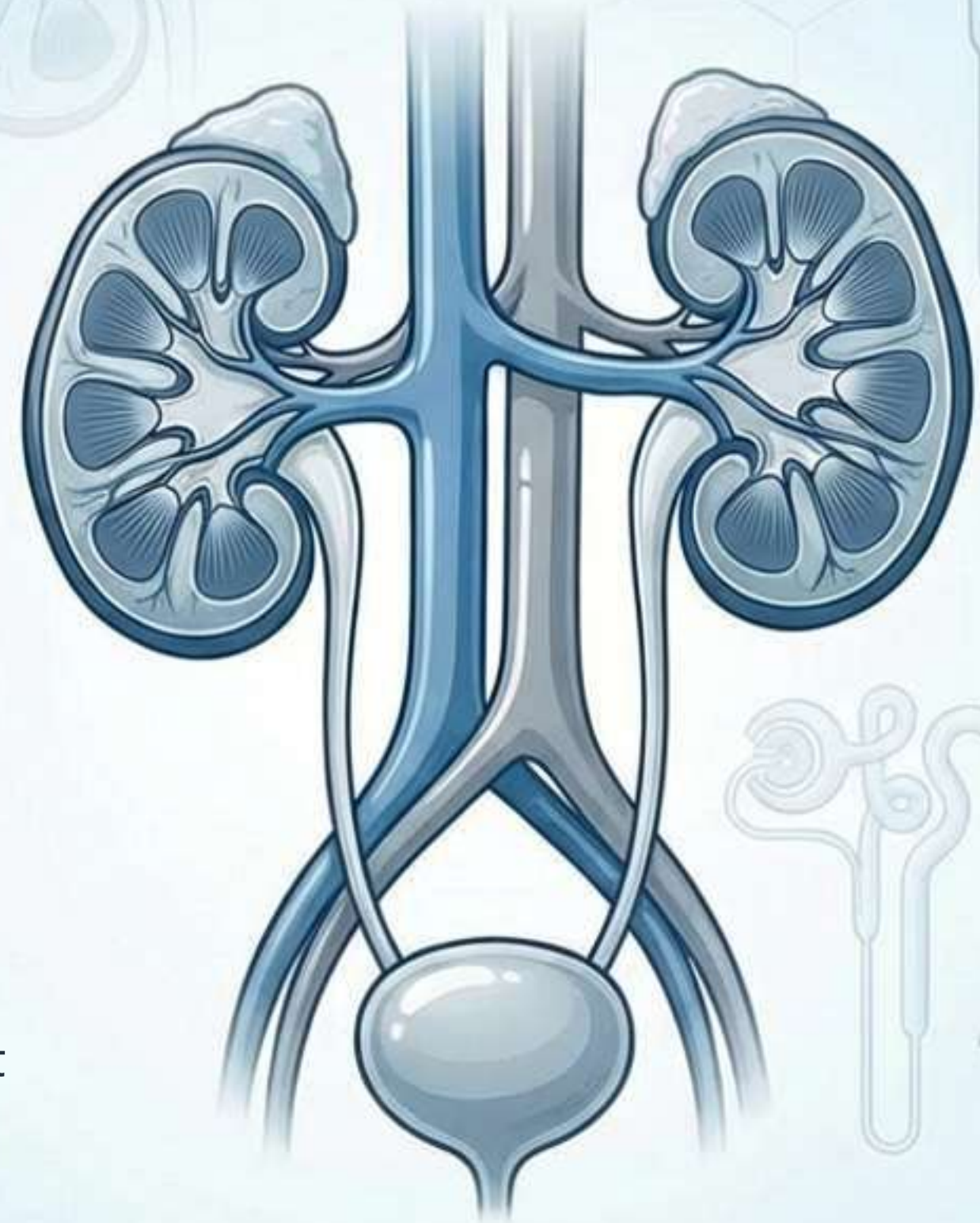


ACUTE KIDNEY INJURY (AKI)

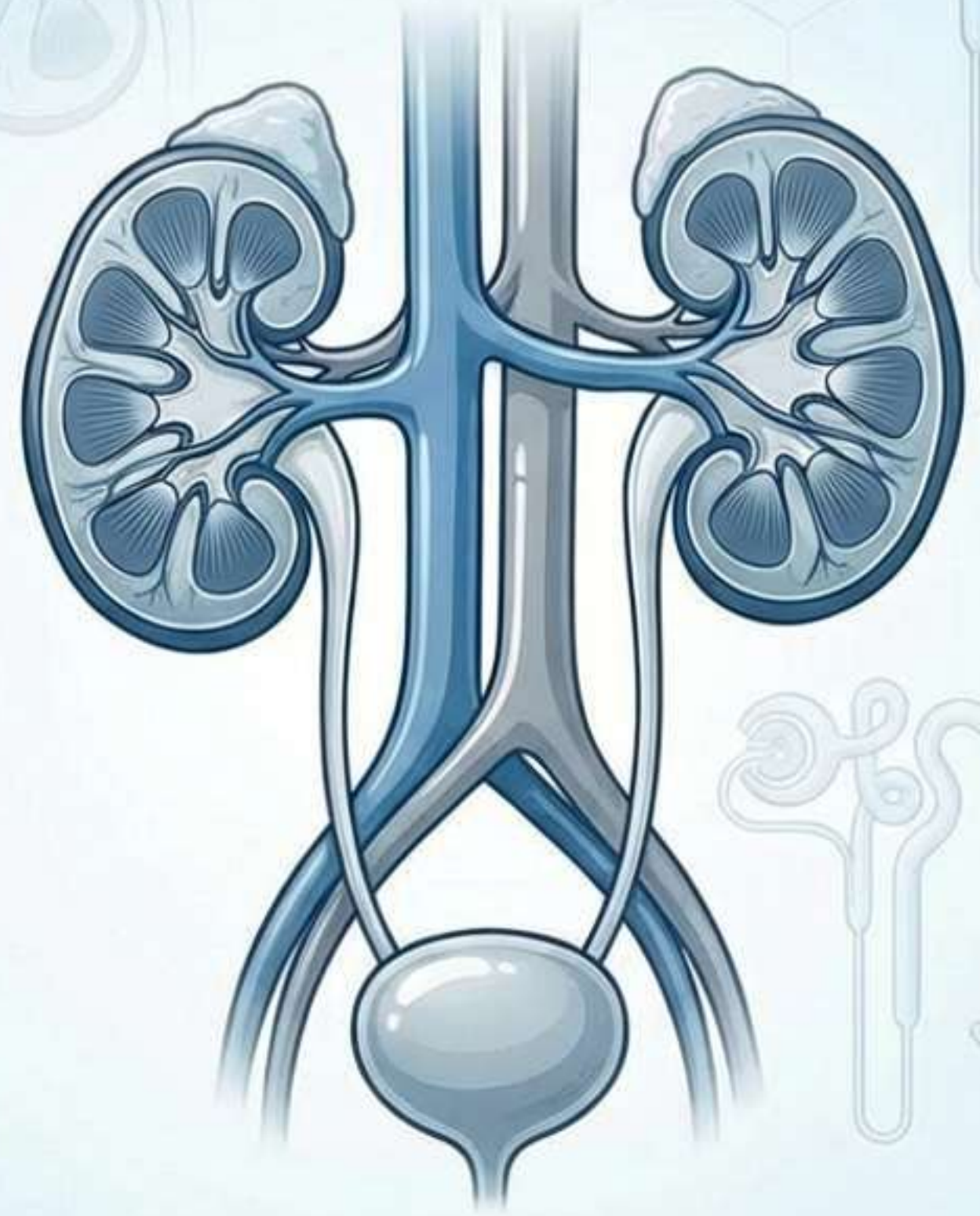
Group C8

Done by : Osama Almaharmeh



ACUTE KIDNEY INJURY (AKI)

It is a sudden decline in kidney function occurring over hours to days, resulting in the accumulation of nitrogenous waste products (urea and creatinine), disturbance of fluid balance, and abnormalities in electrolyte and acid-base homeostasis



KDIGO criteria

Serum Creatinine



rise $\geq 0.3\text{mg/dL}$
within 48h
OR
 $\geq 1.5\times$ baseline
within 7 days

Urine Output



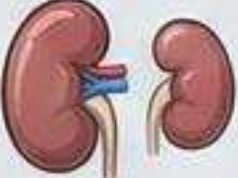
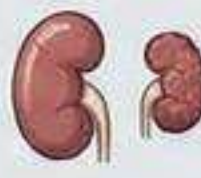
$< 0.5\text{mL/kg/hr}$
for $> 6\text{h}$



Enables early detection before
overt symptoms appear.



AKI vs. CKD: A Comparative Overview

 Acute Kidney Injury (AKI) 	 Chronic Kidney Disease (CKD) 
 Sudden Onset (Hours to Days)	 Gradual Onset (Months to Years)
 Normal Kidney Size	 Shrunk Kidneys
 Absent Anemia/Bone Disease	 Common Anemia/Bone Disease
 Often Reversible	 Irreversible

Note: AKI can worsen pre-existing CKD.



Azotemia vs. Uremia: Key Definitions

Azotemia



Azotemia: elevated urea/creatinine without symptoms.



Uremia



Uremia: clinical syndrome from toxic metabolite accumulation causing encephalopathy, pericarditis, bleeding, anorexia, pruritus. Uremia refers to symptoms, not just lab values.

Uremic Manifestations: System-Wise Effects



CNS (Central Nervous System)

- Confusion
- Seizures
- Coma



CVS (Cardiovascular System)

- Pericarditis
- Arrhythmia



Skin

- Uremic Frost
- Pruritus



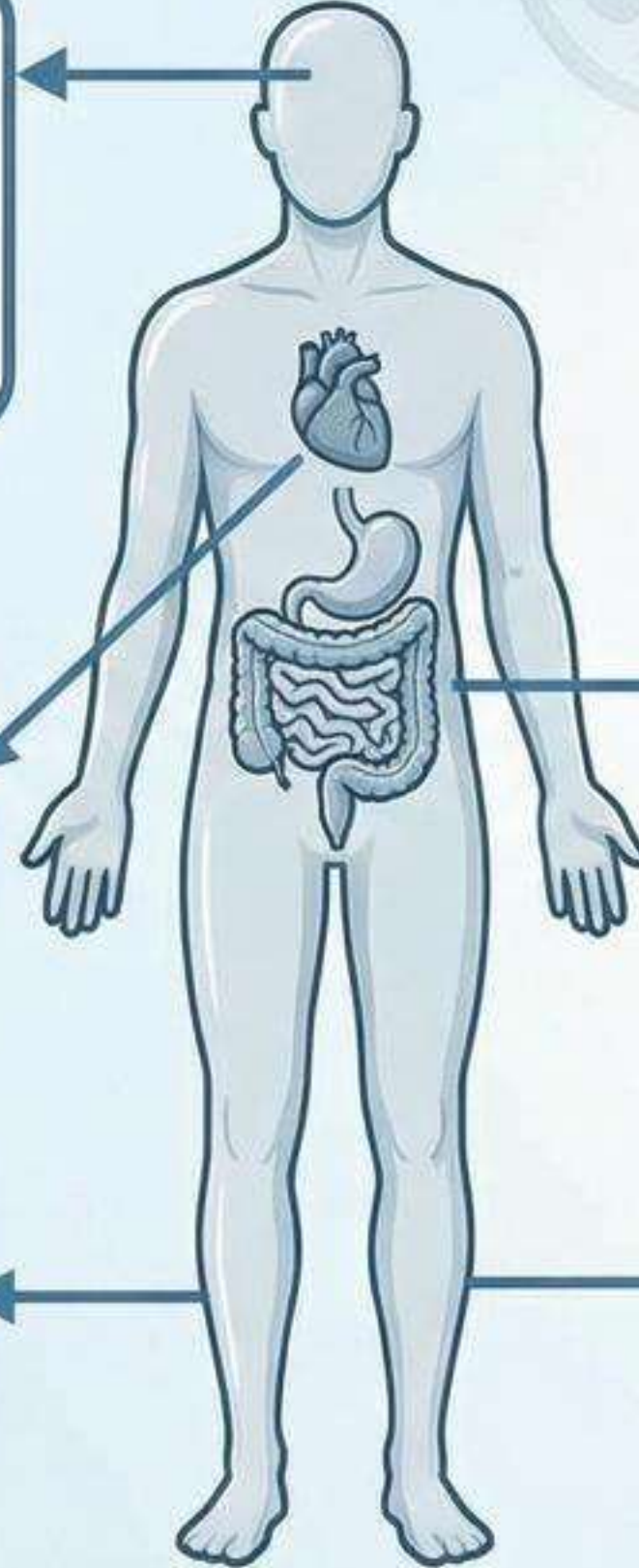
GIT (Gastrointestinal Tract)

