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# Posterior urethral valves in children. Literature review

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## ABSTRACT

The posterior urethral valve is the most common cause of infravesical obstruction in newborn boys with a prevalence of about 1 in 3800. The etiology is multifactorial, associated with impaired embryogenesis in the 4th week of pregnancy with abnormal fusion of the mesonephral ducts with the cloaca. The pathogenesis includes mechanical obstruction of the urethra, leading to increased intravesical pressure, secondary changes in the bladder (detrusor hypertrophy, trabecularity), vesicoureteral reflux (up to 50% of cases) and progressive kidney damage. Prenatal ultrasound can diagnose up to 47% of cases based on the following symptoms: bilateral ureterohydronephrosis, megacystis, keyhole symptom, oligo-/anhydramnion. The basis of treatment is early endoscopic valve ablation in the first months of life and the initiation of drug therapy with M-holinoblockers (oxybutynin), which improves urodynamics and prognosis. If primary ablation is not possible, temporary urine withdrawal (catheterization, cystostomy) is used. Long-term complications include the formation of a "valvular bladder" with neurogenic dysfunction, persistent vesicoureteral reflux, chronic kidney disease (develops in 30–50% of patients), which requires constant dynamic monitoring, urodynamic control, and in some cases, augmentation cytoplasty or renal replacement therapy. Prenatal diagnosis and early minimally invasive intervention remain key to improving outcomes.

**Keywords**: posterior urethral valve, renal failure, valvular bladder, vesicoureteral reflux, valve ablation, children

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# Клапан задней уретры у детей. Обзор литературы

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## РЕЗЮМЕ

Клапан задней уретры — наиболее частая причина инфравезикальной обструкции у новорожденных мальчиков с распространенностью около 1:3800. Этиология мультифакториальна, связана с нарушением эмбриогенеза на 4-й неделе беременности при аномальном слиянии мезонефральных протоков с клоакой. Патогенез включает механическую обструкцию уретры, приводящую к повышению внутрипузырного давления, вторичным изменениям мочевого пузыря (гипертрофия детрузора, трабекулярность), пузырно-мочеточниковому рефлюксу (до 50% случаев) и прогрессирующему поражению почек. Пренатальное ультразвуковое исследование позволяет диагностировать до 47% случаев по признакам: двусторонний уретерогидронефроз, мегацистис, симптом «замочной скважины», олиго-/ангидрамнион. Основой лечения является ранняя эндоскопическая абляция клапана в первые месяцы жизни и начало медикаментозной терапии М-холиноблокаторами (оксибутинин), что улучшает уродинамику и прогноз. При невозможности первичной абляции применяется временное отведение мочи (катетеризация, цистостомия). Долгосрочные осложнения включают формирование «клапанного мочевого пузыря» с нейрогенной дисфункцией, персистирующий пузырно-мочеточниковый рефлюкс, хроническую болезнь почек (развивается у 30–50% пациентов), что требует постоянного динамического наблюдения, уродинамического контроля, а в части случаев — аугментационной цистопластики или заместительной почечной терапии. Пренатальная диагностика и раннее малоинвазивное вмешательство остаются ключевыми для улучшения исходов.

**Ключевые слова:** клапан задней уретры, почечная недостаточность, клапанный мочевой пузырь, пузырно-мочеточниковый рефлюкс, абляция клапана, дети

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## BACKGROUND

The research of infravesical obstruction caused by a urethral valve spans more than 300 years. A congenital posterior urethral valve was first described by Giovanni Battista Morgagni in 1717 [39]. The earliest known mention of posterior urethral valves as valve-like folds of the mucous membrane in autopsy findings dates back to 1802. However, the researcher did not conclude that these findings had any clinical significance [35]. Subsequently, there were isolated reports describing these structures as a result of autopsies [2]. In 1870, research findings on semilunar valves of the urethra in children were published, and in 1875, a comprehensive description of the valves as a pathological phenomenon was provided, along with a hypothesis regarding their origin [48]. The first cases of ante-mortem diagnosis of posterior urethral valves and their successful treatment were described in 1913. A classification of posterior urethral valves in children, which remains relevant to this day, was presented shortly thereafter [54].

