



Episode 150 AKI – A Simple ED Approach

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Defining AKI

The 2012 Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Acute Kidney Injury (AKI) [1] defines AKI by any of the following:

- Increase in serum creatinine by ≥ 0.3 mg/dL (>26.5 $\mu\text{mol/L}$) within 48 hours; or
- Increase in serum creatinine to ≥ 1.5 times baseline, which is known or presumed to have occurred within prior 7 days; or
- Urine volume <0.5 mL/kg/h for 6 hours

Stepwise approach to AKI in the ED

Step 1: Rule out the 2 immediate life-threats

1. **Hyperkalemia** – get ECG, electrolytes off the blood gas
2. **Severe acidosis** – get blood gas

Step 2: Assess for adequate perfusion – are they in shock?

Use your history, physical examination and POCUS to assess for perfusion and treat shock (hemorrhagic, vasodilatory, cardiogenic shock etc.) accordingly.

Step 3: Assess for both pulmonary and peripheral edema

Assess JVP and lungs with POCUS for pulmonary edema, look and palpate for peripheral edema (including pre-tibial edema, sacral edema)

If there is no evidence of pulmonary or peripheral edema, give a fluid challenge.

AKI with adequate perfusion, with pulmonary edema (with or without peripheral edema)

1. 1. Give furosemide 1 mg/kg IV (or 1.5 mg/kg IV if on furosemide already)

2. Think about pulmonary renal syndromes other than CHF (such as anti-GBM disease, ANCA associated vasculitis, circulating immune complex syndromes like lupus), and look for clinical clues (inflammatory arthritis, purpura, Raynaud's, mononeuritis multiplex, uveitis or Sicca syndrome ?)

AKI with adequate perfusion, with peripheral edema but not pulmonary edema

1. Give furosemide 1 mg/kg IV (or 1.5 mg/kg IV if on furosemide already)
2. If no improvement in renal function think about hypovolemia ("pre renal") despite peripheral edema

- o Low serum albumin – treat underlying cause, and consider [hepatorenal syndrome](#) which may require IV albumin
- o Venous insufficiency and/or lymphedema – give crystalloid, consider compression therapy
- o Drug induced edema – give crystalloid, reassess offending drug
- o Severe myxedema – give L-thyroxine and monitor

Step 4: The golden rules of AKI workup

1. Measure a **post-void residual (PVR)** with bladder scan or urethral catheter

2. Get a **urine dip** to look for blood and protein suggestive of **nephritic syndrome**
3. Monitor **urine output** ideally with a urethral catheter
4. **Avoid nephrotoxins** (NSAIDs, ACEi, ARBs, gentamicin etc)

Step 5: Consider imaging for a small subset of post-renal AKI

Radiology department imaging should be reserved for those patients who:

- Do not improve with fluid challenge (making pre-renal less likely),
- Have a normal urine dip (making intra-renal less likely),
- Have a post-void residual <100mL (making BPH less likely)
- Have obvious bilateral hydronephrosis on POCUS

These patients warrant further imaging as they might have a rare post-renal bilateral ureteric obstruction cause of AKI such as obstructive metastatic cancer, lymphoma or a kidney stone with a solitary kidney.

AKI pearls and pitfalls

- BUN:Cr ratio >1 is not reliable at distinguishing pre-renal from renal causes. [2]
- Hematuria and proteinuria are often overlooked or ignored in the ED and should trigger the consideration of nephritic syndrome as an intrarenal cause of AKI,

especially in those patients presenting with acute uncontrolled hypertension

- Urine electrolytes and fractional excretion of sodium are rarely required in the ED as they are nonspecific, very difficult to interpret without further inpatient workup and may be misleading; [3,4] They should be considered, however in patients suspected of hepatorenal syndrome.
- The fluid of choice for most patients with pre-renal AKI is Ringer's Lactate [5]
- Sodium bicarb should be considered in patients with uremic acidosis, however this is usually best suited for the ICU setting [6]
- The most common causes of post-renal AKI are BPH and urethral catheter obstruction; radiology department imaging to rule out a pelvic mass causing bilateral ureteral obstruction should be reserved for those with obvious hydronephrosis on POCUS but little or no post-void residual
- A common pitfall is to attribute AKI to obstructive nephrolithiasis; unless the patient has a solitary kidney, nephrolithiasis rarely causes AKI; look for other causes of AKI instead

Prerenal AKI

Prerenal AKI, caused by decreased renal perfusion, is the most common cause of all AKIs (90%) [7]. Prerenal causes occur in the setting of recent volume losses, such as hemorrhage,

gastrointestinal or urinary fluid losses, sepsis, and recent postoperative courses during which the patient was hypotensive.

Common causes of pre-renal AKI include:

- Volume depletion (renal losses – ie. Diuretics, and extra-renal losses – GI losses, third spacing, hemorrhage)
- Shock of any etiology
- Cardiorenal syndrome

Additional pre-renal causes of AKI to consider include:

- Hepatorenal syndrome
- Abdominal compartment syndrome
- Hypertensive emergency
- Thrombotic thrombocytopenic purpura & hemolytic uremic syndrome

Fluid of choice in AKI

Our experts recommend Ringers Lactate (RL) as the fluid of choice as it has relatively neutral effect on acid-base status and may reduce the risk of further AKI compared to Normal Saline. Consider 1L bolus up front followed by 150mL/hr and aim for a urine output of ≥ 50 mL/hr (≥ 200 mL/hr in rhabdomyolysis). Bicarb should be considered in patients with uremic acidosis based on the BICAR-ICU trial but probably best reserved for the ICU.

