◆ NGN NCLEX · 2026 TEST PLAN · RENAL / GENITOURINARY SYSTEM ◆

Acute Kidney *Injury*

Pre-renal · Intra-renal · Post-renal — A High-Yield Clinical Judgment Study Card

PRIORITY: ABCS + FLUID/ELECTROLYTES | NCJMM INTEGRATED | STUDY SERIES · RENAL

01. Definition & Overview

WHAT IT IS

Acute Kidney Injury (AKI) is a *sudden* (hours to days), potentially reversible decline in kidney function marked by accumulation of nitrogenous waste (BUN, creatinine) and impaired fluid, electrolyte, and acid–base balance.

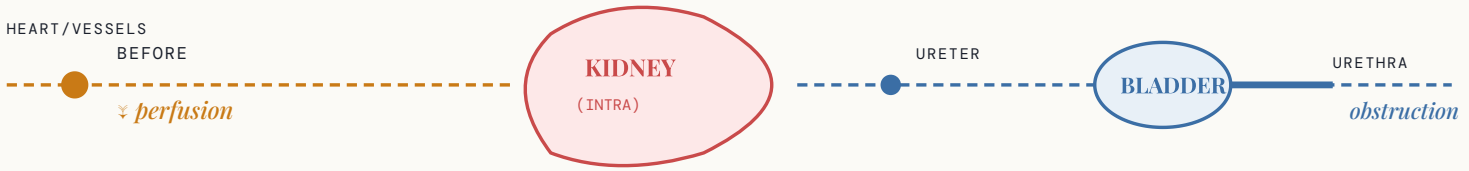
AKI is diagnosed by a **rise in serum creatinine ≥ 0.3 mg/dL in 48 hrs** OR a **urine output < 0.5 mL/kg/hr for ≥ 6 hrs**.

It is classified by **where the problem starts**: before the kidney (Pre), inside the kidney (Intra), or after the kidney (Post). Identifying the category drives treatment — the NCLEX loves this distinction.

≥ 0.3 MG/DL ↑ CREATININE / 48H	**< 0.5** ML/KG/HR URINE ≥ 6H
4 PHASES OF AKI	**3** ETIOLOGIC CATEGORIES

02. The Three Types of AKI

LOCATION, LOCATION, LOCATION



TYPE I

60%
of AKI cases

Pre-renal

"Before the kidney" — a perfusion problem

CORE PROBLEM

↓ Blood flow → kidneys are *starving* but structurally intact. Quickly reversible if perfusion restored; if not → ATN.

COMMON CAUSES

- → Hypovolemia (hemorrhage, dehydration, burns, D/V)
- → Shock (septic, cardiogenic, anaphylactic)
- → Heart failure / MI / low cardiac output
- → Renal artery stenosis / clamping
- → NSAIDs, ACE-I/ARBs (↓ glomerular pressure)

HALLMARK LABS

- → BUN:Creatinine ratio **> 20:1**
- → Urine Na **< 20 mEq/L** (kidney holding Na)
- → Urine specific gravity **> 1.020** (concentrated)
- → FENa **< 1%**

TYPE II

30%
of AKI cases

Intra-renal

"Inside the kidney" — tissue damage

CORE PROBLEM

Direct damage to glomeruli, tubules, or interstitium. **ATN** (acute tubular necrosis) is the #1 cause. Longer recovery; may not fully reverse.

COMMON CAUSES

- → **ATN** — prolonged ischemia or nephrotoxins
- → Nephrotoxins: aminoglycosides (gentamicin), vancomycin, contrast dye, amphotericin B, NSAIDs
- → Glomerulonephritis, lupus, pyelonephritis
- → Rhabdomyolysis (myoglobin-induced)
- → Hemolytic transfusion reaction

HALLMARK LABS

- → BUN:Creatinine ratio **≈ 10–15:1**
- → Urine Na **> 40 mEq/L** (tubules can't reabsorb)
- → Urine specific gravity **≈ 1.010** (fixed/isosthenuric)
- → Muddy-brown casts in urine 🚨

TYPE III

10%
of AKI cases

Post-renal

"After the kidney" — an obstruction

CORE PROBLEM

Urine outflow is blocked → backpressure → hydronephrosis → kidney damage. Most reversible *IF caught early*.

