Post Obstructive Diuresis

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Common case presentation

75-year-old man presents to the emergency room complaining of lower abdominal pain and difficulty urinating. A bladder ultrasound performed by the emergency room physician demonstrates a distended bladder. A catheter is placed, and two liters of urine is immediately drained. You are called to assess if the patient needs to be admitted for monitoring.

I. Receiving the phone call and initial thoughts

a. **Does the patient have a history of urologic diagnoses, e.g. BPH, urinary retention, prostatic surgery, urethra stricture?**
   Acute urinary retention is a common cause of emergency room visits, and most commonly this is caused by prostatic enlargement. Prostatic surgery, endoscopic resection or prostatectomy can lead to bladder neck contracture or anastomotic stricture.
b. **How long has the patient been in retention?**
   Patients with acute urinary retention typically do not have electrolyte abnormalities or complications following relief of urinary obstruction. Patients with longstanding or chronic urinary obstruction are more likely to have electrolyte abnormalities.
c. **What are the vital signs?**
   Hypertension is a sign of volume overload while hypotension can be a sign of urinary infection.
d. **What is his baseline and current renal function?**
   Without prompt drainage, urinary obstruction can impair renal function. Patients with poor baseline renal function will have higher risk of complications and electrolyte abnormalities.
e. **What medications is the patient taking?**
   Certain medications such as anticholinergics and narcotics can result in urinary retention. Diuretics can exacerbate diuresis and worsen electrolyte abnormalities.
f. **What is the patient's cognitive capacity?**
   Patients who are cognitively impaired should warrant additional attention as they will have difficulty at consuming fluids orally to prevent hypovolemia and mental status changes can be associated with electrolyte derangements.

II. Differential Diagnosis

a. **Postobstructive diuresis.**
   This is a physiologic state wherein copious water and salt is eliminated after the relief of severe urinary tract obstruction.[1] It is a relatively common occurrence following relief of bladder outlet obstruction, ureteral obstruction of both kidneys, or ureteral obstruction of a solitary kidney.[2] Postobstructive diuresis is defined as urine output > 200 mL/hour for two consecutive hours or > 3L/24 hours after the obstruction is relieved. Most cases are a self-limited phenomenon and are deemed ‘physiological diuresis'. This is a normal response to solute and water overload and terminates after the patient returns to a euvolemic state. In some cases, patients can experience ‘pathologic’ postobstructive diuresis which can be life-threatening.[3] This is an inappropriate diuresis of water beyond the euvolemic state. The pathophysiology is not fully understood, but can be related to a temporary state of nephrogenic diabetes insipidus (i.e. impaired renal tubular response to anti-diuretic hormone), a decreased medullary concentrating gradient, decreased tubular resorption of sodium and water due to an increased tubular transit flow time, accumulation of ANP, and release of prostaglandins immediately following relief of obstruction.
b. **Central diabetes insipidus.**
c. **Congenital or acquired nephrogenic insipidus**
d. **Psychogenic polydipsia**
e. **High protein or hyperglycemic osmotic diuresis**
f. **Salt-wasting nephropathy**

III. Evaluation

a. **Physical exam - Key Points**
   i. **Vital signs.**
      Fever, tachycardia, or hypotension can be signs of severe infection or sepsis. Patients with urinary obstruction and retention may have severe urinary tract infection.
   ii. **Abdomen.**
      Costovertebral tenderness may be an indicator of pyelonephritis or obstructive uropathy causing hydronephrosis. Suprapubic discomfort may be an indication of cystitis or incomplete emptying of the urinary catheter.
   iii. **Genital.**
      Examination of the penis can rule out severe phimosis or urethral meatal stenosis as a culprit of urinary retention.
   iv. **Rectal Exam.**
      In the absence of severe infection and recent quantification of the prostate size, a DRE may be considered to approximate the size of the prostate.
   v. **Catheter.**
      Review the urine output from the catheter. Hematuria can occur in about 10% of cases

following an even non-traumatic insertion of a urinary catheter for urinary retention.[4] This is typically a mild phenomenon, but can result in clot retention or significant bleeding in patients with coagulopathy or in those on anticoagulation medication. Purulent urine is highly suggestive of infection. Concentrated urine is an indicator of the patient's fluid volume status at the time he went into retention.

b. **Laboratory data**

i. **Serum electrolytes.**
Check a comprehensive metabolic profile every 6-12 hours.

ii. **Complete blood count.**
Leukocytosis may indicate the presence of possible infection.

