Approach and Management of Obstructive Uropathy
INTRODUCTION

• One of the most urgent clinical entity need to be diagnosed\(^1\).

• 10% of the causes of ARF and 4% of CRF\(^2\).

• Symptoms and signs of obstruction are often mild, occurring over long periods of time and requiring a high index of suspicion for diagnosis.

• Early recognition and treatment are the keys to preventing renal loss.

**TERMINOLOGY**

- **Obstructive uropathy** is structural impedance to the flow of urine anywhere along the urinary tract. 
  Walsh, Retnik, Vaughan, & Wein, 1998

- **Hydronephrosis**, which is dilation of the renal pelvis and calyces resulting from obstruction to flow of urine.

- The damage caused by these conditions often leads to **obstructive nephropathy**, that is, damage to the renal parenchyma (both anatomical and functional).
CLASSIFICATION

- Unilateral vs bilateral obstruction
- Degree of obstruction – partial or complete
- Duration – acute or chronic
- Site of obstruction – upper tract or lower tract
ETIOLOGY

• Inflammatory
  • TB, cystic ureteritis, schistosomiasis, amyloidosis
  • Prostatitis, periurethral abscess

• Neoplastic
  • BCa, PCa, primary Ca of ureter, metastatic Ca

• Inherited
  • Ureterocele, obstructive megaureter, retrocaval ureter, posterior urethral valve, PUJO

• Miscellaneous
  • Urolithiasis, PUJO, urethral or ureteral stricture, BPH, GU trauma
ETIOLOGY

- Infants and children
- Adult
- Pregnancy
- Urethral
- Bladder
- Ureteral
- Intra-renal

Common causes of obstruction include urolithiasis, PUJO, BPH, strictures of the ureter or urethra and urinary tract trauma.
### Table 2.5.1 Congenital causes of urinary tract obstruction

<table>
<thead>
<tr>
<th>Region</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal pelvis</td>
<td>Ureteropelvic junction anomalies</td>
</tr>
<tr>
<td></td>
<td>Aberrant renal artery</td>
</tr>
<tr>
<td>Ureter</td>
<td>Collector system duplicity</td>
</tr>
<tr>
<td></td>
<td>Ectopic ureter</td>
</tr>
<tr>
<td></td>
<td>Ureterocele</td>
</tr>
<tr>
<td></td>
<td>Ureterovesical junction anomalies</td>
</tr>
<tr>
<td>Bladder</td>
<td>Neurogenic (spina bifida)</td>
</tr>
<tr>
<td>Urethra</td>
<td>Atresia</td>
</tr>
<tr>
<td></td>
<td>Valves</td>
</tr>
<tr>
<td></td>
<td>Stenosis</td>
</tr>
</tbody>
</table>

### Table 2.5.2 Causes of obstructions developing in existing renal disease

<table>
<thead>
<tr>
<th>Region</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detachment of papillae</td>
<td>Analgesic-induced nephropathy</td>
</tr>
<tr>
<td></td>
<td>Diabetic necrotizing papillitis</td>
</tr>
<tr>
<td>Clots</td>
<td>Polycystic kidney</td>
</tr>
<tr>
<td>Neurogenic bladder</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Ureteral and vesical fibrosis</td>
<td>Renal tuberculosis</td>
</tr>
</tbody>
</table>

### Table 2.5.3 Main causes of acquired obstruction and related sites

<table>
<thead>
<tr>
<th>Region</th>
<th>Site</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal pelvis</td>
<td>Lumen</td>
<td>Stones, clots, neoplasms, urate crystals</td>
</tr>
<tr>
<td></td>
<td>Wall</td>
<td>Ureteropelvic junction obstruction</td>
</tr>
<tr>
<td></td>
<td>Extrinsic</td>
<td>Narrowing due to surgical sequelae</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neoplasms</td>
</tr>
<tr>
<td>Ureter</td>
<td>Lumen</td>
<td>Stones, clots, neoplasms, detached papillae</td>
</tr>
<tr>
<td></td>
<td>Wall</td>
<td>Narrowing induced by trauma, radiation, inflammation</td>
</tr>
<tr>
<td></td>
<td>Extrinsic</td>
<td>Retroperitoneal lesions (fibrosis, neoplastic disease, hemorrhage, sequelae of surgery)</td>
</tr>
<tr>
<td>Bladder</td>
<td>Lumen</td>
<td>Stones, clots, neoplasms</td>
</tr>
<tr>
<td></td>
<td>Wall</td>
<td>Interstitial cystitis, tuberculosis</td>
</tr>
<tr>
<td></td>
<td>Extrinsic</td>
<td>Neurogenic bladder (spinal lesions, diabetes mellitus)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prostatic or cervical cancer</td>
</tr>
<tr>
<td>Urethra</td>
<td>Lumen</td>
<td>Neoplasms, foreign bodies</td>
</tr>
<tr>
<td></td>
<td>Wall</td>
<td>Narrowing (due to trauma, tuberculosis, inflammation)</td>
</tr>
<tr>
<td></td>
<td>Extrinsic</td>
<td>Prostatic hypertrophy or carcinoma</td>
</tr>
</tbody>
</table>
Regardless of the cause, the ultimate effect is the same, an increase in the hydrostatic pressure of the collecting system, which is transmitted into Bowman space.

