

ACUTE KIDNEY INJURY (AKI)

1. DEFINITION

Acute Kidney Injury (AKI) is a sudden decline in kidney function over hours to days, leading to an accumulation of waste products (urea, creatinine), electrolyte imbalances, and fluid dysregulation. It is characterized by:

- An **increase in serum creatinine** (≥ 0.3 mg/dL within 48 hours or ≥ 1.5 times baseline within 7 days).
- A **decrease in urine output** (< 0.5 mL/kg/hr for ≥ 6 hours).

AKI can be **reversible** if diagnosed early and managed appropriately, but severe cases can progress to **chronic kidney disease (CKD)** or **end-stage renal disease (ESRD)**.

2. RISK FACTORS

AKI is more likely in patients with:

Patient-Related Risk Factors

- Pre-existing kidney disease (CKD)
- Diabetes mellitus
- Hypertension
- Elderly age
- Heart failure
- Liver disease (cirrhosis, hepatorenal syndrome)
- Dehydration or hypovolemia
- Sepsis or severe infections
- Autoimmune diseases (e.g., lupus, vasculitis)

Medication & Hospital-Related Risk Factors

- Nephrotoxic drugs (NSAIDs, aminoglycosides, contrast agents, ACE inhibitors, ARBs)
 - Major surgery (especially cardiac surgery)
 - Prolonged hypotension (shock, sepsis, trauma)
 - Exposure to toxins (heavy metals, myoglobin in rhabdomyolysis)
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3. CAUSES

AKI is classified into three major categories:

A. Pre-renal (↓ Renal Perfusion, Most Common Cause - 60-70%)

- Hypovolemia (dehydration, hemorrhage, vomiting, diarrhea)
- Low cardiac output (heart failure, myocardial infarction)
- Systemic vasodilation (sepsis, anaphylaxis)
- Renal artery stenosis

B. Intrinsic Renal (Direct Kidney Damage - 25-40%)

- Acute tubular necrosis (ATN) (ischemia, toxins, contrast agents)
- Glomerulonephritis (autoimmune diseases, infections)
- Acute interstitial nephritis (AIN) (allergic reaction to drugs like NSAIDs, antibiotics)
- Vascular causes (thrombotic microangiopathy, malignant hypertension)

C. Post-renal (Obstruction of Urine Flow - 5-10%)

- Kidney stones (nephrolithiasis)
- Prostate enlargement (BPH, prostate cancer)
- Ureteric obstruction (tumors, fibrosis, strictures)
- Neurogenic bladder dysfunction

4. SYMPTOMS AND SIGNS

General Symptoms

- Decreased urine output (oliguria <400 mL/day, anuria in severe cases)
- Fluid overload symptoms (swelling, pulmonary edema, hypertension)
- Fatigue, weakness, confusion (due to electrolyte imbalances, uremia)

Gastrointestinal Symptoms

- Nausea, vomiting, loss of appetite
- Metallic taste in the mouth (uremia-related)

Cardiovascular Symptoms

- Hypertension or hypotension
- Arrhythmias (due to hyperkalemia)

Neurological Symptoms

- Altered mental status, confusion, seizures (in severe uremia)

Signs on Physical Examination

- Edema (peripheral, pulmonary, or generalized anasarca)
- Crackles in lungs (pulmonary edema)
- Pericardial friction rub (uremic pericarditis, in severe cases)

5. COMPLICATIONS

1. **Electrolyte Imbalances**
 - Hyperkalemia → arrhythmias, cardiac arrest
 - Hyponatremia → confusion, seizures
 - Metabolic acidosis → Kussmaul breathing
2. **Volume Overload**
 - Pulmonary edema, respiratory distress
3. **Uremia**
 - Pericarditis, encephalopathy, bleeding tendency
4. **Infections**
 - Increased susceptibility to sepsis
5. **Chronic Kidney Disease (CKD)**
 - Some patients progress to long-term kidney dysfunction

6. INVESTIGATIONS AND FINDINGS

Laboratory Tests

- **Serum Creatinine & Blood Urea Nitrogen (BUN)** → ↑ (BUN:Creatinine ratio >20:1 in pre-renal AKI)
- **Electrolytes:**
 - ↑ K⁺ (hyperkalemia)
 - ↓ Na⁺ (hyponatremia)
 - ↓ HCO₃⁻ (metabolic acidosis)
- **Urinalysis:**
 - Muddy brown casts → **ATN**
 - RBC casts → **Glomerulonephritis**
 - WBC casts → **Interstitial nephritis**
- **Fractional Excretion of Sodium (FeNa):**
 - <1% in pre-renal AKI
 - >2% in intrinsic AKI

Imaging

- **Renal ultrasound** → to rule out obstruction (hydronephrosis suggests post-renal AKI)
- **CT scan** (if stones or masses are suspected)

Special Tests

- **Renal biopsy** (if glomerulonephritis or autoimmune disease is suspected)

7. MANAGEMENT

General Principles

- Identify and treat the underlying cause
- Monitor urine output and kidney function (daily creatinine, electrolytes, fluid balance)
- Avoid nephrotoxic drugs (e.g., NSAIDs, contrast, aminoglycosides)

1. Pre-Renal AKI (Restore Perfusion)

- IV fluids (normal saline or balanced crystalloids for hypovolemia)
- Blood transfusion if hemorrhagic shock
- Vasopressors (e.g., norepinephrine) in septic shock

2. Intrinsic AKI (Treat Underlying Cause)

- Stop nephrotoxic drugs
- Treat infections aggressively (sepsis management)
- Steroids or immunosuppressants for glomerulonephritis or autoimmune causes

3. Post-Renal AKI (Relieve Obstruction)

- Catheterization for bladder outlet obstruction (e.g., BPH)
- Ureteric stent or nephrostomy for ureteric obstruction
- Surgical removal of stones/tumors if necessary

Supportive Care

- **Electrolyte management:**
 - **Hyperkalemia** → calcium gluconate, insulin + dextrose, sodium bicarbonate, dialysis if severe
 - **Acidosis** → sodium bicarbonate if severe

- **Dialysis Indications ("AEIOU"):**
 - **Acidosis** (severe metabolic acidosis)
 - **Electrolyte** disturbances (refractory hyperkalemia)
 - **Intoxication** (drug toxicity, e.g., lithium, methanol)
 - **Overload** (severe fluid overload, pulmonary edema)
 - **Uremia** (encephalopathy, pericarditis)