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Epidemiological Patterns, Causative Factors, and Pathogenesis of Obstructive Uropathies

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Abstract Obstructive uropathies occupy a significant place among urgent urological conditions and represent one of the leading causes of acute and chronic kidney failure. This review summarizes current data on the epidemiology, major etiological factors, and pathogenetic mechanisms underlying urinary tract obstruction. The most common causes include urolithiasis, benign prostatic hyperplasia, urethral strictures, and iatrogenic injuries. Particular attention is given to clinical and pathophysiological aspects such as impaired urodynamics, reduced tubular reabsorption, development of hyperkalemia and metabolic acidosis, post-obstructive diuresis, and interstitial fibrosis. The importance of early detection and relief of obstruction is emphasized in order to prevent irreversible renal damage and systemic complications. The review highlights the high clinical relevance of timely diagnostic and therapeutic approaches to obstructive uropathies in the context of increasing epidemiological burden.

Keywords Obstructive uropathies, Urolithiasis, Benign prostatic hyperplasia, Urethral stricture, Pathogenesis, Post-obstructive diuresis, Kidney failure, Urology

1. Introduction

Obstructive uropathies occupy a significant place in the structure of emergency urological care, particularly in the elderly population, and are among the leading causes of obstructive acute kidney injury, urosepsis, and persistent loss of renal function [1,2]. The most common etiological factors include urolithiasis, benign prostatic hyperplasia, and urethral strictures [3,4]. Over the past decades, there has been a steady increase in incidence, attributed to population aging, the growing prevalence of metabolic disorders, and the rising number of invasive procedures, which are often complicated by iatrogenic injuries [5,6].

Obstruction at any level of the urinary tract initiates a cascade of pathophysiological changes affecting both urodynamics and the electrolyte and acid–base balance, forming the basis of severe clinical manifestations that require timely diagnosis and correction.

The aim of this review is to systematize current data on the epidemiology, etiological factors, and pathogenesis of obstructive uropathies, with an emphasis on their clinical significance, and to discuss the key pathophysiological mechanisms underlying acute and chronic kidney injury associated with impaired urinary outflow.

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2. Epidemiology and Etiology of Obstructive Uropathies

According to global literature, the estimated incidence of this condition is approximately 1.7 per 1,000 population, accounting for 5–10% of all cases of acute kidney injury (AKI). A clear age-related pattern has been established: the incidence peaks occur in infancy and in older adults [3,4]. Large-scale studies have shown that among elderly patients with AKI, obstruction as the primary cause can account for up to 22% of cases [5]. In men, AKI of obstructive origin is more common, primarily due to gender-specific conditions such as benign prostatic hyperplasia (BPH) and prostate cancer [6].

Within the etiological structure of obstructive uropathy, urolithiasis remains the leading cause, diagnosed in 60–70% of urological patients. The prevalence of urolithiasis demonstrates a steady global upward trend, posing a major public health concern [7]. In the United States, the incidence of urolithiasis has nearly doubled over the past 15 years, and by various estimates, one in eleven individuals has experienced at least one episode of urinary stone disease [8]. Lifetime prevalence in European countries and the United States ranges from 5% to 12%, with a markedly higher occurrence among men (up to 13%) compared to women (around 7%) [9].

In Europe and North America, up to 1.5 million cases of renal colic are reported annually per million population, with these numbers steadily increasing due to changes in lifestyle and diet, as well as population aging and the rising frequency

of metabolic disorders [10,11]. In contrast, the annual detection rate of upper urinary tract stones in Japan is 134.0 per 100,000 population [12], substantially lower than in Western countries. However, South Korea has shown a persistent increase in urolithiasis incidence. According to the study by B.S. Tae et al. [13], based on an extensive database from the Korean National Health Insurance Service, the annual incidence demonstrated a statistically significant upward trend ($p<0.001$), with an 11-year cumulative incidence of 5.71%, significantly higher in men (7.07%) than in women (4.34%). The highest rates were observed in the 60–69 age group, reaching 9.08%. The age-standardized lifetime prevalence was estimated at 11.5%, again higher in men than in women (12.9% vs. 9.8%).

Acute urinary retention (AUR) is one of the most frequent urological emergencies, characterized by the sudden inability to void despite a strong urge to urinate. The predominant causes of AUR include benign prostatic hyperplasia, acute prostatitis, urethral strictures, neurogenic bladder dysfunction, and iatrogenic factors such as the use of certain medications (e.g., anticholinergics, alpha-adrenergic agonists) [14]. The prevalence of AUR increases with age, affecting approximately 1–2% of middle-aged men and reaching 10–15% in men over the age of 70. Notably, up to 90% of AUR cases in elderly patients are attributable to BPH [15].