This reduces the GFR and initiates a cascade of events that, if not reversed, will result in kidney scarring and loss of kidney function.

The extent of kidney function loss and the damage to the physical structures of the collecting system varies depending on the duration and completeness of the obstruction.

The decrease in kidney function is determined by the loss of GFR and the loss of tubular functions.
Vasoconstriction of the renal vasculature causing decreased renal blood flow and decreased GFR triggers the initial sequence of events leading to obstructive nephropathy.

If obstruction is relieved within 24 hours, the vasoconstrictive process can be stopped before significant biochemical and immunological activation, and the potential for full recovery of renal function is good. (Klahr, 1998)

With increased severity and length of obstruction, the prognosis for recovery diminishes. With standing the length of time of obstruction, the ability of the kidney to function immediately following relief is undependable. (Chevalier & Klahr, 1998)
PATHOPHYSIOLOGY

- **Acute - 1 to 2 first hours**
  - In unilateral RBF is reduced in the obstructed kidney, but because of the existence of the contralateral healthy kidney, the GFR remains stable in this phase.
  - In bilateral obstruction, GFR decreases in relevance with the degree of obstruction.
  - Tubular damage starts 5 min after obstruction due to the increasing intratubular pressure. The increased pressure is transmitted to the site of Bowman’s capsule. This results in the decrease of the intravessel hydrostatic pressure and in the decrease of the GRF. Changes in the RBF further reduce the GFR.

- **Intermediate 2 - 5 hours**
  - A progressive decline of up to 50% of RBF due to the increased renal vascular resistance due to the composition of vasoconstrictor agents such as thromboxane A2 and angiotensin II. Since RBF is reduced, the pressure filtration system in the glomerulus is reduced and there is a further decline in GFR.

- **Late - After 24 hours**
  - Fibrosis, atrophy and interstitial cell apoptosis take place in the tubules. This is due to the increased synthesis of metalloproteinases, which results in extracellular fibrin deposition and the activity of macrophages.
  - Atrophy are related to the increased pressure and ischemia of arcuate arteries crossing the base of the renal pyramids.
  - If obstruction is not relieved, cell death occurs within approximately 15 days. These histological lesions remain partially after the relief of the obstruction, explaining in some cases the persistence of renal insufficiency.
HISTORY

- Obstructive nephropathy should be considered especially in uremic patients without a previous history of renal disease, hypertension, or diabetes.

- Gross or microscopic hematuria often is associated with renal calculi, papillary necrosis, and tumors, all of which can cause obstruction.

- Recurrent UTIs should always lead to an investigation for urinary obstruction.

- New-onset or poorly controlled hypertension secondary to obstruction and increased renin-angiotensin has been reported.

- Polycythemia secondary to increased erythropoietin production in the hydronephrotic kidney also has been reported.

- History of recent gynecologic or abdominal surgery can give important clues to the etiology of urinary obstruction.

- Pediatric recurrent infections and voiding dysfunction such as enuresis, incontinence, or urgency should be sought.
PHYSICAL EXAMINATION

• Evaluation for signs of dehydration and intravascular volume depletion; peripheral edema, hypertension, and signs of congestive heart failure from fluid overload may be observed in obstruction from renal failure.

• Palpable kidney or bladder (indicative of a dilated urinary collection system)

• Rectal or pelvic examination to help determine whether enlargement of pelvic organs is a possible source of urinary obstruction.

• Examination of the external urethra for phimosis or meatal stenosis.
DIAGNOSTIC WORKUP

• Early diagnosis is crucial.

• Laboratory studies
  • Urinalysis
  • Serum electrolyte
  • FBC
IMAGING STUDIES

- KUB X-ray can revealed radiopaque urinary tract calculi.

- Goal standard include USG, CT and CTU.
  
  - Ultrasonography is the procedure of choice to determine the presence of hydronephrosis.

  - USG is 98% sensitive for detecting hydronephrosis secondary to obstruction, but the specificity is 78 %.
    
    Koelliker & Cronan, 1997

  - Laing et al reported that when hydronephrosis was diagnosed, there was 35% false negative result in the diagnosis of acute obstructive uropathy.
    
    Laing, Jeffrey, Wing 1985

  - CT equals the accuracy of the IVP in determining the presence of obstruction, but surpasses the IVP in detecting the specific cause of the obstruction.

    Smith et al. 1995
...IMAGING STUDIES

- Others include IVU, MRI or renal scintigraphy or MCUG

- RPG
  - Provides the same information as IVP without dependence upon renal function and does not expose the patient to nephrotoxicity from intravenous dye.

