- Tubulointerstitial nephritis

injury to tubules + interstitium → inflammation
∴ diverse causes
acute or chronic
acute = leukocyte infiltration + focal tubular necrosis
see impaired [ ] ability so { salt wasting, acidosis, polyuria }
+ tubule function
no hallmarks of glom. injury
eg haematuria proteinuria

* pyelonephritis - , staph, E Coli
- haematogenous spread due to bacteraemia
- ascending infection often context vesico-ureteral junction ; E Coli, proteus, enterobacter
occasionally viral - polyoma virus
see: patchy neutrophils ; focal ; if reflux related
= upper and lower poles
spread interstitium to tubules then intraluminal though glomeruli = resistant
heals by scars seen on the cortical surface = fibrous depressions

* hypersens to drugs and toxins
diuretics, penicillins, sulfonamides, NSAIDs allopurinol. Idiosyncratic; not dose related
lag phase
fever, eosinophilia, renal abnorm ± rash
haematuria, mild proteinuria & leukocyturia often with eosinophils

* uate nephropathy - accum @ collecting duct → obstruction eg tLS

- **thrombotic microangiopathies**

damage to endothelium → thrombi @ capillaries + arterioles + fibrin deposition
→ intravascular haemolysis + fragments
∴ anaemia
→ plt consumption ∴ thrombocytopenia
platelets & platelet/fibrin deposits @ renal arteries, arterioles + glomeruli

* HUS
  - paediatric after O157 E Coli → bloody diarrhoea; shiga like toxin damages endothelium
  - adult post O157; APLS; post partum; chemotherapeutics
  - familial deficiency complement factor H so → break down alternative pathway C3 convertase

* idiopathic TTP fever, neurology, anaemia, purpura + renal failure
ADAMTS-13 deficit acquired or inherited → large complexes VWF → plt activation

see plt-fibrin thrombi @ glomeruli