Acute Kidney Injury

(Abrupt rise in serum Cr)

INTRA-renal

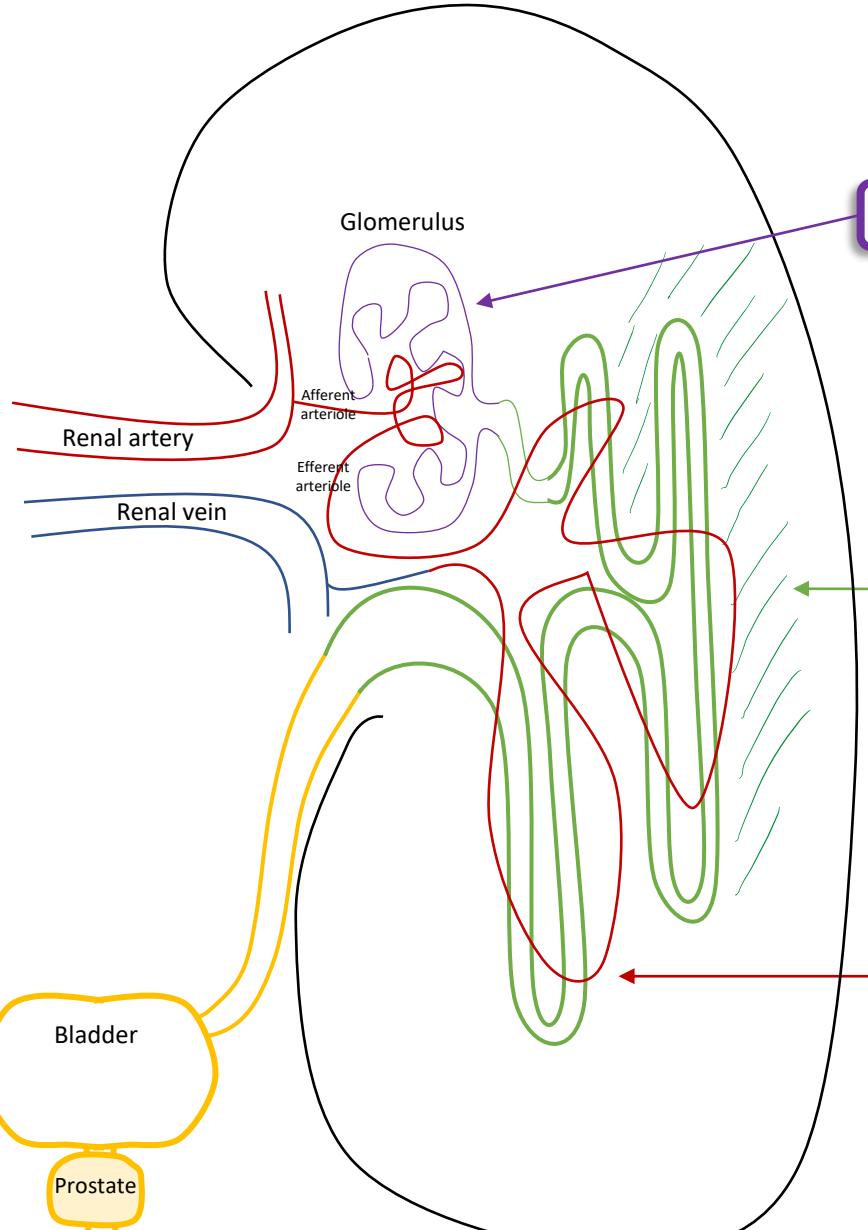
PRE-renal

Glomerular

Tubulointerstitial

Vascular

POST-renal



Acute Kidney Injury

(Abrupt rise in serum Cr)

Key point: eGFR is not meaningful in setting of AKI. eGFR is based on assumption of stable GFR

PRE-renal

Etiologies

↓ Effective Arterial Blood Volume (EABV)

1. Hypovolemia
2. Sepsis
3. Heart failure
4. Cirrhosis

Altered vasculature

1. Renal vein thrombosis
2. Renal vein HTN (CHF)
3. Inta-Abdominal Hypertension (IAH)

Dx

Suggestive history, bland urinalysis, no hydronephrosis and FeNa <1% (if oliguric)