- Nausea
- Metallic Taste
- Vomiting



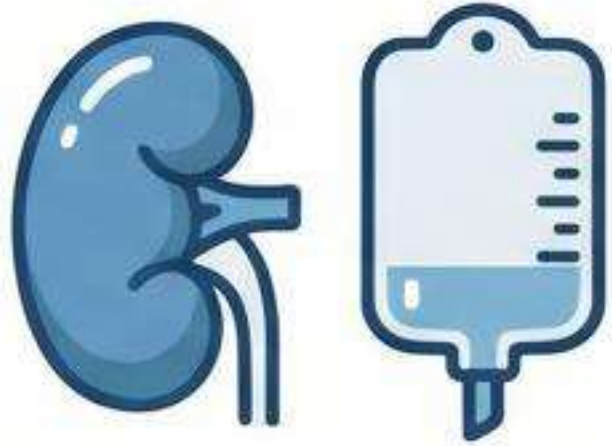
Hematologic

- Bleeding Tendency
- Platelet Dysfunction



AKI Classification by Urine Output

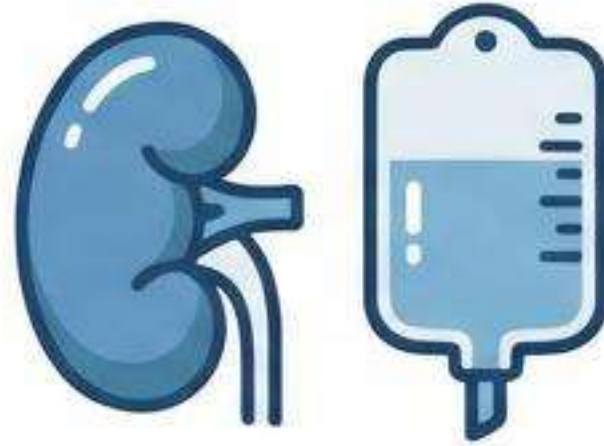
Classification based on daily urine volume, crucial for prognosis.



OLIGURIC AKI

<400 mL/day

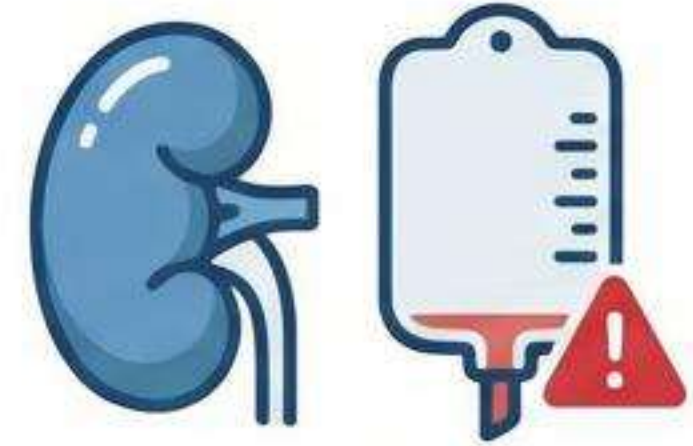
- Commonly indicates significant GFR reduction.



NON-OLIGURIC AKI

Normal urine output

- Impaired filtration persists despite normal volume.



ANURIC AKI

<100 mL/day

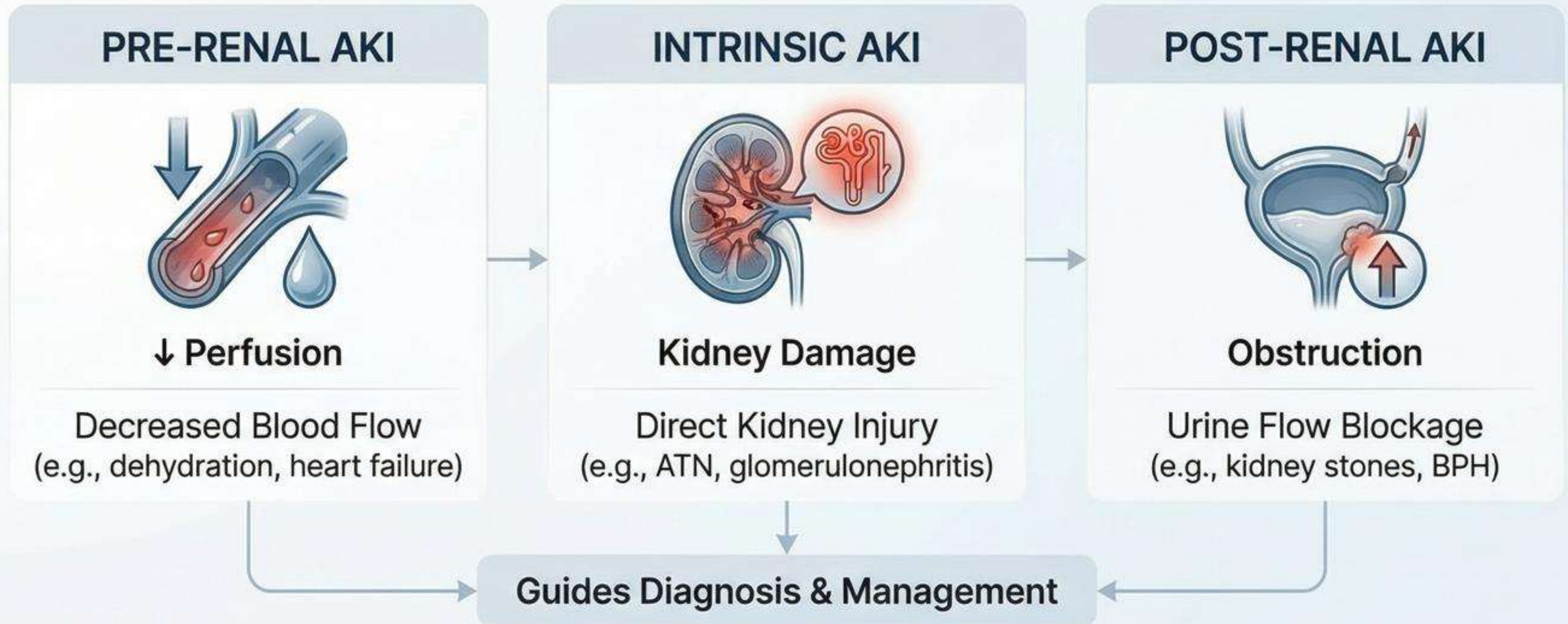
- Suggesting obstruction
- Suggesting cortical necrosis.

OLIGURIA PREDICTS WORSE PROGNOSIS

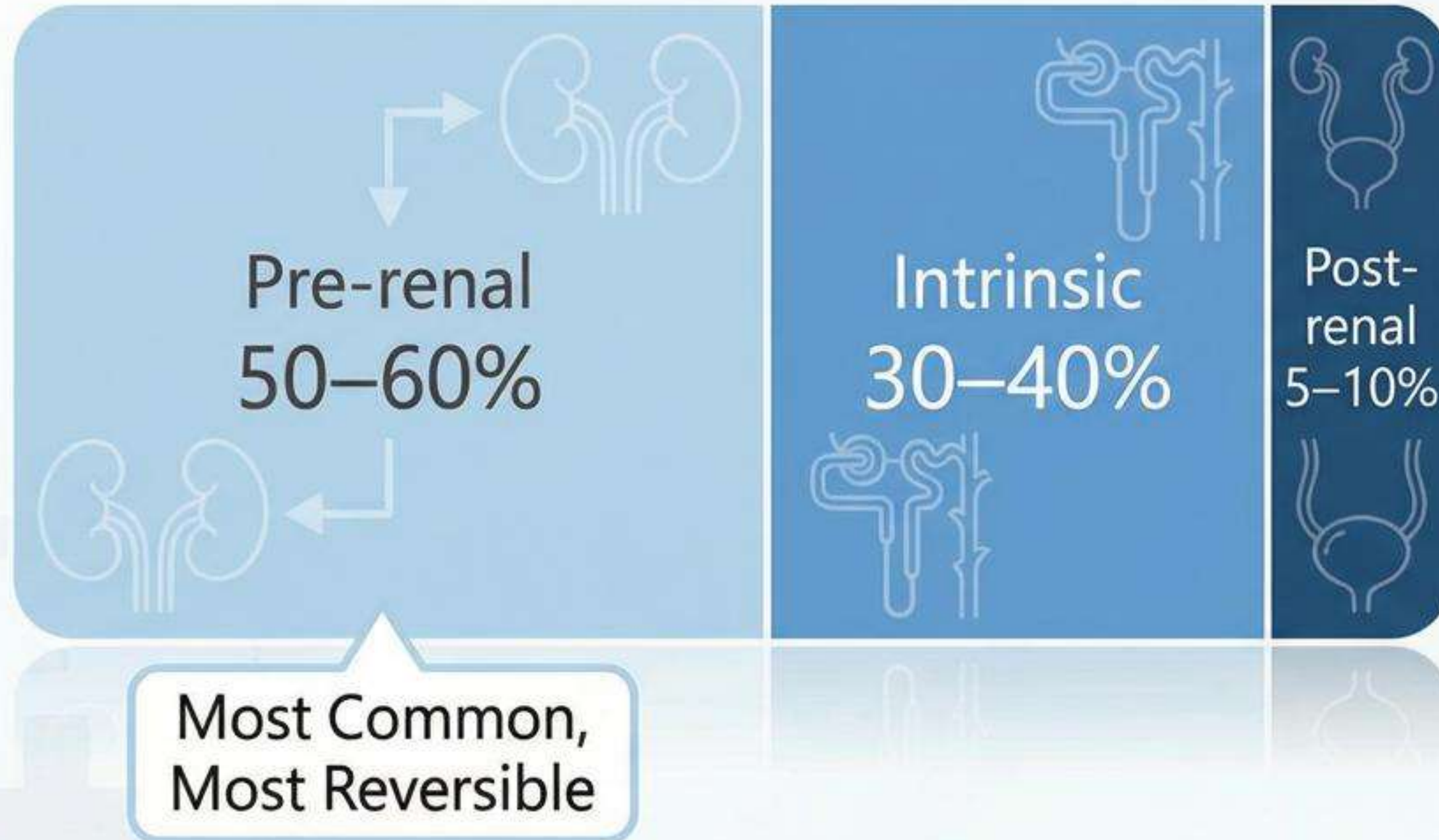
Volume status serves as an important clinical indicator.

