Urinalysis Master Diagnostic Algorithm

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Introduction: The Underutilized Diagnostic Tool

Urinalysis remains one of the most powerful diagnostic tools in clinical medicine, yet is often ordered but not carefully interpreted. A methodical, stepwise approach to urinalysis—dipstick interpretation followed by microscopic analysis—reveals specific pathophysiologic processes occurring within the kidneys and urinary tract. This guide teaches pattern recognition for urinalysis as a diagnostic window into renal disease [1].

> [!important] Key Point Urinalysis is not a screening test—it is a targeted diagnostic tool. The findings must be integrated with BMP, renal function, imaging, and serologic markers to construct a differential diagnosis.

Part 1: Dipstick Analysis

The Seven Key Dipstick Parameters

1. pH (4.5–8.0 normal)

pH	Causes	Clinical Significance
Acidic (< 5.5)	Meat-based diet, metabolic acidosis, diarrhea	Favors uric acid stone formation
Neutral (6.5–7.0)	Mixed diet, normal	Normal range
Alkaline (> 7.5)	Vegetarian diet, UTI (urease-producing organisms), alkali therapy, respiratory alkalosis	Favors phosphate/calcium stone formation (triple phosphate)

Clinical Pearl: Urine pH interpretation requires **timing**—pH changes rapidly if specimen sits at room temperature (bacteria consume acids, pH rises).

2. Specific Gravity (1.005–1.030)

SG Value	Interpretation	Clinical Significance
1.005–1.010 (Low)	Dilute urine	Polyuria, DI, overhydration, acute tubular necrosis
1.010–1.020 (Normal)	Concentrated urine	Normal renal concentrating ability
> 1.020 (High)	Very concentrated urine	Prerenal azotemia, dehydration, SIADH, acute glomerulonephritis

> [!tip] Clinical Pearl High SG with low osmolality is paradoxical and suggests presence of osmotically active particles: glucose (DM, Fanconi), protein (nephrotic syndrome), contrast (post-CT), lipids.

3. Protein (Dipstick: Negative–3+)

Dipstick Result	Approximate Quantity	Pathophysiology
Negative	< 150 mg/day	Normal or excellent renal function
Trace–1+	150–300 mg/day	Borderline; quantify with UPCR or 24h collection
2+	500 mg/day	Likely glomerular disease; warrants investigation
3+	1,000 mg/day	Nephrotic-range proteinuria (> 3.5 g/day)
4+	> 1,000 mg/day	Massive proteinuria; almost always glomerular disease

Dipstick Limitations: - Detects albumin best; misses immunoglobulin light chains, Bence Jones protein - False positives: concentrated urine, alkaline urine, blood contamination - False negatives: dilute urine, non-albumin proteinuria

Quantification Methods: - **24-hour urine protein** (gold standard; cumbersome) - **Spot urine protein-to-creatinine ratio (UPCR)** (preferred; more convenient) - UPCR mg/g = (urine protein mg/dL / urine Cr mg/dL) × 100 - UPCR 100–300 mg/g = microalbuminuria (early diabetic/hypertensive disease) - UPCR > 300 mg/g = macroalbuminuria - UPCR > 3,500 mg/g = nephrotic-range

4. Blood on Dipstick (Negative–3+)

Finding	Interpretation	Differential
Blood + RBCs on microscopy	True hematuria	Glomerular (casts, proteinuria), IgAN, ANCA, post-infectious, trauma, stones, malignancy

Finding	Interpretation	Differential
Blood + NO RBCs on microscopy	Hemoglobinuria (free Hb)	Intravascular hemolysis (malaria, PNH, severe burns)
Blood + NO RBCs + dark urine	Myoglobinuria	Rhabdomyolysis, severe myositis
Blood + lots of RBCs + casts	Glomerulonephritis	PSGN, IgAN, lupus, ANCA-associated

> [!warning] High-Yield Board Point The dipstick is **exquisitely sensitive** for blood (detects heme-containing compounds) but **not specific** (can detect Hb, myoglobin). **Always confirm with microscopy.** If blood 3+ but no RBCs on microscopy → think hemolysis or rhabdo.

5. Leukocyte Esterase (Negative or Positive)

Result	Interpretation	Significance
Positive	WBCs present (from neutrophils)	UTI, pyelonephritis, acute interstitial nephritis, contamination
Negative	No WBCs or very few	Absence of pyuria; helps rule out UTI/AIN

Limitations: False negatives if immunosuppressed (unable to mount WBC response); false positives from contamination.