- APG
  - indicated when the anatomy of the upper urinary tract needs to be assessed but IVU or RPG cannot be used.
  - The Whitaker test

- Bladder scan - PVR
THE WHITAKER TEST

• Also known as the ureteral perfusion challenge, is an invasive provocative maneuver used to differentiate obstructive from non-obstructive uropathy in the presence of a dilated collecting system:

• Patients with dilated renal pelvis and suspected ureteropelvic obstruction,
• Patients with dilated ureters and concern for obstruction,
• Patients following treatment for collecting system obstruction,
• Renal transplant patients with hydronephrosis, and
• Patients with urinary obstruction who do not demonstrate dilatation of the collecting system.
• A normal absolute renal pelvis pressure is less than 25 mm H2O. If the absolute pressure is above 30 mm H2O or an abnormally high resting gradient is detected, we have the diagnosis and no provocaive challenge is needed.

• If the absolute pressure is normal, the test continues by measuring the pressure gradient between the renal pelvis and urinary bladder.

• Pressure gradient:
  • less than 15 mm H2O at maximal fluid challenge
  • above 22 is diagnostic
  • between 15 and 22 are indeterminate.
MANAGEMENT

• The collaborative management of obstructive nephropathy involves primary care providers, nephrologists, urologists, and both advanced practice and staff nurses.

• Recognize and treat life-threatening complications of obstructive uropathy (e.g., pulmonary edema, hypovolemia, urosepsis, hyperkalemia).

• Reestablishment of urinary flow.

• Initiation of specific therapy for obstruction.
ACUTE MANAGEMENT

- Indications for emergency treatment:
  - Bilateral obstruction
  - Urosepsis
  - Uraemia
  - Hyperkalaemia
  - Persistence renal colic
  - Worsening hydronephrosis
  - Worsening renal impairment
TREATMENT

- Medical treatment options depend on cause and include:
  - MET to help facilitate stone passage
  - Increased water intake to prevent stone development
  - Urinary catheterization and combination medication in BPH
  - Immunosuppressants in retroperitoneal fibrosis

- Surgical treatment of obstructive uropathy includes ureteral stents and percutaneous nephrostomy tubes.
JJ stent vs NS

• No difference in efficacy


Pearl MS et al. Optimal methods of urgent decompression of the collecting system for obstruction and infection due to ureteral calculi. J Urology 1998; 160: 1260-4


• Options:
  • Anti-coagulant – JJ stent
  • Urosepsis – NS
  • Malignancy – NS
POST-OBSSTRUCTIVE DIURESIS

• It is characterized by a marked natruresis and diuresis with excretion of large amounts of sodium and water.

• In addition to the potential for severe volume depletion, electrolyte disorders such as hypokalemia, hyponatremia, hypernatremia, and hypomagnesemia may occur.

• Etiology of this massive diuresis and electrolyte loss is multifactorial. It is related to fluid and urea overloads during obstruction and acquired tubular resistance to antidiuretic hormone and aldosterone.

• Treatment of post-obstructive diuresis consists of judicious fluid replacement with 0.45% saline (at a rate slightly less than urine output) and replacement of electrolytes.
PAIN MANAGEMENT

- Pain due to increase pressure in collecting system and due to ureteral wall or renal capsule distension.

- Drugs:
  - NSAIDS
  - Narcotics
  - Alpha blocker

- NSAIDs
  - reduce collective system pressure
  - inhibit PG synthesis
  - reduce RBF
PROGNOSIS

- Age, duration of obstruction, initial renal function and existence of urine extravasation.

- Obstruction remaining for more than 6 weeks results in the emergence of a degree of irreversible chronic renal disease.
  
  - The longer the duration of obstruction, the lower the probability of recovery of any renal function and the lower the resultant GFR.
  - In humans, partial recovery of function frequently is observed after less than 3 weeks of obstruction; however, case reports have noted some return of function after 5 months of obstruction.

- UTIs complicating obstruction further decrease the probability of recovery.
Diagnostic Work-up and Management of Obstructive Uropathy

- Acute Flank Pain
  - Kidney ultrasound
    - Upper Urinary Tract Dilatation
      - MRU
      - IVU
      - Doppler Ultrasound
      - NCCT
      - CT(IC)
        - Fever
        - Vomiting
        - Signs of Sepsis
        - Lower urinary tract malignancy
          - Percutaneous Nephrostomy
          - JJ stent placement
          - Surgical Intervention (ESWL, URS)
        - Coagulopathy Stone
          - (No symptoms)
            - Persistent Pain
              - Renal scintigraphy
                - <10% renal function
                  - Consider Nephrectomy
                - >10% renal function
CONCLUSION

• Obstructive nephropathy is a frequent but preventable disease.

• Time is an important determinant of outcome for patients with obstructive uropathy.

• Obstructions need to be identified and addressed before the progression to obstructive nephropathy.
CASE DISCUSSION

- 62 male
- Lt distal ureteric calculus with mild to moderate HN + HU.
- Background HPT/DM
- Recent MI May 2015 – angioplasty done; on double anti-platelet
- Asymptomatic
- Creatinine 116 to 126
- Stent or not? JJ vs NS
THANK YOU