## ETIOLOGY, EMBRYOLOGY, CLASSIFICATION, AND EPIDEMIOLOGY

The exact cause of posterior urethral valves is unknown, but it is likely a multifactorial embryopathy. Familial inheritance is rare, but such cases have been described [12]. During embryogenesis, the most caudal end of the Wolffian duct opens into the primitive cloaca at the site of the future seminal hillock in the posterior urethra. In healthy males, the residual structures are the posterior urethral folds, known as *plicae colliculi* [41]. Histological examination shows that the posterior urethral valve (PUV) forms around the 4th week of gestation, when the Wolffian duct fuses with the developing cloaca. If the confluence of the mesonephric ducts into the cloaca is abnormal or occurs too distally, normal duct migration is disrupted, and they merge in front of the seminal tubercle, leading to abnormal ridges or folds, which are believed to be the source of 95% of PUVs [24]. This type is called Type I PUV. Although Hugh Young described Type II PUV, most pediatric urologists believe that these are not obstructive valves but simply hypertrophy of the mucosal folds of the bladder neck. Type III valves account for the remaining 5% and consist of a ring-shaped membrane located distally to the seminal tubercle, with a central perforation. Type III valves result from incomplete resolution of the urogenital membrane [24].

**H. Young's classification of posterior urethral valves** [54] includes three types.

1. **Type I valves.** These are found in 90–95% of patients as sail-like folds originating from the urethral crest (*crista urethralis urethrae masculinae* — a longitudinal fold on the posterior wall of the urethra extending from the bladder neck through the prostatic urethra), extend distally, curving around the seminal tubercle on both sides, and merge in front of the membranous portion of the urethra.

2. **Type II valves.** These are natural folds of the mucous membrane extending from the seminal tubercle to the bladder neck. They are rarely detected. Since they almost never affect the passage of urine through the urethra, they are considered a variant of the anatomical norm.

3. **Type III valves.** Diagnosed in 5–10% of cases. They consist of a solid diaphragm or a ring-shaped membrane with a central opening, located at the level of the seminal tubercle or slightly distally along the urethra. They can significantly restrict urination [54].

Currently, it is debatable whether Young's classification is appropriate, since in some cases the valves detected during cystoscopy cannot be classified under any of Young's valve types [3].

In congenital urinary tract obstruction (CUTO), there are urinary system lesions of varying severity, which depend on the degree of obstruction. The pathological process is not limited to transient infravesical obstruction. Congenital obstruction of the urinary tract at a critical stage of organogenesis can have a significant impact on kidney, ureter, and bladder function [11].

Appearance of the posterior urethral valve is the primary pathology causing mechanical obstruction of the urethra, leading to subsequent secondary changes. The severity of these changes depends on the degree and timing of the primary obstruction [32].

From the 20$^{th}$ week of gestation, the fetal kidneys produce 90% of the amniotic fluid. An adequate volume of amniotic fluid is vital for lung and skeletal development [49]. Severe oligohydramnios or anhydramnios, caused by decreased urine output, leads to a reduced uterine cavity. This results in compression of the fetus, which impedes normal growth and expansion of the fetal chest, in turn causing lung hypoplasia and soft tissue deformities [19, 32]. The typical fetal phenotype associated with oligohydramnios/anhydramnios includes low-set ears, wide-set eyes, micrognathia, limb contractures, and clubfoot (Potter's sequence) [16]. An adequate volume of amniotic fluid (produced by the kidneys) is necessary for complete and proper branching of the bronchial tree and the formation of alveoli. Bladder distension and urinary ascites increase the fetal abdominal volume and disrupt the development of the abdominal wall muscles, leading to a wrinkled abdomen. Spontaneous decompression of the upper urinary tract can lead to urinary ascites, paraneural, or perineal urinoma [5, 32]. Severe oligohydramnios, azotemia, and severe pulmonary hypoplasia can result in fetal death.

Obstruction of the posterior urethra leads to increased intravesical pressure, progressive thickening

of the muscles (hypertrophy and hyperplasia), trabeculation, the formation of diverticula, and, in severe cases, paravesical or paraurethral urinoma. Some authors believe that a large bladder diverticulum provides some degree of protection for the upper urinary tract due to a pressure-relief mechanism, which is associated with a good prognosis [8, 41]. However, there are publications reporting that a bladder diverticulum or urinoma did not subsequently result in incidence of end-stage renal failure compared to a group in which these complications did not occur [27]. A bladder with pronounced trabeculation exhibits an increase in collagen and connective tissue elements. This leads to the following changes: high intravesical pressure, increased residual urine volume, elevated pressure during micturition, and bladder dysfunction even after valve ablation [32].

Ureteral dilation may occur in PUV due to vesicoureteral reflux (VUR). It is present in 50% of patients with PUV. VUR may occur either primarily due to pathology of the ureterovesical segment or secondarily due to high intravesical pressure [41]. A significant portion of vesicoureteral reflux resolves after the obstruction is removed [17].

Renal pathology includes hydronephrosis and progressive renal damage. Hydronephrosis is caused by: 1) vesicoureteral reflux; 2) obstruction — high intravesical pressure transmitted to the ureter and kidney via hydrostatic pressure; 3) abnormal development of the ureteric bud, leading to renal dysplasia and dilatation of the collecting system.

Renal damage is caused by the following two factors.

