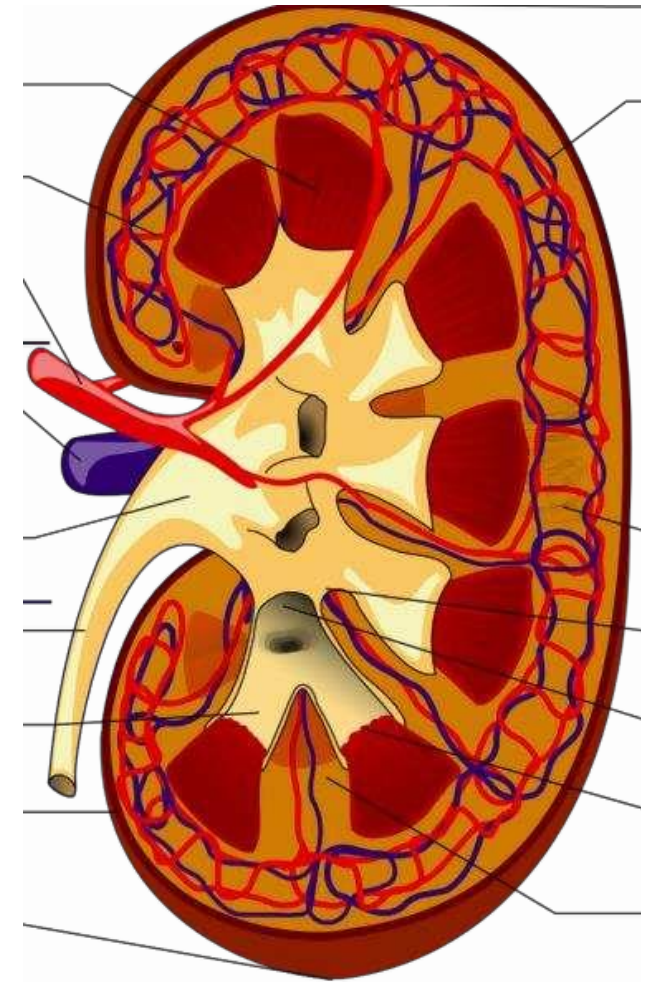


Acute Renal Failure

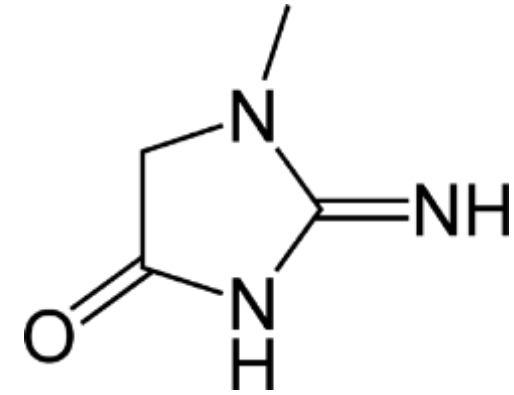
Terms

- **Acute renal failure(ARF)/Acute kidney injury (AKI)**
 - Abrupt decrease in renal clearance over days
 - Sometimes associated with symptoms
 - Many causes
 - Most cases resolve
- Chronic kidney disease/Chronic renal failure
 - Slow, steady deterioration of renal function (years)
 - Usually due to diabetes, hypertension
 - Symptoms only in most severe stages
 - Usually irreversible

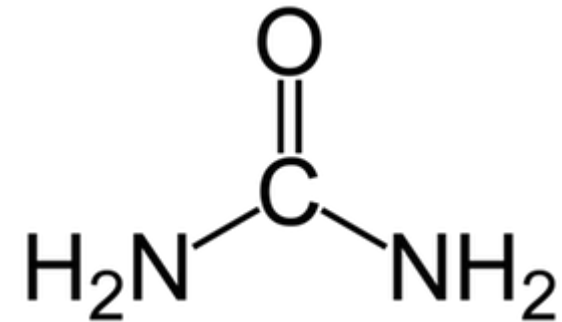


Terms

- Azotemia
 - Elevated BUN and Cr
 - Indicates insufficient filtering of blood by kidneys
- Uremia
 - Azotemia + “uremic” symptoms



Creatinine



Urea

Uremic Symptoms

- Anorexia
- Nausea, vomiting
- Platelet dysfunction (bleeding)
- Pericarditis
- Asterixis
- Encephalopathy



Wikipedia/Public Domain

AKI Clinical Definitions

- **Acute kidney injury**
 - Increase in Cr of ≥ 0.3 over 48 hours
 - Increase ≥ 1.5 x the baseline within past 7 days
 - Oliguria Urine output < 0.5 ml/kg/hr
- Types of AKI
 - Oliguric
 - Non-oliguric
 - Anuric

