Acute Kidney Injury

Formerly known as acute renal failure, acute kidney injury (AKI) is a reversible rapid reduction in glomerular filtration rate (GFR) or kidney function, resulting in an increase in serum blood urea nitrogen (BUN), creatinine, and metabolic waste products (Okusa & Rosner, 2023). If left untreated, AKI can lead to reduced urine output, fluid retention, volume overload, and ultimately irreversible loss of kidney cells and nephrons leading to chronic kidney disease.

Definition (KDIGO, 2012)

The Kidney Disease: Improving Global Outcomes (KDIGO) organization defines AKI as **any of the following**:

- Increase in serum creatinine (SCr) by greater than or equal to 0.3 mg/dL (25.6 µmol/L) within 48 hours
- Increase in SCr greater than or equal to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days
- Urine volume less than 0.5 mL/kg/hour for 6 hours

Classifications of Acute Kidney Injury (Lippincott Advisor, 2024)			
	Prerenal	**Intrarenal (Intrinsic)**	**Postrenal**
Pathophysiology	Decreased blood flow to kidneys (decreased renal perfusion)	Structural injury that causes vessel constriction within the kidney	Blockage along the urinary tract obstructing urine outflow from the kidney

Causes	• Absolute decrease in circulating volume (Banasik, 2022)
	  ○ vomiting, diarrhea
	  ○ hemorrhage
	  ○ burns
	  ○ dehydration
	• Relative decrease in circulating volume
	  ○ Systemic vasodilation and hypotension caused by sepsis, anaphylaxis, anesthesia, drug overdose
		• Tubular
		  ○ Ischemic: acute tubular necrosis, prolonged prerenal failure, transfusion reaction, Rhabdomyolosis
		  ○ Nephrotoxic: prolonged post renal failure, medications (NSAIDS, certain antibiotics, cytotoxic chemotherapeutics, heroin, amphetamines), heavy metals, snake and insect venom,
			• Renal calculi
			• Emboli
			• Prostate enlargement
			• Genetic anatomic narrowing
			• Intra-abdominal tumors
			• Urinary tract strictures
			• Kinked or obstructed indwelling urinary catheters

	○ Third spacing and edema ○ Decreased cardiac output	radiographic contrast media. • Glomerular ○ Acute glomerulonephritis • Interstitial ○ Allergic interstitial nephritis ○ Acute pyelonephritis • Vascular ○ Vasculitis ○ Emboli ○ Nephrosclerosis (from chronic hypertension, hypertensive urgencies and emergencies) • Coagulation defect • Leukemia, lymphoma	

Contrast-induced AKI (CI-AKI) (KDIGO, 2012; Rudnick & Davenport, 2024)

CI-AKI [which may also be referred to as contrast-induced nephropathy (CIN)] may occur in patients who receive iodinated radiocontrast for procedures. Patients should be screened for risk factors such as impaired renal function, advanced age, diabetes, hypertension, congestive heart failure, chronic kidney disease, volume depletion, hemodynamic instability, concurrent nephrotoxic medication use and use of large volume and/or high osmolality contrast agents.

For patients at high risk, the following prevention measures are recommended:

- Verify that contrast material is necessary.
- Consider alternative methods of imaging studies that do not require the use of contrast.
- Use low- or iso- osmolarcontrast medium through an intravenous (IV) route at the lowest dose possible.
- IV fluids (isotonic sodium chloride or sodium bicarbonate) are generally initiated in high-risk patient unless patient is hypervolemic or on hemodialysis despite the lack of evidence to support this strategy as being of benefit (with the exception of patients undergoing coronary angiography).
- Withhold potentially nephrotoxic, non-critical medications for a minimum of 38 hours prior to contrast administration.

For patients not at risk for CI-AKI, these prevention measures are not recommended with the exception of verifying the need for contrast material.

Signs and Symptoms

AKI may not produce signs or symptoms until other organs are affected (Dihn, 2020). Signs and symptoms depend on the etiology and/or complications of AK.

Potential Signs and Symptoms of AKI (Lippincott Advisor, 2024)		
Shortness of breath	Tachycardia	Arrhythmias
Nausea	Hypertension	Muscle cramps
Vomiting	Weakness	Seizures
Edema (abdomen, extremities)	Fatigue	Oliguria or nonoliguria

Diagnosis (Lippincott Advisor, 2024)

Laboratory Tests

- Electrolytes: sodium, potassium, calcium, chloride, phosphorus, bicarbonate
- Blood glucose
- Albumin
- Blood urea nitrogen (BUN) and creatinine
- pH to detect degree of acid-base imbalance
- Urinalysis
- Complete blood count

Imaging

- Ultrasound of kidneys and perirenal structures – detects kidney tissue damage, kidney stones, urinary tract obstruction, or other abnormalities
- Computed tomography (CT) scan
- Renal angiography – examines the blood vessels

Diagnostic Procedures

- Electrocardiography (ECG) to assess for arrhythmias related to electrolyte imbalance
- Kidney biopsy for AKI with no clear etiology; can test for malignancy, vasculitis, nephrotic syndrome and glomerular disease

Stages of Acute Kidney Injury

Stages of AKI (KDIGO, 2012; Palevsky, 2025)	
Stage	**Clinical Manifestations**
1	Any of the following: ▪ SCr increased 1.5 – 1.9 times baseline ▪ SCr increased greater than or equal to 0.3 mg/dL (26.5 µmol/L) ▪ Urine output reduced to less than 0.5 mL/kg/hour for 6 – 12 hours
2	Any of the following: ▪ SCr increased 2.0 – 2.9 times baseline ▪ Urine output reduced to less than 0.5 mL/kg/hour for 12 hours or more
3	Any of the following: ▪ SCr increased 3.0 times baseline

	▪ SCr increased greater than or equal to 4.0 mg/dL (353.6 µmol/L) ▪ Urine output reduced to less than 0.3 mL/kg/hour for 24 hours or more ▪ Anuria for 12 hours or more ▪ Initiation of renal replacement therapy ▪ In patients less than 18 years, decrease in estimated GFR to less than 35 mL/min per 1.73m^2

Complications (Okusa & Rosner, 2023; Mishra et al., 2022)

The following complications require immediate renal replacement therapy RRT/hemodialysis.

