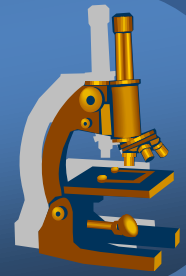
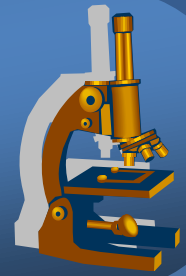


# *Systematic pathology*



Kidney and urinary tract pathology

# *Kidney diseases*



# Congenital diseases



- × **Renal agenesis** (bilateral)

- ⇒ *incompatible with life*

- × **Renal dysplasia** (uni-, bilateral)

- ⇒ *developmental disorder due to abnormal morphogenesis and differentiation. Parenchyma with foci of immature renal tissue.*

- ⇒ *Clinically: diff.dg. x renal tumors of childhood*

# Congenital diseases



## × Horseshoe kidney (*ren arcuatus*)

⇒ *both kidneys fused by their lower poles*

## × Cysts and cystosis 2 main forms :

⇒ *autosomal recessive polycystic kidney disease*

- Infantile, microcystic
- death commonly soon after birth, kidneys completely replaced by multiple cysts < 2mm



# Congenital diseases



## ⇒ *Adult polycystic kidney disease*

- common congenital disease, ↓ of renal function in the 3.- 4. dec., autosomal dominant - gen usually on the short arm of chromosome 16
- *gross*: symmetrical kidney enlargement – length to 30 cm, multiple cysts 0,5-50mm

# Congenital diseases

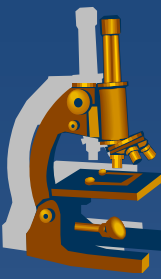
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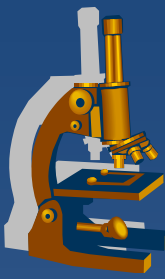


## ⇒ *Solitary kidney cysts*

- accidental finding . Important diff. dg x cystic renal carcinoma

# *Polycystic kidney*





# *Vascular kidney disorders*

## **x Renal artery stenosis**

⇒ *renovascular hypertension (Goldblatt's)*

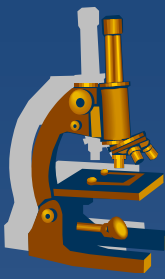
⇒ *pressure ↓ in afferent arterioles*

⇒ *↓ of filtration pressure in the glomerulus*

⇒ *juxtaglomerular apparatus hyperplasia + renin overproduction*

⇒ *blood pressure ↑ - by longer duration - vascular atrophy.*

# *Vascular kidney disorders*



## **x Benign nephrosclerosis**

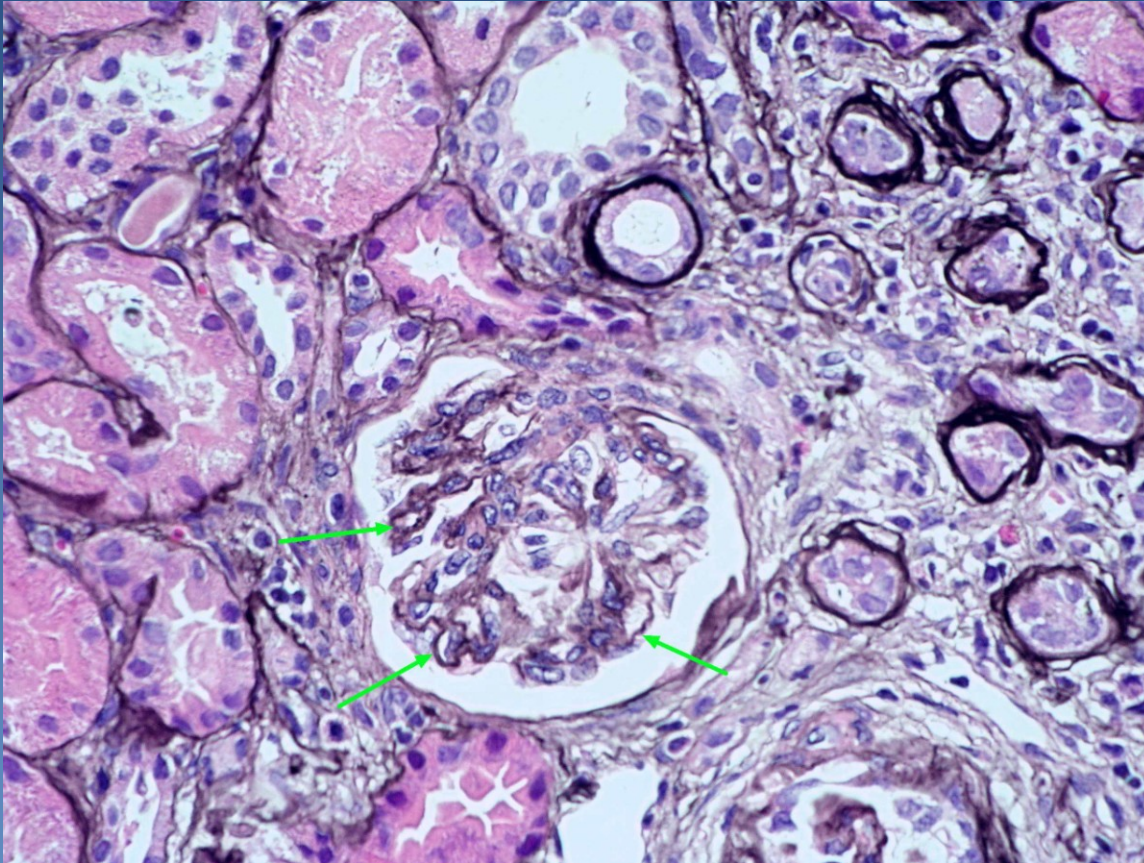
⇒ *by benign (compensated) hypertension*

- gross : symmetrical decrease in size, **fine granulated surface**
- micro : **hyaline insudates in** arteriolar walls, median **hypertrophy + intimal sclerosis**, ischemic changes +/- glomerular loss, vascular atrophy of the tubules, adjacent interstitial fibrosis.



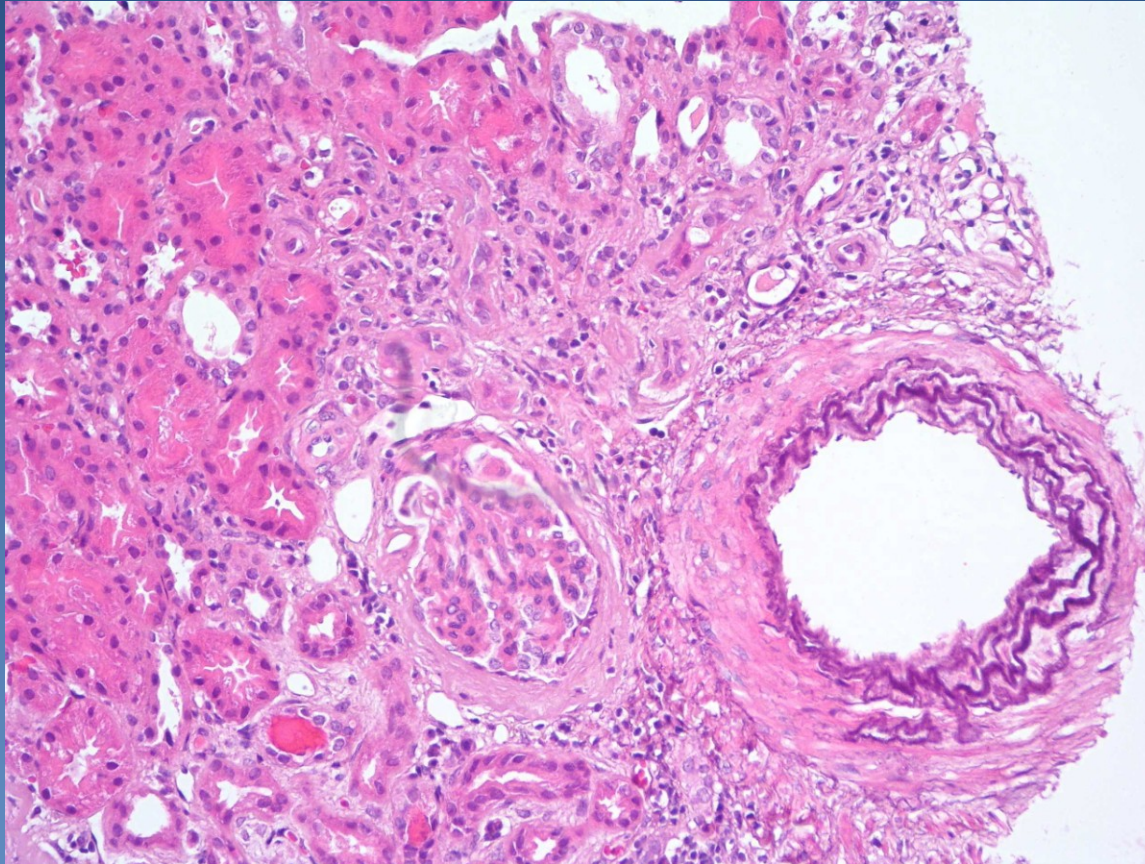
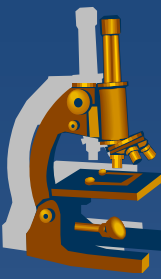