AKI Classification: The Three Main Types

Divides AKI into pre-renal, intrinsic, and post-renal categories, guiding diagnosis and management.



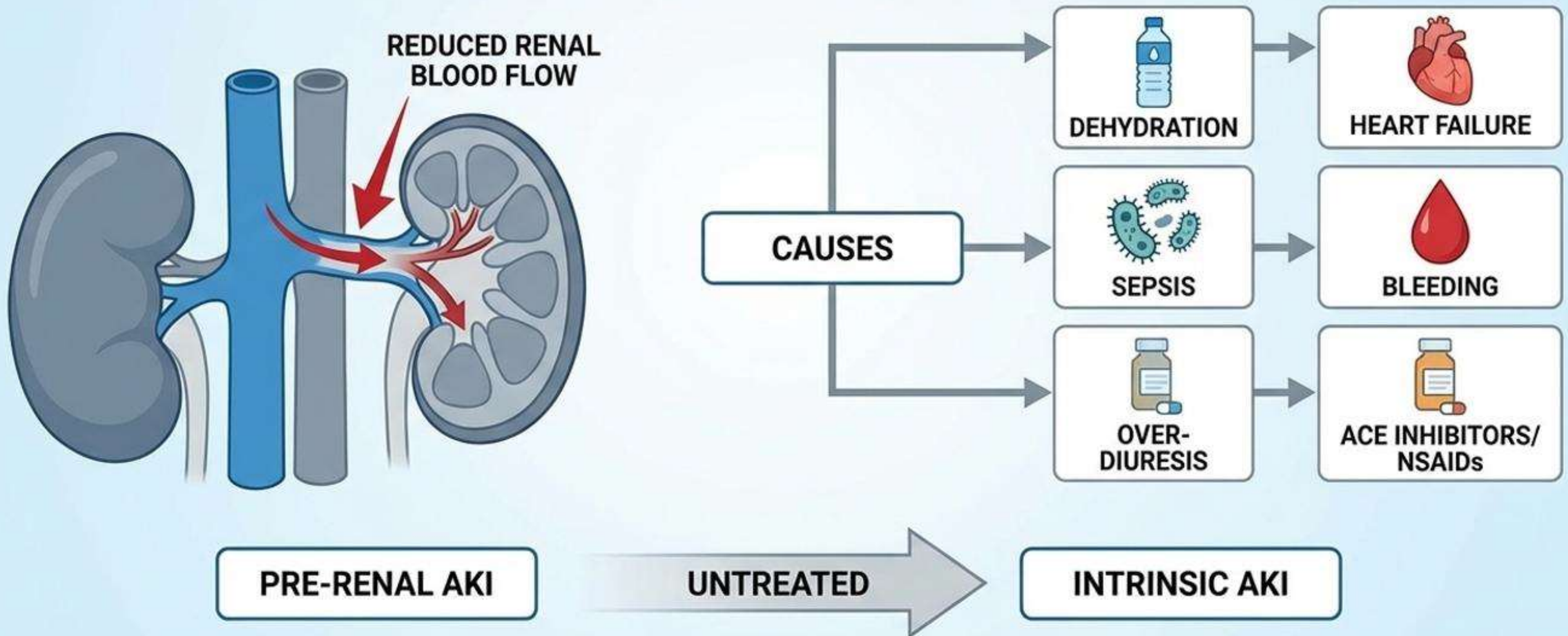
Epidemiology of Acute Kidney Injury (AKI)



- Approximate proportions: pre-renal 50–60%, intrinsic 30–40%, post-renal 5–10%.
- Pre-renal is most common and most reversible.

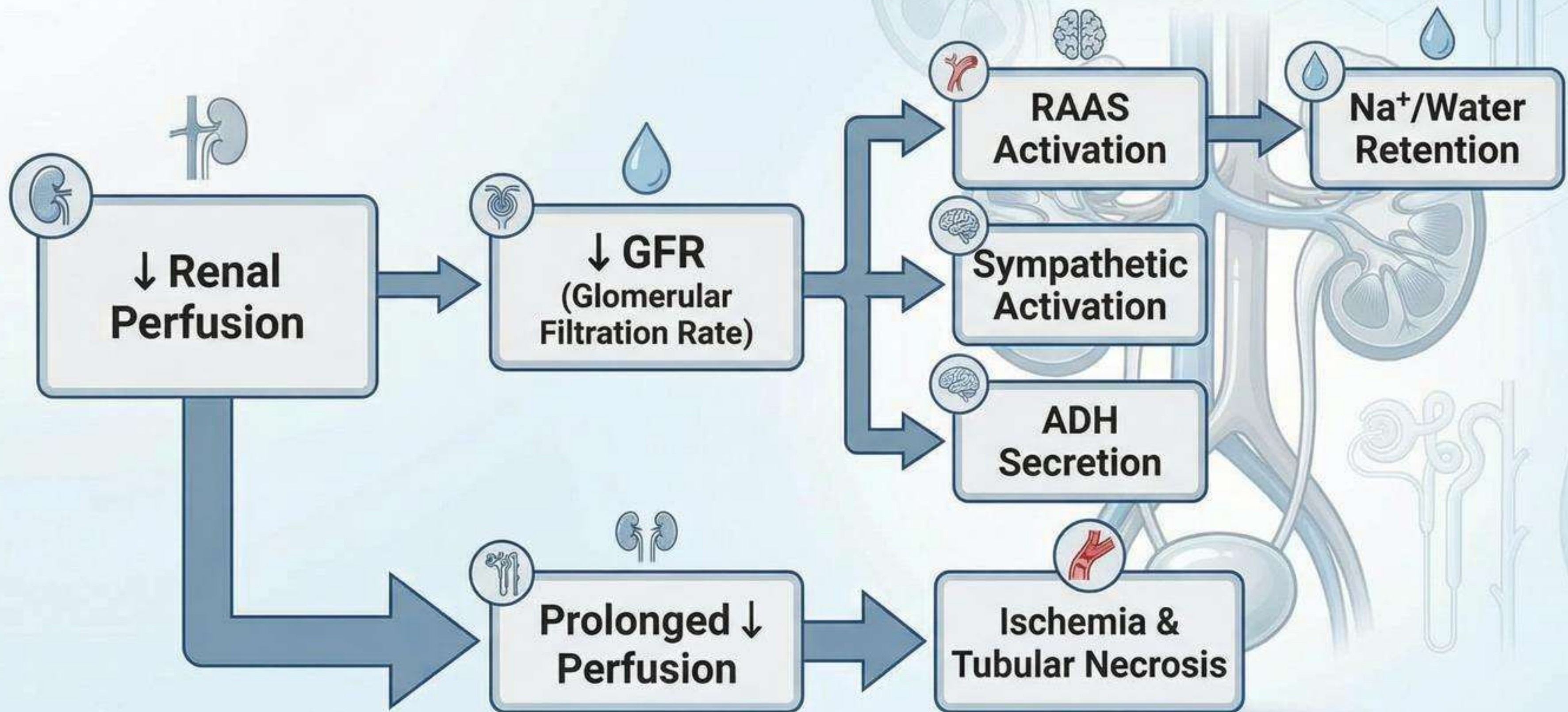
PRE-RENAL ACUTE KIDNEY INJURY

- Pre-renal AKI from reduced renal blood flow without structural damage.



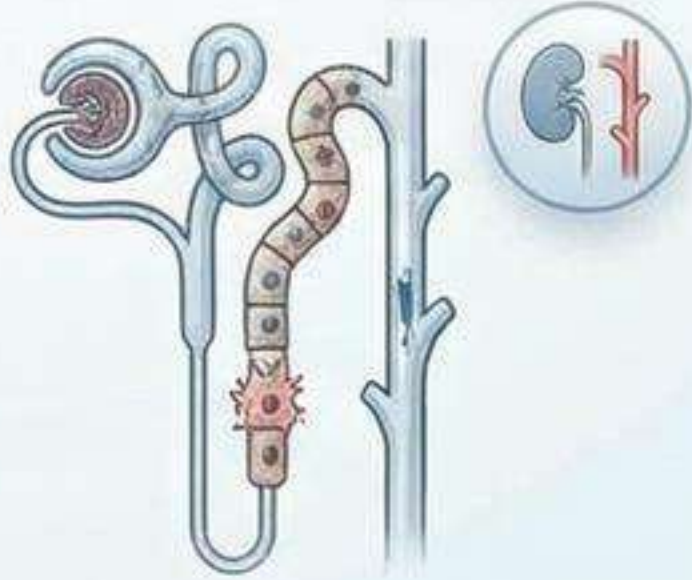
Untreated, it progresses to intrinsic AKI.

Consequences of Reduced Renal Perfusion



Structural Kidney Damage Categories

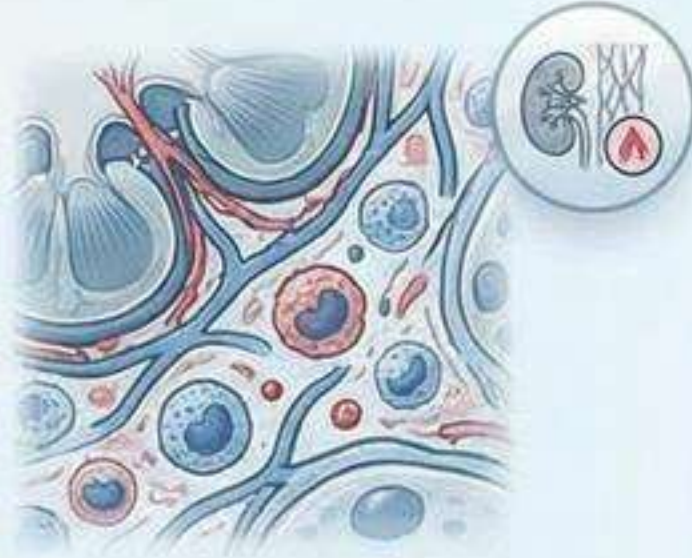
Tubular (ATN)



Acute Tubular Necrosis (ATN)

- Damage to tubular epithelial cells.
- Common causes: Ischemia, nephrotoxins.

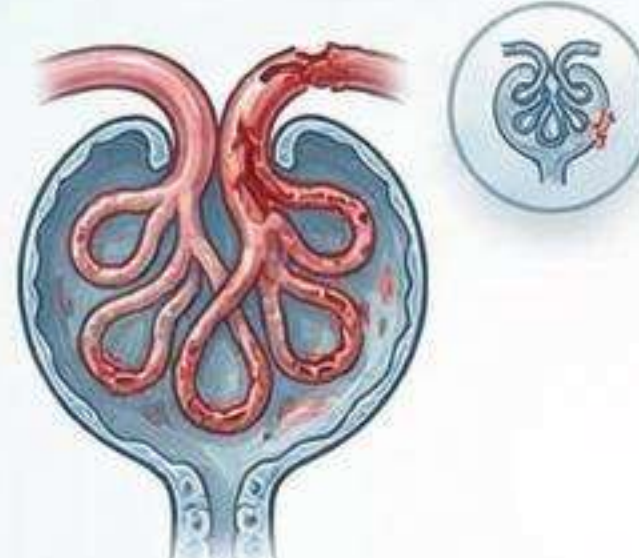
Interstitial (AIN)



Acute Interstitial Nephritis (AIN)

- Inflammation of the renal interstitium.
- Common causes: Drugs, infections.