COMMON CAUSES

- → BPH / prostate cancer (most common in elderly men)
- → Kidney or ureteral stones (calculi)
- → Bladder, cervical, or pelvic tumors
- → Urethral strictures / blocked Foley catheter
- → Neurogenic bladder

HALLMARK LABS

- → BUN:Creatinine ratio variable (often elevated)
- → **Anuria** or alternating oliguria/polyuria
- → Bladder scan ↑ residual volume
- → Hydronephrosis on ultrasound (imaging = key)

03. Pathophysiology Pathways

STEP-BY-STEP

PATHOPHYSIOLOGY

PRE-RENAL → ↓ Blood volume / CO → ↓ Renal perfusion → ↓ GFR → RAAS activation (Na & H_2O retention) →

If unresolved → ATN

INTRA-RENAL

Nephrotoxin / ischemia → Tubular epithelial injury → Cellular debris/casts block tubules → ↓ GFR + back-leak of filtrate → Retention of urea, K^+, acid

POST-RENAL

Outflow obstruction → ↑ Pressure in collecting system → Hydronephrosis → Tubular compression & ischemia → If prolonged → permanent damage

04. The Four Phases of AKI

TIMELINE

Onset / Initiation — I

HOURS TO DAYS

- Kidney injury begins
- UO starts to decline
- Often asymptomatic
- Reversible if caught

Oliguric — II

1-3 WEEKS

- UO < 400 mL/day 🚨
- ↑ BUN, Creatinine, K^+, PO_4
- Metabolic acidosis
- Fluid overload, HTN
- **Highest mortality risk**

Diuretic — III

1-3 WEEKS

- UO 3–5 L/day
- Risk: dehydration
- Risk: hypokalemia, hyponatremia
- BUN/Cr slowly normalize
- **Monitor for hypovolemia**

Recovery — IV

UP TO 12 MONTHS

- GFR gradually returns
- Labs trend toward baseline
- May retain some deficit
- Risk of progression to CKD

05. Signs & Symptoms

CUE RECOGNITION

Early / Mild

- **Oliguria** — UO < 400 mL/day (or < 30 mL/hr)
- **Fatigue, malaise** — often first subtle cue
- Slight ↑ BUN and creatinine
- Mild peripheral edema (ankles, periorbital)

Late / Severe

- **Anuria** — UO < 100 mL/day 🚨
- **Hyperkalemia → peaked T-waves**, dysrhythmias 🚨
- **Pulmonary edema** — crackles, dyspnea, frothy pink sputum 🚨
- Kussmaul respirations (metabolic acidosis)

- Dry mucous membranes (if pre-renal)
- Orthostatic hypotension (pre-renal)
- Flank pain (post-renal from obstruction)

- JVD, ↑ BP, weight gain (fluid overload)
- Confusion, lethargy, seizures (uremic encephalopathy)
- Pruritus, uremic frost, metallic taste, N/V
- Pericardial friction rub (uremic pericarditis)

🚨 **TOP NCLEX RED FLAG — Hyperkalemia**

K^+ > 6.5 mEq/L with peaked T-waves, widened QRS, or bradycardia = *immediate* priority. This is the #1 cause of cardiac arrest in AKI. Act before confirming labs: stop K^+ sources, notify HCP, prepare calcium gluconate (cardiac protection), insulin + D50, sodium bicarb, or kayexalate.

06. Lab Values at a Glance

WHAT TRENDS MATTER

LAB	NORMAL	IN AKI	DIRECTION
BUN	10-20 mg/dL	> 20 mg/dL	↑↑
Creatinine	0.6-1.2 mg/dL	Rises ≥ 0.3/48h	↑↑
Potassium (K^+)	3.5-5.0 mEq/L	> 5.0 🚨	↑↑
Phosphorus	3.0-4.5 mg/dL	> 4.5 mg/dL	↑↑
Calcium	9.0-10.5 mg/dL	< 9.0 mg/dL	↓↓
Sodium	135-145 mEq/L	↓ (dilutional)	↓↓
pH / HCO_3^-	7.35-7.45 / 22-26	Acidosis	↓↓
Hgb / Hct	12-18 / 36-52	Anemia (↓ EPO)	↓↓

GFR	> 90 mL/min	< 60 mL/min	↓

07. Priority Nursing Interventions

ABCS FIRST

A·B·C

Airway → Breathing → Circulation

Pulmonary edema compromises breathing; hyperkalemia compromises circulation. Address these BEFORE routine labs or I&O documentation.