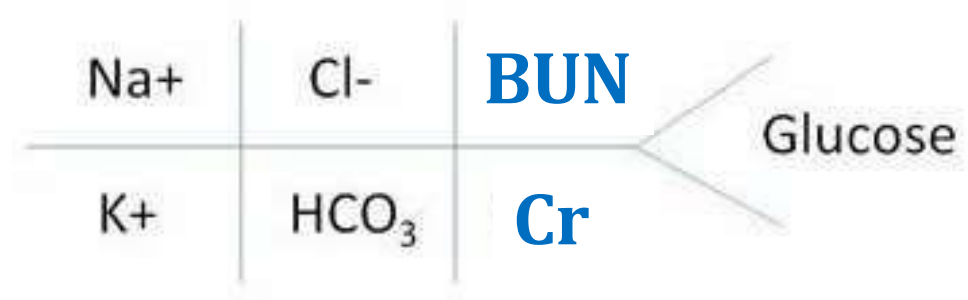
Key Labs

- **Creatinine**

- Freely filtered
- Small amount of secretion

- **Blood urea nitrogen**

- Freely filtered
- Reabsorbed when kidney reabsorbs water
- In acute renal failure both rise (less filtered)
- If kidneys under-perfused: BUN rises more (less filtered, more reabsorbed)



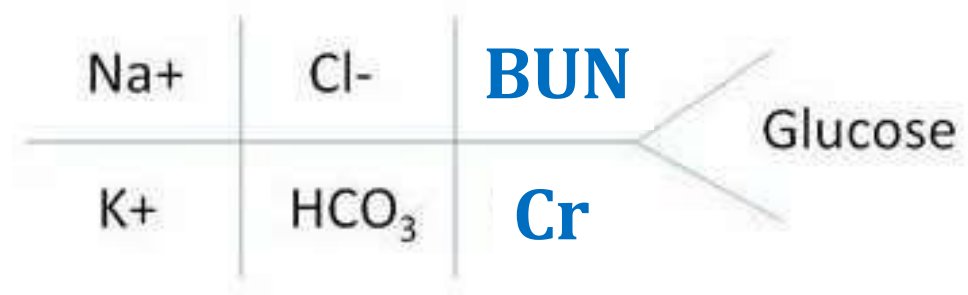
Acute Renal Failure

- **Pre-renal failure:** 60 to 70% cases
 - Insufficient blood flow to kidneys
 - Volume depletion, shock, heart failure
- **Post-renal failure:** 5 to 10% cases
 - Obstruction of urine outflow
 - Need bilateral obstruction
 - Kidney stones, BPH, tumors, congenital anomalies
- **Intrinsic renal failure:** 25 to 40% cases
 - Acute tubular necrosis
 - Glomerulonephritis

Pre-Renal Failure

Serum BUN/Cr

- Decreased blood flow to kidneys
- Less BUN/Cr filtered
- Rising BUN/Cr in blood
- More resorption H₂O
- BUN resorbed with H₂O
- BUN rises >> Cr rises
- Result
 - ↑ Cr
 - ↑↑ BUN
 - ↑ BUN/Cr ratio



Renal Measurements

- **Urinary sodium (U_{Na})**
 - Varies based on intake of sodium and water
 - Low when retaining salt/water
 - < 20 mEq/L is low
- **Fractional excretion of Na (Fe_{Na})**
 - Amount of filtered Na that is excreted
 - Low when kidney retaining salt/water
 - $< 1\%$ is low
- **Urinary osmolarity (U_{osm})**
 - Measure of concentrating ability of kidney
 - High when kidney retaining water
 - > 500 mOsm/kg is high

$$Fe_{Na} = \frac{P_{Cr} * U_{Na}}{P_{Na} * U_{Cr}}$$

****Na measurements not useful
in patients on diuretics**

Pre-Renal Failure

Urinary Findings

- Lots of H₂O and Na resorbed
- Concentrated urine
 - ↑U_{osm}
- Low sodium excretion
 - ↓U_{Na}
 - ↓Fe_{Na}
- Bland sediment
 - No protein
 - No cells
 - Rarely hyaline casts