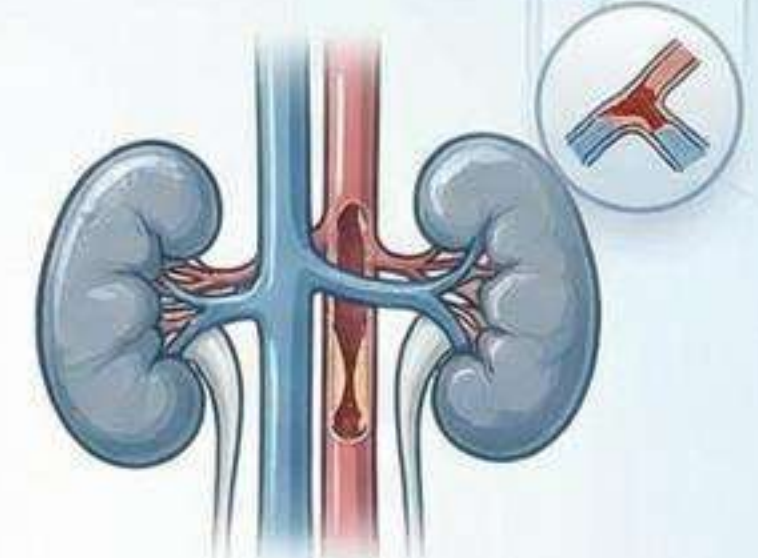
Glomerular (GN)



Glomerulonephritis (GN)

- Inflammation and damage to the glomeruli.
- Common causes: Autoimmune diseases, post-infection.

Vascular



Vascular Injury

- Damage or obstruction of renal blood vessels.
- Common causes: Vasculitis, thromboembolism.

References

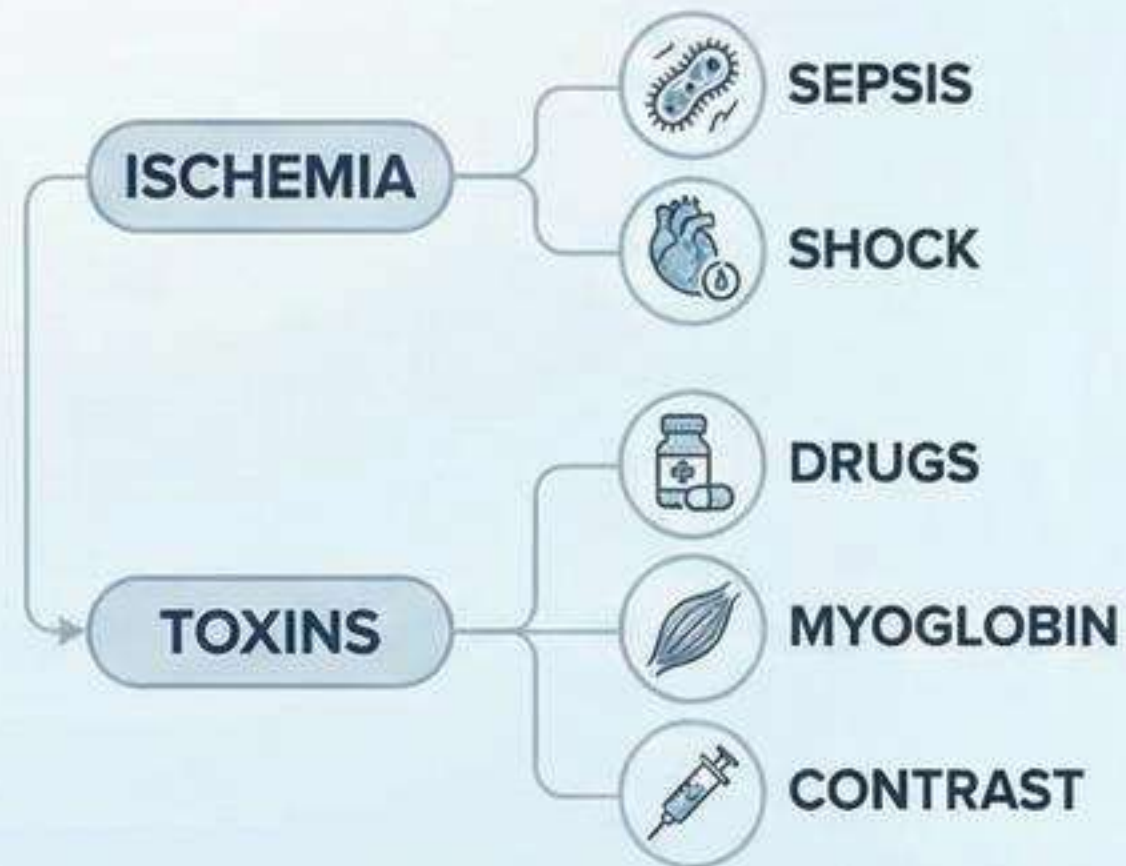
Kumar & Clark intrinsic AKI and Davidson.



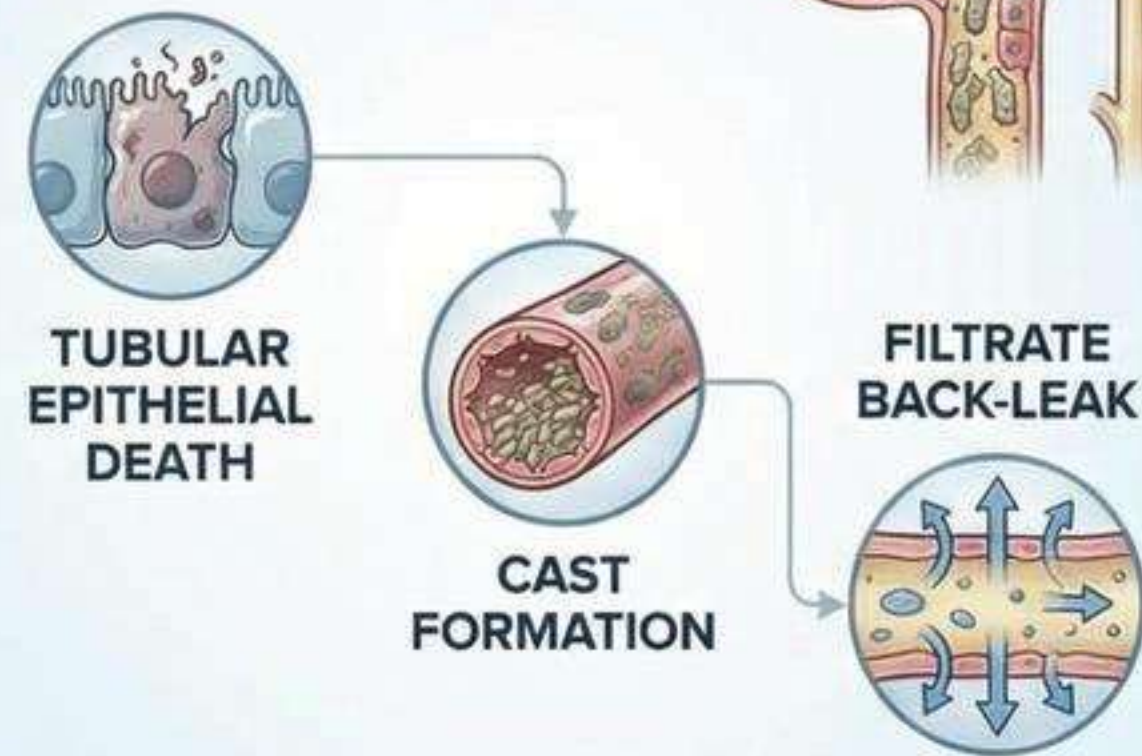
ACUTE TUBULAR NECROSIS (ATN)

Commonest Intrinsic AKI

CAUSES



PATHOLOGY

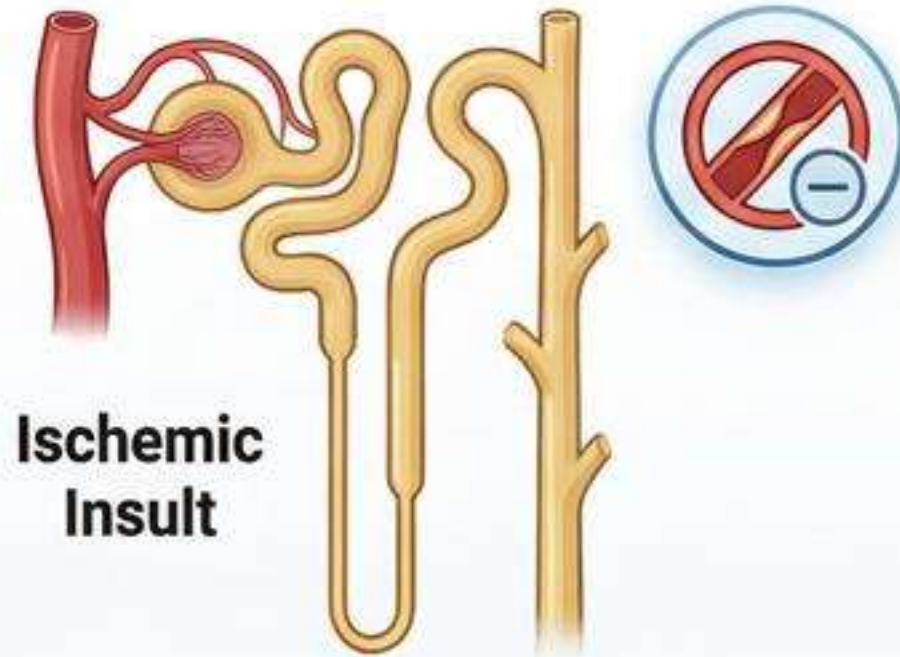


References: Davidson ATN and Kumar & Clark.

Speaker Notes: ATN is the most frequent cause of intrinsic acute kidney injury. Ischemia (from sepsis, shock) and toxins (medications, rhabdomyolysis, contrast media) are the primary insults. The key pathological processes involve the death of tubular epithelial cells, which then slough off into the lumen, forming obstructions, and the back-leak of filtrate across the damaged tubular wall, reducing GFR.

Phases of Acute Tubular Necrosis (ATN)

1. INITIATION PHASE



Ischemic Insult

- **Ischemic insult** (reduced blood flow, toxin exposure).
- Tubule cell injury begins.
- Lasts hours to days.



2. MAINTENANCE PHASE

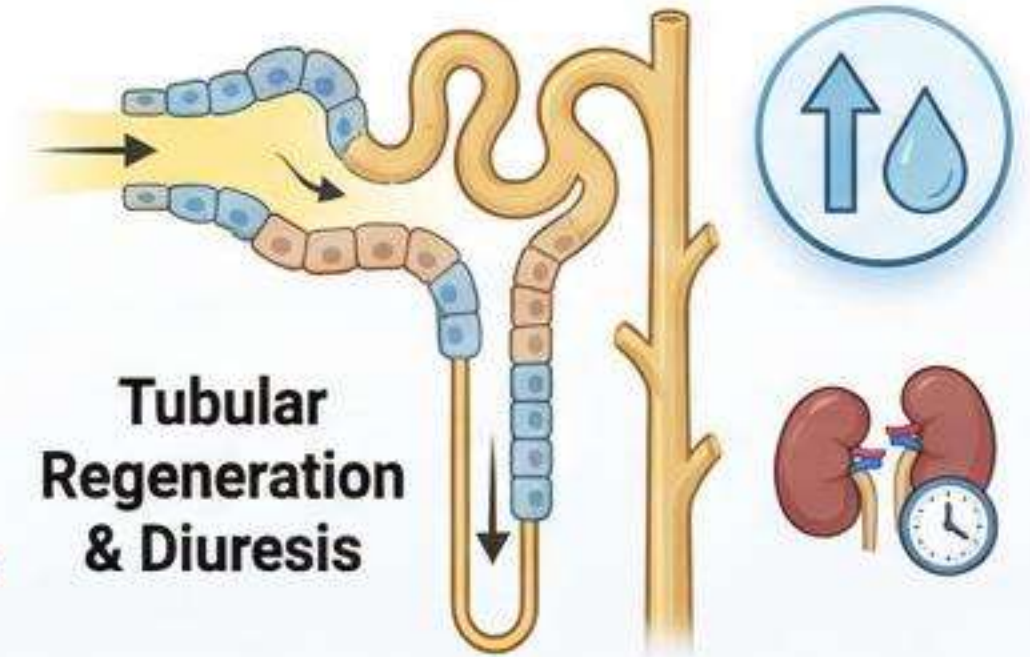


Tubular Injury & Obstruction

- **Oliguria** (decreased urine output).
- **Uremia** (buildup of waste products).
- Fluid and **electrolyte imbalances** (e.g., hyperkalemia, metabolic acidosis).
- Lasts days to weeks.



