Renal, Upper Tract Obstruction

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1. Introduction

Obstruction of the urinary tract results in many functional changes in the kidney (**Table 1**).[1] The severity of these changes depends on the degree of obstruction (partial or complete, unilateral or bilateral), the chronicity (acute or chronic) and the baseline condition of the kidneys including their potential for recovery. Complete obstruction results in progressive injury to the mechanisms of urinary concentration and acidification and can lead to permanent renal damage.[2] The term *hydronephrosis* refers to dilation of the renal pelvis or calyces and may be due to obstruction, but also may be present in the absence of obstruction. The term *obstructive uropathy* refers the functional obstruction of urinary flow and *obstructive nephropathy* is present when the obstruction causes renal damage.[1]

Table 1. Causes of Upper Tract Obstruction

	Congenital	Neoplastic	Inflammatory	Metabolic	Miscellaneous
Renal	• Polycystic kidney • Peripelvic cyst • Ureteropelvic junction obstruction	• Wilms tumor • Renal cell carcinoma • Urothelial cell carcinoma • Multiple myeloma	• Tuberculosis • *Echinococcus* infection	• Calculi	• Sloughed papillae • Trauma
Ureter	• Stricture • Ureterocele • Obstructing megaureter • Retrocaval ureter • Prune-belly syndrome	• Primary ureteral carcinoma • Metastatic carcinoma	• Tuberculosis • Amyloidosis • Schistosomiasis • Abscess • Ureteritis cystica • Endometriosis		• Retroperitoneal fibrosis • Pelvic lipomatosis • Aortic aneurysm • Trauma • Pregnancy
Bladder and Urethra	• Posterior urethral valves • Phimosis • Hydrocolpos	• Bladder carcinoma • Prostate carcinoma • Urethral carcinoma • Penile carcinoma	• Prostatitis • Periurethral abscess		• Benign prostatic hypertrophy • Neurogenic bladder • Urethral stricture

2. Effects on Glomerular Filtration and Renal Blood Flow

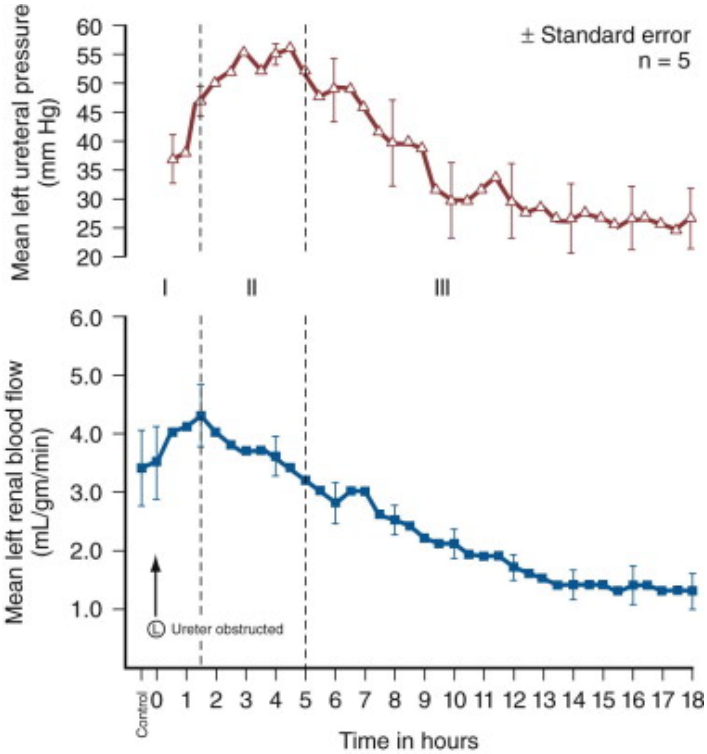

Figure 1: Triphasic relationship between ipsilateral renal blood flow and left ureteral pressure during 18 hours of left-sided occlusion.

The effects of obstruction on glomerular filtration rate (GFR) and renal blood flow (RBF) vary between unilateral and bilateral obstruction. Unilateral obstruction of a solitary kidney behaves as bilateral obstruction.

2.1 Unilateral ureteral obstruction

(see **Figure 1**)[1]

Phase 1: The first phase lasts 1 to 2 hours and both ureteral pressure and RBF rise dramatically. Vasodilation of the afferent arteriole increases RBF which offsets the fall in GFR. This response is mediated by the tubuloglomerular feedback mechanism, prostaglandin E2 and nitric oxide.[1] GFR decreases by 50% in 4 hours, 75% in 12 hours and by 95% within 24 hours.[2]

Phase 2: The second phase lasts 3 to 4 hours and is characterized by a continued rise in ureteral pressure, but RBF begins to decrease. Vasoconstrictors such as angiotensin II and thromboxane A_2 play an important role in this phase. Infusion of an angiotensin-converting enzyme (ACE) inhibitor mitigates the decline in GFR and RBF, providing evidence that angiotensin II is involved.[1]

Phase 3: The third phase begins at about 5 hours after obstruction. RBF and ureteral pressure decline together in this phase. This is due to an increase in afferent arteriole resistance. RBF shifts from the outer cortex to the medullary regions resulting in a lack of perfusion to many glomeruli, thus reducing GFR further.[1] RBF decreases to 70% at 24 hours, 50% at 72 hours, 20% at 2 weeks and 12% at 8 weeks.[3]

