AKI BMP Pattern Recognition Guide

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Overview

Acute kidney injury (AKI) is identified by rapid changes in serum creatinine (SCr) and urine output, but fundamental understanding begins with pattern recognition on the basic metabolic panel (BMP) [1]. Beyond simply noting an elevated creatinine, the experienced clinician extracts critical diagnostic clues from the entire electrolyte profile, osmolar gap, and acid-base status. This guide integrates serologic patterns with clinical context to localize injury and accelerate diagnosis.

Key Clinical Pearl

BMP patterns narrow the differential diagnosis of AKI before ever sending a urine sample. The pattern of electrolyte derangement often reveals the mechanism (prerenal, intrinsic, postrenal) and even the specific etiology (rhabdomyolysis, contrast nephropathy, cardiorenal syndrome).

Part 1: Three-Compartment AKI Framework

Prerenal Azotemia (75–80% of AKI cases)

Definition: Reduced glomerular filtration driven by decreased renal perfusion. Normal tubular function preserved; kidneys avidly reabsorb sodium and water [2].

BMP Pattern: - **Creatinine elevation:** Mild to moderate (usually <3.0 mg/dL unless severe shock) - **BUN elevation:** Disproportionately high relative to creatinine - **BUN:Cr ratio:** >20:1 (classic finder; normal 10:1) - **Sodium:** Normal to low (hyponatremia from volume expansion + water retention) - **Potassium:** Usually normal early; rises only if oliguria develops - **Chloride:** Normal to low - **Bicarbonate:** Normal or slightly elevated (contraction alkalosis possible)

Associated Findings: - Fractional excretion of sodium (FeNa) <1% (sodium-avid state) - Fractional excretion of urea (FeUrea) <35% (preserved tubular reabsorption) [3] - Urine osmolality >500 mOsm/kg (concentrated urine) - Urine sodium <20 mEq/L (avid reabsorption)

Clinical Context: Volume depletion (diarrhea, vomiting, hemorrhage), hypotension, heart failure, sepsis, liver disease.

Intrinsic Renal Disease (10–15% of AKI cases)

Definition: Structural damage to glomeruli, tubules, interstitium, or vasculature. Loss of normal tubular reabsorptive capacity.

BMP Pattern: - **Creatinine elevation:** Rapid rise; may be >3.0 mg/dL - **BUN elevation:** Parallel to creatinine (BUN:Cr ratio ~10:1) - **Sodium:** Often low (tubular dysfunction prevents reabsorption) - **Potassium:** Often elevated (loss of tubular secretion + cell death) - **Chloride:** Low to normal - **Bicarbonate:** Reduced (metabolic acidosis from uremia) - **Anion gap:** Elevated if significant metabolic acidosis

Associated Findings: - FeNa >2% (tubular damage; loose criterion, less specific) - Urine osmolality <350 mOsm/kg (dilute urine) - Urine sodium >40 mEq/L (loss of avidity) - Hematuria and/or proteinuria on urinalysis (suggests glomerular involvement) - Red cell casts (glomerulonephritis)

Clinical Context: Sepsis, rhabdomyolysis, nephrotoxins (aminoglycosides, NSAIDs, ACE inhibitors in acute setting), glomerulonephritis, vasculitis, contrast-induced nephropathy (CIN).

Postrenal Obstruction (5–10% of AKI cases)

Definition: Obstruction downstream of glomerular filtration (renal artery, ureter, bladder, urethra). Pressure builds; tubular reabsorption initially preserved, then lost if obstruction incomplete or partial [4].

BMP Pattern: - **Creatinine elevation:** Variable; depends on completeness and duration of obstruction - **BUN elevation:** Variable; may lag creatinine if obstruction acute - **Potassium:** Often high (impaired secretion, oliguria, possible acute cellular necrosis) - **Sodium:** Variable - **Bicarbonate:** Reduced if prolonged (metabolic acidosis) - **Phosphorus:** Often elevated (impaired excretion)

Associated Findings: - Bladder catheterization shows large post-void residual (if bladder outlet obstruction) - Renal ultrasound shows hydronephrosis (if ureteral obstruction) - History of hematuria with stone, prostate enlargement, or recent procedure

Clinical Context: Nephrolithiasis, prostate cancer, retroperitoneal malignancy, urinary catheter obstruction, neurogenic bladder.

Part 2: Specialized BMP Patterns—Key Etiologies

Rhabdomyolysis Pattern

Muscle injury releases myoglobin and intracellular contents into serum. AKI occurs from pigment precipitation in tubules (especially in acid urine), hypovolemia, and direct tubulotoxicity.