3. RECOVERY PHASE



Tubular Regeneration & Diuresis

- **Diuresis** (increased urine output).
- Gradual restoration of GFR.
- Electrolyte levels normalize.
- Lasts weeks to months.



Renal recovery may be complete or partial.



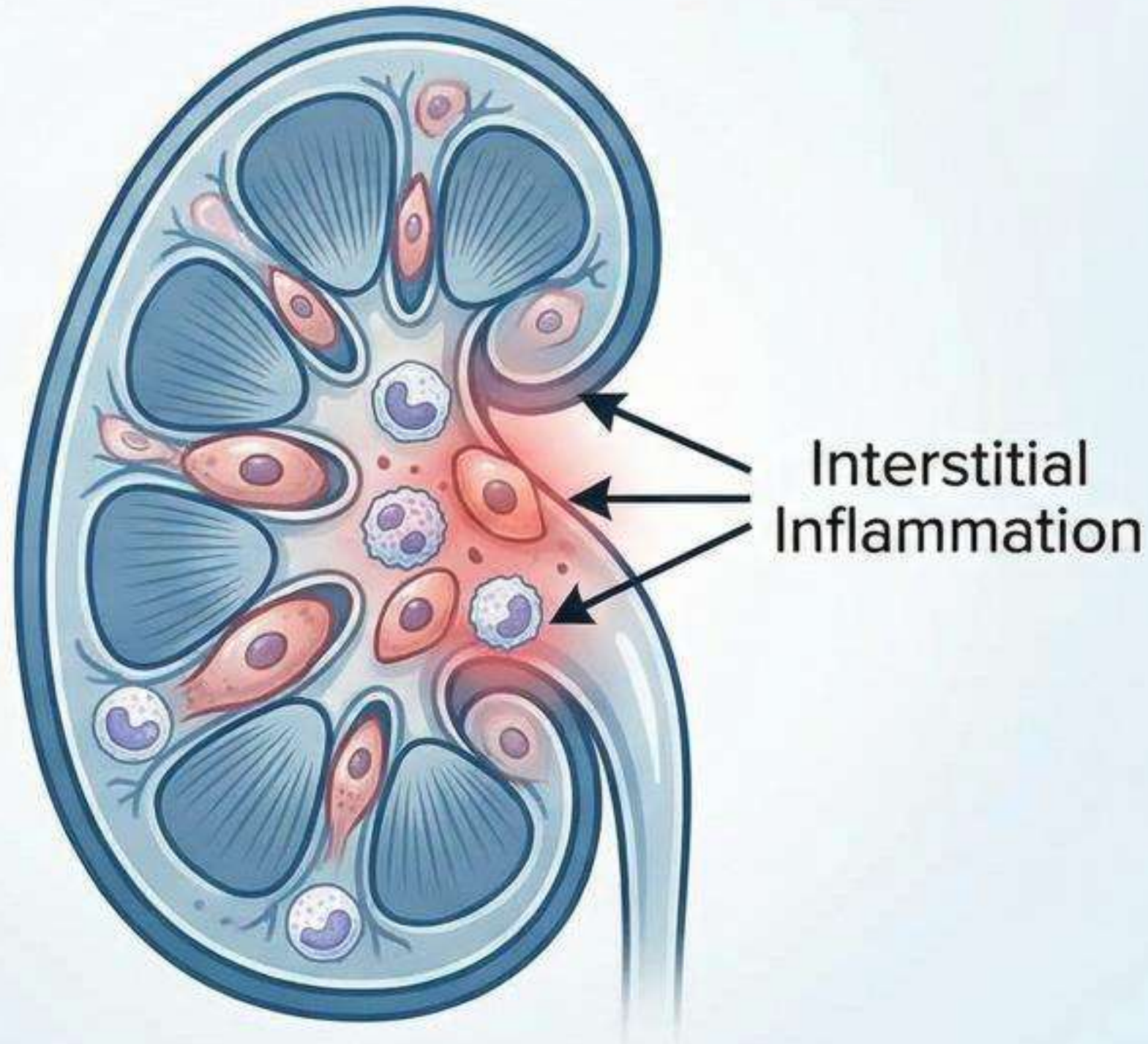
Pre-renal AKI vs Acute Tubular Necrosis

Pre-renal AKI		Acute Tubular Necrosis
<10 mmol/L	Urine sodium	>20 mmol/L
>500 mOsm/kg	Urine osmolality	<350 mOsm/kg
>1.020	Urine specific gravity	<1.010
<1%	Fractional excretion of sodium (FE _{NA})	>1%
Good	Response to fluid challenge	Poor
Usually normal Hyaline/fine granular casts	Urinalysis	Brown granular, epithelial casts Free renal tubular epithelial cells

$$FE_{NA} = (\text{urine sodium/plasma sodium}) / (\text{urine creatinine/plasma creatinine}) \times 100$$

Acute Interstitial Nephritis (AIN)

Immune-mediated renal interstitial inflammation.



Common causes:

- Drugs (NSAIDs, antibiotics, PPIs)
- Infections
- Autoimmune disease



Classic triad:

– Fever



– Rash



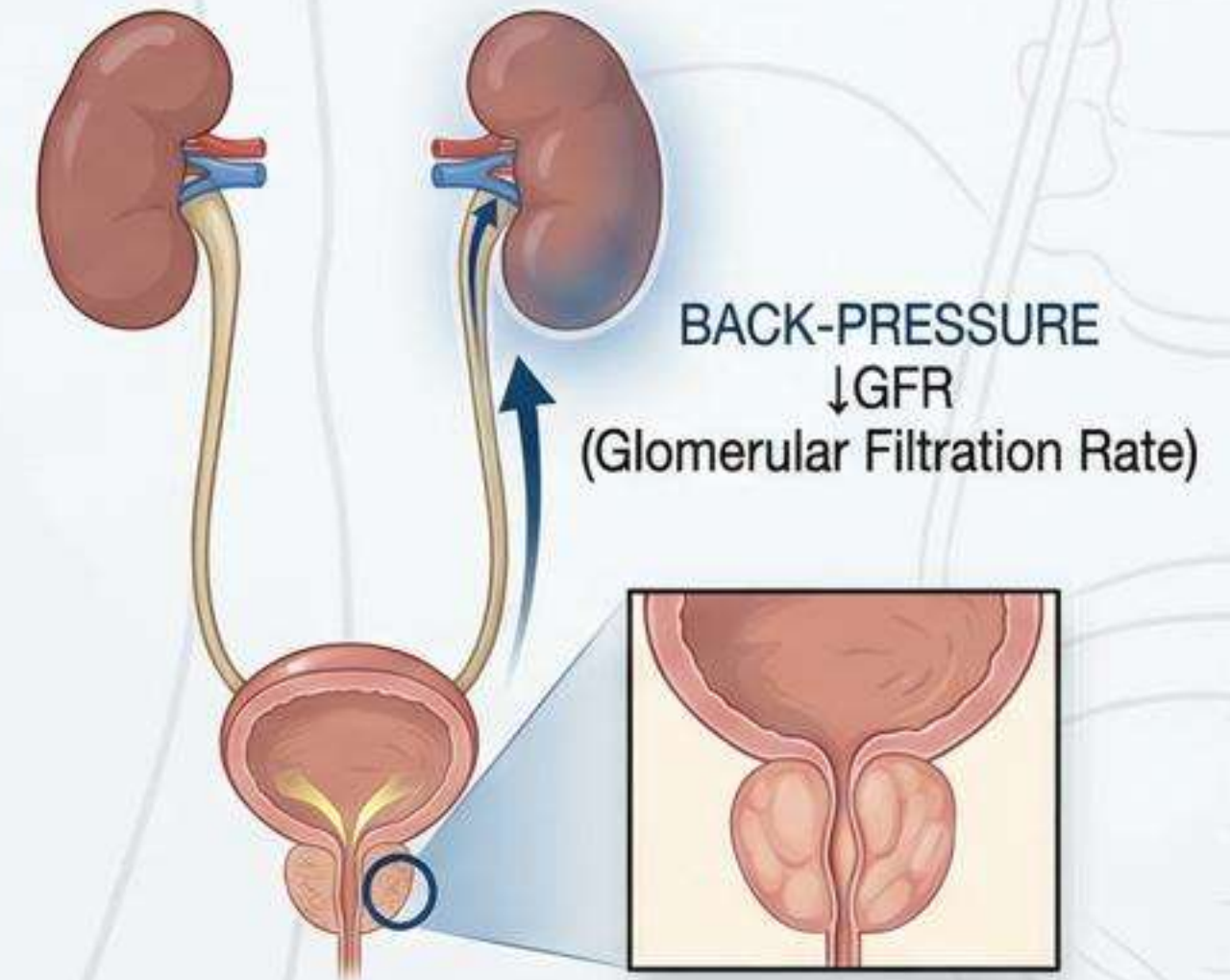
– Eosinophilia (often absent)

Post-renal AKI (Obstruction)

Urinary Flow Obstruction Causing Back-Pressure and ↓GFR

- ✓ Causes:
 - ✓ BPH (Benign Prostatic Hyperplasia)
 - ✓ Kidney Stones (Calculi)
 - ✓ Tumors (Bladder, Ureters, Prostate)
 - ✓ Strictures
 - ✓ Blood Clots

IMPORTANT: Bilateral obstruction is typically required to cause significant AKI.



Obstruction → Increased Tubular Pressure → Reduced GFR → AKI

CLINICAL FEATURES OF ACUTE KIDNEY INJURY (AKI)

Symptoms vary by cause and severity:



Oliguria

(Decreased urine output)



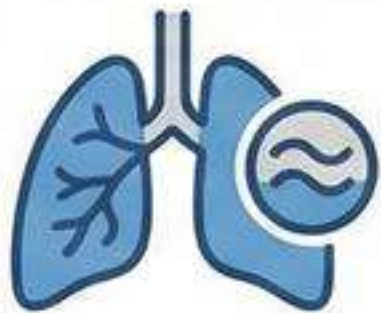
Edema

(Fluid retention)



Fatigue

(General weakness)



Shortness of Breath

(Fluid overload)



Confusion

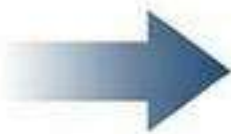
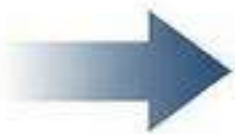
(Uremic encephalopathy)



Nausea

(Uremic toxins)

Post-renal cases may present with:



Urinary Retention

(Inability to empty bladder)



Pain

(Flank, pelvic)

AKI Diagnostic Investigations & Findings



Key tests:



serum urea/creatinine



potassium



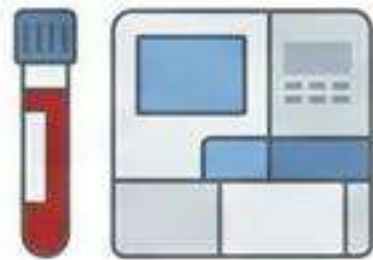
bicarbonate



calcium/phosphate



ABG



Common findings:



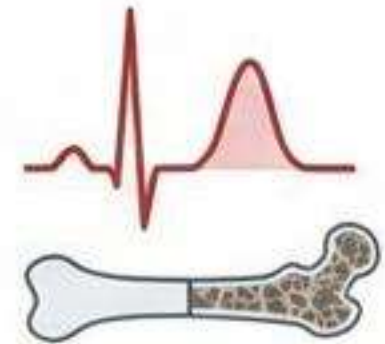
hyperkalemia



metabolic acidosis



hypocalcemia



References: Kumar & Clark AKI investigations and Davidson.