6. Nitrites (Negative or Positive)

Result	Interpretation
Positive	Gram-negative bacteria (E. coli, Klebsiella, Proteus); highly specific for UTI
Negative	Gram-positive bacteria, anaerobes, Pseudomonas (all nitrite-negative); does NOT rule out UTI

Pearl: Positive leukocyte esterase + positive nitrites = high likelihood of bacterial UTI (~95%). Negative nitrites does NOT exclude UTI.

7. Glucose (Dipstick: Negative–4+)

Finding	Threshold	Significance
Negative	< 5 mg/dL	Normal (renal threshold ~180 mg/dL plasma)

Finding	Threshold	Significance
Positive (any grade)	> 5 mg/dL	**Plasma glucose > renal threshold (160–180) OR abnormal proximal tubular reabsorption (Fanconi, SGLT2i)**

Additional Dipstick Parameters

Parameter	Normal	Pathologic	Interpretation
Ketones	Negative	Positive	DKA, starvation ketosis, high-fat diet
Bilirubin	Negative	Positive	Bilirubinuria (conjugated bilirubin in urine); suggests liver disease/biliary obstruction
Urobilinogen	Trace–0.1 mg/dL	Elevated	Hemolysis, liver disease; absent in biliary obstruction

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Part 2: Microscopy Interpretation

Specimen Preparation and Quality

- **Fresh urine only** (< 2 hours old ideally)
- **Centrifuged:** 5 mL at 1,500 rpm × 5 min
- **Low power (LPF, 10×):** 10–15 fields for casts, crystals, large structures
- **High power (HPF, 40×):** 5–10 fields for cells, bacteria, small crystals

Casts: The "Renal Cell Express"

Golden Rule: Finding ANY casts in urine = casts came from kidneys (not contamination)

Cast Types and Pathophysiology

Cast Type	Composition	Clinical Significance	Associated Findings
Hyaline	Tamm-Horsfall (uromodulin) only	Normal if < 2–3/LPF; increased in dehydration, fever, exercise, acidosis	Often asymptomatic
RBC	RBCs trapped in matrix	**ALWAYS pathologic**; glomerulonephritis	Hematuria, proteinuria, dysmorphic RBCs, acanthocytes
WBC	WBCs in matrix	Pyelonephritis, acute interstitial nephritis, lupus	Pyuria, bacteriuria, systemic symptoms

Cast Type	Composition	Clinical Significance	Associated Findings
Muddy Brown/Granular	Cellular debris, degenerated organelles	**Acute Tubular Necrosis (ATN)**; the "muddy brown" is pathognomonic	Acute rise in Cr, FeNa > 2%, muddy casts
Waxy/Broad	Uromodulin + proteinaceous material	**Chronic kidney disease**; indicates slowed urine flow/long tubular transit	Chronic Cr elevation, narrow casts also present
Fatty	Lipids, oval fat bodies (lipid-laden epithelial cells)	**Nephrotic syndrome**	Heavy proteinuria, hyperlipidemia, lipiduria
Crystalline	Specific crystal types (see table below)	Stone-forming tendency; Fanconi; toxic medication (sulfadiazine, ampicillin)	Acidic/alkaline pH, relevant medication history

Cells and Cellular Elements

Element	Normal	Pathologic	Interpretation
RBCs	0–3/HPF	> 3/HPF = hematuria	Glomerular if dysmorphic/acanthocytes; non-glomerular if isomorphic
WBCs	0–5/HPF (female); 0–2/HPF (male)	> 5/HPF = pyuria	UTI, pyelonephritis, AIN, lupus
Epithelial cells (renal tubular)	0–1/HPF	> 2–3/HPF	Tubular injury (ATN, drug toxicity, reflux)
Epithelial cells (squamous)	Common (contamination)	Should be ignored if > 5–10/HPF	Vaginal/skin contamination; invalidates specimen
Bacteria	Absent	Present	UTI if symptomatic + nitrites/LE positive
Yeast	Absent	Rare	Vaginal candidiasis (female) or contamination
Trichomonads	Absent	Rare	Trichomoniasis (sexually transmitted)

[!tip] Clinical Pearl **Dysmorphic RBCs** (irregular shape, membrane protrusions) indicate glomerular origin; **isomorphic RBCs** (uniform, regular shapes) indicate non-glomerular hematuria (stones, tumors, trauma).