Wikipedia/Public Domain

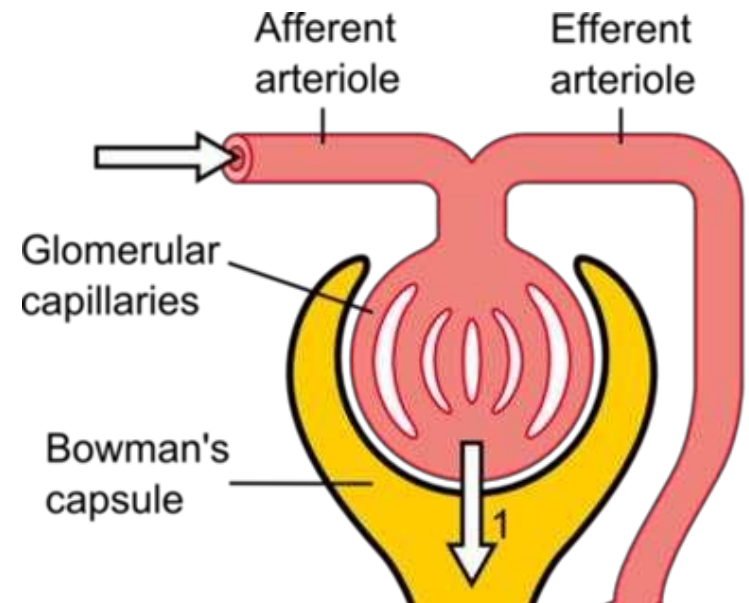
Pre-Renal Failure

	Normal	Pre-Renal Failure
BUN (mg/dl)	20	60
Cr (mg/dl)	1.0	2.0
BUN:Cr	20:1	> 20:1
UNa (mEq/L)	variable	< 20
FeNa (%)	variable	< 1
Uosm (mOsm/kg)	variable	> 500

Pre-Renal Failure

Selected Causes

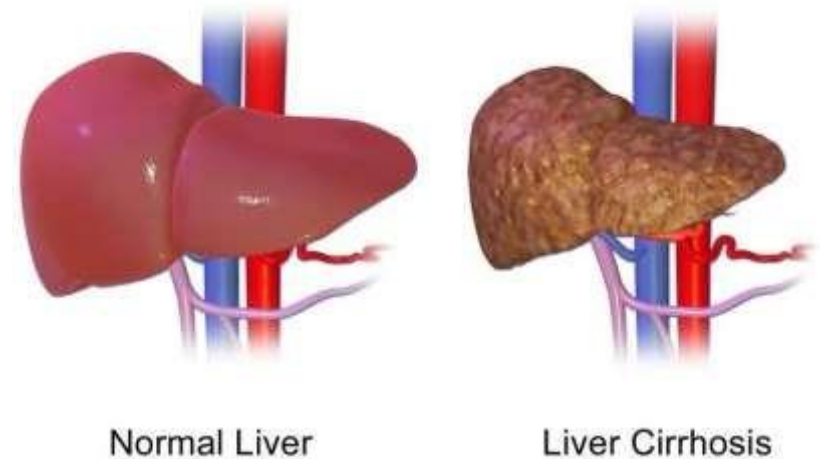
- Hypotension
 - Sepsis, bleeding, anaphylaxis
- Hypovolemia
 - Diuretics, burns
- Renal artery stenosis
- NSAIDs
 - Afferent arteriolar vasoconstriction
- ACE inhibitors
 - Efferent arteriolar vasodilation



Madhero88/Wikipedia

Cardiorenal and Hepatorenal Syndromes

- Occurs with heart failure or cirrhosis
- Renal failure with some pre-renal features
 - Often low urinary sodium
- Normal/bland urinalysis
 - No protein
 - No/few red or white cells
- **Does not respond to fluid administration**



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Post-Renal Failure

a small minority of cases(5-10 %). Post renal azotemia is caused by any decrease in the outflow of the urine . This may come by obstruction of any part of the renal collection systems (renal pelvises to urethra) because that the urine backs up to the kidney and makes high pressure in tubules which leads to a defect in filtering blood in kidney also kidney's resorptive mechanisms damaged/destroyed.

