# Acute Kidney Injury (AKI)

## Definition:

- **Abrupt** and often **reversible** decline in kidney function (hours to days).
- May be associated with **oliguria (<400 mL/day)**.
- **Results in:**
  1. Retention of nitrogenous wastes (urea, creatinine).
  2. Electrolyte and volume dysregulation.

## Epidemiology:

- **Increasing incidence, particularly in:**
  1. Hospitalized acutely ill patients (1 in 5 adults, 1 in 3 children).
  2. Patients undergoing major surgery.

## Diagnostic Criteria (KDIGO):

1. **Creatinine:** Increase ≥ 0.3 mg/dL within 48 hrs OR ≥ 1.5x baseline within 7 days.
2. **Urine Output:** < 0.5 mL/kg/hr for 6 hrs.

## Staging (KDIGO):

| Stage | Creatinine | Urine Output |
|---|---|---|
| 1 | ≥ 0.3 mg/dL or 1.5–1.9x baseline | < 0.5 mL/kg/hr for 6–12 hrs |
| 2 | 2.0–2.9x baseline | < 0.5 mL/kg/hr for ≥12 hrs |
| 3 | ≥ 3x baseline or ≥ 4.0 mg/dL or dialysis initiated | < 0.3 mL/kg/hr for ≥24 hrs or anuria ≥12 hrs |

## Etiology:

1. **Prerenal:**
   - Hypovolemia (Burns, diarrhea, vomiting, bleeding).
   - Shock (septic, cardiogenic).
   - Drugs (NSAIDs, ACEI/ARB, calcineurin inhibitors).
2. **Intrinsic:**
   - **Glomerular:** RPGN, GN.
   - **Tubulointerstitial:** AIN, ATN.
   - **ATN causes:**
     a) **Endogenous toxins:** Hemoglobin, myoglobin, uric acid, Bence-Jones proteins.
     b) **Exogenous toxins:** Aminoglycosides, Amphotericin B, Cisplatin, contrast agents.
   - **Vascular:** Vasculitis, malignant HTN, TTP/HUS, atheroemboli.

③ **POSTRENAL:**

- Bilateral obstruction or unilateral in solitary kidney.
- Causes: BPH, stones, tumors.

## PATHOPHYSIOLOGY PHASES:

| PHASE | FEATURES |
|---|---|
| INITIATION | Injury trigger (hours-days) |
| OLIGURIC | ↓ GFR, fluid overload, electrolyte imbalance (1–3 weeks) |
| DIURETIC | Polyuria with electrolyte loss (~2 weeks) |
| RECOVERY | Normalization (months) |

## CLINICAL PICTURE:

① **Oliguric Phase:** Edema, hyperkalemia, hyponatremia, acidosis.
② **Diuretic Phase:** Dehydration, hypokalemia, hyponatremia.
③ **Other:** Uremic symptoms, volume overload, pericarditis.

## DIAGNOSIS:

### ① AKI VS CKD:

| AKI | CKD |
|---|---|
| Short history, normal-sized kidneys, normal Hb. | Long duration, small kidneys, anemia, osteodystrophy. |

### ② URINALYSIS:

- **Prerenal:** Bland, hyaline casts.
- **ATN:** Muddy brown casts, tubular cells.
- **AIN:** WBC casts without bacteria.
- **GN:** RBC casts, dysmorphic RBCs.

### ③ KEY LABS:

| PARAMETER | PRERENAL | ATN |
|---|---|---|
| **Urine Osmolality (Uosm)** | > 500 mOsm/kg | < 350 mOsm/kg |
| **Urine Sodium (UNa)** | < 20 mEq/L | > 40 mEq/L |
| **FENa %** | < 1% | > 2% |

### ④ IMAGING:

- **Ultrasound:** Normal (prerenal), enlarged (intrinsic), hydronephrosis (postrenal).

### ⑤ RENAL BIOPSY INDICATIONS:

- Persistent AKI, systemic disease, nephritic/nephrotic features, unexplained etiology.

## Management:

### ① General Principles:

- ✧ Identify & treat reversible cause.
- ✧ Avoid nephrotoxins.
- ✧ Monitor fluid/electrolytes/acid-base.

### ② Fluids:

- ✧ **Hypovolemia:** IV fluids.
- ✧ **Hypervolemia:** Diuretics, fluid restriction.

### ③ Electrolytes:

- ✧ **Hyperkalemia:** Diet, medications, dialysis if severe.
- ✧ **Hyponatremia:** Water restriction.
- ✧ **Hyperphosphatemia:** Phosphate binders.
- ✧ **Hypocalcemia:** Treat if symptomatic.

### ④ Acid-Base:

- ✧ Metabolic acidosis: Sodium bicarbonate (avoid if hypervolemic).

### ⑤ Nutrition:

| Patient Category | Protein Needs (g/kg/day) |
|---|---|
| Non-catabolic (No dialysis) | 0.8–1.0 |
| Catabolic (IHD) | 1.0–1.5 |
| Catabolic (CRRT) | up to 1.7 |

### ⑥ Dialysis Indications:

- ✧ Refractory fluid overload.
- ✧ Severe hyperkalemia.
- ✧ Metabolic acidosis (pH < 7.1).
- ✧ Uremic complications (pericarditis, encephalopathy).
- ✧ Certain poisonings (ethylene glycol).