Infectious and inflammatory diseases of the urinary tract also demonstrate high incidence rates. Acute pyelonephritis is diagnosed annually in 15–20 individuals per 10,000 population, while complicated forms of pyelonephritis and urosepsis are associated with mortality rates of up to 20–30%, particularly among elderly patients and individuals with comorbidities [16].

Another significant clinical concern is acute urinary retention (AUR), the prevalence of which reaches 10% in men over the age of 70. This condition considerably impairs patients' quality of life, necessitates urgent and elective surgical interventions, and increases the risk of infectious complications and hospitalizations [16]. Obstructive causes of AUR include any mechanical impediment to urinary outflow within the urethra at or below the bladder neck. In women, the urethra is short and offers a relatively low-resistance pathway, whereas in men it is approximately 20 cm in length, with the most common cause of AUR being compression of the prostatic urethra due to benign prostatic hyperplasia (BPH). BPH accounts for 53% of AUR cases, and autopsy findings indicate an increasing frequency of histological BPH—from 8% in men in their fourth decade of life to 80% in their ninth decade [17,18].

Structural and functional alterations of the urethral epithelium and subepithelial spongy tissue, arising from nonspecific urethritis or trauma, may lead to progressive fibrotic narrowing and the development of strictures. Urethral strictures are among the most ancient urological conditions, referenced in medical sources dating back to antiquity. Evidence of this pathology is found in descriptions of primitive urethral dilators in ancient Egyptian papyri, as well as in Indian and Chinese medical treatises [19]. Historical accounts also indicate that notable figures such as the philosopher Epicurus and Emperor Napoleon suffered from urethral strictures [20].

The prevalence of urethral strictures in men is estimated to range from 229 to 627 cases per 100,000 population [21]. The overall occurrence of the condition is approximately 0.9% [22]. The anterior urethra is most frequently affected (up to 92.2% of cases), with the bulbar segment being the most common site (46.9%). Among men, the incidence rises markedly after the age of 55 and continues to increase steadily thereafter [23,24]. In women, urethral strictures are much rarer, occurring in only 0.08–5.4% of cases, primarily in the context of therapy-resistant lower urinary tract symptoms (LUTS) [25,26].

In men, the primary causes of urethral strictures include idiopathic, iatrogenic, traumatic, inflammatory (e.g., obliterative xerotic balanitis), and infectious factors. Notably, idiopathic strictures—diagnosed even after thorough history-taking—account for 34% of all penile urethral strictures and 63% of all bulbar strictures [27].

The most frequent cause of urethral stricture is iatrogenic stenosis, occurring in 32–79% of cases [28,29]. Iatrogenic injury most often results from bladder catheterization and cystoscopy, surgical treatment for benign prostatic obstruction, local therapy for prostate cancer, and surgical correction of hypospadias [30]. Urethral catheterization (via catheter or cystoscope) accounts for 11.2–16.3% of all stricture cases [29,31], with the bulbar and posterior urethra being the most frequently injured sites [32]. The incidence of stricture formation following catheterization is approximately 3.4% [9], but increases dramatically to 24–78% in cases of intraprocedural urethral injury (false passages) [33,34]. Additional mechanisms of urethral trauma during catheterization include inflation of the catheter balloon within the urethra and prolonged catheter dwell time. The use of smaller-caliber catheters, minimizing catheterization duration, restricting the indications for this procedure, and ensuring proper training of medical and paramedical personnel in urethral catheterization techniques can reduce the risk of this iatrogenic complication [26,35,36].

The second most frequent cause of urethral strictures is surgical intervention for benign prostatic hyperplasia (BPH), accounting for 1.7–13% of all cases [37,38]. Postoperative strictures typically involve the bulbo-membranous urethra, less commonly the fossa navicularis or penile urethra [39], and rarely (0.3–9.7% of cases) the bladder neck [40]. Pathogenetic mechanisms implicated in postoperative stricture development include ischemia due to endoscope sheath compression, inadequate instrument lubrication, rough endoscope manipulation, electrical current dispersion, and thermal tissue injury [40]. Several risk factors for this complication after transurethral prostate surgery have been identified, including operative time exceeding 60 minutes, mucosal injury, postoperative infection, and small prostate volume (<40 g) [41,42].