Crystals: Identifying Stone-Forming Tendency and Toxicity

Crystal Type	pH	Associated Condition	Clinical Pearl
Calcium oxalate	Acidic	Hyperoxaluria, ethylene glycol toxicity	Envelope-shaped (diagnostic); elevated in ethylene glycol poisoning
Calcium phosphate/apatite	Alkaline	Normal variant, stone former	Appear as amorphous granules
Uric acid	Acidic	Gout, tumor lysis, dehydration	Rhomboid or needle-shaped; increase in acidosis
Struvite (magnesium ammonium phosphate)	Alkaline	UTI with urease producers (Proteus); chronic struvite stones	"Coffin-lid" shaped; always indicates infection
Cystine	Acidic	Cystinosis (rare inherited disorder); cystine stones	**Pathognomonic hexagonal shape**; strongly suggests cystinosis
Sulfonamide/Sulfadiazine	Variable	Drug-induced nephropathy (TMP-SMX, sulfadiazine)	Needle-shaped; associated with fever, rash (AIN)
Bilirubin	Any	Hemolysis, liver disease	Rare; biliary obstruction prevents bilirubin excretion
Myoglobin/Hemoglobin	Any	Rhabdomyolysis, hemolysis	Dark (tea-colored) urine; associated with hypokalemia

Bacteria and Organisms

Organism	Morphology	Associated Condition
Bacteria (rods)	Gram-negative coccobacilli	E. coli, Klebsiella, Proteus (UTI)
Cocci	Clusters or pairs	Staphylococcus, Streptococcus
Trichomonads	Motile, flagellate	Trichomoniasis
Yeast	Budding forms	Candidiasis (especially with glycosuria)
Sperm	Motile (fresh specimen)	Semen contamination

Part 3: Systematic Diagnostic Algorithm

The Five-Question Diagnostic Cascade

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URINALYSIS INTERPRETATION FLOWCHART
│
├─ Question 1: Is there HEMATURIA (RBCs > 3/HPF)?
│  ├─ YES → Is there PROTEINURIA and CASTS?
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│   │   ├─ YES → GLOMERULONEPHRITIS (See Differential Table)
│   │   │   └─ Pattern: Dysmorphic RBCs, RBC casts, proteinuria
│   │   │       Differential: IgAN, post-infectious GN, lupus, ANCA
│   │   │
│   │   └─ NO → NON-GLOMERULAR HEMATURIA
│   │       └─ Pattern: Isomorphic RBCs, NO casts, no proteinuria
│   │           Differential: Stones, malignancy, trauma, benign hematuria
│   │
│   └─ NO
│
├─ Question 2: Is there PROTEINURIA (dipstick positive)?
│   ├─ Nephrotic range (> 3.5 g/24h)
│   │   ├─ Pattern: Heavy proteinuria, oval fat bodies, fatty casts, hypoalbuminemia
│   │   │   Differential: Minimal change, FSGS, membranous, diabetic
│   │   │
│   └─ Non-nephrotic (< 3.5 g/24h) with/without RBCs
│       └─ Differential: IgAN, secondary GN, CKD, hypertensive
│
├─ Question 3: Is there PYURIA (WBCs > 5/HPF)?
│   ├─ + Leukocyte esterase + Nitrites → BACTERIAL UTI
│   ├─ + Leukocyte esterase – Nitrites → Viral UTI or Sterile Pyuria
│   │   └─ Sterile pyuria differential: Tuberculosis, AIN, glomerulonephritis
│   │
│   └─ – Leukocyte esterase → Rule out UTI/AIN
│
├─ Question 4: Are there CASTS?
│   ├─ RBC or WBC casts → INTRINSIC RENAL DISEASE
│   ├─ Muddy brown casts → ACUTE TUBULAR NECROSIS (ATN)
│   ├─ Waxy/broad casts → CHRONIC KIDNEY DISEASE
│   └─ Hyaline casts alone → Usually benign (if < 2/LPF)
│
└─ Question 5: Are there CRYSTALS or UNUSUAL ELEMENTS?
    ├─ Calcium oxalate → Stone former, ethylene glycol toxicity
    ├─ Cystine → Cystinosis (rare)
    ├─ Sulfonamide → Drug-induced AIN (TMP-SMX, sulfadiazine)
    └─ Myoglobin + dark urine → Rhabdomyolysis
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Diagnostic Patterns by Clinical Scenario