Intrinsic renal AKI

Intrinsic renal AKI is caused by direct injury to the kidney parenchyma. Intrarenal causes of AKI are usually considered only after pre-renal and post-renal causes have been ruled out. The “can’t miss” diagnosis for emergency physicians in a patient with AKI is *nephritic syndrome*. Thus it is important to get a urine dip to look for protein and blood in all patients with AKI.

Nephritic syndrome presents as: acutely elevated Cr with hypertension, hematuria, proteinuria and no obvious pre-renal or post-renal cause.

Other important causes of renal AKI to consider in the ED include:

- Nephrotoxic medications (ACEi, NSAIDs, Gentamicin etc.)
- Acute Tubular Necrosis (ATN) (rhabdomyolysis, hemolysis, tumor lysis syndrome)
- Renal thrombosis

Other lab test to differentiate pre-renal vs. renal AKI:

Other than ordering a urine dip to assess for intrinsic renal cause of AKI, there little role for measuring BUN in the ED.

BUN:Cr ratio is not reliable at distinguishing pre-renal from renal causes [2]. Similarly, there is a limited role for urine electrolytes in the ED except in suspected of hepatorenal syndrome.

Pearl: Nephritic syndrome presents with acutely elevated Cr, hypertension, hematuria, proteinuria and no obvious pre-renal or post-renal cause.

Post-renal AKI

Post-renal AKI is caused urologic obstruction to urine flow. It’s important to measure a post-void residual in all patients with an AKI. The most common causes are **BPH** and **urethral catheter obstruction**.

Imaging to rule out an obstructive cause should not be performed in every patient with AKI NYD. Imaging should be considered in patients with obvious bilateral hydronephrosis on POCUS but little or no post void residual. These patients warrant further imaging as they might have a rare post-renal bilateral ureteric obstruction cause of AKI such as obstructive metastatic cancer, lymphoma or a kidney stone with a solitary kidney.

Pitfall: A common pitfall is to attribute AKI to obstructive nephrolithiasis. Unless the patient has a solitary kidney, nephrolithiasis rarely causes AKI. Look for other causes of AKI instead.

Role of POCUS in AKI

POCUS can provide valuable information about the overall volume status of a patient by via assessment of the JVP, [IVC](#) and signs of pulmonary edema.

POCUS can often obviate the need for further advanced renal imaging, as it can also provide important sources information pertaining to post-renal/obstructive causes including assessing a

post-void residual, hydronephrosis and absence of ureteric jets. The absence of ureteral jets entering the bladder has a 90% positive predictive value for acute urinary tract obstruction [8].

However, the accuracy of POCUS by ED docs is provider dependent. Using the consensus radiology interpretation of POCUS as the reference standard, emergency physicians had an overall sensitivity of 85.7%, specificity of 65.9% [9] The sensitivity of POCUS for the detection of hydronephrosis was 77.1% and the specificity was 71.8% in a 2020 study of patients with nephrolithiasis [10]. The sensitivity of POCUS improved with worsening degrees of hydronephrosis.

Despite the above limitations, our experts recommend using POCUS to assess for hydronephrosis when the pre-test probability for obstruction gathered from history and physical exam is high.

Rhabdomyolysis and AKI

Studies have shown a correlation of CK levels greater than 5000 IU/L and 50% chance of progression to AKI [11].

When to order CK and other blood tests for rhabdomyolysis

1. Any one risk factor for rhabdomyolysis: trauma/compartament syndrome/crush, extreme exertion, hyperthermia, found down
2. Any one symptom of rhabdomyolysis: muscle pain, muscle weakness, vomiting, dark urine

Note that CK peaks at 24-72hr after the initial insult, so serial CKs are important to consider, especially if the insult was recent. If the CK is >1000, consider **calcium, phosphate and VBG** to estimate severity of rhabdomyolysis and their risk for requiring dialysis.

Management of rhabdomyolysis based on CK levels

1. CK<1000 – oral fluids usually adequate as long as CK is not trending upwards and McMahan score is low
2. CK 1000-5000 – usually requires IV RL and trending of CK and Cr are especially important to determine disposition
3. CK>5000 – usually needs IV RL and admission +/- dialysis if McMahan ≥ 6

The McMahan score estimates severity and requirement for dialysis in rhabdomyolysis patients, which includes age, sex, Cr, Ca, CK, phosphate and bicarb as the score parameters [12].

Urine myoglobin has little role in the ED diagnosis of rhabdomyolysis

There is a limited role for urine myoglobin because the half-life is only 2-3hrs, so a negative result after 4-6 hrs post insult may be misleading [13,14]. In contrast, CK peaks at 24-72 hrs after the initial insult, so may be low or normal in the first few hours.

The indications for dialysis for rhabdomyolysis are the same as for any patient with AKI

Indications for dialysis in rhabdomyolysis are the same as those for any AKI patient; use the mnemonic **AEIOU** [15]:

Acidemia – pH<7.1 despite medical management

Electrolyte abnormalities – hyperkalemia refractory to medical management

Ingestion – nephrotoxic drug ingestion amenable to dialysis

Overload – volume overload resulting in respiratory failure

Uremia with bleeding, pericarditis or encephalopathy

Safe discharge considerations for patients with rhabdomyolysis

Patients are likely safe to be discharged home according to our experts if the underlying cause is identified and reversed, their CK is <1000 and down-trending, their creatinine has normalized and the patient is reliable to continue fluids orally at home.

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