BUN:Creatinine Ratio in AKI Differential Diagnosis

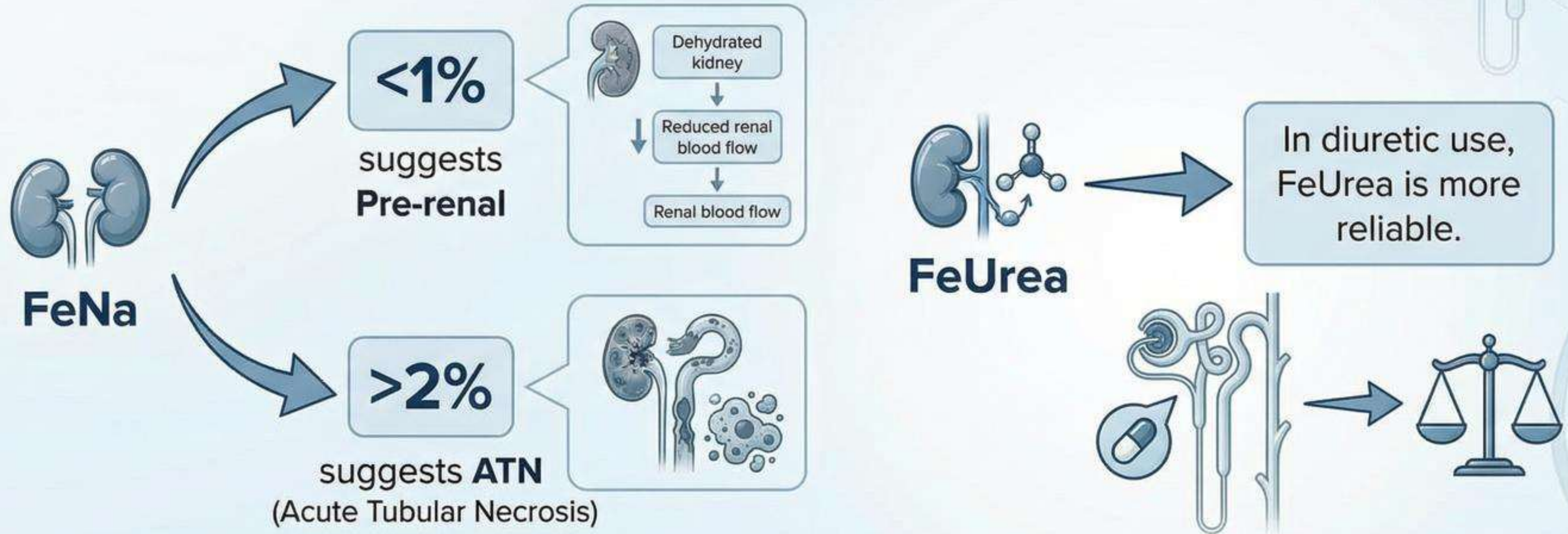
PRE-RENAL AKI	ACUTE TUBULAR NECROSIS (ATN)
 BUN:Creatinine Ratio >20:1	 Normal BUN:Creatinine Ratio
 Suggests pre-renal cause (e.g., Dehydration)	 Seen in ATN (Intrinsic cause)
• Urea rises disproportionately due to increased reabsorption in dehydration	• Urea and Creatinine rise proportionately

References:

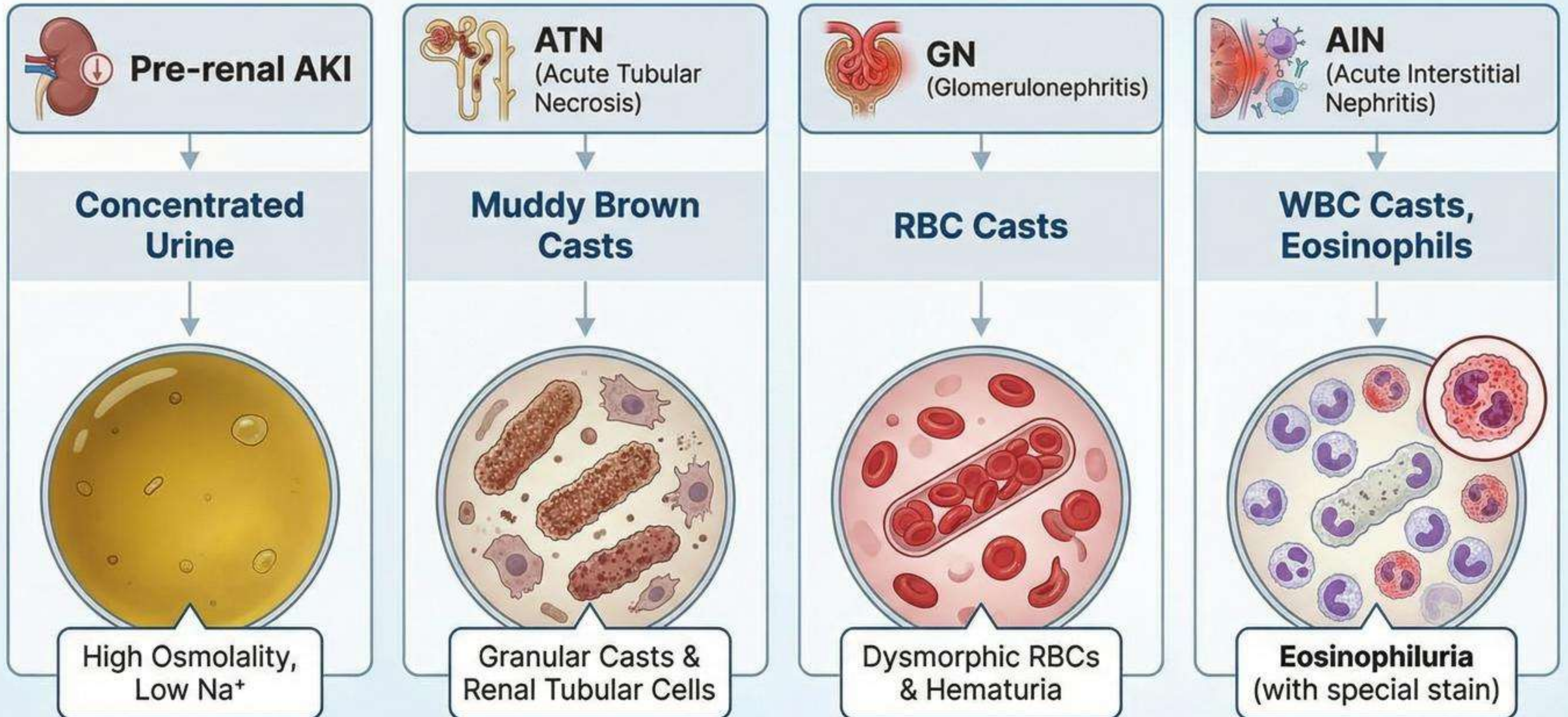
Davidson lab interpretation, Kumar & Clark