Pattern 1: Acute Glomerulonephritis (AGN)

Finding	Significance
RBC casts	Hallmark finding; proves glomerular origin
Dysmorphic RBCs	Indicates glomerular damage
Proteinuria	Often 2–3+ (can be nephrotic; 5–10 g/day)
RBC count	Moderate to numerous

Finding	Significance
WBC casts or RBC casts	Variable
Hyaline casts	Often present

Associated BMP findings: ↑ Cr, ↓ GFR, ↑ K (if severe oliguria), hypervolemia

Differential by serologies/clinical context: - **Post-infectious GN:** Young, recent infection (throat, skin), complement low - **IgA nephropathy:** Recurrent hematuria, IgA dominant on biopsy - **ANCA-associated:** Systemic symptoms, ANCA positive - **Lupus:** Systemic symptoms, ANA/anti-dsDNA positive

Pattern 2: Nephrotic Syndrome

Finding	Significance
Heavy proteinuria	3+–4+ dipstick (> 3.5 g/24h)
Oval fat bodies	Lipid-laden tubular epithelium; pathognomonic
Fatty casts	Lipid-containing casts
No RBC casts	Distinguishes from membranoproliferative GN
Hyaline casts	Often numerous
Minimal hematuria	< 5 RBCs/HPF (absent or trace blood)

Associated findings: Hypoalbuminemia, hyperlipidemia, edema, heavy proteinuria on quantification

Differential: - **Minimal change disease:** Young, sudden onset, excellent prognosis - **Focal segmental glomerulosclerosis (FSGS):** Partially responsive to steroids; worse prognosis - **Membranous nephropathy:** Older, anti-PLA2R antibodies, insidious onset - **Diabetic nephropathy:** Long DM history, concurrent diabetic retinopathy, proteinuria without RBCs

Pattern 3: Acute Tubular Necrosis (ATN)

Finding	Significance
Muddy brown/dark granular casts	**Pathognomonic for ATN**
Renal tubular epithelial (RTE) cells	From desquamated tubules
RTE cell casts	Desquamated cells in casts
Coarse granular casts	Debris from necrotic tubules
Minimal or absent RBCs	Unlike glomerular disease
Minimal proteinuria	< 1+ dipstick (< 500 mg/day)
High urine osmolality	Often > 400 (kidney trying to concentrate)

Associated findings: Acute rise in Cr, oliguria, FeNa > 2%, “dirty” urine appearance

Causes: Ischemia (shock, sepsis), nephrotoxins (aminoglycosides, contrast, NSAIDs with volume depletion), rhabdomyolysis

Pattern 4: Pyelonephritis

Finding	Significance
Pyuria	WBCs > 5/HPF, often numerous
WBC casts	Pathognomonic if present; casts from collecting duct
Bacteria	Rods (gram-negative) most common
Nitrites	Positive if gram-negative organism
Leukocyte esterase	Positive
Proteinuria	Usually trace–1+ (< 500 mg/day)
Hematuria	Trace to 1+ (microscopic)

Associated findings: Fever, flank pain, CVA tenderness, elevated WBC, elevated CRP/ESR

Pattern 5: Acute Interstitial Nephritis (AIN)

Finding	Significance
Sterile pyuria	WBCs without bacteria; LE positive, nitrites negative
WBC casts	If present, confirms renal involvement
RTE cells	Tubular epithelial shedding
Eosinophiluria	(Special stain: Hansel stain); suggests drug-induced
Proteinuria	Usually mild (trace–1+); nephrotic is unusual
Minimal hematuria	May be absent or trace
Normal or mildly elevated casts	Hyaline predominant

Associated findings: Acute Cr rise (often > 50% increase), fever, rash (if drug), recent NSAID/antibiotic use

Causes: NSAIDs, β-lactams (PCN, cephalosporins), diuretics, PTIs (omeprazole), anticonvulsants

Pattern 6: Chronic Kidney Disease (CKD)

Finding	Significance
Waxy/broad casts	Indicate slowed urine flow; advanced CKD
Granular casts	Fine or coarse; reflect chronic tubular atrophy
Mild proteinuria	Usually < 1+ (< 1 g/day), unless secondary cause
Minimal hematuria	Absent or trace; may be present in FSGS/IgAN
Minimal or absent RBC casts	Unlike active glomerulonephritis
Hypocellular sediment	Few cells overall