## ***Benign nephrosclerosis***



Ischemic glomerular changes, „wrinkling“ of the GBM

# ***Benign nephrosclerosis***



# *Vascular kidney disorders*



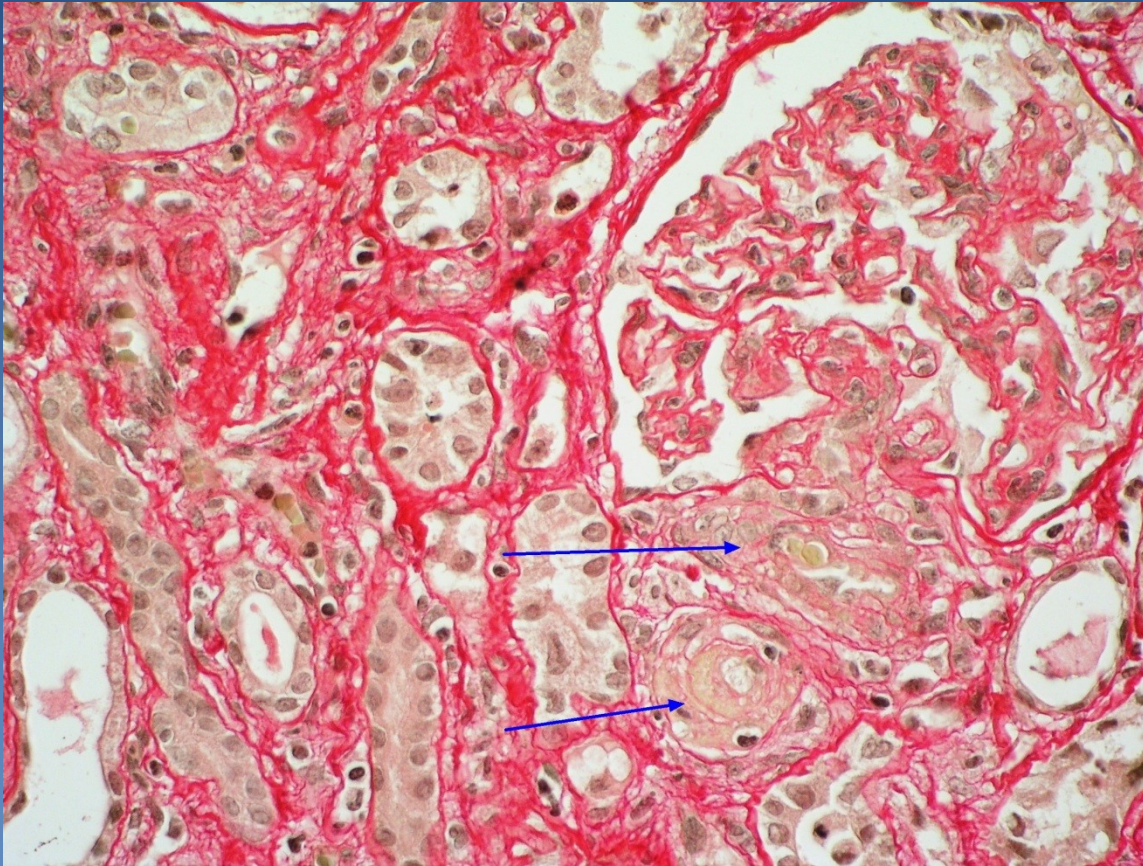
## **x Malignant nephrosclerosis**

⇒ *due to accelerated arterial hypertension (diastole >130mmHg), endothelial damage*

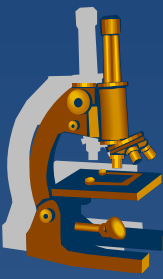
- gross : renal oedema, infarctions possible
- micro: oedema, intimal **muroid seepage** in arteries, fibrinoid necrosis of the arteriolar wall, possible trombi