Distinctive BMP Signature: - **Potassium:** Markedly elevated (massive cell lysis releases K+) - **Phosphorus:** Elevated (released from muscle cells) - **Calcium:** LOW (hyperphosphatemia drives precipitation into muscle; "tertiary" hyperparathyroidism later) - **Uric acid:** Elevated (purine metabolism) - **Creatinine phosphokinase (CK):** Markedly elevated (>1000 U/L, often >10,000) - **Myoglobin:** Present in urine (dark, tea-colored) - **Anion gap metabolic acidosis:** Present (organic acids from muscle necrosis)

Clinical Pearl: A patient with AKI + hyperkalemia + hypocalcemia + hyperphosphatemia should immediately raise suspicion for rhabdomyolysis. Aggressive isotonic saline + sodium bicarbonate to alkalinize urine (pH >6.5) reduces myoglobin precipitation.

References: Crush injury, prolonged immobilization, statins (rare), severe exercise, heat stroke, serotonin syndrome.

Contrast-Induced Nephropathy (CIN) Pattern

Iodinated contrast agents cause AKI via osmotic diuresis, oxidative stress, and direct tubulotoxicity (especially in patients with diabetes, CKD, or dehydration).

BMP Pattern (24–48 hours post-contrast): - **Creatinine elevation:** Mild to moderate; peaks 3–5 days post-contrast - **BUN elevation:** Often parallel (BUN:Cr ~10:1) - **Sodium:** Usually normal to low - **Potassium:** Normal early; rises if oliguria develops - **Osmolality:** May be elevated if contrast agent still circulating (high osmolar contrast agents) - **Anion gap:** Normal (non-anion-gap metabolic acidosis if severe)

Distinguishing Feature: Temporal relationship to contrast administration. BMP worsens predictably 24–48 hours after procedure.

Prevention Strategy: Isotonic saline hydration before and after contrast; avoid nephrotoxic drugs (NSAIDs, ACE-I, metformin temporarily); consider N-acetylcysteine if high-risk.

Hepatorenal Syndrome (HRS) Pattern

Cirrhosis with portal hypertension triggers splanchnic vasodilation and renal vasoconstriction. Type 1 HRS is fulminant (SCr doubles to >2.5 mg/dL within 2 weeks); Type 2 is gradual.

BMP Pattern: - **Creatinine elevation:** Rapid in Type 1; SCr may reach 3–5 mg/dL - **BUN elevation:** Marked (BUN:Cr >20:1) - **Sodium:** LOW (<130 mEq/L); severe hyponatremia common - **Potassium:** Normal or elevated - **Bicarbonate:** Reduced (metabolic acidosis) - **Albumin:** Markedly low (synthetic liver dysfunction) - **INR:** Elevated (coagulopathy from liver disease) - **Ammonia:** Elevated (hepatic encephalopathy risk)

Clinical Pearl: HRS AKI with severe hyponatremia in a cirrhotic patient suggests a grim prognosis. Vasoconstrictors (terlipressin, norepinephrine) + albumin infusion are standard therapy.