iii. **Urine osmolality or specific gravity.**
These tests measure the concentration of eliminated urine and serve to estimate the ability of the kidneys to dilute or concentrate urine in an effort to maintain fluid balance. In most situations, urine specific gravity is a good approximation of urine osmolality (meaning the number of solutes per unit of urine). Situations in which the urine specific gravity does not correlate well with urine osmolality include uncontrolled diabetes mellitus, nephrotic syndrome, saline diuresis, or administration of intravenous radiocontrast material.[5] Urine osmolality serves to measure the solute content of the urine, with urine osmolality of < 150 mOsm/kg representing water diuresis, 150-300 mOsm/kg a mixed diuresis, and 300-500 mOsm/kg representing a solute diuresis. For reference, normal plasma osmolality is approximately 290 mosm/kg. Urine specific gravity is typically available on a urinalysis and 'normal' specific gravity values typically range from 1.010-1.030. Increased urine specific gravity is indicative of concentrated urine while decreased urine specific gravity is indicative of dilute urine.[6]

iv. **Urinalysis and Urine Culture.**
These should be checked in the setting of acute urinary retention as urinary tract infection is both a culprit and result of urinary retention.

c. **Radiologic studies**

i. **Renal bladder ultrasound.**
Even after catheterization and decompression of the bladder, you may consider an ultrasound of the kidneys to rule out obstruction or the presence of hydronephrosis. Possible causes of persistent hydronephrosis following bladder decompression include chronic dilation of the upper urinary tract without obstruction, or secondary obstruction of the ureterovesical junction due to bladder hypertrophy.

ii. **Non-contrast CT scan abdomen and pelvis.**
Non-Contrast CT can be rapidly performed and shows better anatomic detail that either ultrasound or KUB, especially when upper urinary tract obstruction is suspected. Additionally, CT scan can be used to evaluate for renal calculi as a source of upper tract obstruction. Renal function is not impacted when non-contrast imaging protocols are utilized.

IV. Management

a. **Initial management/problem to resolve**
   i. Treatment begins with relief of urinary obstruction, either with insertion of a urethral catheter or suprapubic tube to decompress the bladder in the event of bladder outflow obstruction; or percutaneous insertion of nephrostomy tubes or indwelling ureteral stents in the instance of upper urinary tract obstruction. It was once believed that decompression should be gradual and outflow should be periodically clamped to prevent hematuria and postobstructive diuresis but there is no evidence that this is an effective strategy.[4,7]
   ii. Predicting who will develop pathologic postobstructive diuresis is difficult. Factors that have been implicated include a high serum creatinine level, high sodium bicarbonate level, signs of fluid overload (edema, congestive heart failure, hypertension).
   iii. Consider admission/hospitalization of patients at high risk of developing pathologic post-obstructive diuresis, those with significant derangements in renal function, electrolyte status, or mental status.
   iv. Check initial electrolytes and replete as necessary.

b. **Potential complications**
   i. **Hematuria.**
   Usually self-limited, however can require irrigation.
   ii. **Hypotension.**
   Indicative of under-resuscitation of fluids in the setting of fluid losses.
   iii. **Electrolyte disturbances.**

c. **Specific management plan**
   i. **Maintain urinary drainage.**
   This is not the time for a “trial of void.”
   ii. **Consider hospitalization:**
   Patients at higher risk of developing pathologic postobstructive diuresis, such as higher serum creatinine, higher bicarbonate, fluid overload, or central nervous system changes.
   iii. **Monitor Vitals.**
   Hypotension can occur.
   iv. **Strict monitoring of intake and outputs.**
   v. **Periodic check and repletion of serum electrolytes.**
   (Na, K, urea, creatinine, magnesium, phosphate)
   vi. **Allow the patient unrestricted access to water for drinking.**
   vii. **Intravenous fluid replacement.**
   In general, patients who have normal mental capacity and the ability to drink should have unrestricted access to fluids, predominantly water. IV fluids are administered to patients with inability to tolerate oral fluids, mental status changes, or clinical hypotension.

IV fluid administration is typically either 0.9% normal saline or 0.45% normal saline depending on the clinical scenario. As a rule of thumb, 0.5 mL of IV fluids per 1 mL of urine output should be administered in replacement. Blood pressure and urine osmolality should guide duration and volume of IV fluid administered. The goal is a negative balance: do not replace fluids at equal rate to losses as this may prolong or exacerbate the diuresis.

viii. **Consider daily weights**

ix. **Urine osmolality or urine specific gravity.**
This can be periodically monitored to determine if postobstructive diuresis is improving.

x. **Nephrology consultation.**
In particular for patients with prolonged postobstructive diuresis, complicating medications, or significant electrolyte abnormalities.