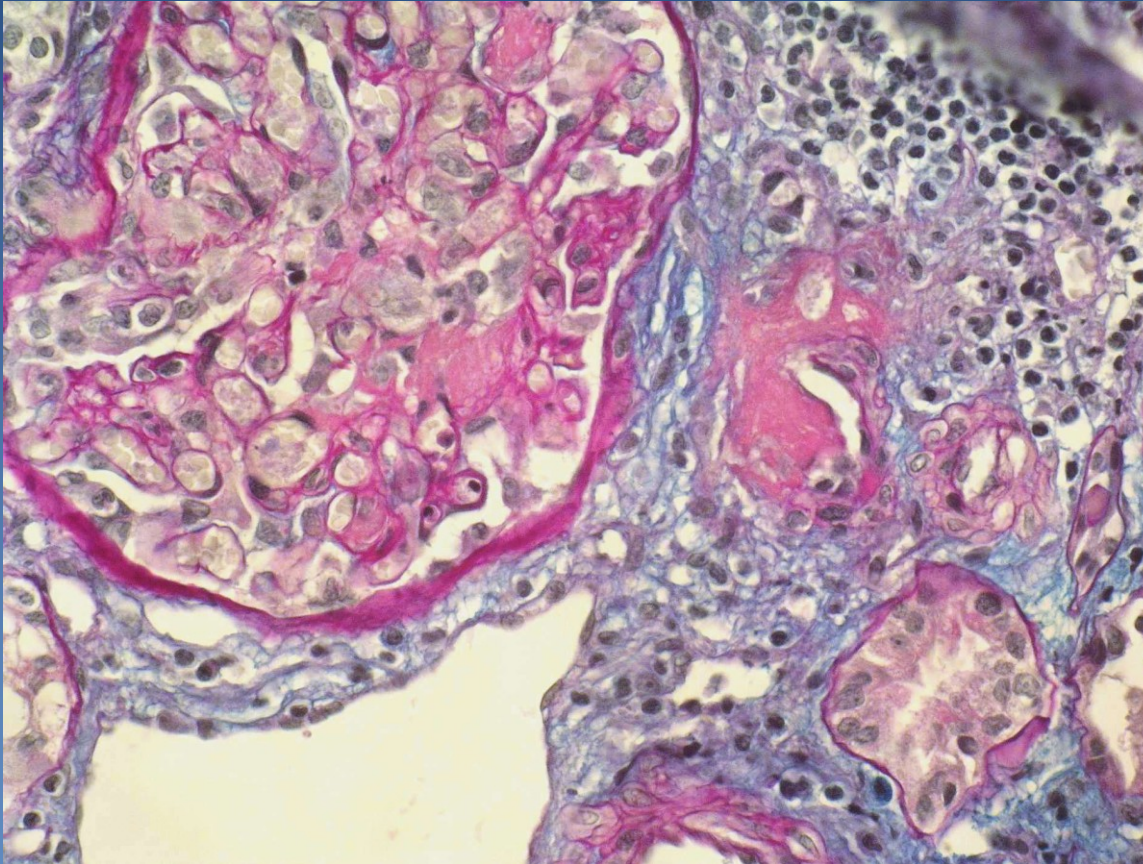
# *Malignant nephrosclerosis*



Significant arteriolar luminal narrowing,  
endothelial oedema



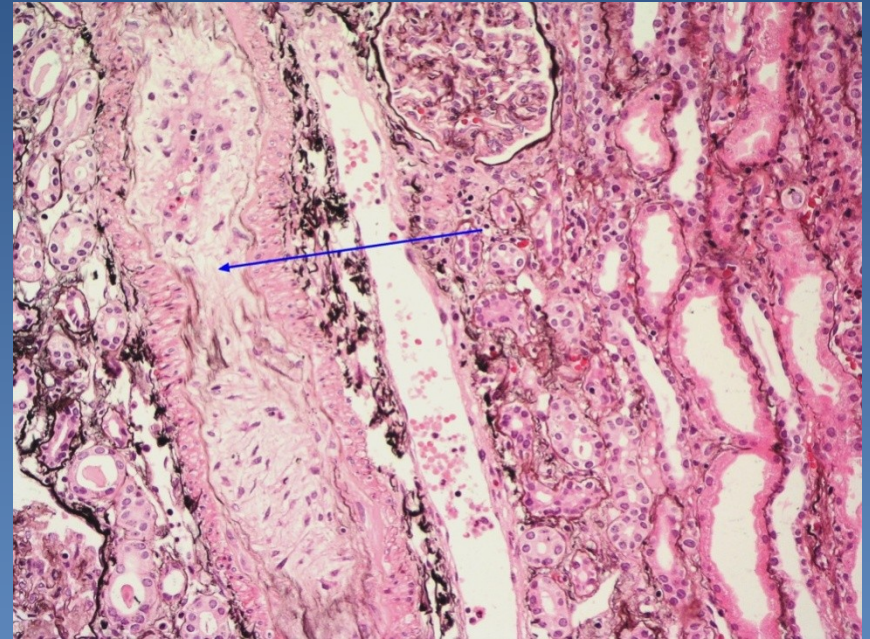
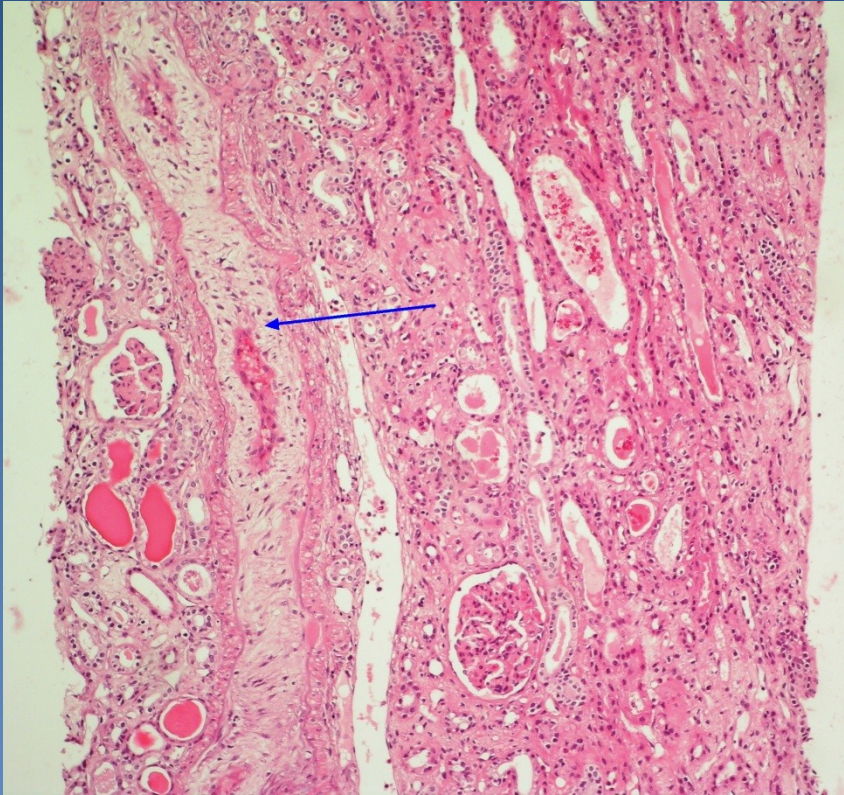
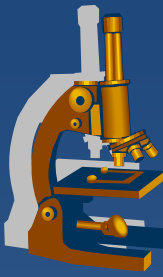
## ***Malignant hypertension***



Fibrinoid necrosis of the hilar arteriole



# *Malignant nephrosclerosis*



**Oedema, mucoid intimal seepage, luminal narrowing in a muscular artery**

# ***Vascular kidney disorders***



## **x Thrombotic microangiopathy (HUS, TTP)**

⇒ *endothelial damage + platelet trombi formation in the systemic microcirculation*

⇒ *platelets consumption*

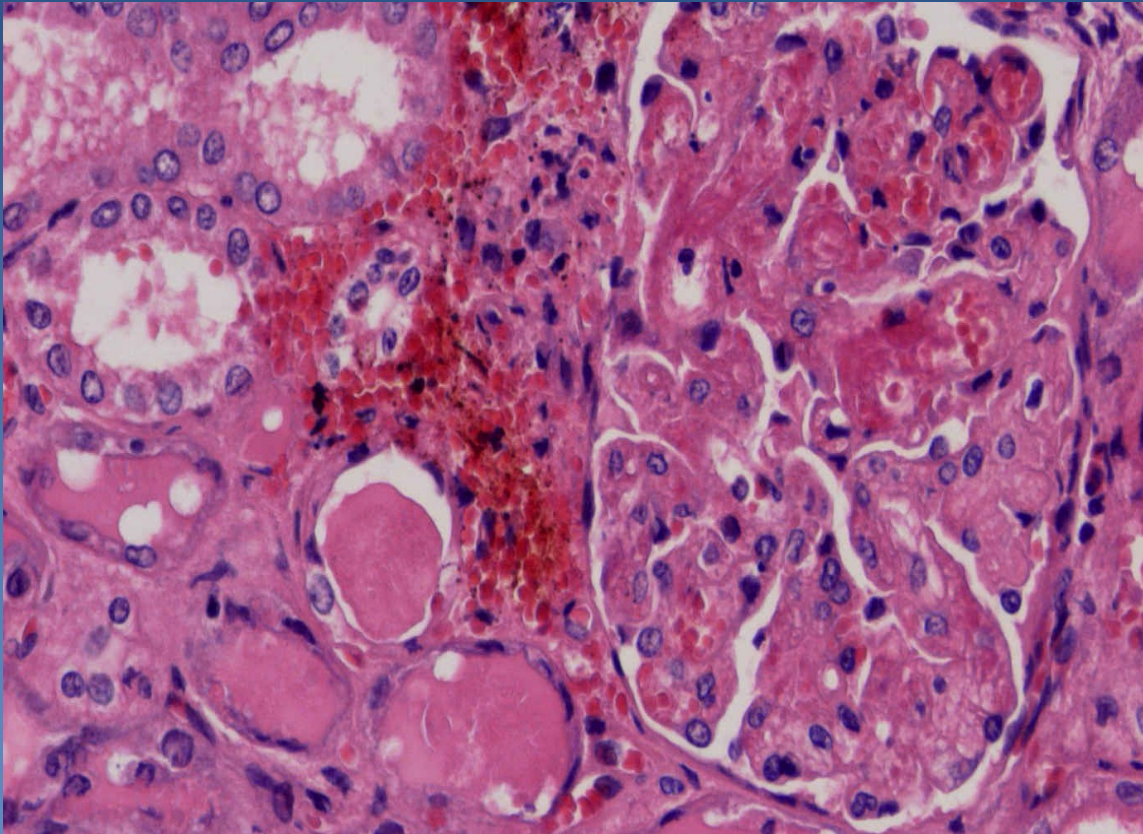
- gross: renal oedema, infarctions possible

- micro: intimal oedema, endothelial distention, platelet trombi, infarctions





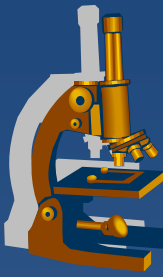
## *Thrombotic microangiopathy*



Luminal thrombi in glomerular capillaries

# *Vascular kidney disorders*

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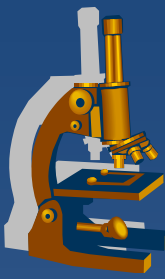
## **x renal infarction**

⇒ *ischemic coagulative necrosis due to blockage of peripherale branches of the renal artery*

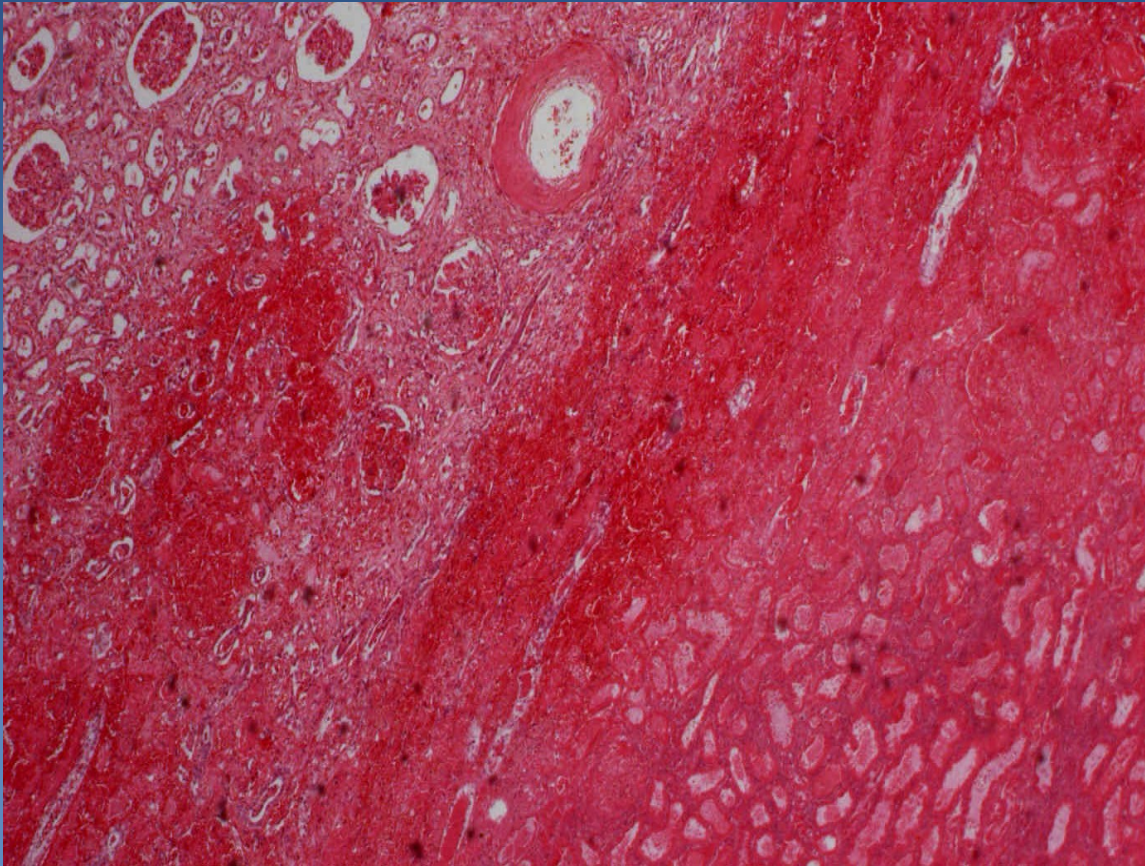
- gross: yellowish conical necrosis
- micro: necrosis with haemorrhagic rim

