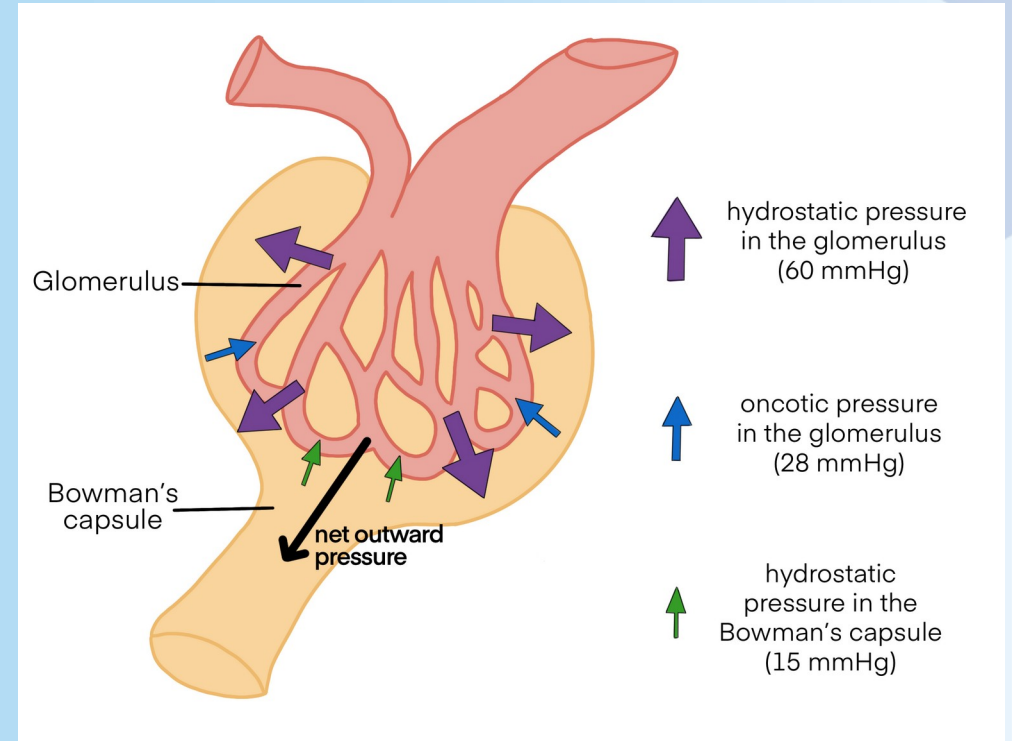


Acute Kidney Injury and Chronic Kidney Disease

By: Thomas Dlugosz

Introduction to GFR

- **Glomerular Filtration Rate (GFR)** measures the flow of plasma across the kidney's glomeruli into Bowman's space per unit time.
- GFR is a measure of kidney function



Introduction to GFR

- In clinical practice, GFR is most commonly estimated by measuring serum levels of creatinine and calculated with the following equation (2021 CKD-EPI):

$$eGFR = 142 \times \min \left(\frac{S_{cr}}{\kappa}, 1 \right)^{\alpha} \times \max \left(\frac{S_{cr}}{\kappa}, 1 \right)^{-1.200} \times 0.9938^{\text{Age}} \times 1.012 \text{ (if female)}$$

Creatinine is a waste product produced by muscles from the breakdown of creatine and creatine phosphate that is readily filtered across the glomerulus and not reabsorbed at all → good marker for function.