POST-renal

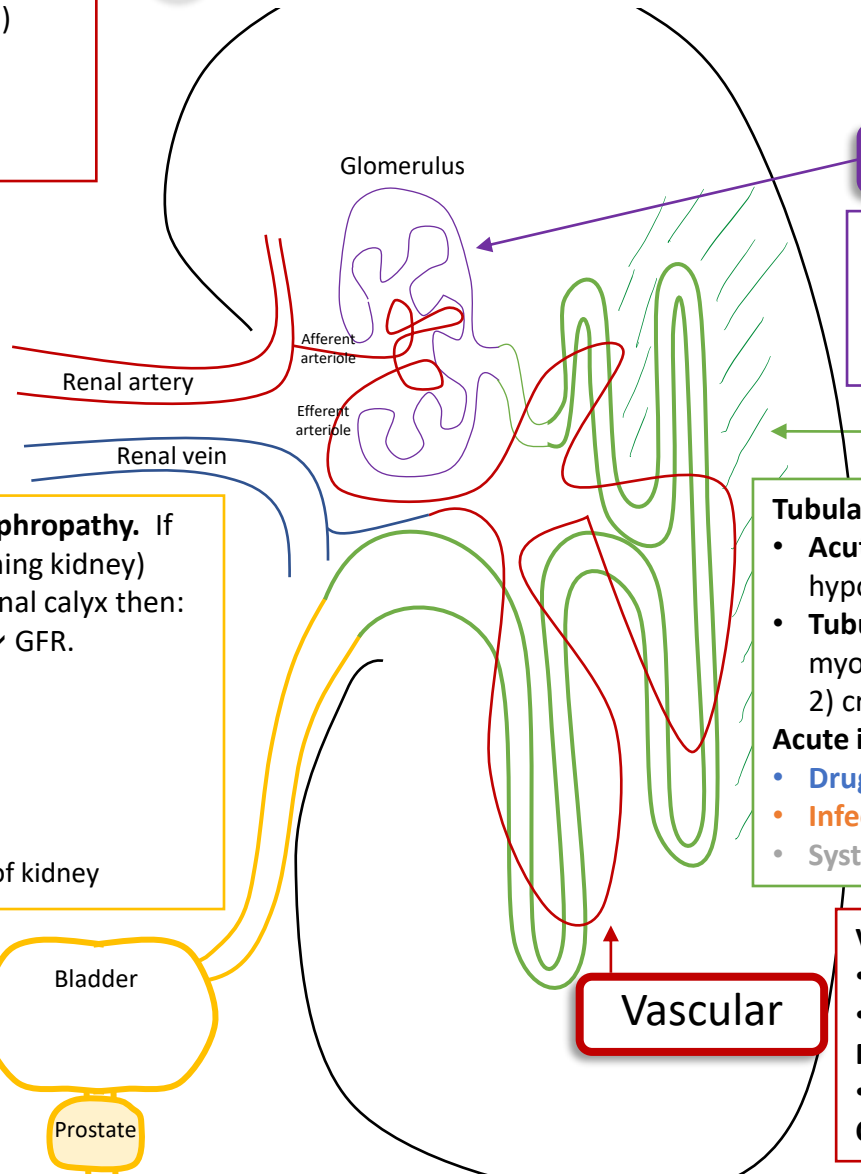
Etiologies

Post-renal AKI manifests through an **obstructive nephropathy.** If the outflow of **BOTH kidneys** (or the single functioning kidney) anywhere from the level of the urethra up to the renal calyx then: ↑ retrograde hydrostatic pressure, and resultant ↓ GFR.

Urethra ->	**Bladder ->**	**Ureter**
- Stricture	- Clot	- Calculi
- BPH	- Malignancy	- Int/ext malignancy or LAN
	- Neurogenic	- Retroperitoneal fibrosis

Dx

Suggestive history and hydronephrosis on ultrasound of kidney



INTRA-renal

Broad differential broken down anatomically by **glomerular**, **tubulointerstitial** and **vascular**.

Dx

1. "Active" urine sediment (supports intra-renal)
2. Elevated FeNa while oliguric (rules out pre-renal)
3. Normal kidney US (rules out post-renal).

Glomerular

Acute Glomerularnephritis (GN): both **non-crescentic** (e.g. class IV lupus nephritis or infection-related) and **crescentic GN or "RPGN"** (grouped into *immune-complex*, *pauci-immune* and *anti-GBM*)
Dx: dysmorphic RBCs / RBC casts, proteinuria, leukocyturia, +/- HTN

Tubulointerstitial

Tubular

- **Acute tubular necrosis (ATN)** *most common*: 1) ischemic (prolonged hypoperfusion) or 2) *toxic (including iodinated contrast)*
- **Tubular obstruction** 1) cast nephropathy (pigments - myoglobin/hemoglobin or proteins - light-chains/paraproteins) or 2) crystals (uric acid, acyclovir, ethylene glycol, MTX).

Acute interstitial nephritis

- **Drugs** - antimicrobials, NSAIDs, PPIs, immunotherapy
- **Infection** – pyelonephritis, *Legionella, Leptospira*, *CMV*
- **Systemic diseases** - Sjogren's, sarcoidosis

Vascular

Vasogenic (change in tone of afferent or efferent arterioles)

- Constriction of afferent – e.g. NSAIDs, calcineurin inhibitors (CNIs)
- Dilation of efferent arteriole – e.g. ACE inhibitors, ARBs

Microangiopathic Hemolytic Anemia (MAHA)

- TTP/HUS, scleroderma renal crisis, malignant HTN, HELLP).

Cholesterol Emboli