# *Renal infarction*





## ***Renal infarction***



coagulative necrosis

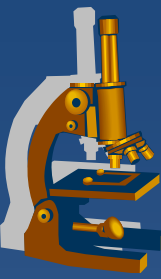


# *Glomerular diseases*

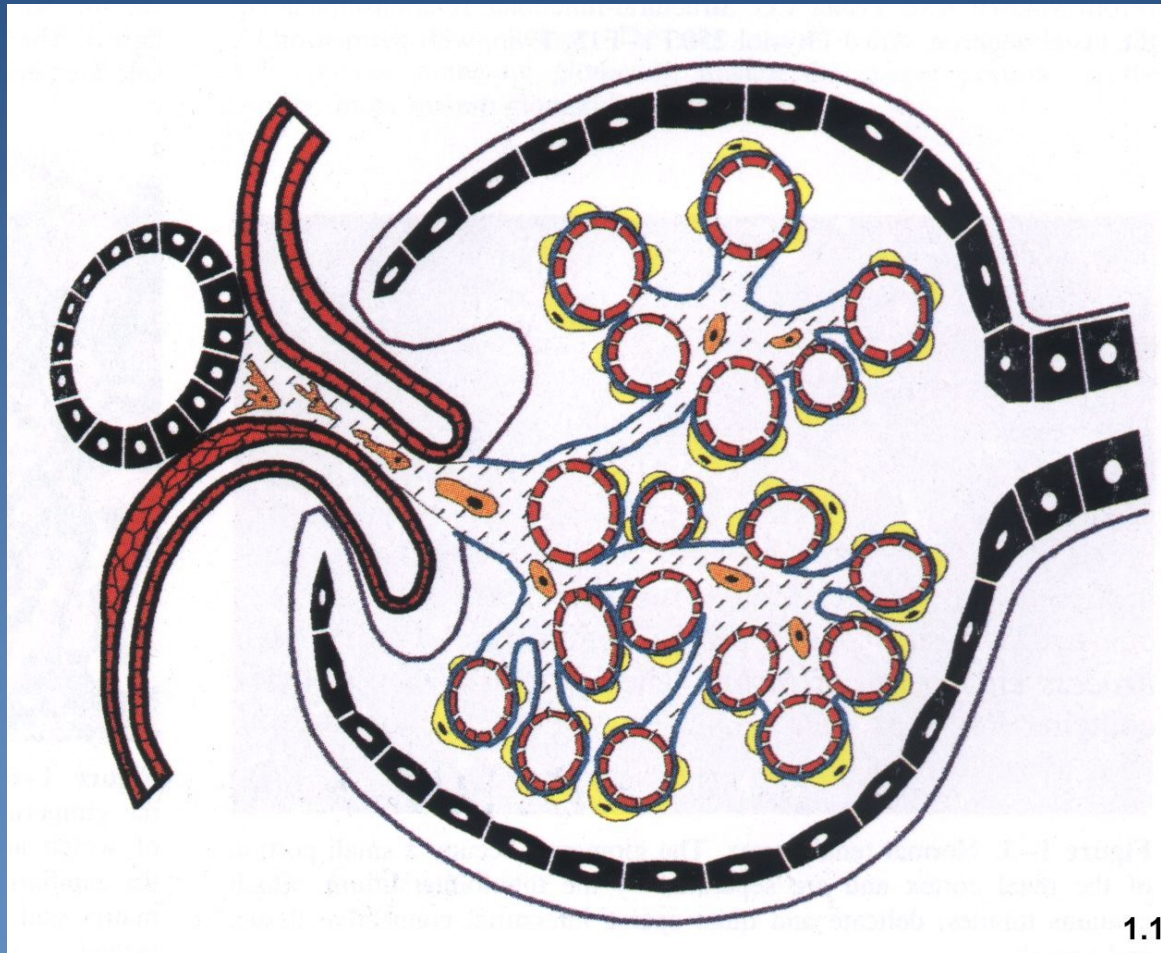
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- × **Glomerular damage caused by various factors**
  - ⇒ *vascular changes*
  - ⇒ *metabolic diseases*
  - ⇒ *familiar diseases*
  - ⇒ *immune-mediated disorders*

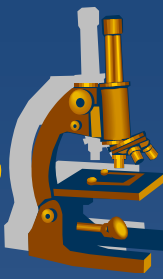


# Normal glomerulus



# ***Mechanism of the glomerular damage***

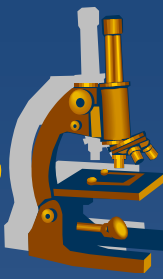
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- x Immune-mediated damage**
  - ⇒ *circulating immune complexes*
  - ⇒ *in situ immune complexes*
  - ⇒ *anti-GBM antibodies*
  - ⇒ *antineutrophilic antibodies*

# ***Mechanism of the glomerular damage***

---



## **x Non-immunological damage**

⇒ *haemodynamic factors*

⇒ *hypertension*

⇒ *ischemia*

# ***Glomerular reaction to the damage***



## **x proliferation:**

⇒ *hyperplasia of mesangial, endothelial, epithelial cells – hypercellularity. Epithelial cells (podocytes) may be a part of crescents filling the Bowman's capsule.*

## **x exudation:**

⇒ *leukocytes + fibrin*

## **x thickening of the glomerular capillary wall**

⇒ *usually due to deposition of immune complexes and/or GBM reaction*

# ***Glomerular reaction to the damage***



## **× sclerosis:**

⇒ *eosinophilic material consisting of the mixture of collapsed membranes, mesangial matrix and plasmatic proteins. PAS + silver impregnation highly positive*

## **× hyalinosis:**

⇒ *foci of refractive amorphous material comprising insudated plasmatic proteins and lipoproteins (PAS intensive positivity, silver impregnation negative)*

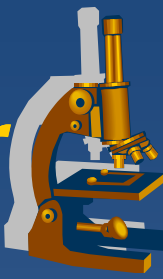
# *Clinical presentation of the glomerular disorders*



- ⇒ *According to the number of affected glomeruli*
  - *diffuse changes (> 50% of gl.)*
  - *focal changes*
  
- ⇒ *According to the extent of glomerular lesion*
  - *global changes (the whole gl.)*
  - *segmental changes*

# ***Clinical presentation of the glomerular disorders***

---



## **x nephritic syndrome:**

⇒ *acute gl. damage, hematuria, proteinuria, oligouria, oedema, hypertension*

## **x nephrotic syndrome:**

⇒ *severe proteinuria with protein loss > 3,5g/24h, hypoalbuminemia, decrease of production of concentrated urine, oligouria → anuria, ↑ azotemia*



# *Clinical presentation of the glomerular disorders*

---



## **x acute renal failure:**

⇒ *sudden decrease of production of concentrated urine, oligouria → anuria, ↑ azotemia*

## **x chronic renal failure:**

⇒ *gradual loss of renal functions*

# ***Glomerular diseases classification***

---



- ✘ Mostly according to the clinical signs
- ⇒ *Glomerulopathy with proteinuria or nephrotic syndrome*
- ⇒ *Glomerulopathy with isolated or predominant hematuria*

# ***Glomerular diseases classification***

---



- ⇒ ***Glomerulopathy with acute nephritic syndrome***
- ⇒ ***Glomerular/kidney involvement by SLE***
- ⇒ ***Chronic glomerulonephritis***