↑ Serum Creatinine = ↓ eGFR

↑ Age = ↓ eGFR

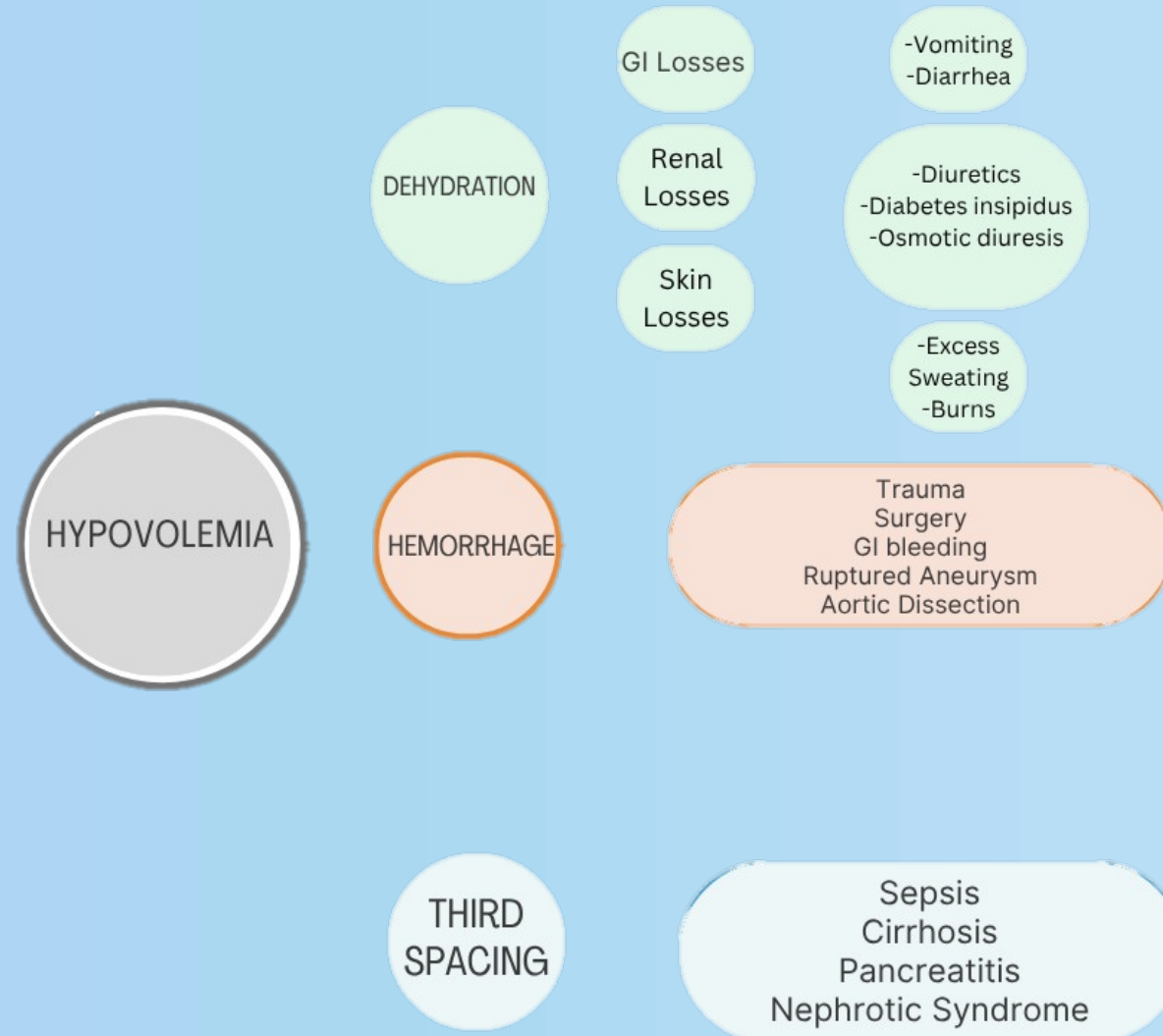
Definition of AKI

- AKI is an abrupt decline in kidney function characterized by elevation of serum creatinine levels and decreased GFR or oligouria
- We have 3 kinds of AKI
 - **Prerenal**- Renal hypoperfusion
 - **Intrarenal**- Direct damage to kidney tissue itself
 - **Postrenal**- Obstruction or impairment of urine outflow

Prerenal AKI



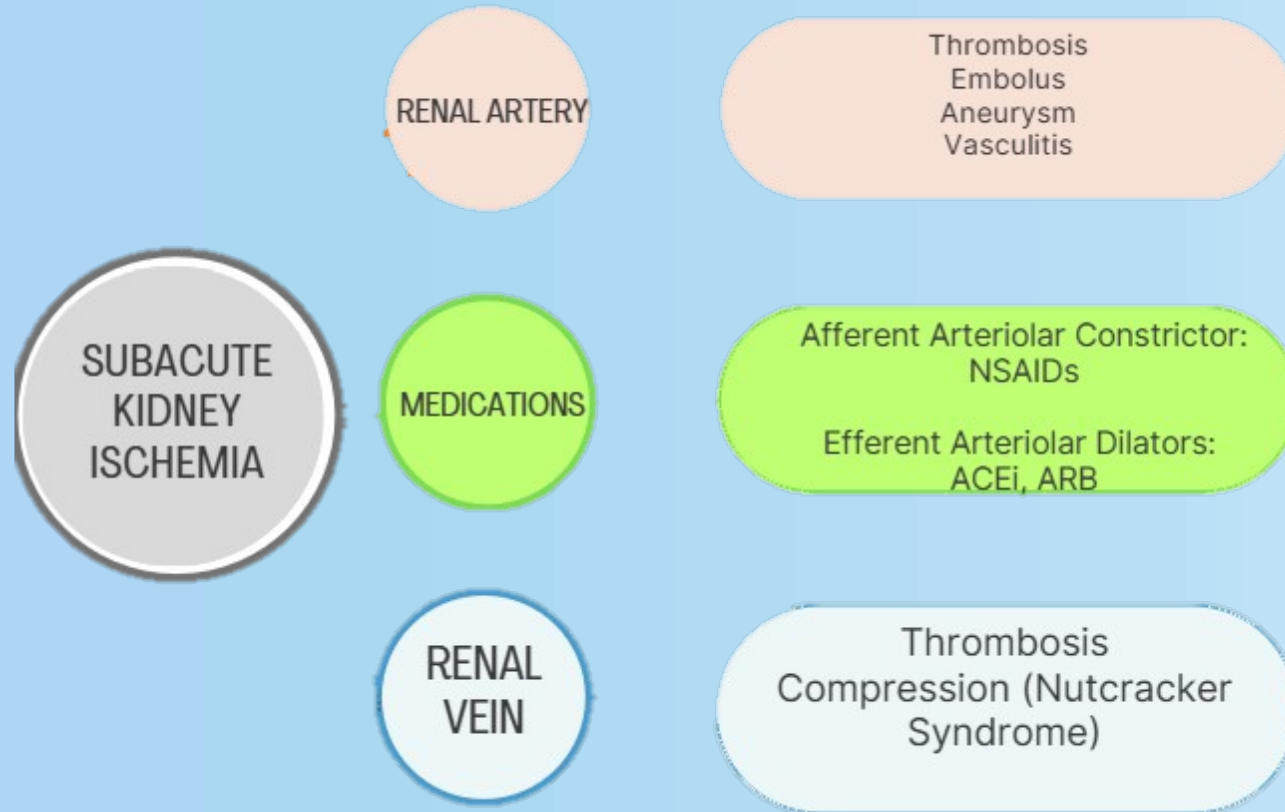
Anything that causes hypoperfusion to the kidney can cause Prerenal AKI



