

Acute Kidney Injury: Treatment Depends on the Cause

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Q I have a patient with a discharge diagnosis of community-acquired acute kidney injury. What does this mean? What do I do now?

Acute kidney injury (AKI) refers to an abrupt decrease in kidney function that is possibly reversible or in which harm to the kidney can be modified.^{1,2} AKI encompasses a broad spectrum of conditions affecting the kidney—including acute renal failure, since even “failure” can sometimes be reversed.¹ Criteria for AKI and its severity can be found in the Table.¹

AKI can be either community-acquired (CA-AKI) or hospital-acquired (HA-AKI).^{1,2} In the United States, CA-AKI occurs less frequently than HA-AKI, although cases are likely underreported.¹ Evaluation and management are similar for both.

The etiology of the AKI must be determined before treatment of the cause or precipitating factor can be attempted. Causes of AKI can be classified as *prerenal* (up to 70% of cases), *intrinsic*, or *postrenal*.¹

Most AKI cases have a prerenal origin.³ Prerenal AKI occurs when there is inadequate blood flow to the kidneys, leading to a rise in blood urea nitrogen (BUN) and serum creatinine (SCr) levels. Reduced blood flow can be caused by

- Diuretic dosing
- Polypharmacy (diuretics, angiotensin-converting enzyme inhibitors [ACEIs]/ angiotensin receptor blockers [ARBs], and/or NSAIDs are common culprits)
- Congestive heart failure exacerbation
- Volume depletion through vomiting or diarrhea
- Massive blood loss (trauma).³

Postrenal causes of AKI include any type of obstructive uropathy. Intrinsic causes involve any condition within the kidney, including interstitial nephritis or acute tubular necrosis. Use of antibiotics (eg, high-dose penicillin or vancomycin) is included in this category.

Obtaining an accurate medical history and exam-

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TABLE

Defining Acute Kidney Injury

Criteria: Patients must have one of the following

- Increase in SCr \geq 0.3 mg/dL within 48 h
- Increase in SCr \geq 1.5 \times baseline that is known or presumed to have occurred within the past 7 d
- Urine volume $<$ 0.5 mL/kg/h for 6 h

Severity

Stage 1 1.5-1.9 \times baseline SCr or \geq 0.3-mg/dL increase in baseline SCr

Stage 2 2.0-2.9 \times baseline SCr

Stage 3 3.0 \times baseline SCr or increase in SCr to \geq 4.0 or renal replacement therapy (eg, dialysis)

Abbreviation: SCr, serum creatinine.

Source: KDIGO. *Kidney Int.* 2012.¹

ining the patient’s fluid status are critical. Although numerous novel biomarkers have been investigated for detection of AKI, none are yet in wide use. The primary assessment measures remain a serum panel to evaluate SCr and BUN levels; an electrolyte panel to assess for abnormalities; a complete blood count to assess for anemia caused by a less likely source; urinalysis; and imaging to assess for abnormalities or structural changes.

Urinalysis. Urine often holds the key to diagnosis of AKI. Notably in a prerenal injury, its specific gravity will be elevated, but the rest of the urine will likely be bland.³

Urinalysis is helpful for ruling out intrinsic causes of AKI. Patients with intrarenal AKI will have abnormal urine sediment; for example, red blood cell casts are found in glomerulonephritis; granular casts in cases of acute tubular necrosis; and white blood cell casts and eosinophils in acute interstitial nephritis.⁴

Imaging. The most commonly used imaging for AKI is retroperitoneal ultrasonography of the kidneys, ureters, and bladder, which provides information on the size and shape of the kidneys and can detect

stones or masses. It also detects the presence or absence of hydronephrosis, which can occur in postrenal injuries.

Currently, no definitive therapy or pharmacologic agent is approved for AKI; treatment focuses on reversing the cause of the injury. In the immediate aftermath of AKI, it is important to avoid potentially nephrotoxic medications, including NSAIDs. Minimize the use of diuretics and avoid ACEIs and ARB therapy; these can be reintroduced after lab results confirm that the AKI has resolved with a stabilized SCr.

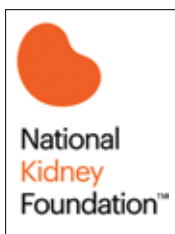
Practice guidelines recommend prompt follow-up at 3 months in most cases of AKI.¹ Providers should obtain a metabolic panel and perform a urinalysis to evaluate for chronic kidney disease (CKD), because almost one-third of patients with an AKI episode are

newly classified with CKD in the following year.⁵ Earlier follow-up (< 3 months) is warranted if the patient has a significant comorbidity, such as congestive heart failure.^{1,2}—**CS**

CR

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