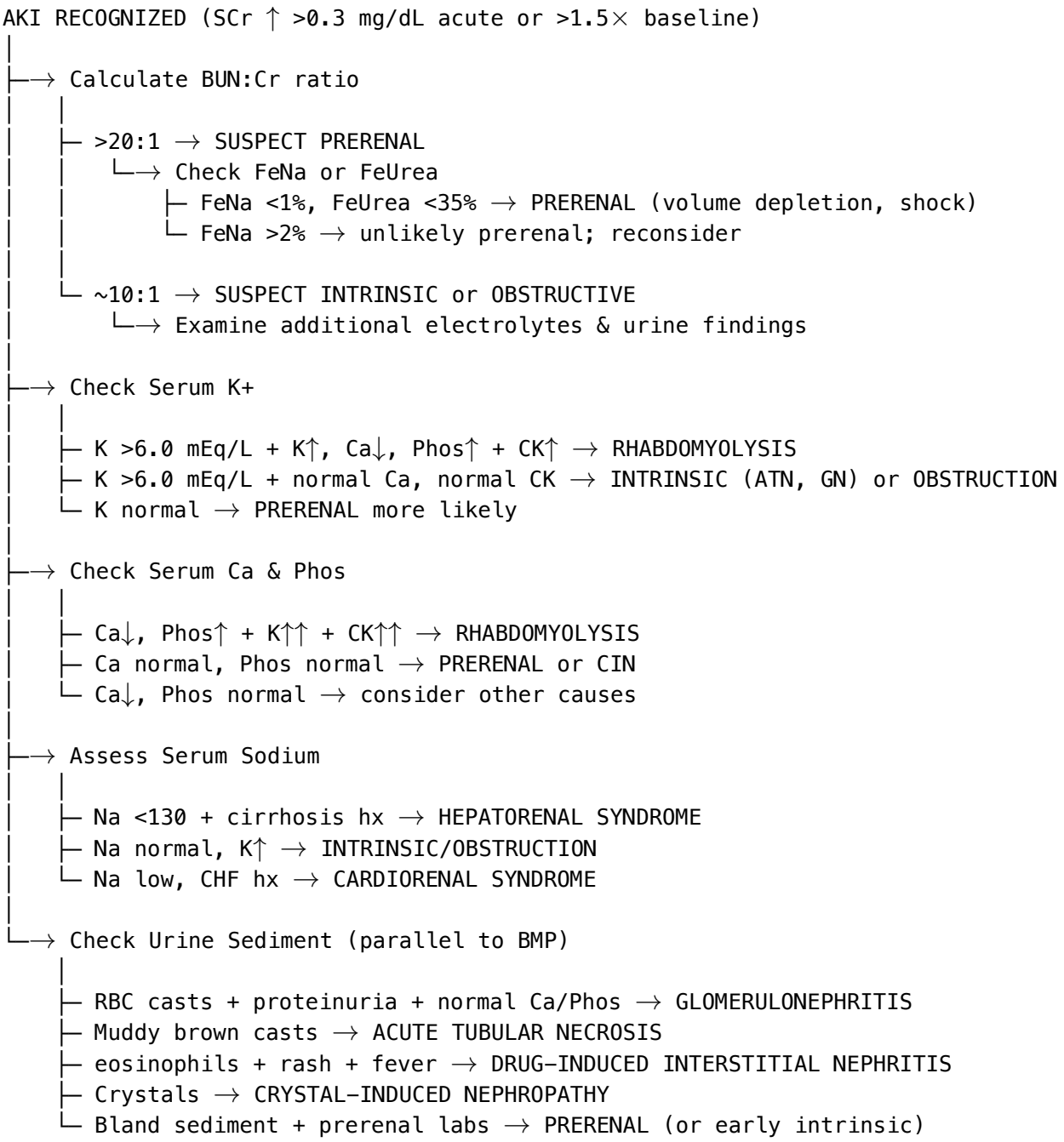
Cardiorenal Syndrome Pattern

Acute decompensated heart failure (ADHF) or cardiogenic shock reduces cardiac output and renal perfusion. Diuretic use complicates interpretation.

BMP Pattern: - **Creatinine elevation:** Mild to moderate (usually <2.5 mg/dL unless shock) - **BUN elevation:** Disproportionate (BUN:Cr >20:1) from poor perfusion + diuretics - **Sodium:** LOW (common; SIADH from hypoperfusion + diuretics) - **Potassium:** Often normal early, but rises if loop diuretics cause hypokalemia reversal - **Bicarbonate:** Reduced if pulmonary edema with respiratory acidosis, or lactic acidosis from shock - **Lactate:** Elevated if cardiogenic shock (anaerobic metabolism)

Distinguishing Feature: Elevated BNP or NT-proBNP; echocardiography shows reduced ejection fraction or diastolic dysfunction.

Part 3: Diagnostic Decision Tree



Part 4: Interpretation of FeNa and FeUrea

Fractional Excretion of Sodium (FeNa)

Formula:

$$\text{FeNa} = \frac{\text{Urine Na} \times \text{Serum Cr}}{\text{Serum Na} \times \text{Urine Cr}} \times 100$$

Interpretation: - **FeNa <1%:** Sodium-avid state; suggests prerenal AKI (sensitivity 90%, specificity 82% in unconfounded cases) [3] - **FeNa 1–2%:** Equivocal; requires clinical context - **FeNa >2%:** Tubular damage; suggests intrinsic AKI or recovery phase of prerenal AKI

Confounders (reduce FeNa sensitivity/specificity): - **Concurrent diuretic use:** Diuretics block proximal tubular reabsorption even in prerenal states; FeNa rises to >1% despite prerenal physiology - **Chronic kidney disease (CKD):** CKD kidneys have impaired reabsorptive capacity; FeNa may be elevated even in prerenal AKI superimposed on CKD - **Acute glomerulonephritis:** Massive proteinuria and hematuria alter electrolyte handling; FeNa may be low despite intrinsic disease - **Contrast-induced nephropathy:** FeNa may be low despite tubular injury

Clinical Pearl: FeNa is most reliable in the absence of diuretics and in patients without significant proteinuria.