Prerenal AKI



Anything that causes hypoperfusion to the kidney can cause Prerenal AKI

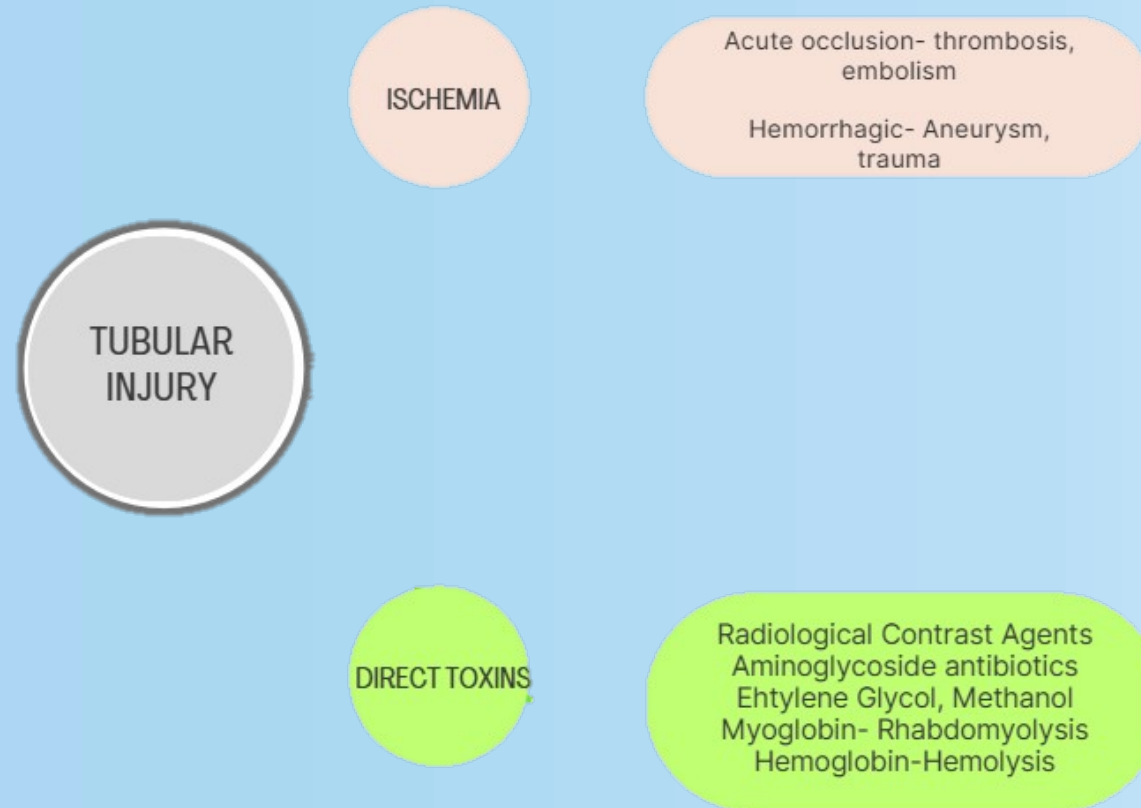


Nuance: Timeframe is important - Acute events tend to lead to intrarenal AKI
Prerenal AKI may progress to intrarenal AKI.