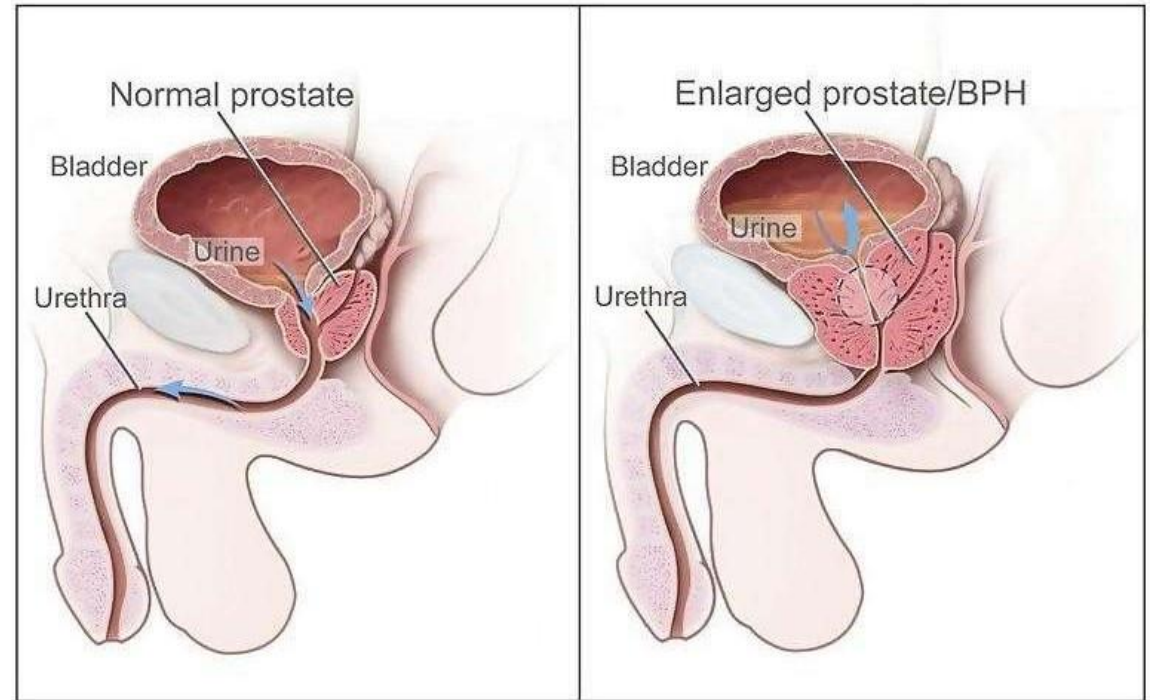
Post-Renal Failure

- Obstruction to outflow
- Urine backs up
- High pressure in tubules
- Kidney cannot filter blood
- Kidney's resorptive mechanisms damaged/destroyed

Post-Renal Failure

Selected Causes

- Prostate enlargement
- Prostate cancer
- Ureteral stones
- Neurogenic bladder
- Medications



National Cancer Institute

Clinical presentations

(key clinical feature : anuria) Clinical Presentation: Patients may experience a distended bladder in prostatism or neuro-logic disorders. Urine output may diminish or cease, preceded by incomplete voiding in prostate or bladder diseases. Patients may have pain over the bladder (prostatism) or flanks (stones)

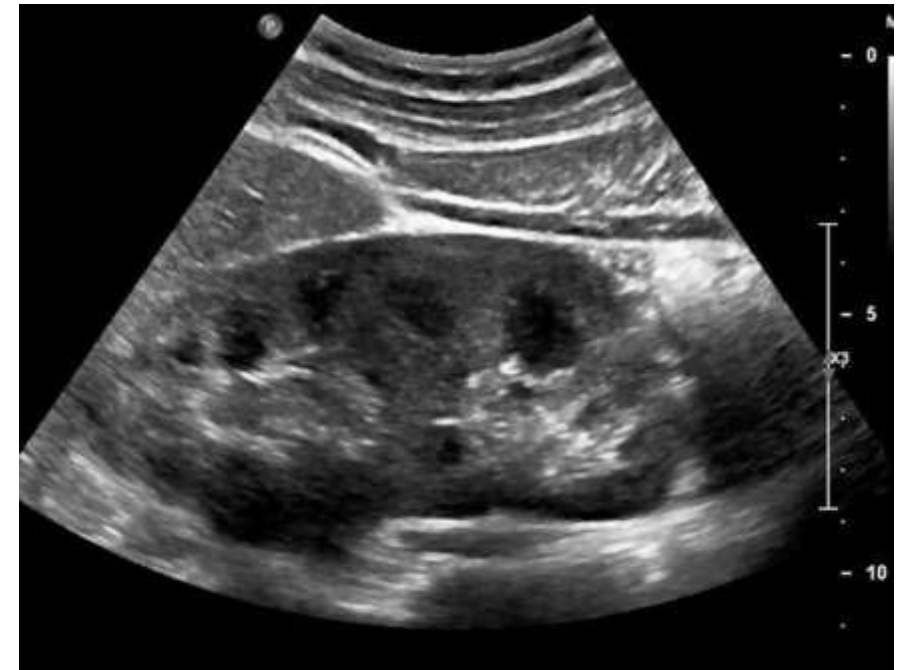
Diagnosis

The serum creatinine elevates unless the disease is unilateral.

- BUN and creatinine will initially elevate in a ratio 20:1 as it does with prerenal azotemia.
- Later the BUN: creatinine ratio will lower to 10:1. The urinalysis is variable, from normal (neurogenic bladder) to hematuria (stones, bladder cancer, clots)

Post-Renal Failure

- Key clinical feature: anuria
- **Test of choice: ultrasound**
 - Bladder ultrasound (bedside)
 - Renal ultrasound
 - Bilateral hydronephrosis
 - Hydroureter
- **Urinary catheterization**
 - Diagnostic and therapeutic
 - Relieves bladder outlet obstruction



Post-Renal Failure

- Lots of variation in lab values based on tubules
- Early post renal → tubular function intact
- Late → high pressure disrupts tubular resorption
- Urine chemistries variable

Na+	Cl-	BUN	Glucose
K+	HCO ₃	Cr	

Acute Tubular Necrosis/Injury

Acute tubular necrosis (ATN) is characterized by damage to tubular epithelial cells and abrupt decline in renal function. There is the shedding of granular muddy brown casts and tubular epithelial cells into the urine. ATN is the most common cause of acute kidney injury and may cause oliguria (urine output of <400 mL/day). It is the most common cause of AKI in hospitalized patients. It is associated with high morbidity and mortality.

