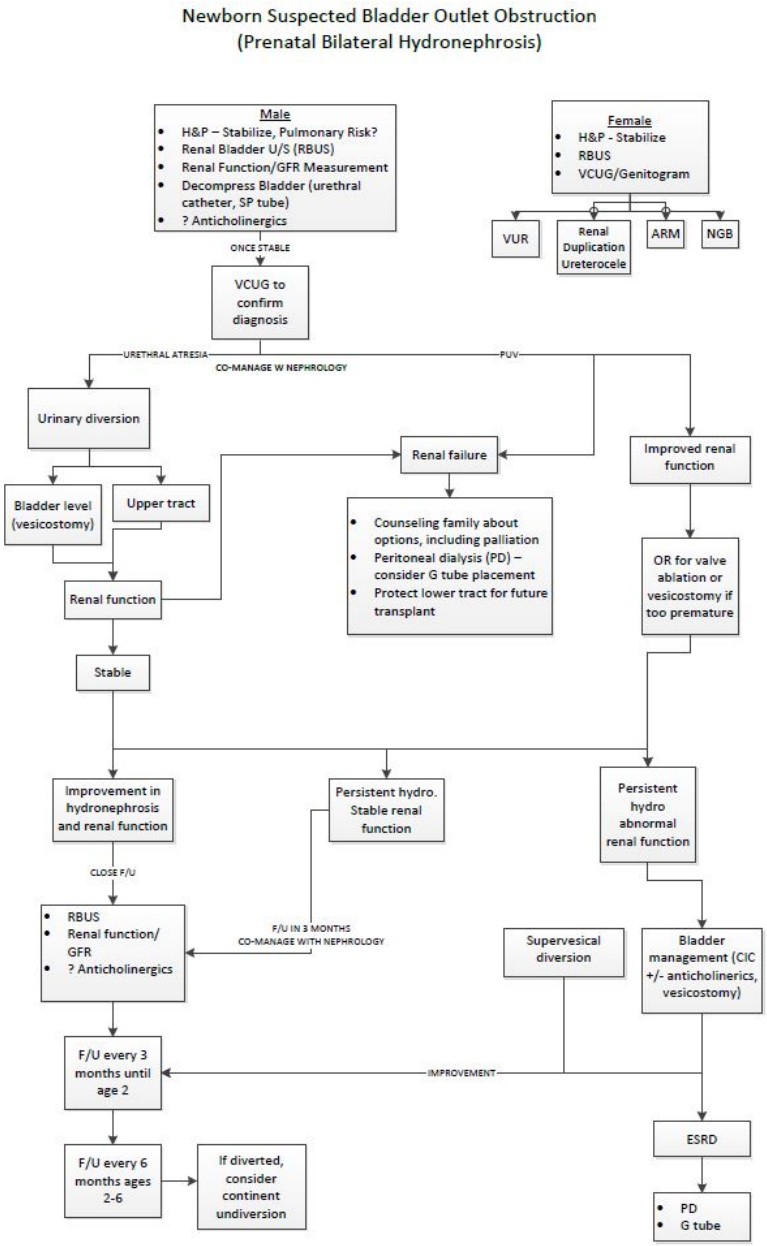

Figure 6: Diagnostic and treatment pathway in the neonate with suspected bladder outlet obstruction. Frequent assessments of renal function and upper tract appearances are advocated along the care path to guide additional interventions versus close observation.

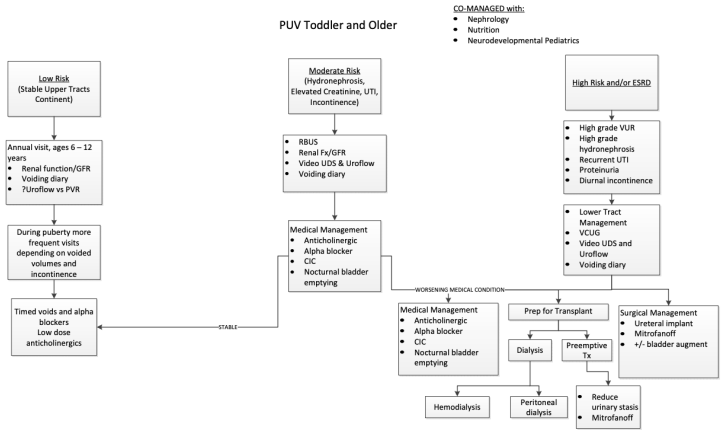

Figure 7: PUV care pathway in a non-neonate, older patient stratified based upon low, intermediate or high risk grouping for abnormal bladder and renal function.

Figure 6 is a sample diagnostic and treatment pathway in neonate with bladder outlet obstruction. Reassessment of renal function and upper tract dilation is advocated along the care path to help determine whether surgical interventions are performed or close observation pursued. **Figure 7** illustrates potential care pathway in a non-neonate older patient with PUV stratified based upon low, intermediate or high risk grouping for abnormal bladder and renal function.

Reference:[42]

Videos

Posterior urethral valve on cystoscopy

Aggressive diagnosis and treatment for PUV as an etiology for VUR or urge incontinence in children

Posterior Urethral Valve Ablation

Presentations

Posterior Urethral Valve and Other Urethral Obstruction Presentation 1

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