Fractional Excretion of Urea (FeUrea)

Formula:

$$\text{FeUrea} = \frac{\text{Urine Urea} \times \text{Serum Cr}}{\text{Serum Urea} \times \text{Urine Cr}} \times 100$$

Advantages over FeNa: - **Urea reabsorption** is less affected by diuretics (occurs in medullary collecting duct via urea transporter UT-A) - FeUrea **<35%** suggests prerenal physiology even in diuretic-treated patients - More accurate in **CKD patients** with AKI

Cutoff: FeUrea <35% = prerenal; >35% = intrinsic [3]

Part 5: Algorithm for Special Electrolyte Patterns

Pattern	BMP Findings	Likely Diagnosis	Next Step
K↑↑↑, Ca↓, Phos↑, CK↑↑	Hyperkalemia + hypocalcemia + hyperphosphatemia	Rhabdomyolysis	Check myoglobin; CK >1000; start aggressive saline + bicarb
K↑, Na normal, Cr ↑ parallel BUN	Hyperkalemia with normal BUN:Cr	ATN or obstruction	Urine sediment (muddy casts = ATN; absence = obstruction)
BUN ↑↑ » Cr, Na low	Severe azotemia + hyponatremia	Hepatorenal syndrome	Albumin level, INR, portal HTN signs

Pattern	BMP Findings	Likely Diagnosis	Next Step
Cr ↑ 24–48 h post-contrast	Temporal relationship to contrast	CIN	Hydration status; hold metformin
Cr ↑, Osm high	Elevated serum osmolality	Osmotic AKI (contrast, sorbitol, glucose)	Check glucose; look for contrast in recent imaging
Cr ↑, RBC casts on UA	Creatinine + hematuria + proteinuria	Glomerulonephritis	Serologic workup: ANA, ANCA, anti-GBM, complement
Cr ↑ + Rash + Fever + eosinophil-uria	Hypercreatininemia + systemic signs	Drug-induced interstitial nephritis	Eosinophils on urine sediment; hold implicated drug

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Part 6: BMP-Guided Clinical Decision-Making

When to Obtain Urine Electrolytes

- Prerenal pattern on BMP (high BUN:Cr, normal K) to confirm with FeNa/FeUrea
- Equivocal presentation (BUN:Cr borderline)
- Post-diuretic to assess tubular response

Do NOT order if clinical picture is obvious (e.g., patient on 10 liters IV NS with rising Cr and urine output <0.5 mL/kg/h = obvious prerenal; treatment is fluids, not urine studies).

When to Obtain Urine Sediment

- All AKI cases where diagnosis is not immediately obvious
- Suspected glomerulonephritis (proteinuria + hematuria)
- Suspected crystal nephropathy (urate, myoglobin, sulfonamides)
- Suspected AIN (eosinophiluria)

When to Order Imaging (Renal Ultrasound)

- Suspect postrenal obstruction (elevated Cr, anuria, abdominal pain, stone hx)
- BMP shows K ↑ + normal or low BUN:Cr (obstruction pattern)
- No response to fluid resuscitation despite prerenal pattern
- Post-void residual >100 mL on catheterization

When to Order Renal Biopsy

- Rapid AKI + proteinuria + RBC casts despite normal serum complement (suggests lupus or membranoproliferative GN)

- AKI with negative serologies but clinical suspicion for vasculitis (ANCA-negative pauci-immune RPGN)
- CKD with superimposed AKI and new proteinuria (recurrent disease or secondary GN)

Part 7: Key Warnings

Warning 1: FeNa is Not Infallible

A patient with **prerenal AKI on ACE inhibitor therapy** may have FeNa >1% because the RAAS is already blocked, reducing tubular reabsorption capacity. Clinical context trumps a single lab value.

Warning 2: Rhabdomyolysis Can Present Insidiously

Creatinine may rise more slowly than CK or myoglobin. A patient with mild initial Cr rise but **CK >5,000** should trigger aggressive volume expansion and urine alkalinization immediately, before AKI progresses.

Warning 3: Osmolar Gap Signals Toxin

If serum osmolality (measured) is >10 mOsm/kg higher than calculated osmolality:

$$\text{Calculated Osm} = 2[\text{Na}] + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}$$

Suspect **methanol, ethylene glycol, propylene glycol** (IV lorazepam), or mannitol toxicity. These can cause AKI via osmotic effects + direct toxicity.

Warning 4: Severe Hyponatremia in Cirrhosis is an Emergency

Serum Na <120 mEq/L in a cirrhotic patient with AKI signals **hepatorenal syndrome Type 1**. Prognosis is poor without vasoconstrictors + albumin. Terlipressin or norepinephrine should be started immediately.

Related Resources

- ← Back to Acute Kidney Injury Hub
- ← Back to Nephrology Hub
- Back to Medical Education Dashboard
- Electrolyte & Acid-Base Disorders Hub

References

[1] Kellum JA, Lameire N; KDIGO AKI Work Group. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). *Crit Care*. 2013;17:204. PubMed

[2] Waikar SS, Bonventre JV. Creatinine kinetics and the definition of acute kidney injury. *J Am Soc Nephrol*. 2009;20:672–679. PubMed

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