The term tubular necrosis is a misnomer, as cellular necrosis is usually minimal. Acute tubular injury is a frequently used term by pathologists to describe this lesion.

Etiology

Acute tubular necrosis happens due to sudden ischemic or toxic events or sepsis.

Ischemic-

Anything causing reduced renal perfusion-

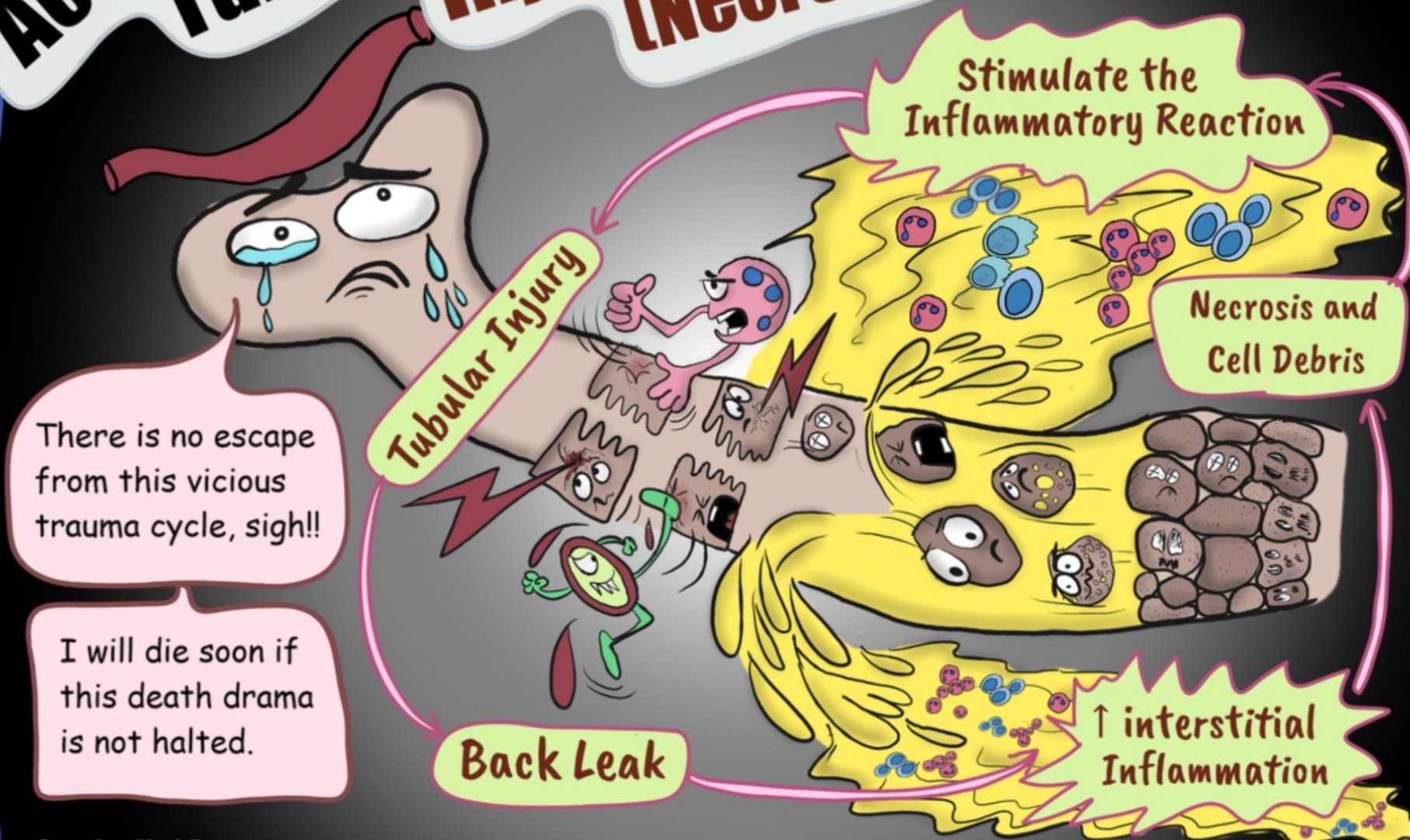
Prerenal azotemia and ischemic acute tubular necrosis have the same spectrum of causes. Any factor that leads to prerenal azotemia can lead to ischemic acute tubular necrosis.