# *Glomerular diseases classification*



## **x primary x secondary GN**

⇒ *primary GN – disorder limited to the kidney, without systemic disease*

⇒ *secondary GN – part of other disease ( SLE, hepatitis C, neoplasia, ... )*

# ***Glomerulopathies manifestated by proteinuria/nephrotic sy***



## **Proteinuria with nephrotic syndrome**

Minimal change disease

Focal segmental glomerulosclerosis

Membranous glomerulopathy

Amyloidosis

Diabetic nephropathy

# *Glomerulopathies with proteinuria/nephrotic sy*



## **x** Minimal glomerular change disease

⇒ *mostly in children's age*

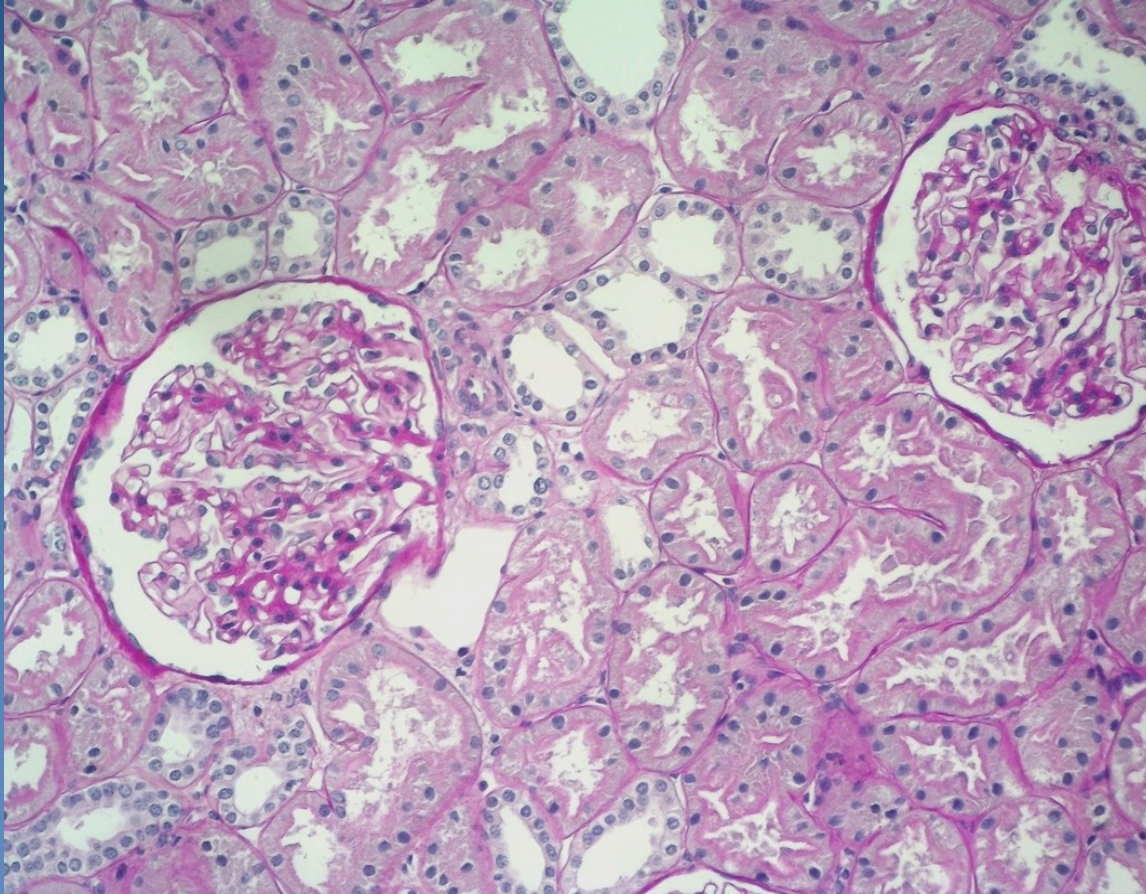
⇒ *heavy selective proteinuria (albuminuria)*

⇒ *nephrotic syndrome responsive to steroid therapy*

⇒ *normal renal functions*

- LM: normal glomerular morphology
- IMF: negative, without immunodeposits
- EM: diffuse fusion of podocytes' foot processes

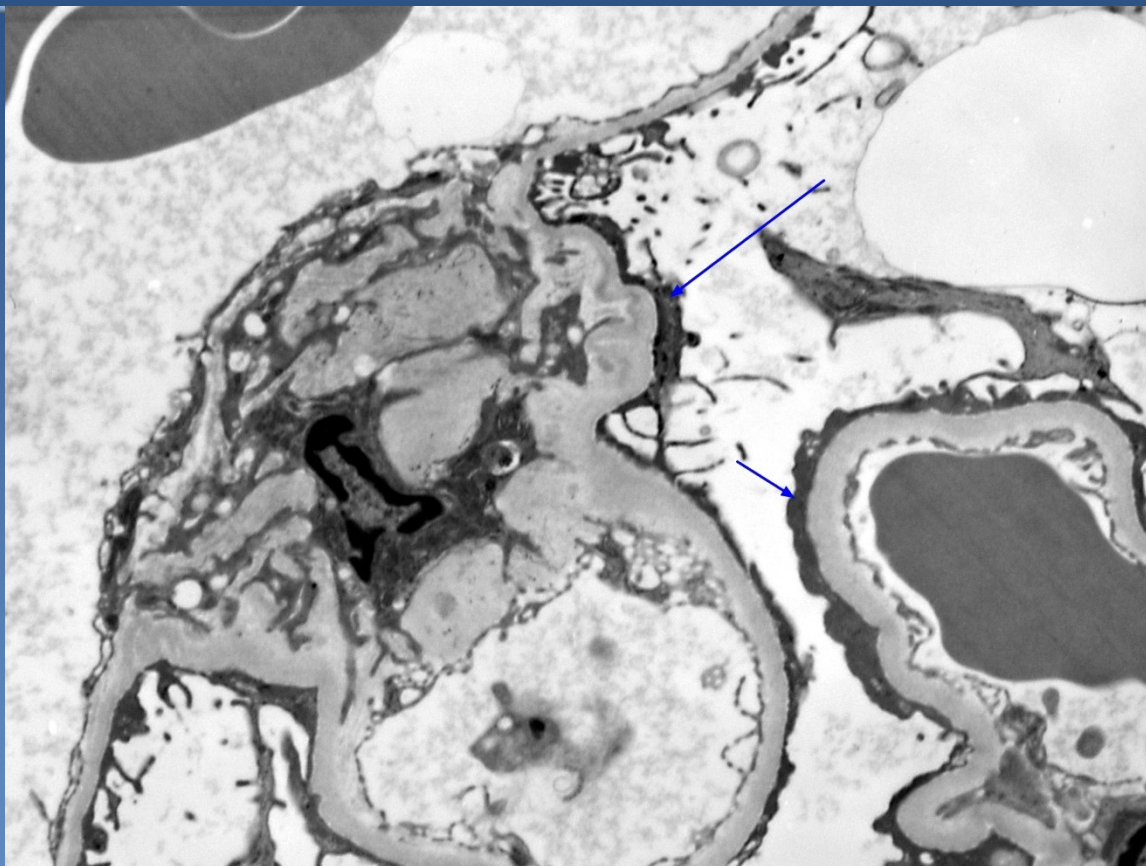
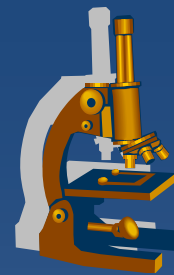
# *Minimal glomerular change disease*



Normal glomerular morphology

# *Minimal glomerular change disease*

*(EM)*



diffuse fusion of podocytes' foot processes



# ***Glomerulopathies with proteinuria/nephrotic sy***



## **x Focal segmental glomerulosclerosis (FSGS)**

⇒ *children, adults (↑ incidence)*

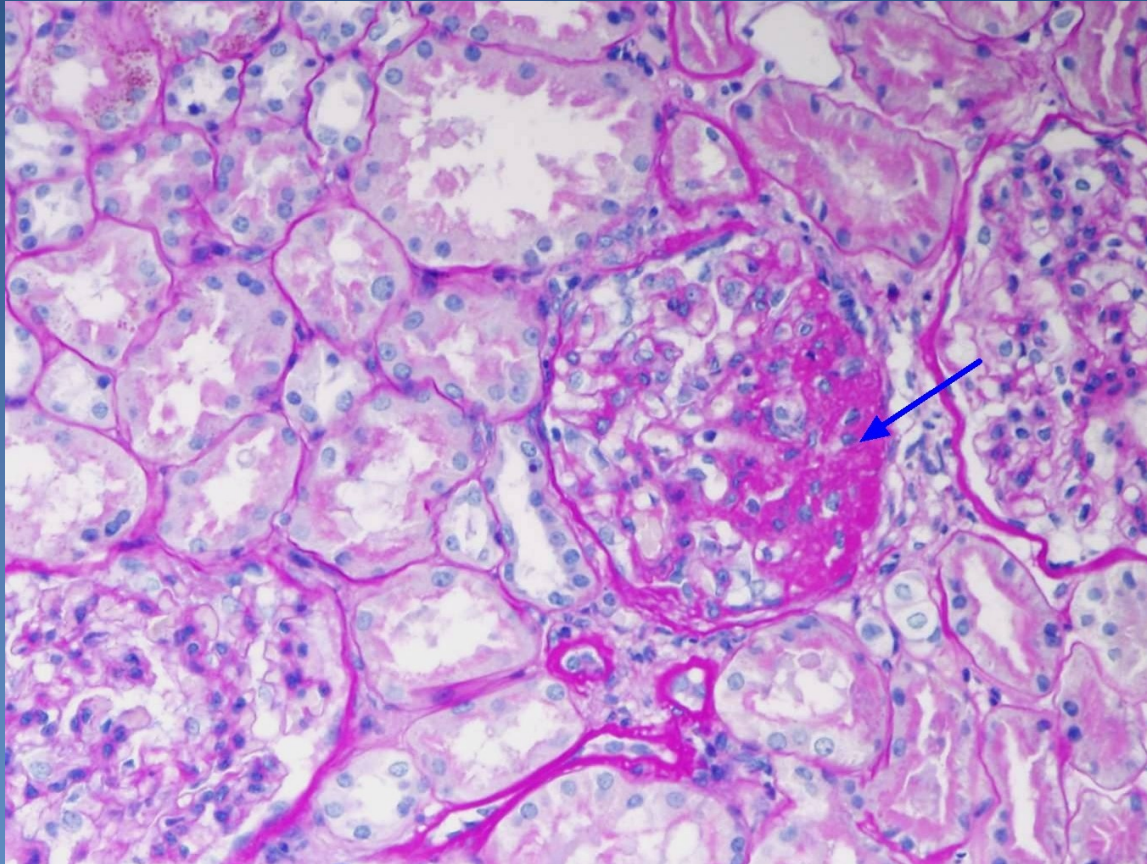
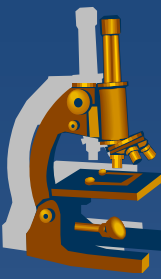
⇒ *non-selective proteinuria up to nephrotic type*