Intrarenal AKI



Anything that directly damages kidney tissues can cause intrarenal AKI



Intrarenal AKI



Anything that directly damages kidney tissues can cause intrarenal AKI

A large, light grey circle with a thin white border, centered on the slide.

GLOMERULONEPHRITIS

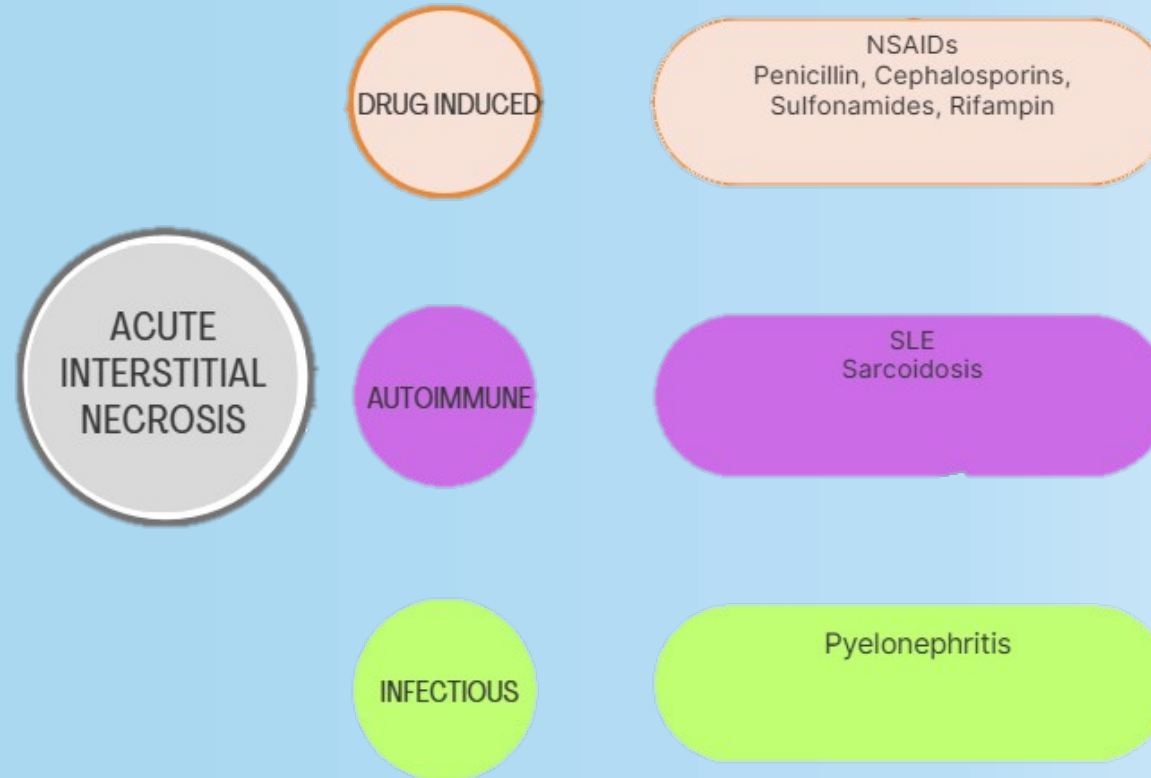
Intrarenal AKI



Anything that directly damages kidney tissues can cause intrarenal AKI

Acute Interstitial Nephritis is thought to be a hypersensitivity reaction