- severe trauma
- blood loss
- acute pancreatitis
- septicemia
- microscopic polyangiitis
- malignant hypertension
- thrombotic microangiopathies
- mismatched blood transfusions and other hemolytic crises (hemoglobinuria)
- myoglobinuria (crush syndrome)

Nephrotoxic substances

- Heavy metals – mercury and lead poisoning
- Organic solvents- carbon tetrachloride
- Drugs- Gentamycin, cisplatin
- Radiographic contrast agents
- Crush injury (myoglobinuria)
- Hemoglobinuria (hemolytic anemia)

Acute Tubular Injury (Necrosis)



Tubulointerstitial nephritis

Tubulointerstitial nephritis (**TIN**) or Tubulointerstitial diseases can be of two types- Primary and Secondary

Secondary tubulointerstitial disease: Inflammation or fibrosis of the renal interstitium and atrophy of the tubular compartment **following the glomeruli or vascular diseases**.

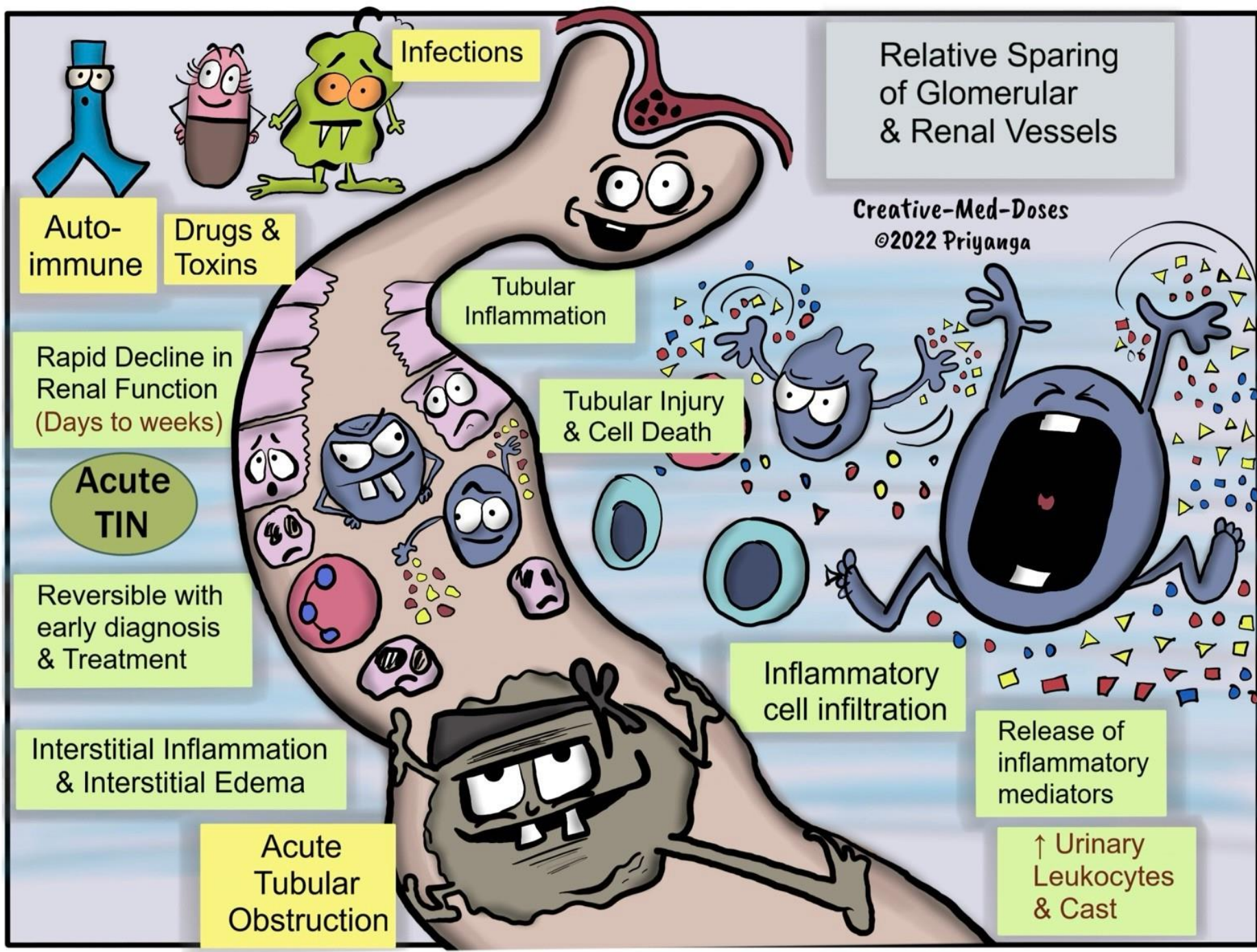
Primary Tubulointerstitial Nephritis (TIN)- **primarily affects the tubules and interstitium**, with relative **sparing of the glomeruli and renal vessels**. Glomerulus and renal vessels get involved in the later stages of the disease.

Primary Tubulointerstitial nephritis can be acute and chronic tubulointerstitial nephritis (TIN).

Acute Tubulointerstitial Nephritis (ATIN)-Tubulo-interstitial inflammation causes a rapid decline in renal function within days to weeks. It is reversible with early diagnosis and prompt treatment.