It has also been observed that the overall incidence of urethral and bladder neck strictures is not significantly

influenced by the type of energy source used (monopolar or bipolar resection, holmium enucleation, thulium ablation, photoselective vaporization). Nonetheless, a relatively higher rate of complications has been reported following monopolar resection [38], while the lowest rates are noted after aquablation, intraprostatic device implantation, water vapor thermal therapy, and prostatic artery embolization [41].

The incidence of vesicourethral anastomotic stricture following radical prostatectomy, according to recent case series, ranges from 1% to 3% [43,44]. In most cases, anastomotic stenosis develops within the first two years after surgery [45]. Predisposing factors include predictors related to the extent and nature of the surgical intervention, the patient's oncologic status, and overall comorbid condition. Among the most significant surgical risk factors are prolonged operative time, presence of an anastomotic leak, intraoperative bleeding, omission of neurovascular bundle preservation, and limited surgeon experience (<40 procedures per year) [46]. Despite the variety of surgical techniques, none has been shown to significantly reduce the risk of anastomotic stricture formation, although some data suggest a slightly lower incidence after robot-assisted prostatectomy [47]. Moreover, no association has been found between the risk of this postoperative complication and the method of suture placement for anastomosis construction (continuous vs. interrupted) or the type of absorbable material used (monofilament, braided, or barbed) [48].

Oncologic risk factors include high-grade malignancy, locally advanced disease, and salvage prostatectomy. Patient -associated risk factors comprise large prostate volume, ischemic heart disease, obesity, arterial hypertension, diabetes mellitus, prior prostate surgery, and advanced age [43,44,46].

High-energy modalities for prostate cancer treatment, such as external beam radiotherapy, brachytherapy, and ablative technologies, also increase the risk of urethral stricture formation. Four-year follow-up data indicate an incidence of 1.5% after external beam radiotherapy and 1.9% after brachytherapy [49]. These strictures are typically located in the bulbo-membranous and prostatic urethra [50]. In contrast to radical prostatectomy, radiation-induced strictures may occur in the late postoperative period, necessitating long-term follow-up for comprehensive risk assessment [51]. For ablative techniques, the incidence of strictures after cryoablation and high-intensity focused ultrasound (HIFU) is approximately similar, ranging from 1.1% to 3.3% [52]. Combined use of high-energy modalities, particularly in salvage therapy settings, is associated with a markedly increased risk of urethral strictures, reaching up to 30% [53]. However, delaying adjuvant or salvage radiotherapy for up to nine months after prostatectomy, when oncologically feasible, may help reduce the incidence of this complication [54].

Trauma ranks third among the causes of urethral strictures in adults [55], affecting both the anterior and posterior urethra. Approximately 10% of all pelvic fractures are accompanied by posterior urethral injury, most commonly resulting from road traffic accidents and falls from height [56].

Sexually transmitted infections (STIs) associated with urethritis have long been recognized as one of the classic etiologies of urethral strictures. In developing countries, infection-related strictures remain relatively common; however, in economically developed nations, the incidence has significantly declined due to effective STI prevention measures and early initiation of therapy, currently accounting for only 0.9–3.7% of all urethral stricture cases [55].

In women, 48.5% of urethral strictures are attributed to idiopathic factors, while 24.1% result from iatrogenic causes, primarily urethral catheterization or prior surgical interventions involving the urethra, including diverticulectomy, fistula repair, and anti-incontinence procedures. Traumatic urethral injuries account for 16.4% of cases. Rare causes of female urethral strictures include radiation therapy and infections [26].

Other causes of obstruction include phimosis and paraphimosis, while in women, pelvic organ prolapse (bladder, rectum, or uterus) may also contribute [57]. Prolonged lower urinary tract obstruction can lead to bladder stone formation, which in turn may cause acute obstructive urethral injury [58]. Extraurethral causes, such as compression of the urethra by retroperitoneal masses or impacted feces, should also be considered.