1) Renal dysplasia is likely caused by damage during the early fetal period or abnormalities in embryogenesis. It manifests as microcysts, which are found primarily in the peripheral cortical zone. Histological findings include disorganization of the renal parenchyma, the presence of embryonic tubules and cysts, as well as the presence of mesenchymal connective tissue. These changes are irreversible and cause renal failure.
2) Obstructive uropathy is kidney damage caused by infravesical obstruction, leading to dysfunction of both the glomeruli and tubules, with a decrease in glomerular filtration rate, reduced kidney function, fibrosis and sclerosis of the renal parenchyma, and tubular damage, which causes impaired urine concentration and acidification. Consequently, diuresis increases, sodium loss rises, and electrolyte imbalances occur. These changes are potentially reversible [22].]

The incidence of PUV is approximately 1 in 3,800 live-born boys. PUV is the most common cause of infravesical obstruction in newborn boys [13].

## DIAGNOSIS

Early diagnosis of PUV has become possible with advances in prenatal ultrasound (US) of the fetus. Currently, up to 46.9% of PUV cases are detected during prenatal screening [37]. Suspected PUV before the 24$^{th}$ week of pregnancy is associated with an increased risk of perinatal mortality and renal impairment [30]. Key ultrasound findings associated PUV include bilateral hydronephrosis, megacystis with wall thickening (>3 mm), and posterior urethral dilation (keyhole sign) [9, 15]. Oligo/anhydramnios may be an important prognostic sign [14].

Postnatal diagnosis involves ultrasound, voiding cystourethrography, and videocystoscopy, which remains the “gold standard” for confirming the diagnosis.

## TREATMENT

**Prenatal treatment.** Based on pathophysiological considerations, fetal surgery and the vesicoamniotic shunting technique were once viewed with great promise. However, a randomized international multicenter study showed inconclusive results regarding actual long-term improvement in renal function in patients who received this treatment. Moreover, the complication rate ranged from 21% to 59% [40]. Two additional intrauterine techniques have recently been proposed: valve ablation via cystoscopy and vesicostomy via open fetal surgery. However, both techniques are associated with high maternal and perinatal morbidity [31, 51]. Maternal complications include premature rupture of the amniotic membranes, while fetal complications include migration of the vesicoamniotic shunt, eversion of the intestinal loop and omentum into the amniotic cavity, urinoma, and intestinal perforation. In 50% of cases, pregnancy was terminated due to fetal death following fetal intervention [51].

At present, there are no clear parameters for predicting future renal function. Fetal surgery may be offered in individual cases as an alternative treatment option after detailed consultation with parents [24].

**Postnatal treatment. T**he early stage of surgical treatment for posterior urethral valves involved extensive, traumatic open procedures on the urethra. Along with the valve, the hypertrophied bladder neck was sometimes removed, leading to total urinary incontinence [42]. Kholzov’s perineal urethroplasty was performed until the 1990s [2].

The modern era of surgical PUV treatment began with video-endoscopic technologies in pediatric urology. Today, newborns with suspected PUV are monitored in specialized multidisciplinary centers during the prenatal period. In a smaller percentage of cases, PUV is diagnosed at a later age due to the presence of heterogeneous urinary symptoms, such as urinary tract infections, urinary incontinence, a weak urine stream,

and enuresis. It is believed that these patients may have a better renal outcome [13]. However, researchers have not found significant differences in long-term outcomes between patients with early and late diagnosis of PUV. The only real difference between the two groups concerned urinary tract infections, which were more common in the group with postnatal diagnosis [50]. In contrast, data have been published indicating that patients with late diagnosis of PUV have a worse prognosis due to prolonged renal exposure to higher blood pressure [46].

A newborn with suspected PUV requires bladder drainage via transurethral catheterization as the primary intervention at birth. Subsequently, voiding cystourethrography should be performed to confirm the diagnosis, provided that patient's condition permits this examination. Once the patient's condition has stabilized (electrolyte, respiratory, and renal disorders) video-cystoscopy can confirm the results of voiding cystography and transition from a diagnostic procedure to a therapeutic one. Endoscopic ablation or resection of the valve should be performed [21]. Valve ablation in the first few months of life yields better long-term outcomes in terms of urodynamics, vesicoureteral reflux, and improvement or elimination of ureterohydronephrosis [21, 34, 53]. Early valve ablation is not feasible in some newborns due to small urethral caliber. In such cases, gradual urethral dilation should be performed by sequentially replacing urethral catheters with larger ones [53]. Methods for PUV destroying include electrocoagulation, a "cold" knife, or laser incision/fulguration. After valve ablation, a urethral catheter of appropriate diameter is placed in the bladder, and drug therapy with M-cholinoblockers (oxybutynin) is initiated at a dose of 0.2 mg per kilogram three times daily [4, 7].