2.2 Bilateral ureteral obstruction

In the early portion of bilateral ureteral obstruction, RBF increases only slightly for approximately 90 minutes. This is likely mediated by nitric oxide. This is followed by an extreme decline in RBF that is much greater than in unilateral obstruction. The involved vasoconstrictors are thromboxane A_2, endothelin and angiotensin II.[1] Medullary blood flow decreases greatly, thus the shift of blood flow in bilateral obstruction is opposite of unilateral obstruction.[1] Ureteral pressure is much higher in bilateral obstruction and remains elevated for at least 24 hours.[1] These differences are hypothesized to be due to an accumulation of vasoactive substances that do not normally accumulate in unilateral obstruction because they would be excreted by the contralateral kidney. Atrial natriuretic peptide (ANP) is one of these substances.[1]

3. Cellular and Molecular Changes

During obstruction, renal tubular cells atrophy and ultimately die via apoptosis. Glomerular cells appear to be resistant to

obstruction-induced apoptosis[3]. Tubular cell apoptosis is mediated by cysteine aspartate-specific proteinases (caspases) which generate significant inflammatory responses and cytokine release. TNF-α is a directly cytotoxic cytokine that also induces apoptosis and plays a key role in tubular cell death after obstruction[3].

Continued urinary tract obstruction leads to the development of tubulointerstitial fibrosis and interstitial inflammation which results in eventual permanent changes to the structure of the kidney. In addition to TNF-α, other important cytokines and growth factors that a play a role in these changes are TGF-β, angiotensin II and NFκB. Obstruction also causes accumulation of extracellular matrix which further contributes to fibrosis[3].

4. Postobstructive Diuresis

After the release of bilateral ureteral obstruction, urine flow increases 3 to 10 – fold. This diuresis, often termed *postobstructive diuresis,* is typically physiologic in that water and endogenous salts retained during the obstruction are excreted. However, a pathologic diuresis occurs when the kidneys excrete salt and water in excess of that retained during the obstruction[3]. There are 4 proposed mechanisms of postobstructive diuresis.[1,2,3]

1. Impaired sodium reabsorption due to tubular damage
2. Impaired urinary concentration ability
3. Solute diuresis due to excretion of retained urea
4. Presence of a circulating natriuretic factor (ANP)

Postobstructive diuresis does not appear to occur after the release of unilateral ureteral obstruction, although there have been cases reported[2]. Patients who are susceptible to pathologic postobstructive diuresis usually exhibit signs of fluid overload prior to relief of the obstruction. Serum electrolytes, blood urea nitrogen and creatinine should be monitored and the frequency of monitoring should depend on risk factors, mental status, renal function and electrolyte status. The clinically stable patient with good cognitive function should be given free access to fluids. If the patient is unable to take in liquids, then they should receive intravenous fluids, but below the normal maintenance rate. The majority of patients have a physiologic diuresis and this is self-limiting. However, if a pathologic diuresis develops then patients can become hypovolemic and develop severe electrolyte abnormalities[1].

5. Diagnosis and Treatment

5.1 Diagnosis

Many diagnostic tests can be uses in the evaluation of upper urinary tract obstruction including ultrasonography, retrograde and antegrade pyelography, excretory urography, nuclear renography, computed tomography, and magnetic resonance imaging. The advantages and disadvantages of each are outlined in **Table 2**.[1]

The Whitaker test is was first described in 1973 and involves the measurement of renal pelvic pressure during infusion of saline into the collecting system through a percutaneous needle or nephrostomy tube at a fixed rate of 10 mL/min[1]. Intravesical pressure is monitored via a catheter in the bladder and the difference between the measured intrapelvic and intravesical pressures is used to determine true intrapelvic pressure. A true intrapelvic pressure of less than 15 cm H_2O is considered normal, greater than 20 cm H_2O indicates obstruction, and between 15 and 20 cm H_2O is equivocal.[1]

Table 2. Overview of Imaging Modalities Used to Diagnose Upper Tract Obstruction.

Imaging Modality	Advantages	Disadvantages
Ultrasonography	No ionizing radiation or iodinated contrast, inexpensive & widely available.	Identifies hydronephrosis, but provides no functional information.
Excretory Urography	Provides both anatomic and functional information & delayed images show anatomic level of obstruction.	Risk of contrast nephropathy.
Retrograde and antegrade pyelography	Accurately defines anatomy & can be used in renal insufficiency.	Invasive.
Nuclear Renography	No iodinated contrast & useful functional information.	Lacks anatomic information.
Computed Tomography	Superior anatomic information due to cross sectional imaging.	Ionizing radiation & risk of contrast nephropathy.
Magnetic Resonance Imaging	Superior anatomic information due to cross sectional imaging.	Time consuming, costly & risk of nephrogenic systemic fibrosis with gadolinium agents.

5.2 Treatment

Treatment of upper tract obstruction involves addressing the underlying etiology and renal drainage. Immediate renal drainage should be pursued for obstruction that is symptomatic, complicated by infection, bilateral, or resulting in renal failure[1]. The kidney can be drained promptly using either indwelling ureteral stents or percutaneous nephrostomy tubes. The choice between the two methods depends on the clinical setting. Indwelling stents are associated with bothersome urinary symptoms such as dysuria, frequency and urgency. Percutaneous nephrostomy tubes require an external bag to collect urine. Despite these downsides, studies have not demonstrated a difference in health-related quality of life[3] for urgent relief of stone related obstruction, the clinical efficacy, availability and patient preference is the same regardless of the method chosen, but indwelling stent placement is more costly by a factor of 2[4]. For patients with uncorrected coagulopathy, indwelling stent placement should be the procedure of choice. Ureteral stenting is likely not as effective in treating patients with extrinsic compression[1]. Finally, most ureteral stents are placed under anesthesia and the patient's ability to tolerate anesthesia should be considered.

6. Abbreviations:

GFR: Glomerular Filtration Rate
RBF: Renal Blood Flow
ACE: Angiotensin converting enzyme
ANP: Atrial natriuretic peptide

References

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