⇒ *nephrotic syndrome, steroid-resistant*

⇒ *gradual progression to the renal failure*

- LM: Focal segmental sclerotic and hyaline gl. changes due to capillary loops collapse and mesangial expansion
- IMF: negative, without immune deposits
- EM: fusion of podocytes' foot processes and podocytes' detachment from the GBM

# FSGS



Segmental sclerosis of the capillary tuft

# *Glomerulopathies with proteinuria/nephrotic sy*

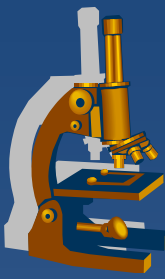


## **x** Membranous glomerulopathy

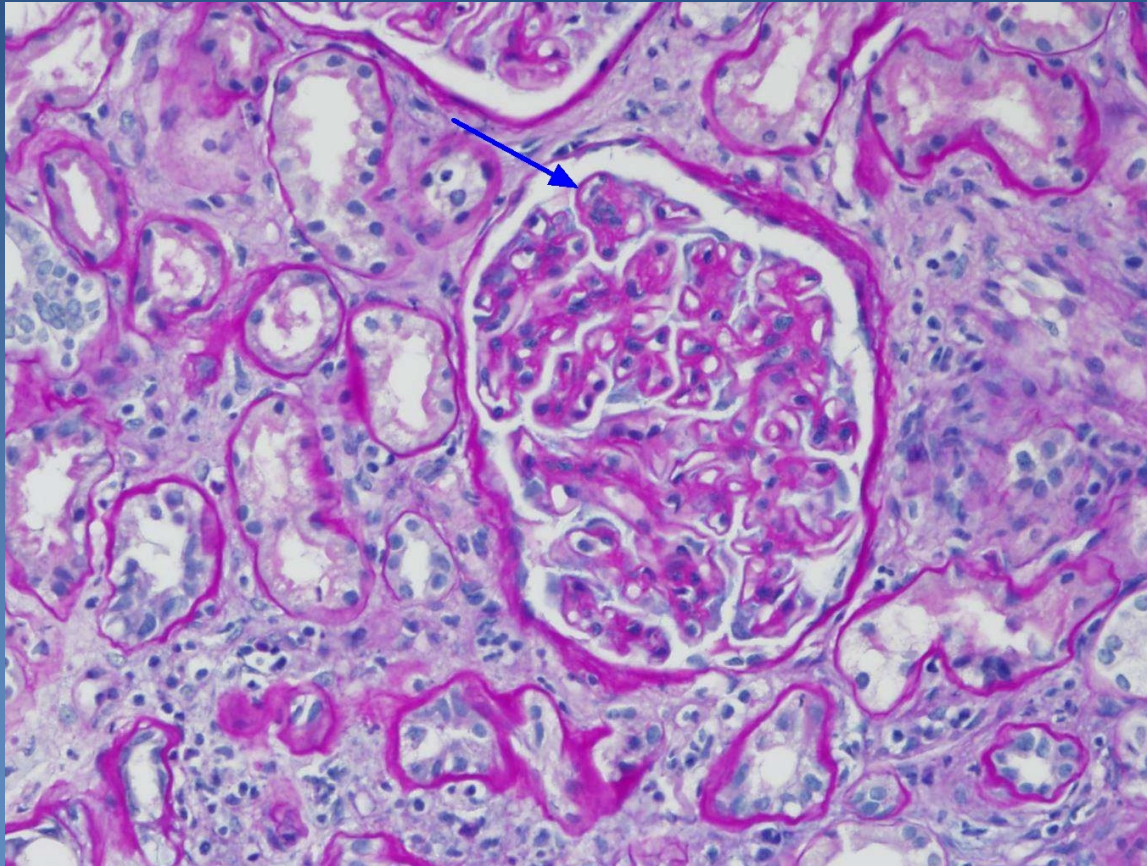
⇒ *immune complex-mediated glomerulopathy, mostly in adults.*

⇒ *proteinuria of nephrotic type, hematuria.*

- LM: diffuse and global gl. involvement, normocellular. Deposition of immune complexes on the outer aspect of the glomerular basal membrane (GBM), thickened in further stages.
- IMF: granular deposits along GBM (IgG, C3)
- EM: subepithelial electron-dense immune deposits



