Kidney Pathology

4 sites → pathology

- blood vessels: autoregulation, bp, ischaemia
- glomerulus: selectivity 7 - 70 kDa
- Tubule: absorb filtrate
- interstitium compact few cells or fibrosis

Acute renal failure
- 50% from baseline - maybe acute on chronic
- failure of renal excretory function due to depression of eGFR often oliguria and uraemia
  multifactorial

Pre-renal (75%) ← spectrum to ATN due to ischaemia
* generalised hypoperfusion
  - cardiogenic
  - hypovolaemia = most common cause
  - vasodilatory
* ↓ renal perfusion - ~~if renal~~
  - bilateral thrombi or single functioning kidney
  - dissection
  - occlusion
  - suprarenal clamp
* inability to autoregulate
  - NSAIDs
  - ACEI generally ↓ renal blood flow
* hepatorenal syndrome

Why susceptable
- medulla on limits due to vasa recta and a-v shunting of O_2 due to countercurrent mechanism
- autoregulation: maintain RBF + GFR in narrow limits despite ABP variation - stabilises filtered load of solute

  if RBF ↑ constrict affeent arteriole → ↑ resistance → ↓ flow; myogenic element - stretch, cation channels open → depol → Ca^{++} entry → constrict
  Tubuloglomerular feedback - Na^+ load at macula densa via ATP, adenosine + thromboxane

↑sens to TGF volume:
PGE_2, adenosine, ang II
more sens at lower blood volumes

Expect fluid resus → ↑ renal perfusion → ↑ urine production and ↓ urea/creatinine

Renal causes:
(1) ATN (vascular directly nephrotoxic)
(2) Glomerulonephritis
(3) vasculitis
(4) tubulointerstitial nephritis
(5) thrombotic microangiopathies
(6) myeloma

• ATN - direct toxicity → endogenous - myoglobin
- haemaglobin
- Ig light chains
- crystal depos'n phosphate urate
↓
exogenous (broad)
aminoglycosides; vancomycin
radiocontrast media
ciclosporin

cellular level ↑ Ca^{++} ↑ ROS ↓ ATP ↑ enzyme act'n
• apoptosis +/- necrosis
• reversible structural changes
* failure of Na reabs → ↑ Na @ MD → tgfb → ↓ flow ↓ GFR
* shed cells → lumen ↑ P_b → ↓ GFR
* expresses adhesion molecules → inflammation
* interstitial oedema → leak back to tubule → ↓ GFR
retain BM then able → repithelialisation and ∴ recovery of function