-NSAIDS can cause both pre-renal AKI and Intrarenal AKI



Postrenal AKI Mechanism

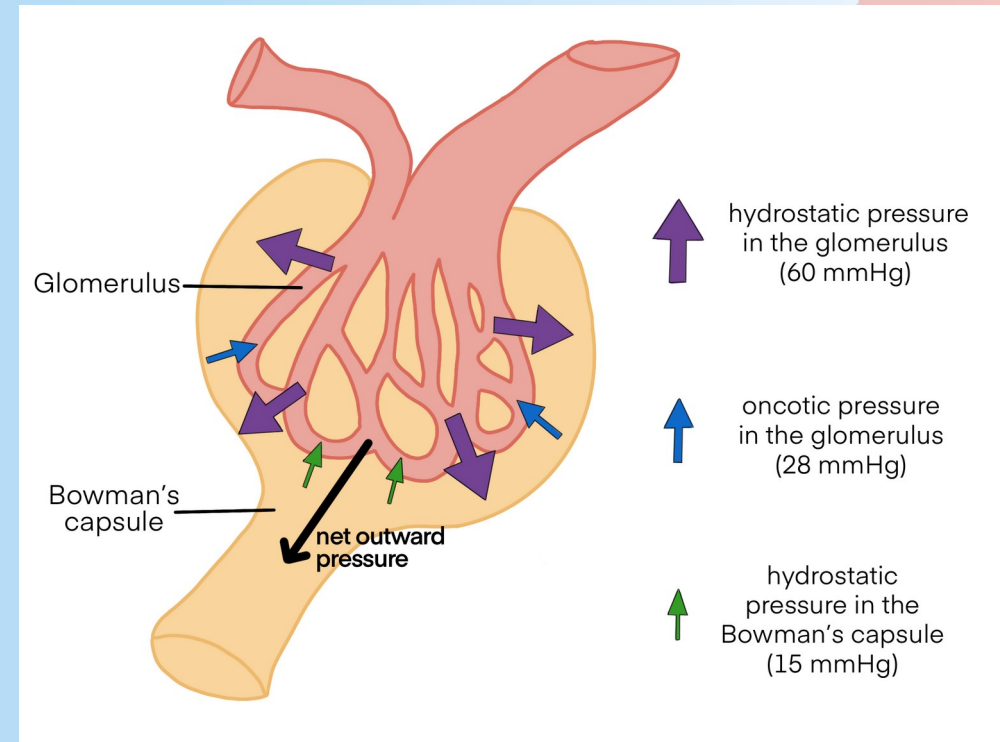


Anything that obstructs or impairs urine outflow can cause postrenal AKI

Starling Forces across the glomerulus is a way to calculate GFR

Any kind of obstruction or impairment of urine outflow will increase hydrostatic pressure in Bowman's capsule → direct decrease in GFR → risk of AKI

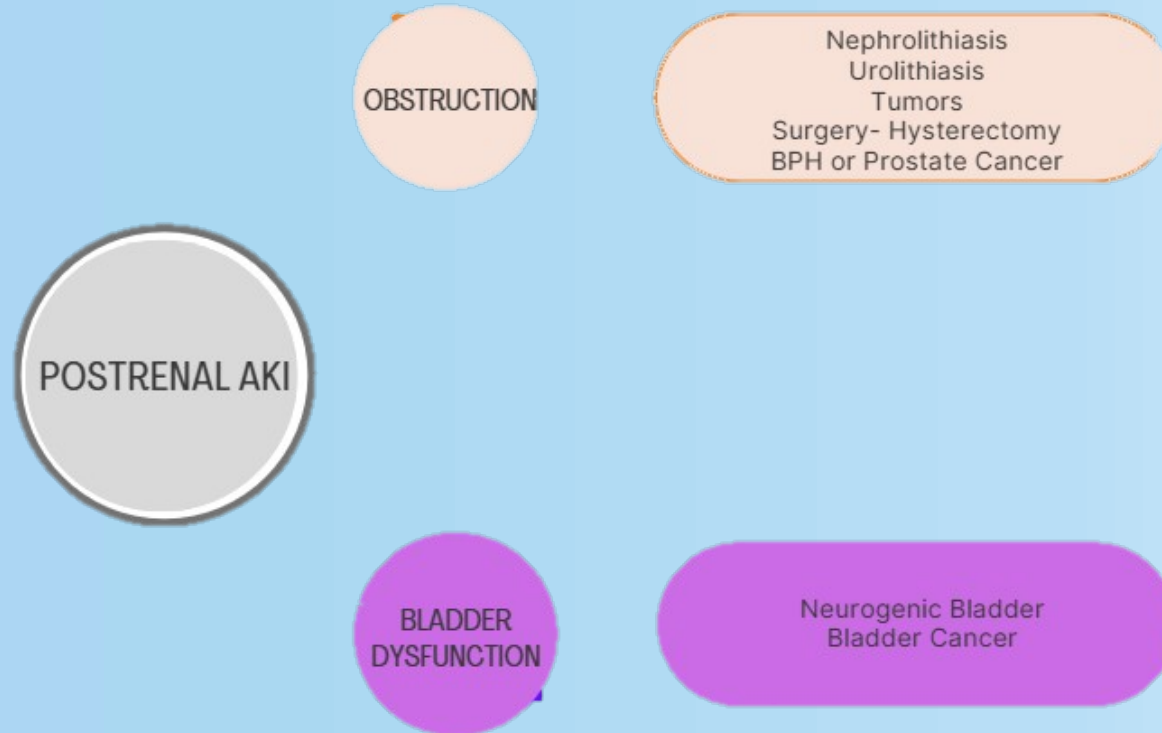
Obstruction may occur anywhere from urethra up to renal pelvis