TYPE	PRIORITY INTERVENTIONS
Pre-renal	**Restore perfusion.** Administer IV fluids (isotonic — NS or LR) as ordered. Treat underlying cause (blood products for hemorrhage, vasopressors for shock). Monitor BP, HR, CVP, and hourly urine output via Foley. Hold nephrotoxic meds (NSAIDs, ACE-I/ARBs). Strict I&O and daily weights.
Intra-renal	**Remove the insult and support the kidney.** Stop all nephrotoxins immediately. For contrast-induced: hydrate before/after dye. For rhabdo: aggressive IV fluids + monitor CK. Adjust all meds for renal function. Prepare for possible dialysis (especially CRRT if unstable). Protect from infection.
Post-renal	**Relieve the obstruction — fast.** Insert/irrigate Foley catheter (check for blockage first), assist with cystoscopy/stenting/nephrostomy tube, or prepare for surgery. Bladder scan to confirm retention. Monitor for *post-obstructive diuresis* — huge UO after relief → replace fluids & electrolytes.
All AKI	**Universal priorities:** Daily weights (same scale, same time, same clothes — best indicator of fluid status). Strict I&O every hour in acute phase. Monitor K^+ and cardiac rhythm continuously. Fluid restriction in oliguric phase (typically 600 mL + prev 24-hr UO). Low-K^+, low-Na^+, low-phosphorus, moderate-protein renal diet. Cluster care to promote rest. Skin care for uremic pruritus. Monitor for signs of dialysis need: **AEIOU** — *A*cidosis, *E*lectrolytes, *I*ntoxication, *O*verload, *U*remia.

08. Complications & Management by Type

WHAT CAN GO WRONG

TYPE	MAJOR COMPLICATION	MANAGEMENT
Pre-renal	**Progression to ATN** if perfusion not restored. Shock, multi-organ failure, death.	Rapid volume resuscitation; vasopressors if MAP < 65; treat sepsis/hemorrhage; hold nephrotoxins; continuous hemodynamic monitoring.
Pre-renal	**Electrolyte shifts** (hyponatremia from RAAS water retention; early hyperkalemia if low UO).	Serial electrolytes q4–6h; cautious Na^+ correction; treat hyperkalemia per K^+ algorithm; cardiac monitoring.
Intra-renal	**Hyperkalemia & dysrhythmias** 🫀 — #1 cause of death in AKI.	Calcium gluconate (cardiac membrane protection — doesn't lower K^+); insulin + D50 (shifts K^+ intracellularly); sodium bicarb if acidotic; kayexalate PO/PR; **dialysis** if refractory.
Intra-renal	**Pulmonary edema / fluid overload** — crackles, dyspnea, frothy sputum.	High Fowler's position, O_2, IV loop diuretics (furosemide) if still producing urine, fluid restriction, daily weights; dialysis/ultrafiltration if unresponsive.
Intra-renal	**Metabolic acidosis** — Kussmaul respirations, confusion.	Sodium bicarbonate IV as ordered; monitor ABGs; treat underlying cause; dialysis if pH < 7.1.
Intra-renal	**Uremia** — encephalopathy, pericarditis, bleeding (platelet dysfunction), pruritus, anemia.	Dialysis (definitive); phosphate binders with meals; epoetin alfa for anemia; skin care; seizure precautions; monitor pericardial rub/effusion; bleeding precautions.
Post-renal	**Hydronephrosis & permanent damage** if obstruction not relieved quickly.	Emergent Foley / nephrostomy / stent / stone removal / TURP; renal ultrasound to confirm; serial creatinine post-relief.
Post-renal	**Post-obstructive diuresis** — massive UO after relief → hypovolemia, hypokalemia, hyponatremia.	IV fluid replacement (often mL-for-mL with UO); aggressive electrolyte repletion (K^+, Na^+, Mg^{2+}); hourly VS and I&O; daily weights; hold diuretics.
Post-renal	**UTI / urosepsis** from stagnant urine.	Urine culture before antibiotics; broad-spectrum then targeted abx; sepsis bundle if unstable; maintain catheter patency with sterile technique.

09. Pharmacology

DRUGS TO GIVE & AVOID

Furosemide (Lasix)

LOOP DIURETIC

MECHANISM:
Blocks $Na^+/K^+/2Cl^-$ reabsorption in Loop of Henle → diuresis.

USE:
Fluid overload in non-oliguric AKI (patient must still produce some urine).

SIDE EFFECTS:
Hypokalemia, hypotension, ototoxicity (infuse slowly, no faster than 20 mg/min).

WATCH:
K^+ levels; daily weights; orthostatic BP.

Calcium Gluconate

CARDIAC PROTECTION

MECHANISM:
Stabilizes myocardial membrane against hyperkalemia. *Does NOT lower K^+.*

USE:
Hyperkalemia with ECG changes (peaked T-waves, widened QRS).

