Oliguria

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Common case presentation

You are called by the emergency room PA about a 72-year-old man who has not urinated in 24 hours. There is no record of his baseline renal function, but his current serum creatinine is 4.

I. Receiving the phone call and initial thoughts

a. **Is this obstructive uropathy?**
   There are prerenal, renal, and postrenal causes of azotemia. As a urologist, the most important information you can provide to the consulting physician is whether the lack of urine output is post-renal (i.e. obstructive).
b. **What is the patient's urologic history?**
   Nephrolithiasis can rarely be bilateral or cause urinary retention if the calculus gets stuck in the urethra. Patients on medications of benign prostatic hypertrophy (e.g. tamsulosin, finasteride, etc.) should prompt you to think of BPH as the culprit and consider the catheter you want to use in case for urinary drainage.
c. **What is the patient's baseline renal function?**
   This is relevant for defining acute kidney injury.

II. Differential Diagnosis

a. **Anuria**
b. **Oliguria**
   Defined as a urine output of < 500cc in 24 hours (adult) or <0.5mL/kg/hour (child or adult) or < 1 mL/kg/hr in a neonate
c. **Urinary retention**
   Prostate hypertrophy, urethral stricture, malignancy
d. **Obstructive uropathy of the upper tracts**

Common causes include nephrolithiasis, crystals (acyclovir, indinavir), clots, tumors

e. **Prerenal azotemia**
   Causes include sepsis, neurogenic shock, hemorrhagic shock, hypovolemia, cardio-renal syndrome, hepato-renal syndrome, abdominal compartment syndrome, medications that cause intrarenal vasoconstriction (non-steroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, cyclosporine, tacrolimus)
f. **Renal azotemia**
   Possibilities include glomerulonephritis / vasculitis, interstitial nephritis, pyelonephritis, viral infection (EBV, CMV, HIV), thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, rhabdomyolysis, myeloma, ischemic renal tubular damage, nephrotoxic medications, tumor lysis, renal vein thrombosis, malignant hypertension, sclerodermal renal crisis, renal atheroembolic disease, renal infarction
g. **Bladder rupture**
   Uncommon scenario but should be cognizant of possibly with trauma or in the setting of neobladder. Patients will often have a rise in serum creatinine due to reabsorption of urine through peritoneum in a ‘pseudo-azotemia’.

III. Evaluation

a. **Physical Exam**
   i. **Vitals**
      Tachycardia, and hypotension may be an indicator of hypovolemia or shock. Fever may be an indicator of infection which can be a source of sepsis, direct infection of the kidneys, or glomerular disease. Hypertension can be associated with pain or volume overload
   ii. **Lungs**
      Rales or crackles may be an indicator of pulmonary edema and volume overload
   iii. **Abdomen**
      Costovertebral angle tenderness may be an indicator of pyelonephritis or hydronephrosis. Suprapubic fullness or discomfort may be an indicator of bladder distention and/or infection. Ascites, caput medusa, and spider angioma can be a sign of liver disease
   iv. **Genitourinary**
      Look for meatal stenosis, digital rectal exam to estimate prostate size, high riding prostate in setting of trauma
   v. **Extremities**
      Peripheral edema can be associated with congestive heart failure, fluid volume overload
   vi. **Skin**
      Rash can indicate drug reaction and should prompt consideration of interstitial nephritis
b. **Labs**

i. **Serum electrolytes.**

1. **Serum creatinine**
   The Kidney Disease Improving Global Outcomes (KDIGO) classification of acute kidney injury is: serum creatinine of 0.3 ml/dl or more in 48 hours, rise to at least 1.5 fold from baseline within 7 days, or urine output of < 0.5mL/kg/hour for 6 hours[1]
2. **Serum blood urea nitrogen**
3. **Serum potassium**
   This is important to check and correct in the setting of renal failure. Hyperkalemia can be deadly.
4. **Urine microscopy**
   Renal tubular cells represent acute tubular injury. Leukocytes are indicative of inflammation. Red cell casts are diagnostic of glomerular disease. White cell casts are indicative of renal infection. Hyaline casts are a non-specific indicator of renal disease. Granular casts are indicative of more significant renal disease. The presence of renal tubular cells indicates acute tubular injury, while “muddy brown casts” are necrotic tubular cells indicating acute tubular injury. Dysmorphic red cells are indicative of glomerular disease (or a non-fresh urine sample) [1]
5. **Urine electrolytes**
   - Fractional excretion of sodium (FeNa). FeNa = (Urine [Na+] / plasma [Na+]) / (urine [creatinine] / plasma [creatinine]). A ratio of < 1 indicates the kidney is able to resorb sodium and the etiology is likely prerenal. A ratio of > 2 indicates the etiology likely is renal. FeNa is a less reliable measure of prerenal azotemia in patients on diuretics and the fractional excretion of urea may be helpful in this setting (<35 indicates a prerenal cause) [2,3]