Postrenal AKI



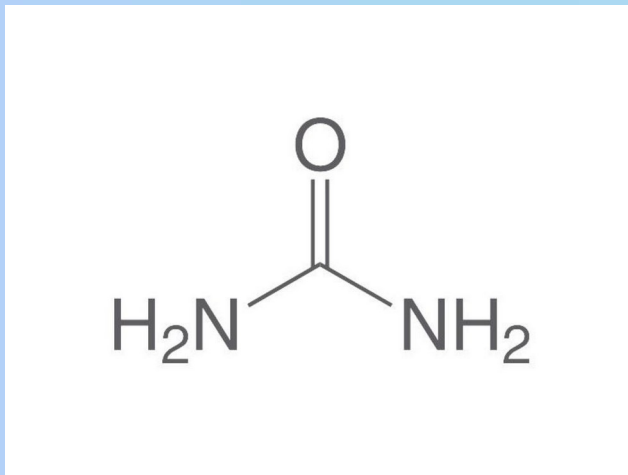
Anything that obstructs or impairs urine outflow can cause postrenal AKI



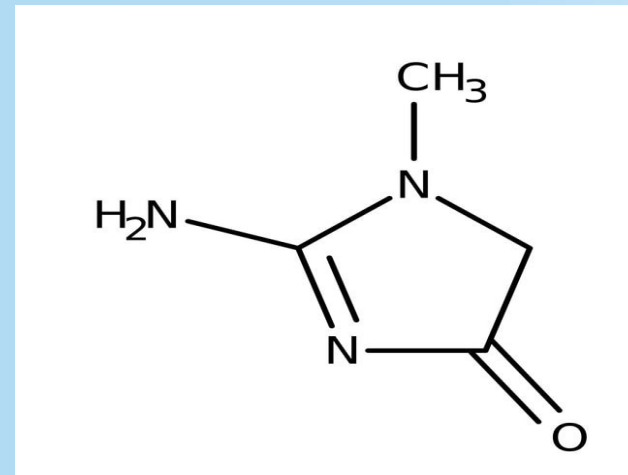
Laboratory Findings of AKI and Complications

Azotemia (by definition)- elevated nitrogen-containing waste products in the blood

Urea



Creatinine

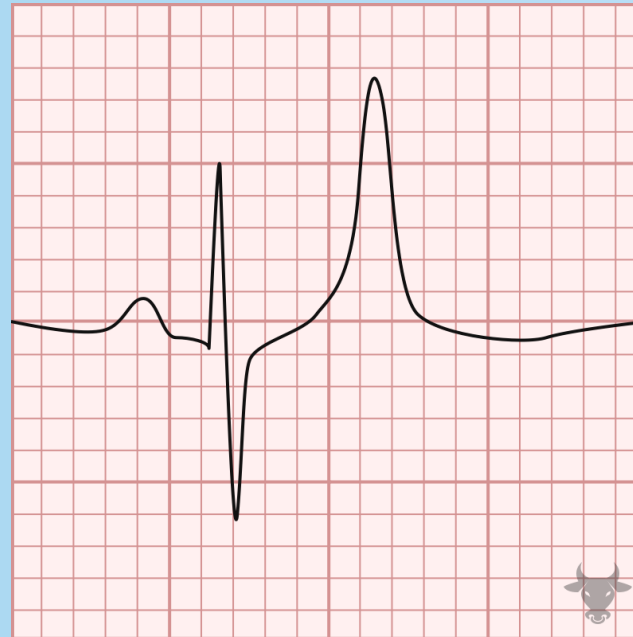


Uremia= Symptomatic azotemia

Laboratory Findings of AKI and Complications

Hyperkalemia- Mainly because of impaired glomerular filtration

Complications: Arrhythmia



Laboratory Findings of AKI and Complications

- **Hyperphosphatemia:** Mainly because of impaired filtration across glomeruli
- **Hypocalcemia:** Mainly because of hyperphosphatemia!



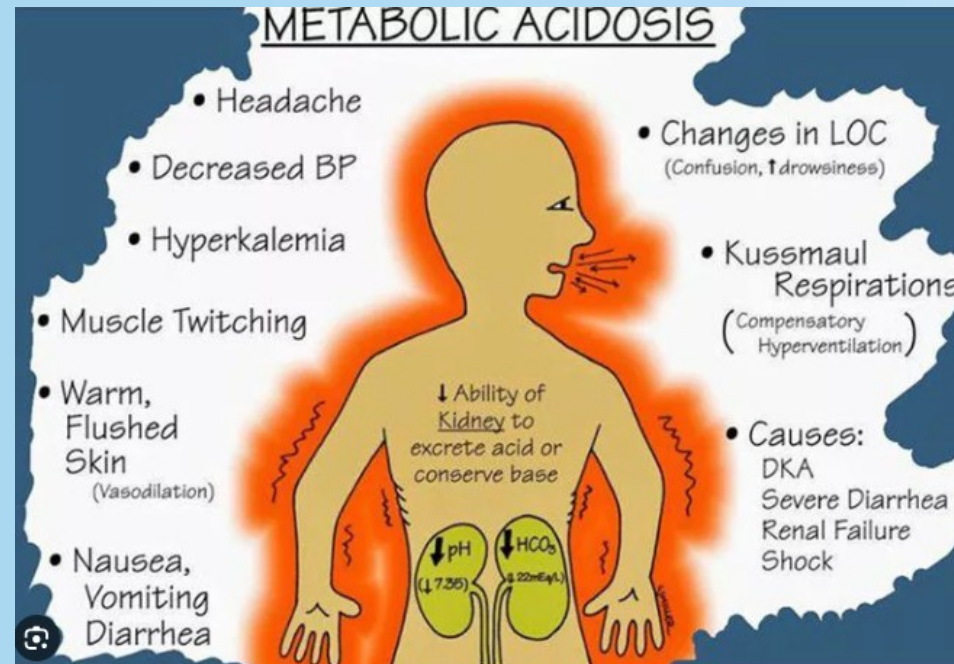
Hypocalcemia means low **ionic (free)** calcium presence in blood!