Causes and Pathogenesis of ATIN

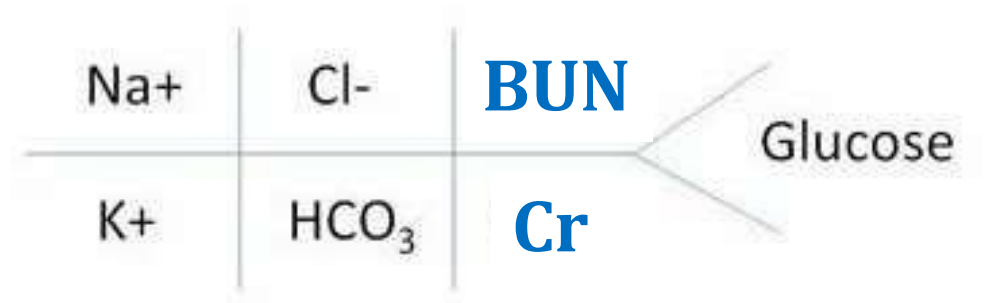
Drug-induced TIN



Intrinsic Renal Failure

Serum BUN/Cr

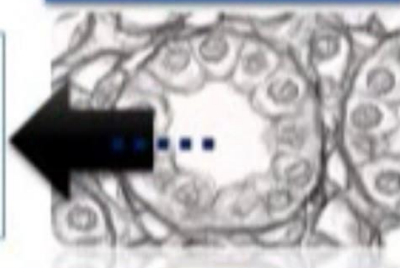
- Kidney cannot filter blood normally
- Less BUN/Cr filtered
- Rising BUN/Cr in blood
- No extra rise in BUN from $\uparrow$ resorption
- Normal ratio BUN:Cr (20:1)



Pre-Renal

Renal

1 - Na
2 - H₂O
3 - Urea



1 FENa



2 Urinary Sodium



3 U/P Osmolality



4 U/P Creatinine



5 Oedema

Easier

6 Improvement

Rapid
Improvement

Intrinsic Renal Failure

Urinary Findings

- Urine: kidney cannot resorb water/Na
- Uosm not high (can't concentrate urine)
- U_{Na} high (can't resorb Na)
- Fe_{Na} high (can't resorb Na)
- Often abnormal sediment
 - Protein
 - Red/white cells
 - Casts



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Intrinsic Renal Failure

	Normal	Intrinsic Failure	Pre-Renal Failure
BUN (mg/dl)	20	40	60
Cr (mg/dl)	1.0	2.0	2.0
BUN:Cr	20:1	20:1	> 20:1
UNa (mEq/L)	variable	> 40	< 20
FeNa (%)	variable	> 2	< 1
Uosm (mOsm/kg)	variable	< 350	> 500

Pre, Intrinsic, Post Problems

- Diseases often cross boundaries
 - Pre-renal → ATN
- Diuretics obscure urine findings
- Pre-existing chronic renal disease

Acute Renal Failure

Management

- Pre- and Post-Renal Failure: **correct underlying cause**
 - Volume depletion
 - Heart failure
 - Ureteral obstruction
- Monitor for dialysis indications
- Measures to correct underlying causes of acute kidney injury (AKI) should begin at the earliest indication of kidney dysfunction. Serum creatinine does not rise to abnormal levels until a large proportion of the renal mass is damaged, because the relationship between the glomerular filtration rate (GFR) and the serum creatinine level is not linear, especially early in disease. Indeed, the rise of serum creatinine may not be evident before 50% of the GFR is lost.

- Maintenance of volume homeostasis and correction of biochemical abnormalities remain the primary goals of treatment and may include the following measures:
- Correction of fluid overload with furosemide
- Correction of severe acidosis with bicarbonate administration, which can be important as a bridge to dialysis
- Correction of hyperkalemia
- Correction of hematologic abnormalities (eg, anemia, uremic platelet dysfunction) with measures such as transfusions and administration of desmopressin or estrogens

Volume overload

Furosemide can be used to correct volume overload when the kidneys are still responsive; this often requires high intravenous (IV) doses. Furosemide plays no role in converting an oliguric AKI to a nonoliguric AKI or in increasing urine output when a patient is not hypervolemic. However, response to furosemide can be taken as a good prognostic sign.