Associated findings: Chronic Cr elevation, small kidneys on imaging, anemia (CKD-related)

Urine Electrolytes and Functional Indices

Urine Sodium (UNa) and Chloride (UCl)

Finding	Interpretation	Clinical Significance
UNa < 10–20 mEq/L	Sodium avid	Pre-renal azotemia, hepatic encephalopathy, heart failure
UNa > 40 mEq/L	Sodium wasting	Intrinsic renal disease, diuretic use, advanced CKD
UCl < 10 mEq/L	Chloride-responsive alkalosis	Vomiting, NG suction (volume depletion)
UCl > 10 mEq/L	Chloride-resistant alkalosis	Primary hyperaldosteronism, diuretic use

Fractional Excretion of Sodium (FeNa)

$$FeNa = \frac{(UNa \times PlasmaCr)}{(PlasmaNa \times UrineCr)} \times 100$$

FeNa	Interpretation	AKI Type
< 1%	Sodium avid; intact tubular function	**Pre-renal azotemia** (volume depletion, shock, heart failure)
> 2%	Sodium wasting; tubular dysfunction	**Intrinsic renal disease** (ATN, GN, AIN, contrast nephropathy)
1–2%	Gray zone; clinical judgment required	Early ATN or severe pre-renal

> [!warning] High-Yield Board Point FeNa < 1% in oliguria strongly suggests pre-renal; FeNa > 2% suggests intrinsic disease. However, FeNa can be falsely low in heart failure/cirrhosis and falsely high in elderly/CKD. **Clinical context is paramount.**

Urine Osmolality (Uosm)

Uosm	Interpretation	Significance
> 500 mOsm/kg	Concentrated urine	Intact renal concentrating ability; pre-renal pattern
250–500 mOsm/kg	Moderately concentrated	Intermediate; assess clinical context
< 300 mOsm/kg	Dilute urine	Loss of concentrating ability; intrinsic disease, DI, overhydration

Urine Anion Gap (UAG) [2]

$$UAG = (UNa + UK) - UCl$$

UAG	Interpretation	Differential
Negative (< 0)	GI bicarbonate loss	Diarrhea (especially small bowel) → appropriate renal acidification
Positive (> 0)	Renal tubular dysfunction	RTA (Type 1, 2, 4); reduced GFR; kidney cannot acidify urine

Clinical use: Differentiates causes of non-gap metabolic acidosis

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Board-Level Clinical Vignettes

Vignette 1: Hematuria with Casts

A 24-year-old male college athlete presents with dark urine × 2 days post-streptococcal pharyngitis. Dipstick: 3+ blood, 2+ protein. Microscopy: Numerous dysmorphic RBCs, RBC casts, WBC casts, no bacteria.

Diagnosis: Post-infectious glomerulonephritis (PSGN)

Key findings: RBC casts (pathognomonic for GN) + dysmorphic RBCs + proteinuria + recent infection

Next steps: Serologies (ASO titer, strep Ag), complement levels (C3 often low), renal ultrasound, possibly biopsy if atypical presentation

Prognosis: Excellent in children/young adults; most recover spontaneously; dialysis if severe oliguria

Vignette 2: Heavy Proteinuria with Oval Fat Bodies

A 38-year-old woman with 2-week history of leg swelling and dark urine. Dipstick: 4+ protein. Microscopy: Numerous oval fat bodies, fatty casts, rare RBCs (< 1/HPF), no casts except fatty.

Diagnosis: Nephrotic syndrome

Likely etiology: Membranous nephropathy (most common in adults), minimal change (if young/sudden), FSGS (if African American or progressive)

Supporting findings to order: - Serum albumin (expect < 3.5 g/dL) - Lipid panel (expect elevated cholesterol/triglycerides) - 24-hour urine protein (should be > 3.5 g) - Renal ultrasound (normal-sized kidneys rule out advanced CKD) - Serologies: ANA, anti-PLA2R (membranous marker), hepatitis B/C, malignancy screening

Next: Kidney biopsy to establish diagnosis and prognosis

Vignette 3: Muddy Brown Casts in Oliguria

A 52-year-old male post-major surgery (day 1 post-op) with oliguria (urine output 200 mL/12h). Dipstick: Trace protein, trace blood. Microscopy: Numerous muddy brown granular casts, RTE cells, minimal RBCs, no bacteria.