Key Takeaways

1. Post Obstructive Diuresis is uncommon following relief of urinary tract obstruction.
2. Most patients are able to replace fluid losses due to excessive diuresis with access water and oral fluid intake.
3. Patients requiring intravenous fluid replacement should receive 0.9% or 0.45% saline at a rate of 0.5 mL per 1 mL of urine output.
4. The goal of fluid replacement is hemodynamic support with a negative overall fluid balance.

Seminal Reference AUA Reference

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Figures

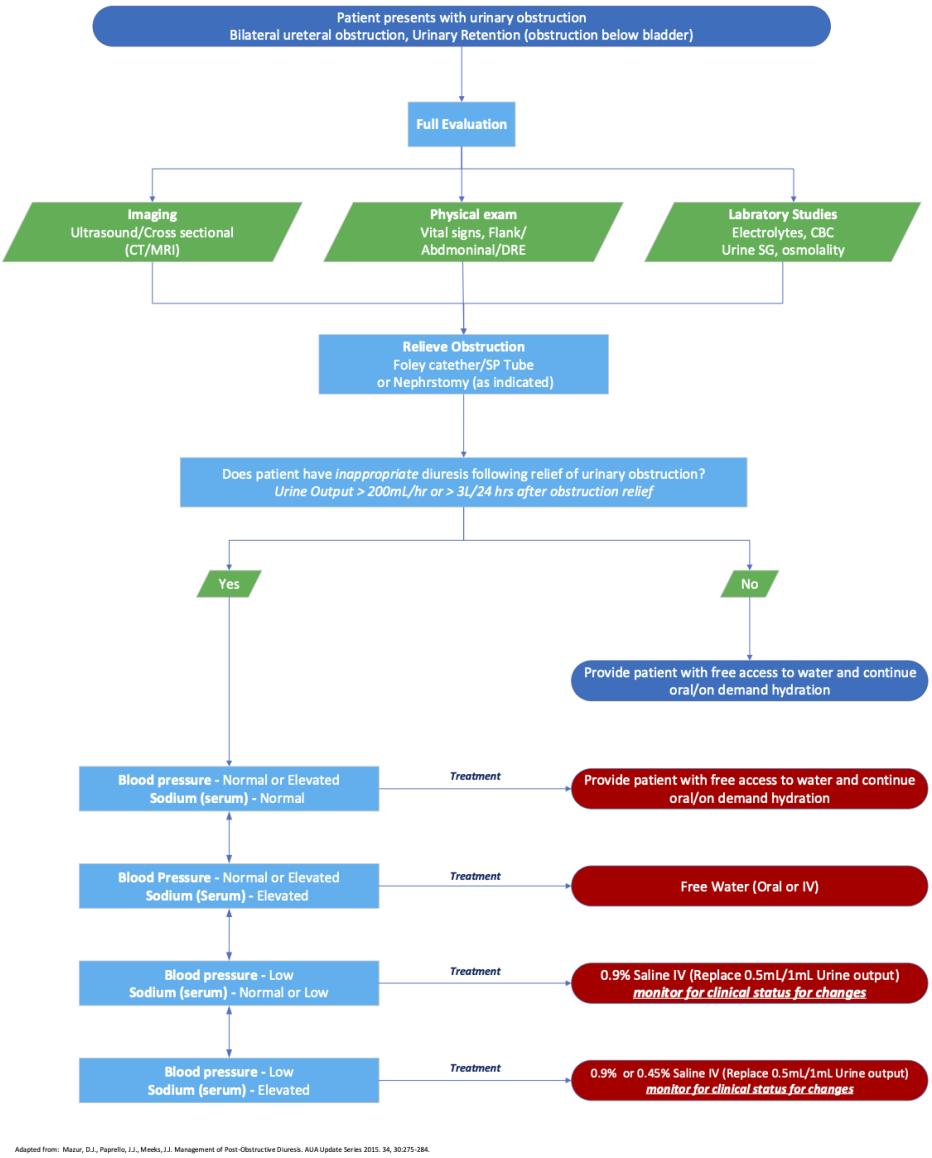

Figure 1: Management of Post-Obstructive Diuresis

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