SIDE EFFECTS:
Bradycardia if pushed fast; extravasation = tissue necrosis.

WATCH:
Continuous cardiac monitoring; give over 2–5 min IV.

Insulin (Regular) + D50

K^+ SHIFTER

MECHANISM:
Drives K^+ into cells. Dextrose prevents insulin-induced hypoglycemia.

USE:
Acute hyperkalemia (temporary effect — 4–6 hrs).

SIDE EFFECTS:
Hypoglycemia, rebound hyperkalemia.

WATCH:
Blood glucose q1h × 6; repeat K^+ level.

Sodium Polystyrene (Kayexalate)

K^+ BINDER

MECHANISM:
Exchanges Na^+ for K^+ in the gut; K^+ excreted in stool.

USE:
Non-emergent hyperkalemia.

SIDE EFFECTS:
Constipation, bowel necrosis (esp. post-op), Na^+ overload.

WATCH:
Bowel sounds; contraindicated in ileus or bowel obstruction.

Sevelamer / Calcium Carbonate

PHOSPHATE BINDER

MECHANISM:
Binds dietary phosphorus in the GI tract → excreted in stool.

USE:
Hyperphosphatemia in AKI/CKD.

SIDE EFFECTS:
Constipation, hypercalcemia (with calcium-based).

Epoetin Alfa (Epogen / Procrit)

ERYTHROPOIETIN

MECHANISM:
Stimulates RBC production — replaces EPO the damaged kidneys can't make.

USE:
Anemia of AKI/CKD.

SIDE EFFECTS:

WATCH:
GIVE WITH MEALS.
Monitor Ca^{2+} & PO_4.

Hypertension, clotting, seizures.

WATCH:
BP before/after doses; check Hgb — do not exceed 11 g/dL.

AVOID — Nephrotoxic Agents

⚠ WITHHOLD

NSAIDS
(ibuprofen, ketorolac) — ↓ renal blood flow.

AMINOGLYCOSIDES
(gentamicin, tobramycin) — tubular toxicity.

VANCOMYCIN
— monitor trough levels.

IV CONTRAST
— hydrate before/after; consider N-acetylcysteine.

ACE-I / ARBS
in acute setting — ↓ glomerular pressure.

Dopamine (low-dose / "renal dose")

VASOPRESSOR

MECHANISM:
At low doses, historically thought to ↑ renal perfusion. *Evidence now shows limited benefit.*

USE:
Pre-renal AKI with hypotension (as vasopressor, not for "kidney protection").

SIDE EFFECTS:
Tachycardia, dysrhythmias, ischemia with extravasation.

WATCH:
Central line preferred; titrate to MAP.

10. NCLEX Test Traps

WHERE STUDENTS MISS POINTS

Trap #1 — "Pushing fluids" is not universal

IV fluids **help pre-renal AKI** but can cause **pulmonary edema** in the oliguric phase of intra-renal AKI. Always identify the *type* before picking fluid orders.

Trap #2 — Calcium gluconate does NOT lower K^+

It **protects the heart** from hyperkalemia's dysrhythmias. To actually *lower* K^+ you need insulin/D50, kayexalate, bicarb, or dialysis.

Trap #3 — Daily weight beats I&O

The **most reliable** indicator of fluid status in AKI is **daily weight** (same scale, same time, same clothes). 1 kg gained ≈ 1 L retained.

Trap #4 — Oliguric ≠ Anuric

Oliguria = **< 400 mL/day**. Anuria = **< 100 mL/day**. Sudden anuria in the older adult male → think **post-renal** (BPH/obstruction) first.

Trap #5 — Don't assume "low protein for everyone"

AKI diet is **moderate protein** (1.0–1.5 g/kg), not zero. Severe restriction causes muscle breakdown → *more* nitrogenous waste.

Trap #6 — Hold the ACE, not the β-blocker

ACE-I/ARBs ↓ glomerular filtration pressure — **held in acute AKI**. β-blockers are generally continued unless bradycardic/hypotensive.

Trap #7 — Peaked T-waves = ACT

Don't wait for the next labs. Peaked T-waves + known renal disease = treat empirically for hyperkalemia. **Recognize → intervene → reassess.**

Trap #8 — Post-obstructive diuresis is a trap

After relieving an obstruction, UO can hit **5–10 L/day**. Patient can become hypovolemic fast. *Replace fluids and electrolytes — don't celebrate yet.*

11. NCJMM Clinical Judgment Framework

THINK LIKE A NURSE

NCJMM CLINICAL JUDGMENT

R — Recognize Cues
Oliguria, ↑ BUN/Cr, peaked T-waves, edema, recent contrast or nephrotoxic med, hypotension, BPH history.