Diagnosis: Acute tubular necrosis (ATN) — ischemic type

Mechanism: Perioperative hypotension + major surgical stress → ischemia to renal tubules

Confirm with: FeNa > 2%, urine osmolality < 400, rapid Cr rise

Management: 1. Aggressive IV hydration with normal saline (restore perfusion) 2. Avoid nephrotoxins (NSAIDs, ACE-I, aminoglycosides) 3. Monitor K, Mg, PO4 (hyperkalemia risk with oliguria) 4. Prepare for dialysis if refractory hyperkalemia, pulmonary edema, or severe acidosis 5. Most ATN improves within 5–7 days; trend urine output, daily weights

Poor prognostic sign: Ongoing muddy casts at day 3–5 post-op indicates severe injury

Vignette 4: Sterile Pyuria Post-Antibiotic

A 68-year-old female post-TMP-SMX for UTI (started 2 weeks ago) presents with fever, rash, acute Cr rise from 1.0 to 2.1. Dipstick: 1+ protein, trace blood, LE positive, nitrites negative. Microscopy: WBCs 15/HPF (no bacteria), occasional RTE cells, hyaline casts, no RBC casts.

Diagnosis: Acute interstitial nephritis (AIN) — drug-induced (TMP-SMX)

Classic triad: Fever + rash + acute kidney injury post-drug exposure

Confirm with: - Urine eosinophils (Hansel stain; eosinophiluria suggests drug-induced) - Absence of RBC casts (distinguishes from GN) - Peripheral eosinophilia (not always present) - Renal ultrasound (may show interstitial edema)

Management: 1. **Immediately stop TMP-SMX** (causative agent) 2. Supportive care, hydration 3. Corticosteroids if severe (prednisone 1 mg/kg/day × 3 days, taper over 2–4 weeks); benefit greatest if started early 4. Monitor Cr daily; most improve within 5–7 days after drug discontinuation 5. Avoid NSAIDs, ACE-I (further stress kidney)

Vignette 5: Cystine Crystals in Asymptomatic Adolescent

A 16-year-old male routine physical. Dipstick: Negative. Microscopy: Several **pathognomonic hexagonal cystine crystals**, otherwise negative findings.

Diagnosis: Cystinosis — autosomal recessive disorder of cystine transport

Significance: Cystine crystals = diagnostic for cystinosis; patient likely has progressive renal disease ahead

Next steps: 1. Check plasma cystine (elevated confirms cystinosis) 2. Slit-lamp examination for corneal cystine deposition 3. Referral to genetics and nephrology 4. Consider cysteamine therapy (chelating agent) to reduce intracellular cystine 5. Monitor for other manifestations: growth retardation, photophobia, hypothyroidism, ESRD by adolescence/early adulthood

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Integration with BMP and Renal Function

Complete Picture: Urinalysis + BMP + Renal Function

Clinical Scenario	UA Pattern	BMP Pattern	GFR/Cr	Diagnosis
Acute GN	RBC casts, dysmorphic RBCs, 2+ protein	Normal K initially; ↑ Cr if severe	Acute ↓ GFR	Post-infectious or primary GN
Nephrotic syndrome	Heavy proteinuria, oval fat bodies	↓ Albumin, ↓ Ca, ↑ lipids	Normal–mildly ↓	FSGS, membranous, minimal change
ATN (ischemic)	Muddy brown casts, RTE cells, trace protein	↑ K, ↑ PO4, ↑ BUN/Cr ratio	Rapidly ↑ (doubling)	Post-shock, post-op, sepsis
Pre-renal azotemia	Concentrated urine, hyaline casts	Normal electrolytes, ↑ BUN/Cr ratio	Mildly ↑	Volume depletion, heart failure, sepsis
AIN	Sterile pyuria, WBC casts, ±eosinophils	Normal electrolytes	Acute ↑	Recent drug exposure
Chronic GN/CKD	Waxy casts, mild proteinuria	Chronic ↑ K, ↑ PO4, ↑ PTH (if severe)	Chronically ↓	IgAN, FSGS, hypertensive, diabetic

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Related Resources

- BMP Pattern Recognition Master Guide

- Acute Kidney Injury Evaluation Protocol
- Glomerulonephritis: Differential and Workup
- Nephrotic Syndrome Comprehensive Approach
- UTI and Pyelonephritis Management

Last updated: 2026-02-28 | Board-review quality reference. Urinalysis interpretation requires integration with clinical context, BMP findings, and renal function trends.