## ***Membranous glomerulopathy***

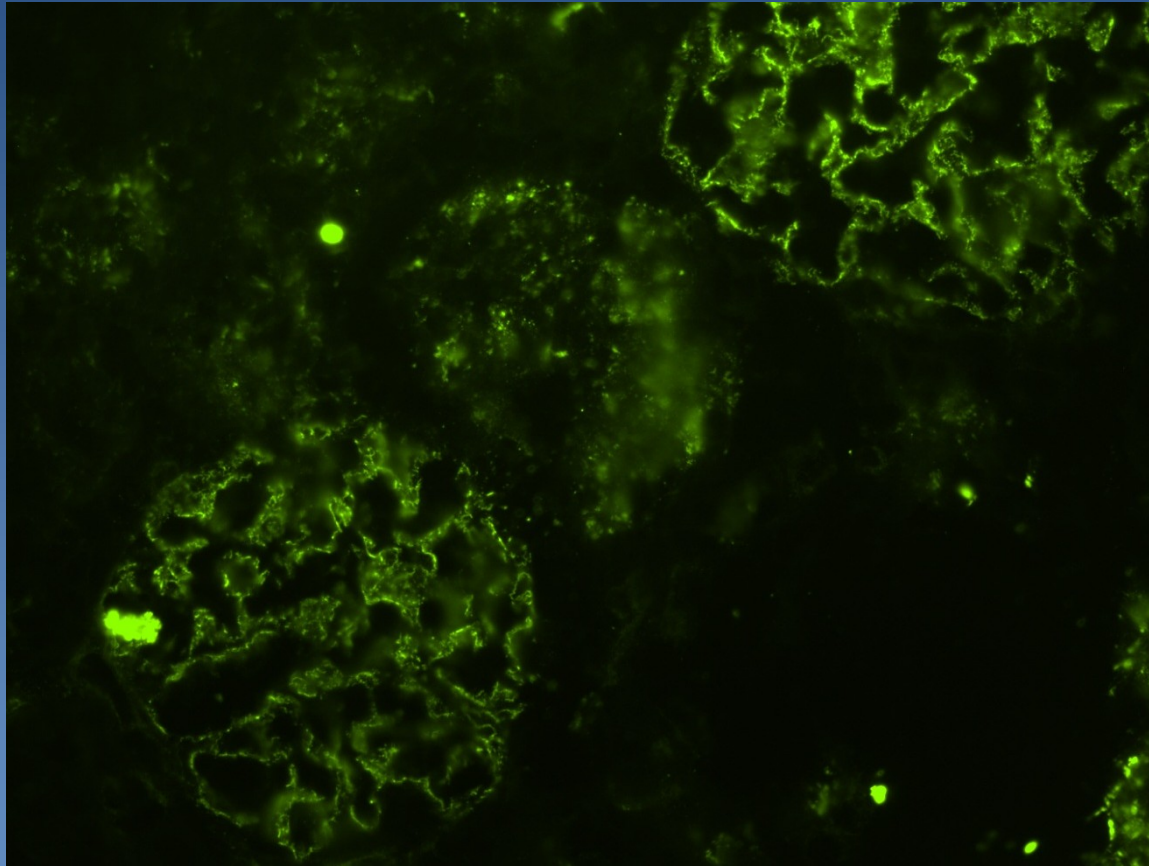


Diffuse GBM thickening

Glomerulus without inflammation or proliferation

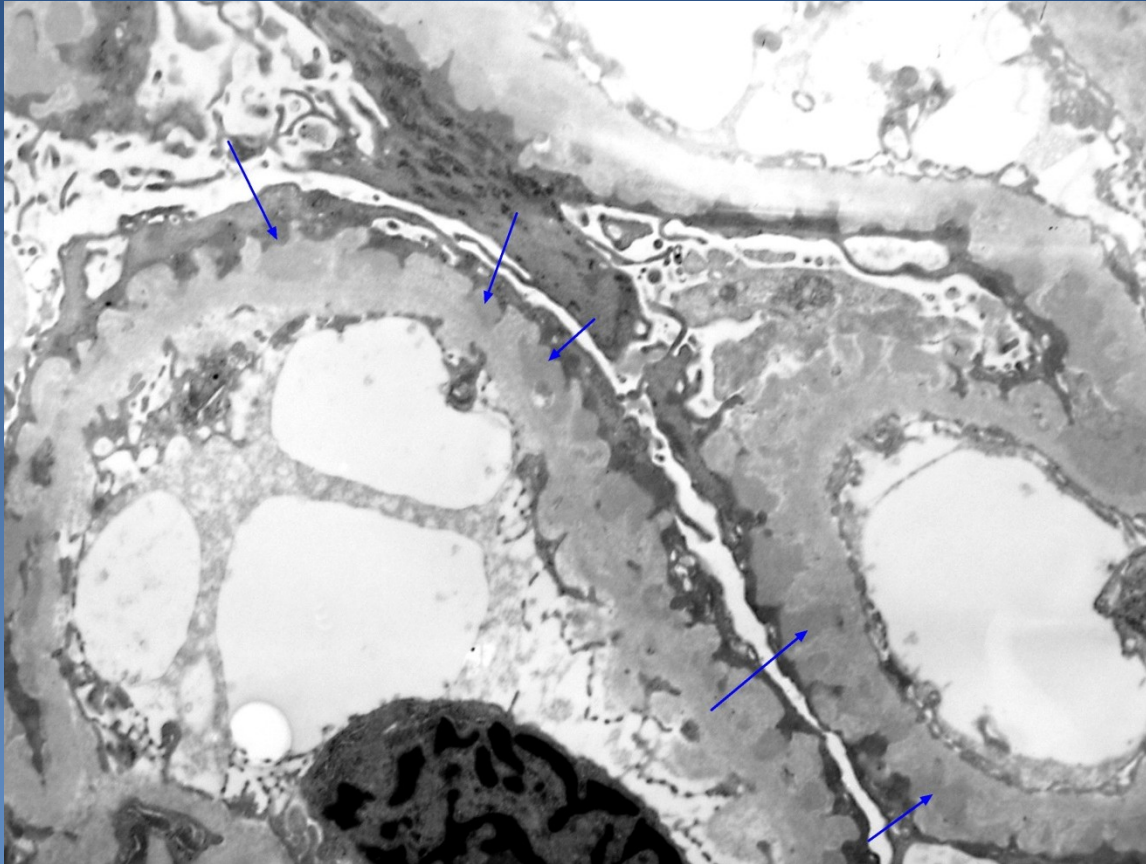


# **Membranous glomerulopathy (IMF)**



Granular deposits along the GBM in IgG

# **Membranous glomerulopathy (EM)**



Diffuse subepithelial (outer aspect of the GBM) immune deposits



# *Glomerulopathies with proteinuria/nephrotic sy*

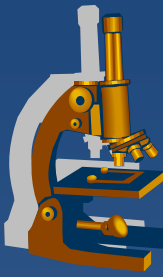


## *x Amyloidosis*

- ⇒ extracellular deposition of pathological fibrillary protein with typical staining features*
- ⇒ systemic amyloidoses most clinically important*
- ⇒ 4 main groups:*
  - AA amyloidosis (SAA protein precursor) in chronic diseases (RA, IBD, ...)*

# *Amyloidosis*

---



- **AL amyloidosis** (precursor - plasma cell product) in monoclonal plasma cell disorders
- **hereditary amyloidosis**: genetically determined protein defect ( transthyretin)
- **amyloidosis associated with haemodialysis**

# Amyloidosis



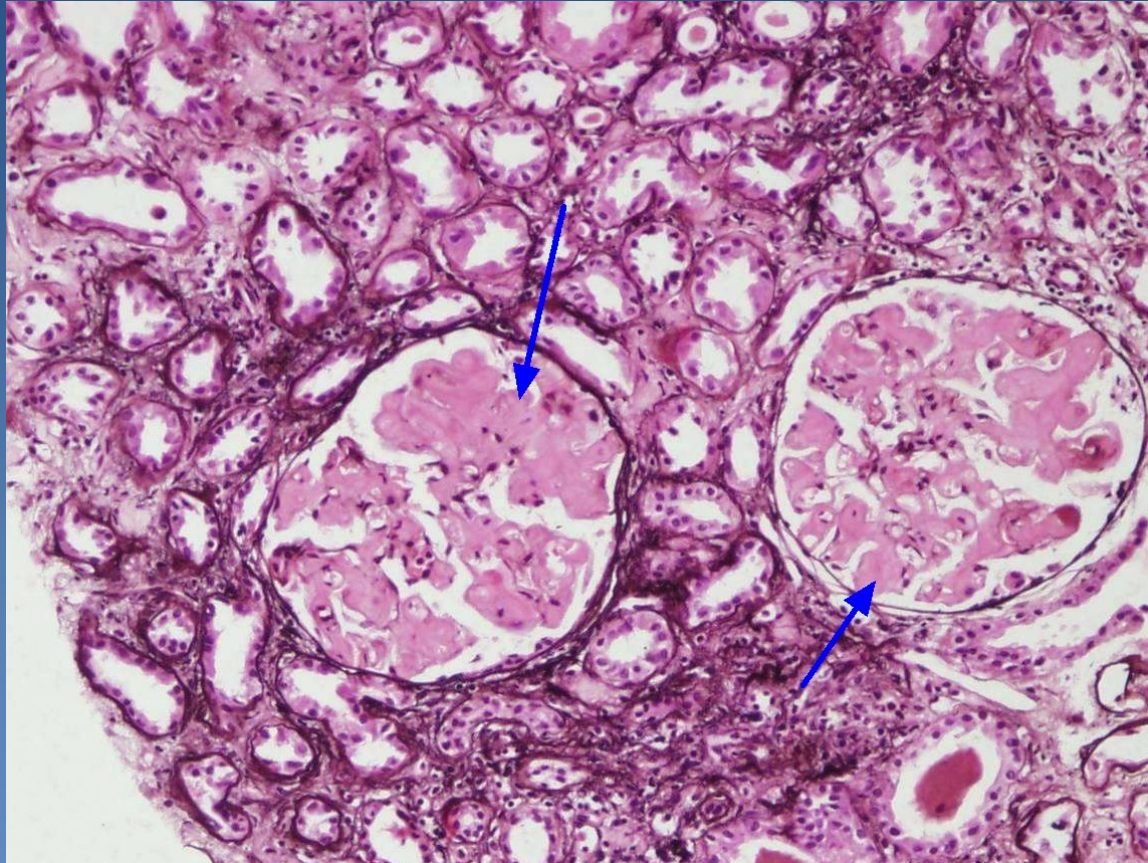
## ⇒ *proteinuria with nephrotic syndrome*

- LM: structure-less eosinophilic masses in the glomeruli, tubules, intersticium and vessels

Positive Congo red staining, green dichroism in polarisation

- IMF: positivity of AA amyloid, light chains
- EM: non-branching, randomly orientated fibrils, size of 6-13nm

