Acute Kidney Injury (AKI)

AKI is a sudden decline in kidney function, leading to waste accumulation, fluid/ electrolyte
imbalances, and systemic effects.

Definition and Epidemiology

- **Definition:** AKI is defined by KDIGO criteria: ↑ serum creatinine (SCr) by ≥0.3 mg/dL within 48h, ↑ SCr by ≥1.5x baseline within 7 days, or urine output <0.5 mL/kg/h for 6h.
- **Prevalence:** ~10-15% of hospitalized patients; ~50% in ICU.
- **Risk Factors:** Age >65, CKD, diabetes, sepsis, hypovolemia, nephrotoxins (e.g., NSAIDs, contrast), substance use (cocaine, alcohol).

Pathophysiology

- **Mechanisms:** Reduced glomerular filtration rate (GFR) due to hypoperfusion (prerenal),
direct renal injury (intrinsic), or obstruction (postrenal).
- **Effects:** Azotemia, uremia, acidosis, hyperkalemia, fluid overload.
- **Key Pathway:** Tubular injury (e.g., acute tubular necrosis, ATN) from ischemia or toxins
leads to ↓ GFR.

Causes by Category

Category	Causes	Notes
Prerenal	-Hypovolemia: Dehydration, hemorrhage, GI losses. -↓ Cardiac Output: Heart failure (cardiorenal syndrome), sepsis (e.g., E. coli bacteremia). -Renal Hypoperfusion: NSAIDs, ACEi/ARBs, cocaine (vasoconstriction).	FeNa <1%, urine osmolality >500 mOsm/kg.
Intrinsic	-ATN: Ischemia (prolonged prerenal), nephrotoxins (contrast, aminoglycosides). -AIN: Allergic (beta-lactams, NSAIDs), infections (e.g., pyelonephritis). -Glomerulonephritis: RPGN (e.g., ANCA vasculitis), post-infectious (e.g., strep). -Rhabdomyolysis: Trauma, substance use (cocaine, alcohol).	FeNa >2%, muddy brown casts (ATN), myoglobinuria (rhabdomyolysis).

Category	Causes	Notes
Postrenal	-Obstruction: BPH, stones (e.g., from infection), malignancy (e.g., prostate cancer). -Neurogenic bladder: Spinal injury.	Hydronephrosis on ultrasound, urine output variable.
Rare	-Hemolytic Uremic Syndrome (HUS): E. coli 0157:H7 (infectious). -Tumor Lysis Syndrome: Chemotherapy (uric acid nephropathy).	Schistocytes (HUS), ↑ uric acid (TLS).

AKI Staging (KDIGO Criteria)

Stage	Serum Creatinine (SCr)	Urine Output
1	↑ SCr by 0.3 mg/dL in 48h or 1.5-1.9x baseline	<0.5 mL/kg/h for 6-12h
2	SCr 2.0-2.9x baseline	<0.5 mL/kg/h for ≥12h
3	SCr ≥3x baseline or ≥4 mg/dL or RRT initiation	<0.5 mL/kg/h for ≥24h or anuria ≥12h

Clinical Presentation

- **Symptoms:** Fatigue, nausea, decreased urine output, confusion (uremia), dyspnea (fluid overload).
- **Exam:**
  - **Prerenal:** Dry mucous membranes, tachycardia, JVD/edema (cardiorenal).
  - **Intrinsic:** Rash (AIN), flank pain (rhabdomyolysis), edema (glomerulonephritis).
  - **Postrenal:** Suprapubic pain, palpable bladder.
  - **Substance Use:** Cocaine (hypertension, rhabdomyolysis), alcohol (dehydration).
- **Red Flags:** SCr >4 mg/dL, K+ >6.5 mEq/L, anuria, pulmonary edema.

Diagnostic Workup

- **Labs:**
  - **Serum Creatinine (SCr):** ↑ SCr confirms AKI; trend for staging.
  - Cystatin C detects AKI earlier, reflects GFR accurately, and is less influenced by non-renal factors than creatinine.
  - BUN/Cr Ratio: >20:1 (prerenal); <15:1 (intrinsic).
  - **Electrolytes:** Hyperkalemia (K+ >5.5 mEq/L), acidosis (HCO3- <22 mEq/L).
  - CPK (rhabdomyolysis), lactate (sepsis), urine culture (pyelonephritis), blood culture (sepsis).

- **Urinalysis:**
  - **Prerenal:** Bland, urine osmolality >500 mOsm/kg.
  - **Intrinsic:** Muddy brown casts (ATN), RBC casts (glomerulonephritis),eosinophils (AIN).
  - **Postrenal:** Hematuria (stones).
  - **FeNa:** <1% (prerenal), >2% (ATN).
- **Imaging:** Renal ultrasound (hydronephrosis), CT (stones).
- **Biopsy:** If glomerulonephritis suspected (e.g., ANCA vasculitis).
- **Key Tip:** FeNa unreliable if on diuretics; use FEurea (<35% prerenal).

Evaluation Flowsheet: AKI Workup

- **Step 1:** Confirm AKI (KDIGO): ↑ SCr ≥0.3 mg/dL in 48h or ≥1.5x baseline, or oliguria.
- **Step 2:** Assess volume status: Hypovolemic → Prerenal (FeNa <1%); JVD/edema →Cardiorenal.
- **Step 3:** Check for obstruction: Ultrasound → Postrenal → Relieve obstruction.
- **Step 4:** Urinalysis and labs:
  - Muddy brown casts → ATN.
  - RBC casts → Glomerulonephritis (check ANCA, strep titers).
  - CPK ↑ → Rhabdomyolysis (cocaine).
  - Urine culture → Pyelonephritis.
- **Step 5:** Treat complications (e.g., hyperkalemia, acidosis).