3. Pathophysiological Consequences of Urinary Tract Obstruction

The level of urinary tract obstruction significantly influences the pathogenesis, clinical course, diagnostic strategy, and choice of treatment in obstructive uropathy. Accurate localization of the obstruction site is a key step in both diagnosis and subsequent therapeutic planning. From a functional perspective, three principal levels of obstruction are distinguished [59,60]:

- Intrarenal level – obstruction located within the calyceal–pelvic system. The most common causes include congenital ureteropelvic junction (UPJ) stenosis, renal pelvic calculi, renal tumors, or blood clots.
- Ureteral level – obstruction occurring within the lumen or wall of the ureter. Causes include ureteral calculi, strictures, extrinsic compression (e.g., pelvic tumors, lymphadenopathy), as well as sequelae of surgical interventions or radiation therapy.
- Post-vesical level – obstruction located distal to the bladder. Typical causes include benign prostatic hyperplasia (BPH), prostate cancer, urethral strictures, neurogenic bladder dysfunction, and anatomical anomalies (e.g., posterior urethral valves in children).

From a surgical standpoint, intrarenal and ureteral obstructions are classified as upper urinary tract obstruction, while post-vesical lesions are categorized as lower urinary tract obstruction [3].

The urinary tract, from the renal papillae to the ureteral orifices in the bladder, constitutes a peristaltic system approximately 25–30 cm in length. Myoblast-like interstitial

cells, particularly concentrated in the renal papillae, play a crucial role in maintaining this function. These cells exhibit pacemaker activity, enabling the ureter to function as a manometric amplifier that generates a stable peristaltic pressure wave propagating toward the bladder [61].

The velocity of peristaltic wave conduction along the ureter ranges from 2 to 5 cm/s. Initially, intraluminal pressure measures only 0–5 cm H_2O, gradually rising to 20–60 cm H_2O near the ureteral orifice [62]. In vivo studies suggest that ureteral peristalsis occurs within a specific anatomical structure referred to by some authors as the "ureteral displacement sheath", which ensures stable transmission of the pressure wave [63].

Disruption of this anatomical structure in cases of external obstruction, such as retroperitoneal fibrosis, results in extrinsic compression of the ureter and impaired urine flow from the kidneys to the bladder. In contrast, in intraluminal causes (e.g., calculi or strictures), the peristaltic wave is directly blocked within the ureteral lumen. The nature and degree of obstruction determine the specific pathophysiological mechanisms involved and, consequently, the clinical manifestations. Hemodynamic alterations in the kidneys are most pronounced in cases of complete or bilateral urinary tract obstruction, when compensatory mechanisms are insufficient to maintain normal organ function [64]. Complete and sudden obstruction more often results in acute kidney injury, whereas partial and progressive obstruction is usually associated with a gradual decline in function, leading to chronic obstructive uropathy [3].

It is important to note that the consequences of obstruction extend far beyond acute impairment of renal function. They include electrolyte imbalances, infectious complications, and structural damage to the renal parenchyma, which in some cases become irreversible [65].

Acute upper urinary tract obstruction leads to impaired urine outflow, followed by an increase in intraluminal pressure that is transmitted retrogradely toward the pelvicalyceal system and further to the renal tubular apparatus. This disrupts intratubular fluid dynamics and causes tissue injury, accompanied by morphofunctional changes in both the renal parenchyma and the urinary tract [66]. Initially, a compensatory hemodynamic response develops: under the influence of locally released prostaglandin E_2, renal blood flow increases, helping to maintain glomerular filtration. However, this phase is short-lived, and within 1–2 hours renal blood flow begins to decline, while intratubular pressure continues to rise. Approximately 3–4 hours later, under persistent elevated pressure, there is a marked reduction in renal perfusion with redistribution from the cortical to the medullary regions [66].

In patients with bilateral obstruction or obstruction of a solitary functioning kidney, two phases of hemodynamic change occur [67]. The first phase is characterized by a transient (up to 90 minutes) increase in renal blood flow, followed by a sharp decline, similar to the mechanism described above. However, unlike unilateral obstruction, in bilateral cases the redistribution of intrarenal blood flow occurs in the opposite direction—from the medulla to the cortex. If not promptly relieved, these vascular disturbances lead to ischemic nephron injury, the development of acute tubular necrosis, and, with prolonged persistence, irreversible renal damage [68].

The pathophysiological consequences of urinary tract obstruction affect three key aspects of renal tubular function: sodium transport, urine concentrating ability, and acid excretion [69]. These impairments are most pronounced in bilateral obstruction, although to varying degrees they may also occur in unilateral cases.

One of the earliest functional alterations in obstructive uropathy is a reduction in the expression of aquaporins —tubular proteins responsible for water reabsorption. Experimental in vivo models have demonstrated that aquaporin levels can decrease by more than 50% even seven days after relief of the obstruction [70]. This mechanism is considered one of the key factors underlying post-obstructive polyuria, in which the kidney's ability to concentrate urine is impaired.