Fractional Excretion of Sodium (FeNa) & Urea (FeUrea) in AKI Diagnosis



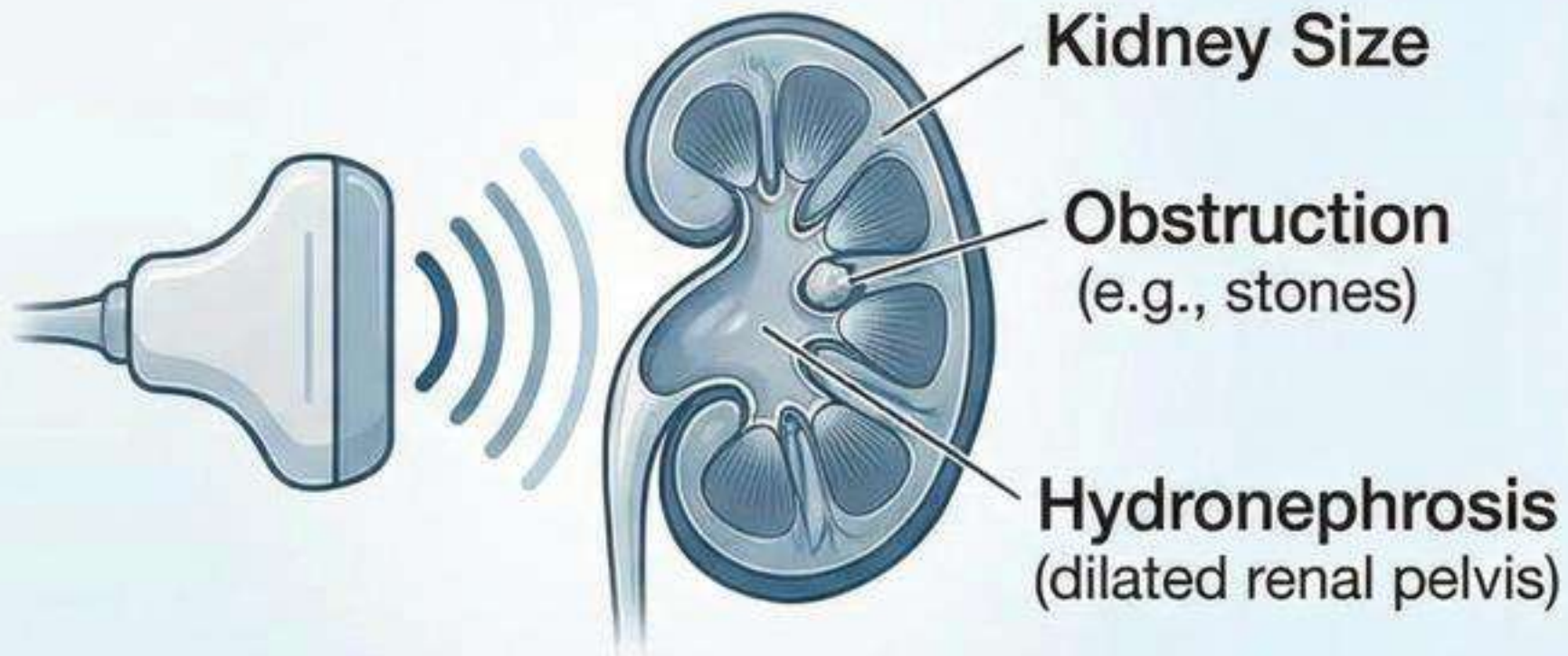
Urine Findings by Condition



IMAGING IN ACUTE KIDNEY INJURY



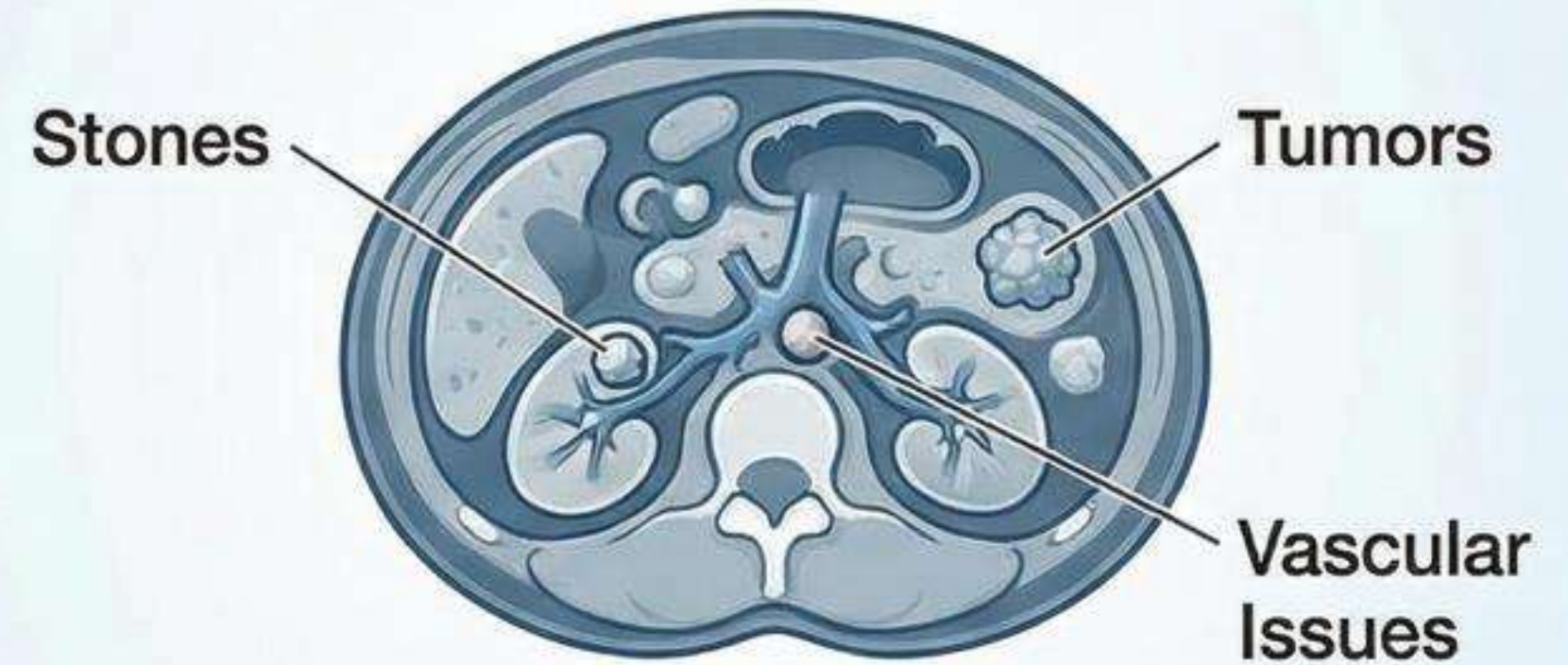
RENAL ULTRASOUND



- Assesses kidney size, obstruction, hydronephrosis.



COMPUTED TOMOGRAPHY (CT)



- Used if stones or tumors suspected.



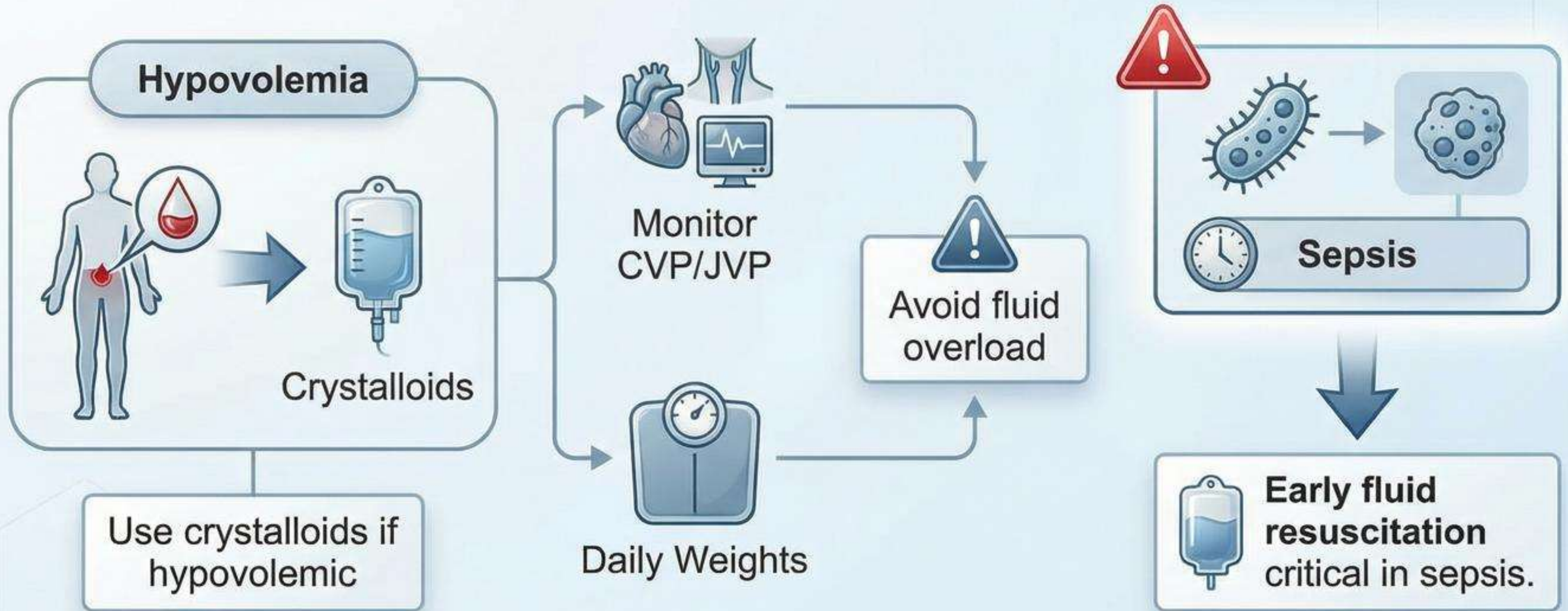
References: Davidson Imaging and Kumar & Clark.

AKI MANAGEMENT

Six Principles



Management: Fluid Balance Strategy



References: Kumar & Clark and Davidson.



Emergency Treatment: Addressing Life-Threatening Complications (e.g., Severe Hyperkalemia) in AKI

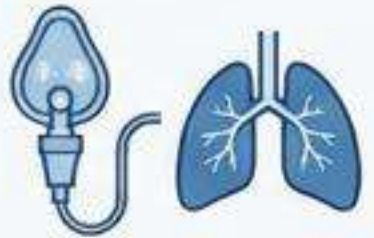
Initial Stabilization (Cardioprotection & Cellular Shift)



- **IV Calcium Gluconate:** Stabilizes cardiac membrane (does not lower K^+).

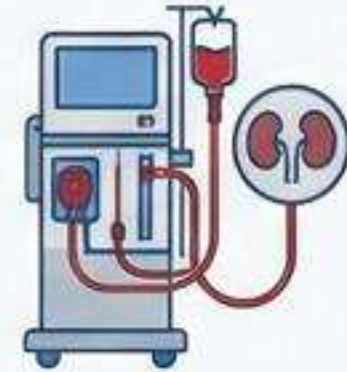


- **Insulin + Glucose:** Promotes intracellular K^+ shift.



- **Salbutamol (Nebulized/IV):** Promotes intracellular K^+ shift (beta-2 agonist).

Refractory Cases (Persistent/Severe Hyperkalemia)



- **Dialysis:** Definitive removal for refractory or life-threatening cases.

References: Davidson's Principles and Practice of Medicine (Emergencies); Kumar and Clark's Clinical Medicine.

Speaker Notes: This slide focuses on the immediate, emergency management of life-threatening hyperkalemia, a common and critical complication of AKI. Emphasize that IV calcium is for cardiac protection, while insulin/glucose and salbutamol shift potassium into cells. Dialysis is the definitive treatment for potassium removal when other measures fail or in severe cases.

MANAGEMENT OF SEVERE METABOLIC ACIDOSIS IN AKI

TREATMENT PROTOCOL



Severe metabolic acidosis: Use sodium bicarbonate.

↳ **Refractory cases:** Dialysis.



**SEVERE
METABOLIC
ACIDOSIS
(pH < 7.1)**



**SODIUM
BICARBONATE
INFUSION**



REFRACTORY?

YES

NO



DIALYSIS



**MONITOR &
REASSESS**

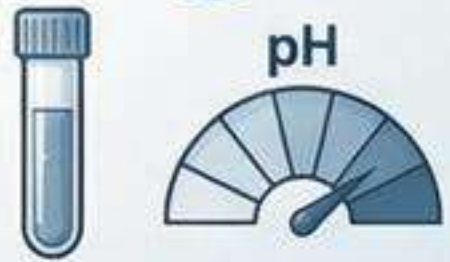


REFERENCES: Kumar & Clark and Davidson.

Mnemonic: AEIOU (Indications for Dialysis)

References: Davidson's Principles and Practice of Medicine, Kumar & Clark's Clinical Medicine.

A



Acidosis:

- Metabolic acidosis (e.g., pH < 7.1) refractory to medical management.

E



Electrolytes:

- Severe, persistent hyperkalemia (e.g., K⁺ > 6.5 mmol/L) or other significant imbalances.

I



Intoxication:

- Dialyzable toxins (e.g., salicylates, lithium, methanol, ethylene glycol).

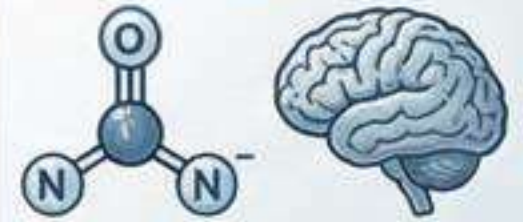
O



Overload:

- Volume overload refractory to diuretics, causing pulmonary edema or respiratory distress.

U



Uremia:

- Signs of severe uremia (e.g., pericarditis, encephalopathy, neuropathy).