**Urinary diversion.** In some cases, primary valve ablation is not possible. Under such circumstances, urinary diversion must be performed using a urethral catheter, suprapubic cystostomy, or temporary vesicostomy. The choice of urinary diversion method remains a matter of debate [34, 45]. Recent studies suggest that vesicostomy does not improve the prognosis or reduce the incidence of renal failure in patients with severe upper urinary tract dysfunction, high-grade vesicoureteral reflux, or recurrent urinary tract infections [45].

**Intermittent catheterization.** In cases of persistent voiding dysfunction, chronic urinary retention, recurrent urinary tract infections, or persistent bladder-dependent neurogenic bladder dysfunction, patients are recommended to undergo clean intermittent catheterization of the bladder. Studies show that clean intermittent catheterization reduces the frequency of complications associated with neurogenic dysfunction of the valve bladder and stabilizes renal function or delays the need for renal replacement therapy [38].

**Augmentation cystoplasty.** Regardless of the treatment administered, approximately one-third of patients with a posterior urethral valve develop chronic renal failure [18]. The problem is exacerbated by polyuria and the development of a high-pressure, low-capacity valve bladder [36]. Augmentation cystoplasty allows to create a low-pressure reservoir with sufficient capacity, thereby relieving patients of urinary incontinence and stabilizing or improving renal function, which, in turn, allows to delay or prevent the need for kidney transplantation in these children [10].

**Treatment of VUR.** Despite the significant prevalence of VUR in PUVs (48–66% [24]), spontaneous resolution of reflux occurs in 27–79% of cases, ranging from 2 weeks to more than one year after valve ablation [26, 43]. The EAU–ESPU guidelines recommend preventive use of antibiotics in boys with severe VUR and PUV [23]. Circumcision may have a beneficial effect on reducing the number of febrile urinary tract infections (UTIs) [25]. When patients have persistent VUR after PUV destruction and experience recurrent episodes of febrile UTIs or progressive renal failure, surgical treatment is indicated (endoscopic correction or ureteral reimplantation) [6, 33]. Early creation of a continent appendicovesicostomy using the Mitrofanov technique may yield good results in patients with a congenital ureteral malformation who develop a manifest urinary tract infection [44].

## OUTCOMES

**Valve bladder syndrome.** The concept of the valve bladder was formulated in the 1980s. It was described as a thick-walled, poorly distensible bladder with high resting pressure, leading to ureterohydronephrosis and renal failure [44]. A series of urodynamic studies in these patients identified three main urodynamic patterns, which represent the progression of the same neurogenic bladder dysfunction, changing with the patients' age [20, 29]. In infants, there is a reduction in functional bladder capacity and detrusor overactivity. Over the next 1–3 years, bladder capacity increases while detrusor hyperactivity persists, and residual urine volume rises. By ages 4–12, bladder capacity also increases, detrusor hyperactivity resolves, detrusor contractility decreases, and residual urine volume increases [47].

**Renal failure.** In children with VUR, there are two main factors that trigger the development of chronic kidney disease (CKD): impaired nephrogenesis and neurogenic bladder dysfunction, which may persist even after transurethral valve resection [1, 44]. Various studies report that chronic kidney disease develops in approximately 30–50% of patients with PUV. Recent reports from multicenter cohort studies in the United States, involving 274 [38] and 685 [28] patients, have

provided new data on chronic kidney disease (CKD) and end-stage renal disease in children with PUV. By ages 1, 5, and 15 years, 7%, 12%, and 20% of children, respectively, required renal replacement therapy. In another study involving 35 infants, by age 10 years, 50% of children had developed stage II–IV CKD, and 15% required renal replacement therapy [18]. Another study covering the period from 2002 to 2011 found that 11% of children on renal replacement therapy in North America were diagnosed with PUV, highlighting the high social and economic burden of this disease [52]. Although figures vary across different reports, the proportion of patients with CKD (up to 50%) and end-stage renal disease (up to 20%) is sufficiently high to warrant ongoing monitoring by a pediatric nephrologist, including urodynamic studies for all children with PUV. Adverse prognostic factors for the development of CKD include: early onset of PUV, serum creatinine levels exceeding 1 mg/dL, grade III or higher VUR, recurrent febrile urinary tract infections, renal dysplasia, and neurogenic bladder dysfunction [18].

## CONCLUSION

Thus, posterior urethral valve is a serious congenital anomaly that can lead to progressive damage involving the kidneys and bladder. Current treatment strategies are based on early diagnosis using prenatal ultrasound and minimally invasive endoscopic ablation of the valve during the first months of life. Despite improved methods for this procedure, a significant proportion of patients remain at risk of developing chronic kidney disease and bladder dysfunction, which requires long-term multidisciplinary follow-up. Further research is needed to standardize treatment approaches and improve long-term outcomes in children.

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