- Fluid overload resistant to medical therapy and causing cardiopulmonary compromise
- Severe Hyperkalemia
    - For any serum potassium greater than 6.5 mEq/L
    - Hyperkalemia associated with cardiac arrhythmias or muscle weakness
    - Hyperkalemia greater than 5.5 mEq/L associated with tissue breakdown or significant gastrointestinal bleeding
- Uremic complications uncontrolled with medical therapy such as pericarditis, seizure, or unexplained change in mental status
- Severe metabolic acidosis (pH less than 7.1) uncontrolled with medical therapy
- Acute poisoning with dialyzable poisons and toxins (e.g., metformin, methanol, ethylene glycol)

Treatment

The goal of therapy is to prevent life-threatening complications and limit further damage to the kidneys (Dihn, 2020). Treatment of AKI is mainly supportive, to preserve volume homeostasis and correct biochemical abnormalities.

- **Treat the underlying cause.**
    - Manage hypotension; administer vasopressor to improve renal perfusion and achieve hemodynamic targets
    - Correct volume depletion in patients with vomiting, diarrhea, hypotension, tachycardia, or oliguria
        - Avoid fluid therapy in patients with pulmonary edema or anuria.
        - Administer 1-3 liters crystalloids individualized to correct volume deficit, followed by maintenance isotonic fluids at 75 mL/hour for volume responsive patients.
    - Urology and/or interventional radiology consultation for patients with hydronephrosis due to urinary tract obstruction.
- **Prioritize glycemic control and nutritional support.**
    - Target plasma glucose between 110 and 149 mg/dL.
    - Restrict salt intake.
    - Low potassium, low phosphate diet.
    - Nutrional requirements will vary based on patient's underlying disease, severity of illness, and co-morbidities; unless higher or lower caloric intakes are indicated, nutritional goals should be approximately 25 to 30kcal/kg per day (Okusa & Rosner, 2023).
- **Administer medications.**
    - All medications should be reviewed, and dose adjusted based on GFR.

  - Diuretics are recommended *only* to manage volume overload (e.g., 80-100 mg IV furosemide) in patients who are not anuric; unless the patient requires fluid overload management, diuretics are not recommended to treat AKI.
  - Vasodilators do not show improvement for AKI patients; dopamine, fenoldopam, and/or natriuretic peptides are not recommended as treatment to improve kidney function.
  - Replenish electrolytes and treat acid-base imbalance.
  - The following medications can worsen AKI. Discuss discontinuing these agents with the healthcare provider.
    - Nonsteroidal anti-inflammatory drugs (NSAIDs)
    - Angiotensin-converting enzyme (ACE) inhibitors
    - Angiotensin receptor blockers (ARBs)
    - Nephrotoxins (aminoglycoside antibiotics, piperacillin-tazobactam, amphotericin B, tenofovir, nephrotoxic chemotherapy)
- **Institute renal replacement therapy (RRT) as ordered.**
  - RRT is indicated immediately for life-threatening changes in fluid, electrolyte, and acid-base balance.
  - For patients with volume overload who have anuria for more than 24 hours, who fail to respond to diuretics, or whose response to diuretics is not enough to avoid worsening hypervolemia, RRT is also indicated.
  - Types:
    - Hemodialysis (intermittent) is recommended for patients that can tolerate a rapid removal of toxins.
    - Continuous renal replacement therapy (CRRT) is recommended for hemodynamically unstable patients.
    - Peritoneal dialysis is rarely used for AKI.
  - The recommended dialysate buffer solution is bicarbonate to correct acidosis, reduce lactate levels and improve hemodynamic stability.
  - Anticoagulant therapy is recommended to prevent clotting of the filter.
    - For intermittent RRT with low risk for bleeding, use unfractionated or low-molecular weight heparin.
    - For CRRT with bleeding risk, regional citrate anticoagulation is recommended; if citrate is contraindicated, use heparin.
    - If heparin-induced thrombocytopenia (HIT) develops, discontinue all heparin and use a direct thrombin inhibitor, or Factor Xa.
- **Surgery**
  - Insertion of vascular access port as needed for dialysis.
    - Choice of access is uncuffed, non-tunneled catheter
    - Choice of vein in order of preference: right jugular vein, femoral vein, left jugular vein and subclavian vein
  - A nephrostomy tube or ureteral stent may be needed to relieve obstruction

Nursing Interventions

- Monitor vital signs including pulse oximetry.
- Maintain continuous cardiac monitoring; assess for rhythm changes that may signal hyperkalemia (bradycardia, peaked T waves).
- Perform daily weights.
- Assess for edema.

- Insert an indwelling bladder catheter to monitor urine output.
- Calculate intake and output each shift or more frequently in ICU.
- Assess lung sounds.
- Monitor level of consciousness and perform neurologic examination.
- Keep head of bed elevated, unless contraindicated.
- For patients with AKI due to prerenal factors such as dehydration, administer IV fluids as ordered.
- For patients with volume overload, ensure fluid restriction and/or administer diuretics as ordered.
- Encourage a low sodium, low potassium diet.

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