Complications: Tetany, muscle spasms, seizures

Laboratory Findings of AKI and Complications

- **Metabolic acidosis:** impaired excretion of protons in distal tubule and collecting duct, reduced filtering of organic acids

Symptoms:



Chronic Kidney Disease

Chronic Kidney Disease (CKD)

- CKD is defined by the presence of kidney damage or an eGFR under 60 mL/min/1.73m² for 3 months or more, regardless of cause.

The 6 CKD categories, known as stages 1 through 5, are described below (stage 3 is separated into 3a and 3b):

- G1: GFR 90 mL/min/1.73 m² and above with evidence of kidney disease, such as hematuria or proteinuria
- G2: GFR 60 to 89 mL/min/1.73 m²
- G3a: GFR 45 to 59 mL/min/1.73 m²
- G3b: GFR 30 to 44 mL/min/1.73 m²
- G4: GFR 15 to 29 mL/min/1.73 m²
- G5: GFR less than 15 mL/min/1.73 m² or treatment by dialysis

The 3 levels of albuminuria include an ACR:

- A1: ACR less than 30 mg/g (<3.4 mg/mmol)
- A2: ACR 30 to 299 mg/g (3.4-34 mg/mmol)
- A3: ACR greater than 300 mg/g (>34 mg/mmol)

Causes of CKD



Any kidney disease and any disease affecting renal perfusion can potentially cause CKD

Diabetes Mellitus

Hypertension

Glomerulonephritis

Polycystic Kidney
Disease

AKI

Pyelonephritis

Autoimmune
Disease (Lupus)

Vasculitis

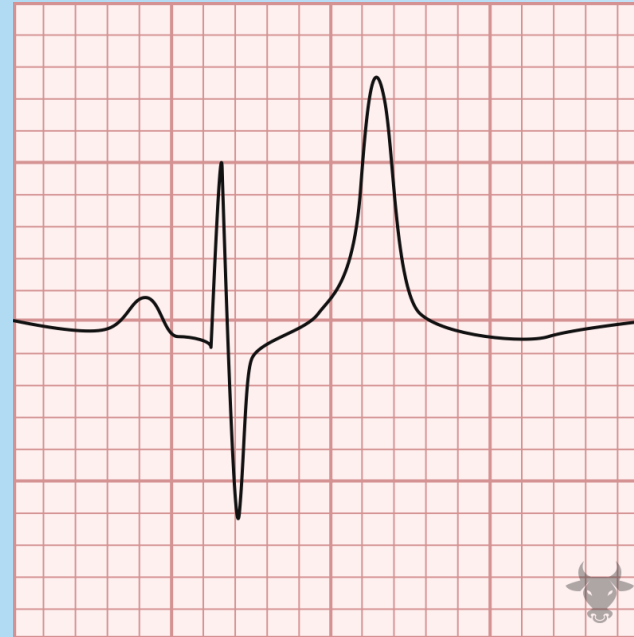
Chronic
Drug/Toxins
(NSAIDS!)