In addition, obstruction affects sodium transport: within just 24 hours of urinary outflow impairment, a decrease in the expression of sodium channels is observed, leading to defective reabsorption and the development of natriuresis, which clinically manifests as a salt-wasting syndrome [71].

One of the most serious electrolyte disturbances in obstruction is hyperkalemia. Reduced glomerular filtration leads to decreased potassium clearance, and subsequent hyponatremia in the distal nephron (as a consequence of natriuresis) disrupts the sodium–potassium exchange mechanism, making effective urinary potassium excretion impossible [72]. Moreover, obstruction also impairs the kidney's acid-excretory function. The secretion of hydrogen ions in the distal tubules is reduced, which may clinically present as type I (distal) renal tubular acidosis [6].

If urinary tract obstruction persists without adequate intervention, it inevitably leads to the development of interstitial fibrosis, loss of tone (atony) of the pelvicalyceal system, and ultimately to end-stage renal disease (ESRD). This process may take anywhere from several days to several months, depending on the severity and duration of the obstruction. Experimental models have shown that after ureteral ligation, residual renal function can be maintained for at least two weeks, but generally not beyond four weeks [73].

Interestingly, even with complete obstruction, renal function does not cease immediately. Once a critical intrapelvic pressure is reached, a phenomenon known as pyelointerstitial backflow occurs—urine begins to seep from the renal pelvis into the interstitial tissue and is subsequently drained via lymphatic vessels [74]. This protective mechanism partially offsets the pressure rise within the system and explains why spontaneous rupture of the pelvicalyceal system in obstruction is extremely rare [75].

Relief of obstruction leads to partial or complete restoration of urinary outflow and initiation of reparative processes. Recovery of renal function may occur relatively rapidly due to functional hypertrophy of the remaining intact

nephrons. However, complete normalization of kidney function is not always achieved—partial recovery remains a common outcome, particularly in cases of prolonged or bilateral obstruction [76].

Approximately two-thirds of patients with obstructive uropathy develop a polyuric phase following relief of obstruction. This represents a physiological renal response aimed at restoring circulating volume and homeostasis. The mechanism is similar to that observed in the polyuric phase following recovery from acute tubular necrosis. In such cases, diuresis is driven by the osmotic excretion of previously retained metabolites—urea, creatinine, and other nitrogenous compounds—and usually resolves spontaneously within 24 hours [1].

However, when polyuria is pronounced or prolonged, it may represent a pathological condition known as post-obstructive diuresis (POD). POD is defined as a urine output exceeding 200 mL/h for two consecutive hours or a total daily urine output greater than 3 liters [77]. In most cases, POD does not persist for more than 48 hours. The principal mechanisms of POD are considered to include impaired urine-concentrating ability due to decreased aquaporin expression and loss of vascular tone in the renal glomeruli, as well as tubular transport dysfunction caused by apoptosis of renal tubular epithelial cells [78].

The incidence of POD varies according to the severity and duration of the obstruction: up to 2% of cases in complete unilateral obstruction and up to 50% in bilateral obstruction [79]. Major risk factors for POD include complete or chronic obstruction, markedly elevated serum creatinine, and a large volume of residual urine in the bladder at presentation. For example, residual bladder volumes greater than 1500 mL have been strongly associated with the development of POD [77,80]. Given the potential consequences, including fluid–electrolyte imbalance and hypovolemia, clinicians are advised to closely monitor urine output dynamics and serum electrolyte levels in patients at high risk for POD.

4. Conclusions

This review confirms that obstructive uropathies represent not only one of the most common causes of urgent urological consultations but also a significant clinical and social problem, often leading to severe complications, including end-stage renal disease. Impaired urinary outflow, regardless of the level of obstruction, initiates a complex cascade of pathophysiological responses involving hemodynamic disturbances, tubular injury, electrolyte and metabolic disorders, as well as infectious and inflammatory complications.

The relevance of this issue is increasing in the context of an aging population, a higher prevalence of comorbid conditions, and the expanding use of invasive diagnostic and therapeutic procedures, which contribute to a growing proportion of iatrogenic causes of obstruction. Current epidemiological trends indicate a steady rise in the incidence of urolithiasis, benign prostatic hyperplasia, and urethral strictures. These trends underscore the need to enhance clinician awareness, develop and implement standardized protocols for early detection, risk stratification, and prevention of complications, and optimize patient management strategies for obstructive uropathies across all stages of medical care.

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