ii. **Complete blood count with differential**
Leukocytosis may indicate infection. Anemia may be indicative of a prerenal etiology (i.e. hemorrhage)

c. **Imaging**
If physical exam is not obvious for urinary retention, this is key to determining if the patient has postrenal azotemia

i. **Bladder scan**
Useful to determine if there is bladder distention or a residual volume

ii. **Renal ultrasound**
May indicate bladder distention, hydronephrosis, or both

iii. **CT Scan**
If hydronephrosis is seen, a non-contrast CT scan may be useful in identifying cause of obstruction (i.e. stone or mass)

d. **Additional Tests**

i. **Measurement of intraabdominal pressure**
If there is suspected intraabdominal compartment syndrome, consider serial

measurements of intraabdominal pressure[4]

ii. **Central venous or pulmonary artery catheter**
to assess volume status

iii. **Pulmonary artery catheter**
to assess cardiac output

iv. **Testing of fluid responsiveness (bolus of normal saline)**

v. **Renal Biopsy**
Reserved for patients in whom prerenal and postrenal causes of AKI have been excluded and when cause of renal source is unclear. This may be a useful test as it may prompt the use of immunosuppressive medications or plasmapheresis. Typically, this will be coordinated after consultation with nephrology

vi. **Cystatin C**
Serum cystatin C can be used to estimate GFR similar to serum Creatinine but is not typically filtered into urine therefore not subject to reabsorption through the peritoneum. Patients with pseudoazotemia due to urine leak will have a discrepancy between GFR measured by Cystatin C compared to creatinine.[5,6]

IV. Management

a. **Initial management**
The management will depend on the cause of azotemia. Prerenal causes of AKI are typically managed with intravenous fluid administration and congestive heart failure is managed with diuretics and afterload reduction. Renal causes of AKI should prompt consultation with nephrology and/or internists.

i. For urinary retention, prompt catheter drainage is required. Most men with urinary retention have this due to BPH. A regular 14-18Fr Foley catheter can be tried, but if there is resistance at the level of the prostate then a coude catheter is preferred. If urethral catheterization is not possible, then a suprapubic catheter is warranted. Even without urinary retention, it may be advisable to place a urethral catheter to facilitate monitoring of urine output

ii. Don’t forget to irrigate the catheter if you do not get any output

iii. If there is obstructive uropathy involving the upper tracts without urinary retention, then placement of indwelling ureteral stents or percutaneous nephrostomy tubes is warranted

iv. **Avoid nephrotoxic drugs**

1. The list of nephrotoxic drugs is extensive. Commonly used drugs include NSAIDS, radiocontrast, ACE inhibitors, angiotensin receptor blockers, aminoglycosides, vancomycin, platinum-based chemotherapy, amphotericin B, the polymyxins, proton pump inhibitors, calcineurin inhibitors, lithium [7]

v. **Avoid hypotension**

vi. **Renal Replacement Therapy**

Continuous venovenous hemofiltration or hemodialysis may be required if there are refractory electrolyte abnormalities (i.e. hyperkalemia), fluid overload, uremic encephalopathy, pericarditis, pleuritic, or removal of certain toxins that cannot be managed conservatively

b. **Potential complications**
    i. Postobstructive diuresis is a known complication after relief of obstructive uropathy
    ii. Chronic kidney disease is a possible sequalae of acute kidney injury[8]
    iii. Uremia – anorexia, nausea, vomiting, weakness, myoclonic jerks, seizures, confusion, coma, uremic pericarditis
    iv. Electrolyte abnormalities – most concerning are hyperkalemia, hyponatremia, hypocalcemia, hyperphosphatemia, metabolic acidosis
    v. Fluid overload – signs include weight gain, peripheral edema, pulmonary edema, respiratory distress
c. **Specific management plan**
    Ultimately depends on the cause of oliguria/anuria. Assuming the source is postrenal (obstructive), patients should be worked up for the etiology and treated appropriately. Patients who have had urinary retention due to BPH should have a post-void residual checked following a trial of void. Patients treated for upper tract obstruction should have renal ultrasonography performed afterwards to ensure that there is no evidence of residual obstruction

Key Takeaways

- It is important to differentiate between prerenal, intrinsic, and postrenal causes of oliguria as this will dictate overall management.
- Labs, including urine microscopy and electrolytes, as well as imaging, can help differentiate causes.
- In obstructive etiologies, catheter placement for urinary retention or placement of a ureteral stent or percutaneous nephrostomy tube may be necessary. Patients should be monitored for post-obstructive diuresis and will require follow-up imaging to ensure resolution of obstruction.

Videos

AUA Core Curriculum: Oliguria

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