Treatment

- **General Principles:** Address underlying cause, avoid nephrotoxins (e.g., NSAIDs, contrast), monitor SCr, urine output, electrolytes daily.
- **Prerenal:**
  - IV fluids (NS 500-1000 mL boluses, titrate to euvolemia).
  - Stop ACEi/ARBs, NSAIDs; treat cause (e.g., antibiotics for sepsis).
  - **Cardiorenal AKI: Optimize cardiac output:** Afterload reduction (hydralazine, isordil), diuresis, inotropes if in shock (e.g.,dobutamine 2-5 mcg/kg/min) Avoid over-diuresis (worsens hypoperfusion).
- **Intrinsic:**
  - **ATN:** Supportive (fluids, stop nephrotoxins), dialysis if severe (see below).
  - **AIN:** Stop offending drug (e.g., beta-lactams), steroids (prednisone 1 mg/kg/day)
    if severe.

    - **Glomerulonephritis:** Immunosuppression (e.g., cyclophosphamide for ANCA
      vasculitis).
    - **Pyelonephritis:** Antibiotics (e.g., ceftriaxone 1 g IV q24h), fluids.
    - **Rhabdomyolysis:** Aggressive IV fluids (NS 200-300 mL/h, target urine output
      >200 mL/h), consider bicarbonate (if pH <7.1) – discuss with nephrology
- **Postrenal:** Relieve obstruction (foley catheter for BPH, nephrostomy for stones).
- **Complications:**
    - **Hyperkalemia:** Calcium gluconate (stabilize cardiac membrane) insulin/ dextrose (shift potassium into cells), Lokelma (get rid of potassium through GI tract), dialysis if refractory.
- **Acidosis:** Bicarbonate if pH <7, dialysis if refractory
- **Fluid Overload:** Diuretics (furosemide 40 mg IV), dialysis if refractory.
- **Substance Use:** Cocaine (stop use, fluids for rhabdomyolysis), alcohol (fluids, monitor for withdrawal).

Indications for Dialysis

- **Mnemonic:** AEIOU - Nephrology should be involved before this stage
    - **Acidosis:** Severe metabolic acidosis (pH <7.1) unresponsive to bicarbonate.
    - **Electrolytes:** Hyperkalemia (K+ >6.5 mEq/L or EKG changes: peaked T waves, QRS widening) refractory to medical therapy.
    - **intoxication:** Toxins cleared by dialysis (e.g., methanol, ethylene glycol).
    - **Overload:** Fluid overload causing pulmonary edema, unresponsive to diuretics.
    - **Uremia:** Uremic symptoms (encephalopathy, pericarditis, bleeding) or BUN >100
      mg/dL.
- **Timing:** Urgent if life-threatening (e.g., K+ >6.5 with EKG changes); otherwise, assessdaily for need.
- **Key Tip:** Consult nephrology early; CRRT in hemodynamically unstable patients. Will
  need HD catheter placed.

Examples

- **Case 1:** Prerenal AKI (Dehydration)
- **Presentation:** 70 y/o M, vomiting/diarrhea, SCr 2.1 (baseline 1.0), FeNa <1%, urine osmolality 600 mOsm/kg.

- **Interpretation:** Prerenal AKI (↑ SCr 2.1x baseline, FeNa <1%). Stage 2 AKI (SCr 2.0-2.9x baseline).
- **Management:** IV NS (1 L bolus, then 100 mL/h), monitor SCr, stop NSAIDs.

- **Case 2:** Cardiorenal AKI (Heart Failure)
- Presentation: 65 y/o M, HF exacerbation, JVD, edema, SCr 2.5 (baseline 1.2), FeNa <1%, BUN/Cr 25:1.
- **Interpretation:** Prerenal AKI (cardiorenal syndrome, ↓ cardiac output), Stage 2AKI (SCr 2.0-2.9x baseline).
- **Management:** Optimize cardiac output (afterload reduction, diuresis, inotropes if in shock), monitor SCr.

- **Case 3:** Intrinsic AKI (Contrast-Induced ATN)
- **Presentation:** 60 y/o F, post-PCI, SCr 3.0 (baseline 1.2), muddy brown casts, FeNa >2%.
- **Interpretation:** Intrinsic AKI (ATN, contrast-induced), Stage 3 AKI (SCr ≥3xbaseline).
- **Management:** Supportive (IV NS 100 mL/h), stop nephrotoxins, monitor for dialysis need.

- **Case 4:** Postrenal AKI (BPH)
- **Presentation:** 65 y/o M, anuria, SCr 4.5, ultrasound shows hydronephrosis.
- **Interpretation:** Postrenal AKI (obstruction from BPH), Stage 3 AKI (SCr >4 mg/dL).
- **Management:** Foley catheter, urology consult, monitor SCr post-relief.

Complications

- **Short-Term:** Hyperkalemia, acidosis, uremic encephalopathy, pulmonary edema.
- **Long-Term:** Progression to CKD, dialysis dependence (~10-20% of severe AKI).

Prognosis

- **Mortality:** ~20-50% in ICU patients with AKI, higher with dialysis.
- **Recovery:** Prerenal often reversible; intrinsic/postrenal variable.

Key Pearls

- Use KDIGO criteria to diagnose AKI.
- FeNa <1% → Prerenal; >2% → Intrinsic (ATN).
- Treat hyperkalemia (K+ >6.5) urgently.
- Avoid nephrotoxins; monitor SCr daily.
- Screen for infections (sepsis, pyelonephritis) and substance use (cocaine).

References

- **UpToDate:** “Acute Kidney Injury” (2025).
- **KDIGO Guidelines:** AKI Management (2023).
- **NEJM:** “Acute Kidney Injury” (Bellomo, 2012)