Kidney Stones

Clinical Manifestations of CKD

Electrolyte Abnormalities

Hyperkalemia - arrhythmia risk



Clinical Manifestations of CKD

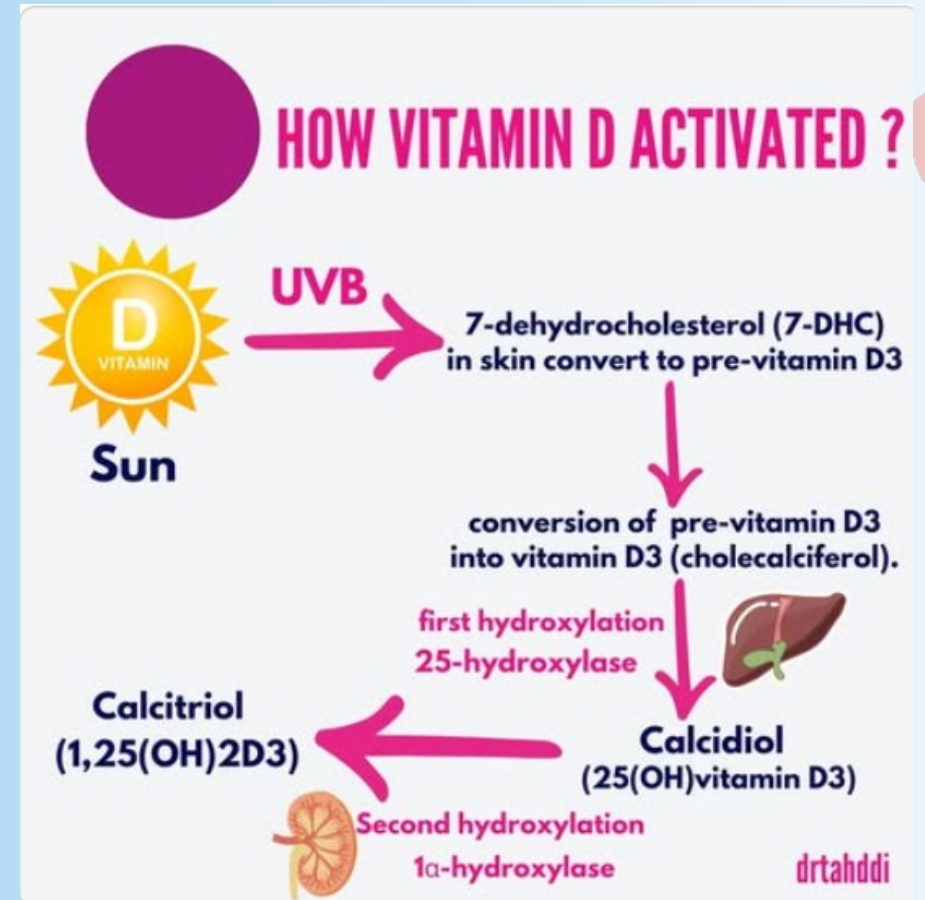
- **Hyperphosphatemia**: Mainly because of impaired filtration across glomeruli
- **Hypocalcemia**: Mainly because of hyperphosphatemia and loss of vitamin D activation



Complications: Tetany, muscle spasms, seizures, metastatic calcification

Clinical Manifestations of CKD

Vitamin D deficiency- Loss of Proximal tubular cells result in inability to convert Vitamin D into an active form → impaired Calcium absorption from intestines → hypocalcemia



Clinical Manifestations of CKD

Secondary Hyperparathyroidism:

Hypocalcemia is detected by the parathyroid glands → PTH release → Calcium resorbed from bones into the blood → renal osteodystrophy (renal mineral bone disease)

Labs: ↑ PTH , ↑ PO_4 , ↓ Ca^{2+} (or normal)

Complications: Osteoporosis, fractures, bone pain and deformity

Metastatic Calcification: Calcium phosphate deposition on arteries and skin (calciophylaxis)

Growth Stunting: Partially because of bone abnormalities



Clinical Manifestations of CKD

Anemia

- CKD → gradual loss of erythropoietin synthesizing cells → decline of RBC synthesis → **normocytic normochromic anemia**
- CKD is a chronic inflammatory state → IL-6 produced → increased hepcidin production → Iron sequestration into macrophages and decreased intestinal absorption → functional iron deficiency → **potential to progress to microcytic hypochromic anemia**

Symptoms: tiredness, pallor, feeling cold



Clinical Manifestations of CKD

Uremia

- CKD → impaired excretion of toxic nitrogenous metabolites → **uremia**

Platelet Dysfunction- Uremic toxins impair platelet aggregation

Consequences: epistaxis, gingival bleeding, easy bruising, prolonged bleeding time (normal apTT and PT)

Pruritus- Uremic toxins accumulate in the skin and irritate it

Consequence: Itchy skin



Clinical Manifestations of CKD

Fibrinous Pericarditis- Uremic toxins damage endothelium → increased endothelial permeability → clotting factors escape into pericardial space → inflammation activates clotting cascade → **fibrin deposition on pericardium**

Consequences: Pericardial friction rub

Encephalopathy- Uremic toxins directly cause global cerebral dysfunction

Consequences: confusion, seizures
asterixis



Clinical Manifestations of CKD

Cardiovascular Disease

- Hypertension- Solute retention + RAAS activation + arterial calcification
- Atherosclerosis- Uremic toxins + inflammatory cytokines cause endothelial dysfunction + hypertension accelerate atheromas
- Valvular Calcification
- Heart Failure



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