→

A — Analyze
Classify: Pre (perfusion)? Intra (toxin/ATN)? Post (obstruction)? Correlate labs, UO, med history, and hemodynamics.

→

P — Prioritize & Act
Treat the cause (fluids, stop toxin, relieve obstruction); address hyperkalemia; restrict fluids if overloaded; prep for dialysis if AEIOU.

→

E — Evaluate
UO ≥ 30 mL/hr, K^+ normalizing, BUN/Cr trending down, weight stable, no crackles, rhythm stable, mentation clear.

12. Patient Education

TEACH-BACK READY

Medications & Nephrotoxins

- ✓ Avoid OTC NSAIDs (ibuprofen, naproxen, ketorolac) — use acetaminophen
- ✓ Tell every HCP and dentist about your kidney history before any prescription
- ✓ Check with pharmacist before herbal supplements
- ✓ Stay hydrated before and after any scan with IV contrast
- ✓ Take phosphate binders *with* meals

Diet & Fluid Limits

- ✓ Low sodium (< 2 g/day) — read food labels
- ✓ Low potassium — avoid bananas, oranges, potatoes, tomatoes, salt substitutes
- ✓ Low phosphorus — avoid dairy, cola, nuts, processed foods
- ✓ Moderate protein as prescribed — don't self-restrict
- ✓ Fluid restriction as ordered — track intake including ice, soup, popsicles

Daily Self-Monitoring

When to Call 911 / the HCP

- ✓ Weigh daily — same scale, same time, same clothing
- ✓ Report weight gain > 2–3 lb in 24 hrs or 5 lb in a week
- ✓ Track urine output if instructed
- ✓ Check BP daily; log readings
- ✓ Report swelling of ankles, face, or hands

- ✓ Shortness of breath, pink frothy sputum, or new chest pain
- ✓ Muscle weakness, palpitations, or irregular heartbeat
- ✓ Sudden drop in urine output or complete inability to void
- ✓ Confusion, extreme fatigue, or seizure activity
- ✓ Fever, chills, or flank pain (possible infection/obstruction)

13. Memory Boosters

LOCK IT IN

DIALYSIS CRITERIA

A · E · I · O · U

When to initiate dialysis

- **A** **Acidosis** — pH < 7.1 refractory to bicarb
- **E** **Electrolytes** — K^+ > 6.5 or refractory to treatment
- **I** **Intoxication** — dialyzable toxin (lithium, ASA, methanol)
- **O** **Overload** — pulmonary edema unresponsive to diuretics
- **U** **Uremia** — encephalopathy, pericarditis, bleeding

THREE TYPES

P · I · P

Pre · Intra · Post — location tells the story

- **P** **Pre** = Perfusion problem (before) → *give fluids*
- **I** **Intra** = Inside injury (ATN) → *stop the toxin*
- **P** **Post** = Plumbing blocked (after) → *relieve obstruction*

NEPHROTOXINS

"A-NSAID"

Common kidney-killers to avoid/monitor

- **A** **A**minoglycosides (gentamicin, tobramycin)
- **N** **N**SAIDs (ibuprofen, ketorolac)
- **S** **S**ulfas & contrast agents
- **A** **A**CE inhibitors / ARBs in acute setting

HYPERKALEMIA TX ORDER

"C · BIG · K · Drop"

Sequence for treating a peaked T-wave

- **C** **Calcium gluconate** — protect the heart *first*
- **B** **Bicarb** — if acidotic
- **I** **Insulin + D50** — shift K^+ into cells
- **G** **Glucose check** q1h × 6

I IV contrast dye

D Diuretics (overuse → volume depletion)

K **Kayexalate** — remove K^+ via gut

D **Dialysis** — if refractory

PHASES

"O · D · R"

After Onset, the body walks through these

O **Oliguric** — UO ↓, K^+ ↑, fluid overload, HIGHEST risk

D **Diuretic** — UO 3–5 L/day, watch for dehydration & ↓ K^+

R **Recovery** — slow return toward baseline over months

URINE CLUES

"Muddy = Intra"

Spot the type from the urinalysis

- **Concentrated, low Na^+** → Pre-renal (kidney still trying to save water)
- **Muddy-brown casts, high Na^+, fixed SG** → Intra-renal (ATN)
- **Hematuria or crystals + hydronephrosis** → Post-renal (stones/obstruction)