Hyperkalemia

Hyperkalemia in patients with AKI can be life-threatening. Approaches to lowering serum potassium include the following:

- 1 Decreasing the intake of potassium in diet or tube feeds
- 2 Exchanging potassium across the gut lumen using potassium-binding resins
- 3 Promoting intracellular shifts in potassium with insulin, dextrose solutions, and beta agonists Instituting dialysis

Acute Renal Failure

- Management
 - **Stop/hold drugs that alter renal function**
 - Diuretics
 - ACEi and ARBs
 - NSAIDs
 - Metformin: risk of lactic acidosis in ARF

Indications for Dialysis

- Acidemia
- **Electrolytes (hyperkalemia)**
- Intoxication (overdose dialyzable substance)
- **Overload of fluid (CHF)**
- Uremic symptoms
- Must be refractory to medical therapy



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AKI of Unclear Etiology

Evaluation

- **History and physical exam**
 - Known heart failure/cirrhosis
 - Volume depletion/overload
- Medication review
- Identify baseline Cr

AKI of Unclear Etiology

Evaluation

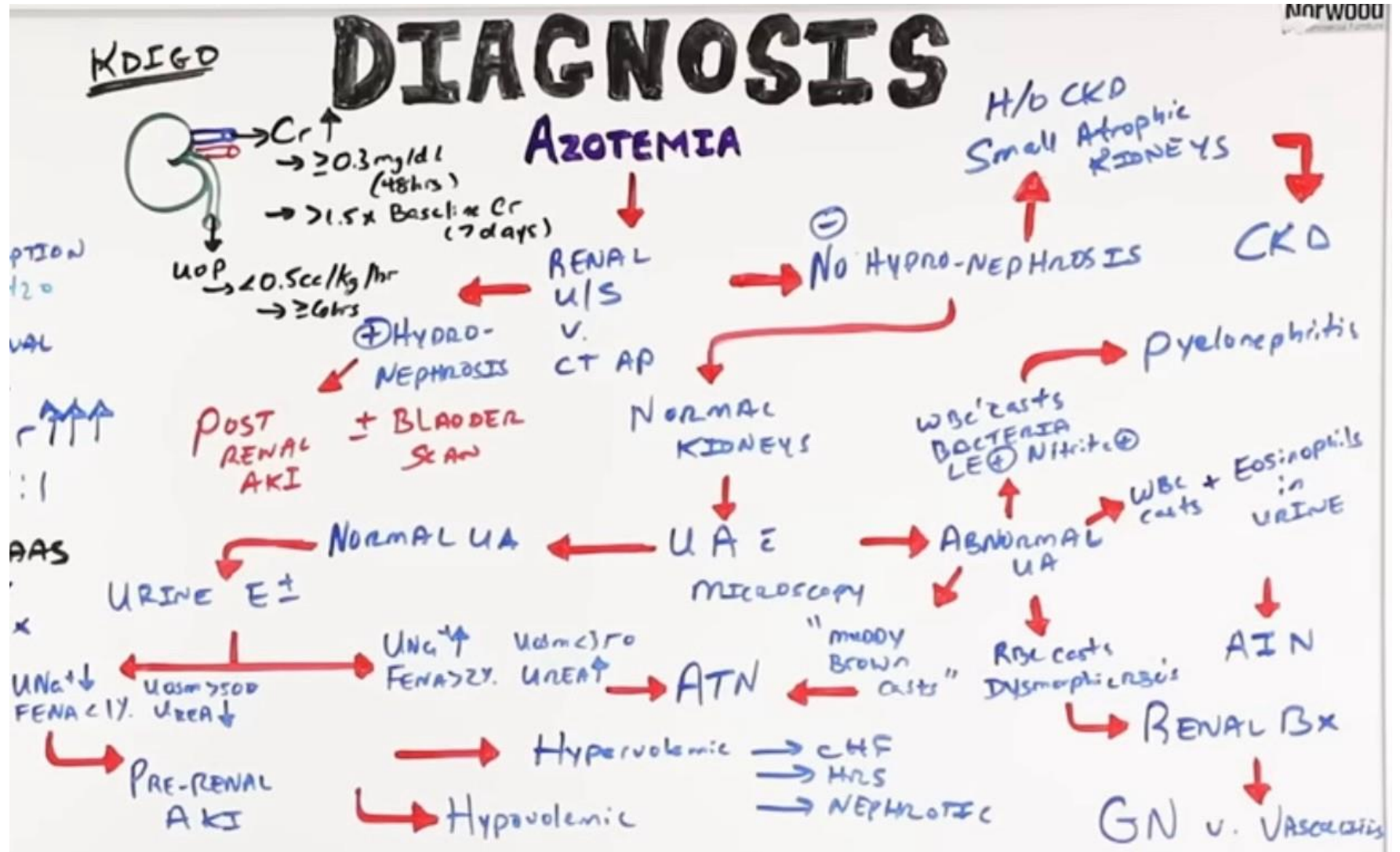
- Bladder or renal ultrasound
- Urinalysis
 - Bland = no casts, few cells
- Urine sodium
- Urine osmolarity
- Fractional Excretion of Na (FeNa)
 - Serum Na and Cr
 - Urine Na and Cr

$$\text{Fe}_{\text{Na}} = \frac{\text{P}_{\text{Cr}} * \text{U}_{\text{Na}}}{\text{P}_{\text{Na}} * \text{U}_{\text{Cr}}}$$

Acute Renal Failure

Monitoring

- BUN/Cr
- Daily weight
- Ins and outs
- Blood pressure
- Electrolytes



Done by :

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