Consider Dialysis



COMMON NEPHROTOXINS

Drug Classes & Examples

• NSAIDs



• Aminoglycosides



• Amphotericin B



• Cisplatin



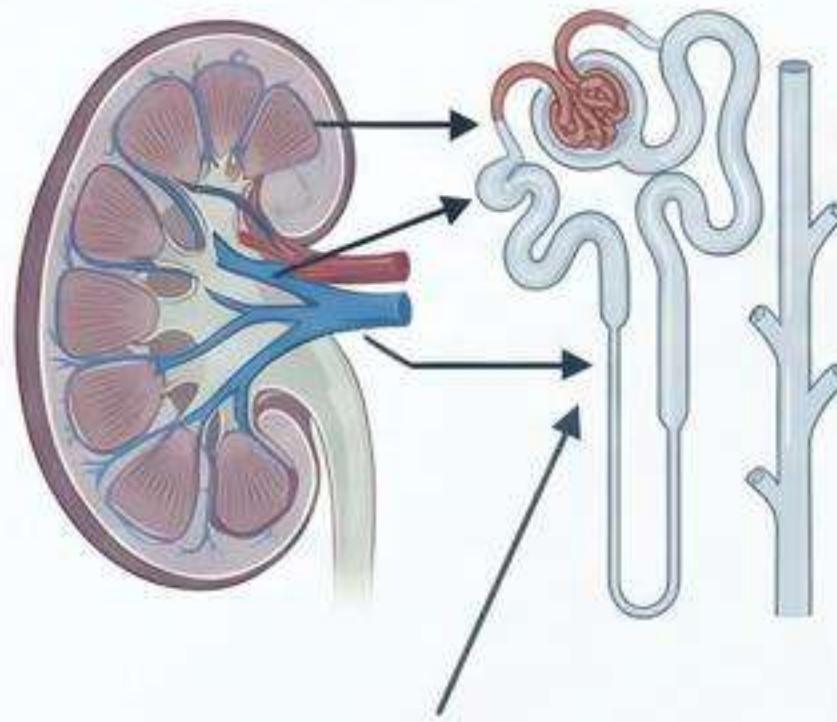
• Contrast Agents



• ACEi



Mechanisms of Injury



Mechanisms of Injury
Tubular Necrosis,
Interstitial Nephritis,
Hemodynamic Changes



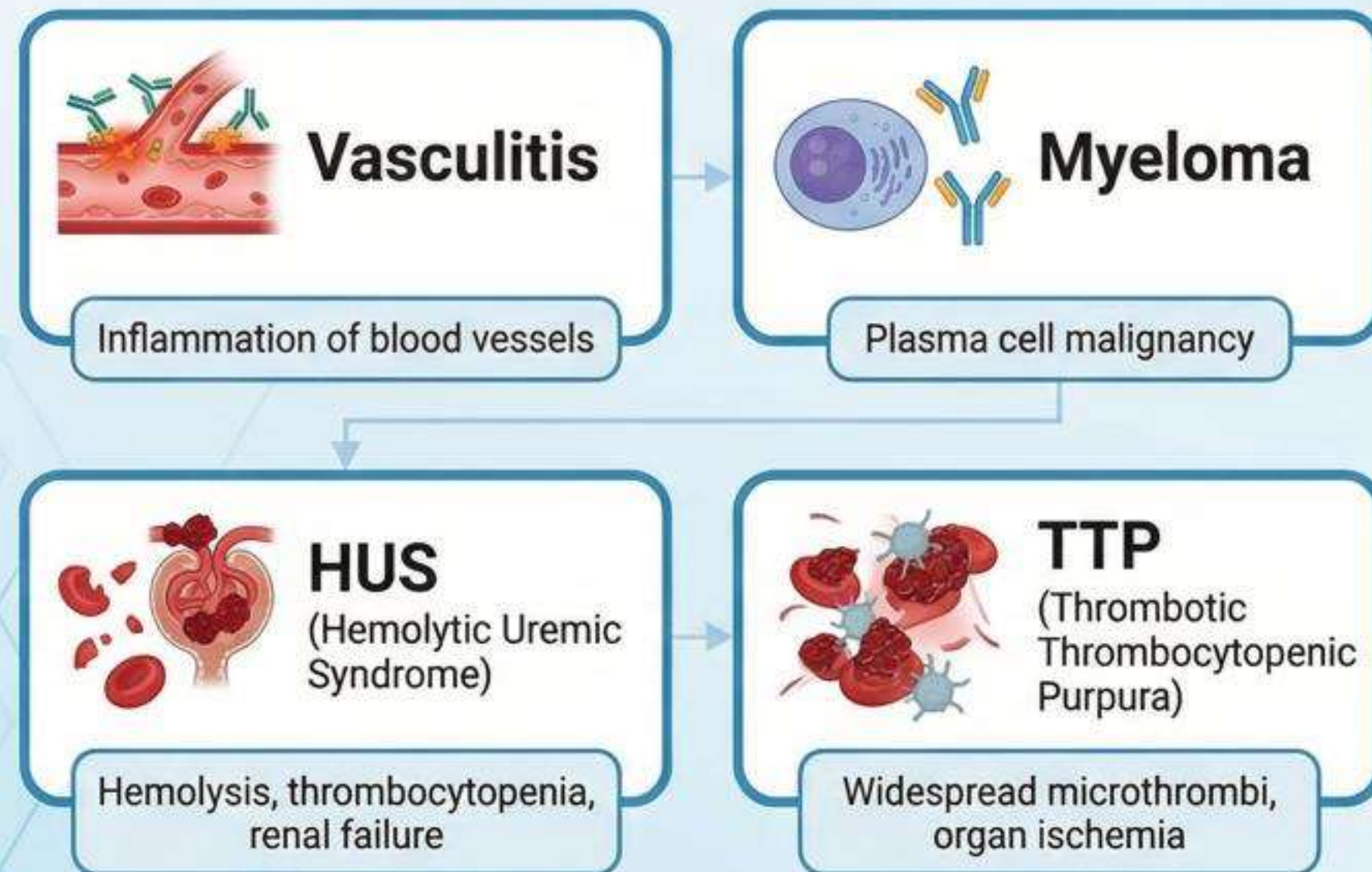
Always review
medications

Key Action: Comprehensive
medication review is crucial
in AKI management.

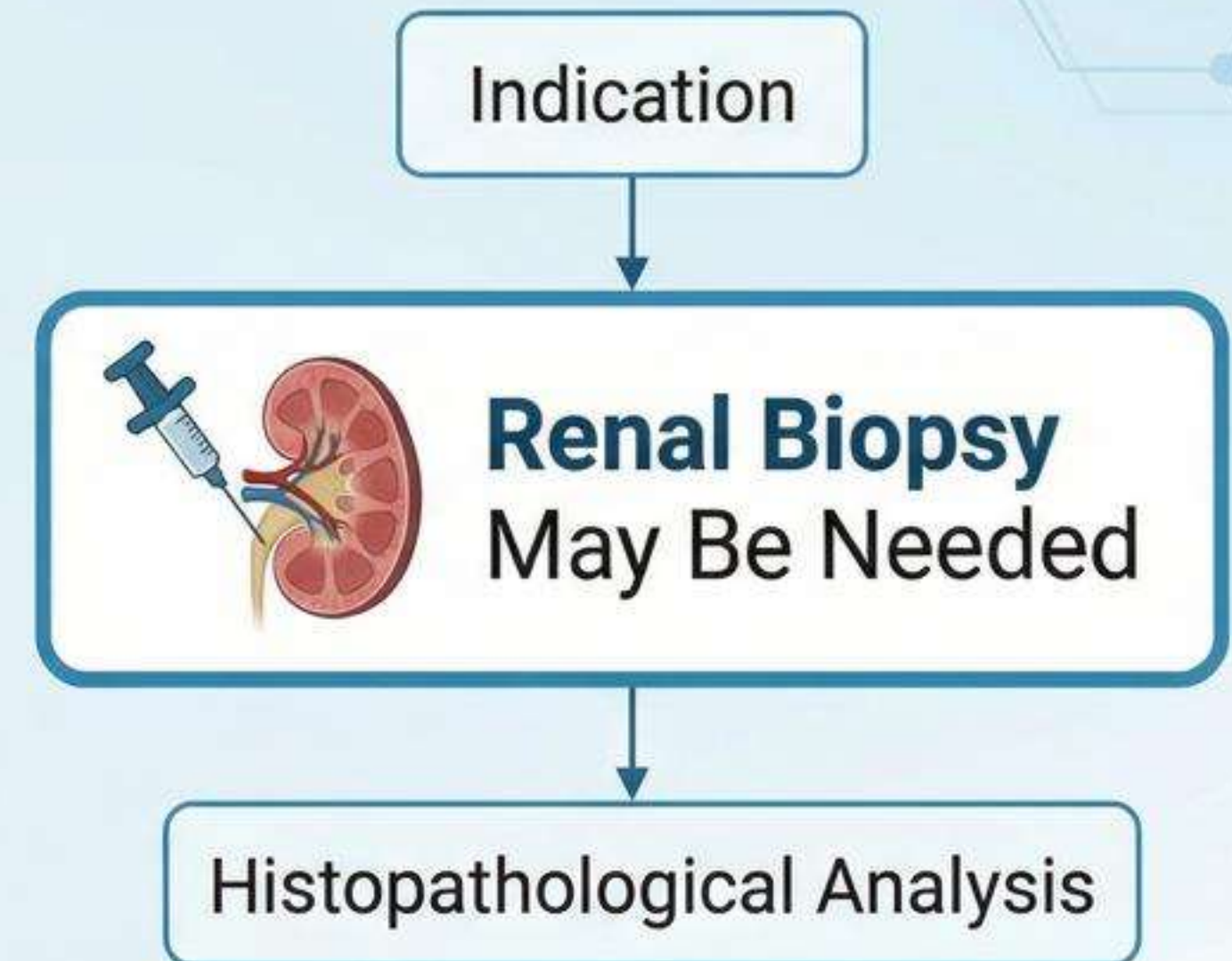
Consider Vasculitis, Myeloma, HUS, TTP

Renal biopsy may be needed

Differential Diagnoses to Consider



Diagnostic Procedure



References:

Kumar & Clark's Clinical Medicine

Davidson's Principles and Practice of Medicine

Acute Kidney Injury (AKI) - Prognosis

Good Prognosis



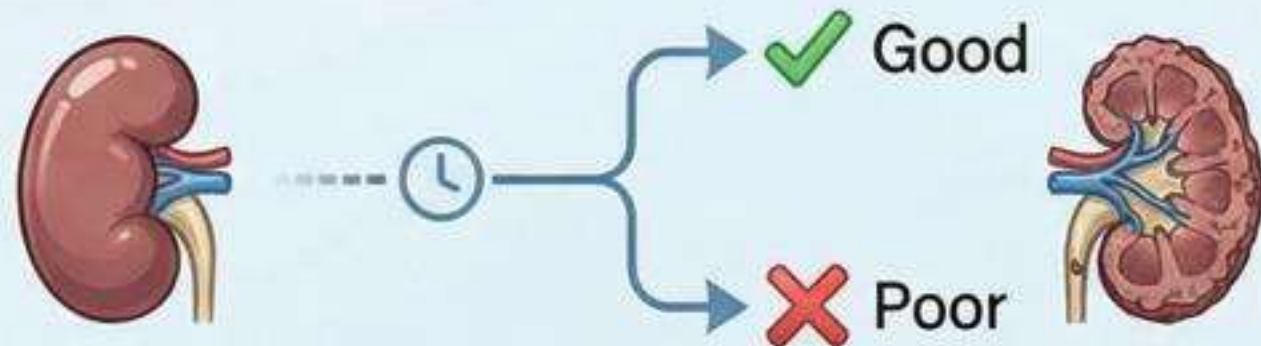
early diagnosis



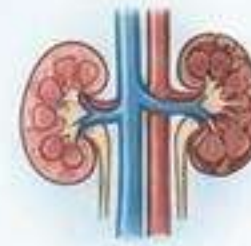
pre-renal cause



no comorbidities



Poor Prognosis



ATN (Acute Tubular Necrosis)



sepsis



elderly



dialysis requirement

Major Complications



Arrhythmias

Potentially life-threatening heart rhythm disturbances.

- Electrolyte imbalances (e.g., hyperkalemia)
- Cardiac conduction issues



Pulmonary Edema

Accumulation of fluid in the lungs, causing respiratory distress.

- Fluid overload
- Impaired gas exchange



Encephalopathy

Brain dysfunction due to toxin buildup.

- Uremic toxins accumulation
- Altered mental status
- Seizures



Infection

Increased susceptibility to bacterial and viral infections.

- Immune suppression
- Catheter-related infections
- Pneumonia



GI Bleeding

Bleeding in the gastrointestinal tract.

- Uremic gastropathy
- Coagulopathy