# Amyloidosis

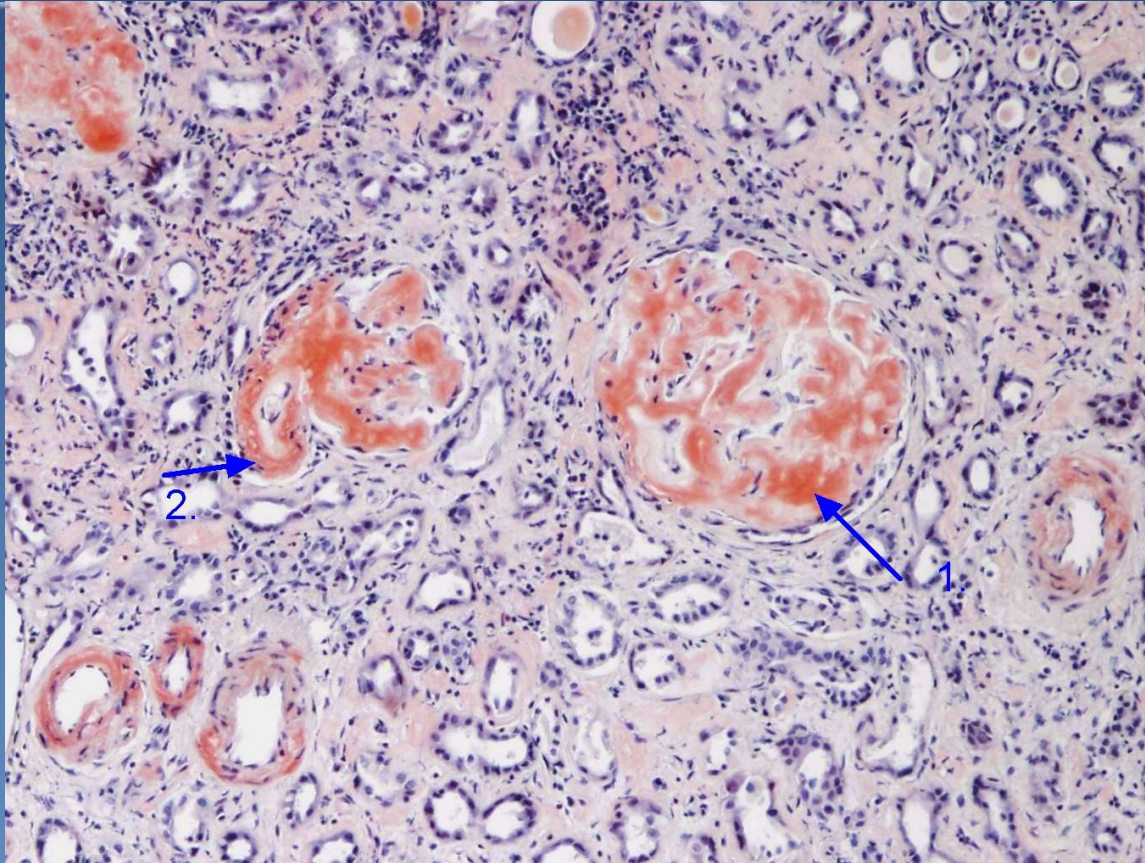


Amyloid deposition in the glomerulus



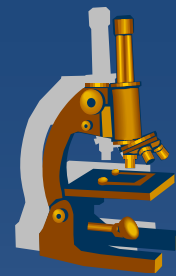


# Amyloidosis



Congo red-positive amyloid deposition in the glomerulus

# *Glomerulopathies with proteinuria/nephrotic sy*



## *x Diabetic glomerulopathy*

⇒ *renal involvement by diabetic microangiopathy*

⇒ *proteinuria of nephrotic type*

- LM: thickening of GBM, mesangial expansion by PAS positive mesangial matrix, mildly increased cellularity, glomerular enlargement – **diffuse diabetic glomerulosclerosis**



# *Diabetic glomerulopathy*



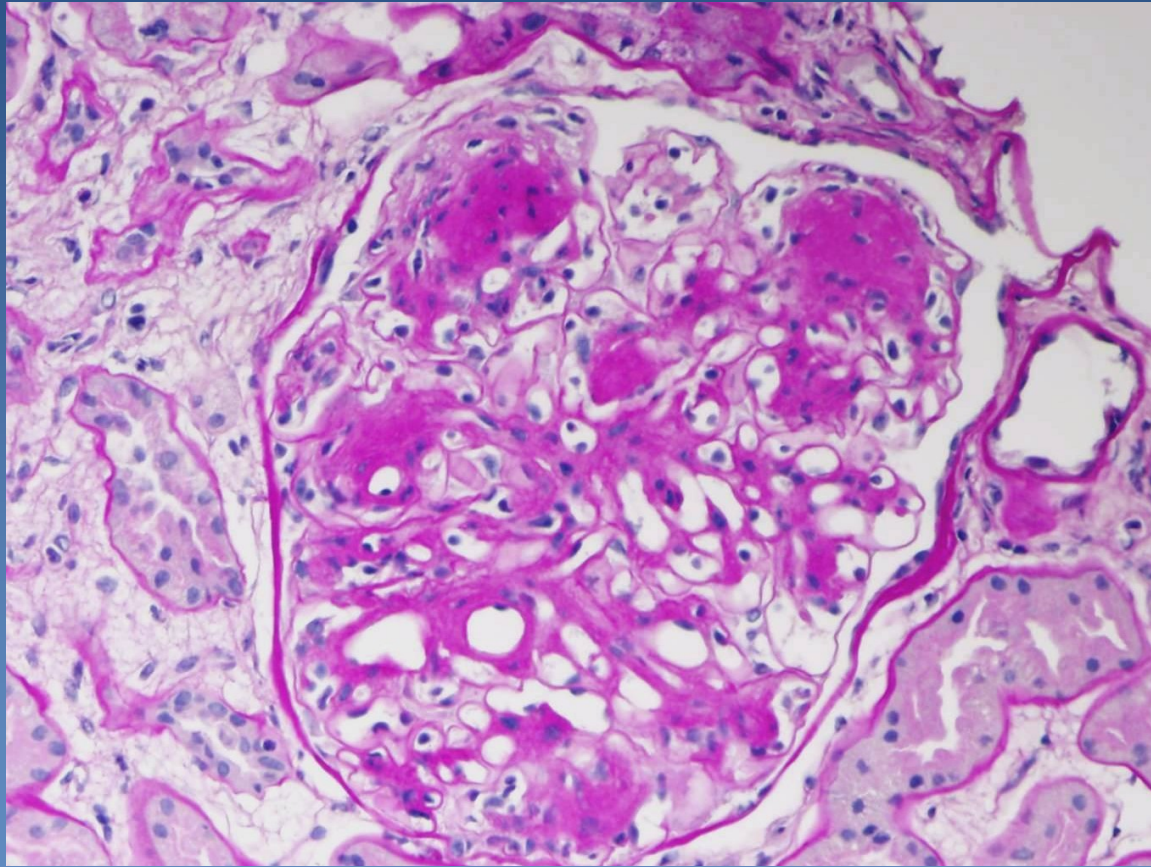
- later homogenous eosinophilic nodular formations, mesangial cells pushed to the periphery – **nodular diabetic glomerulosclerosis** .

Hyalinne insudations in arterioles

- IMF: without immune deposits
- EM: thickening of GBM



# *Diabetic glomerulopathy*



Mesangial nodules

# *Glomerulopathies with haematuria*



Glomerulopathies with isolated or prevalent haematuria

IgA nephropathy (Berger's disease)

Henoch-Schönlein purpura

Alport syndrome / thin basement membranes sy

# ***Glomerulopathies with haematuria***



## **x IgA nephropathy (Berger's disease)**

- ⇒ immune complex-mediated disorder with raised levels of circulating IgA***
- ⇒ IgA mesangial deposits by chronic GIT, respiratory tract mucosal inflammations, liver cirrhosis***
- ⇒ episodic macroscopic haematuria in coincidence with respiratory infection***

# ***Glomerulopathies with haematuria***

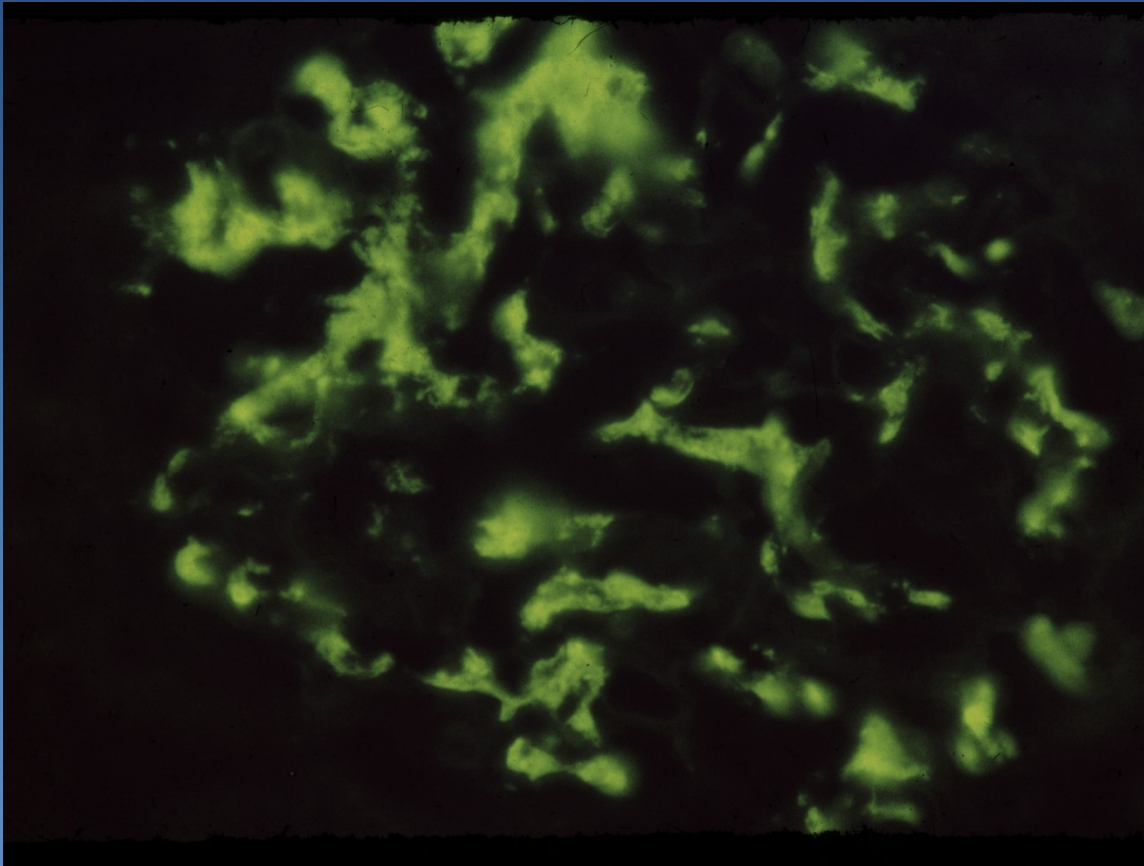


- LM: mesangial proliferation
- IMF: mesangial IgA granules
- EM: Mesangial and paramesangial ID

## **x Henoch-Schönlein purpura**

- ⇒ *extensor skin vasculitis with purpuric rash, GIT manifestations, arthralgia*
- ⇒ *renal involvement - IgA nephropathy*

# *IgA nephropathy IMF*



Mesangial IgA immune deposits



# ***Glomerulopathies with haematuria***



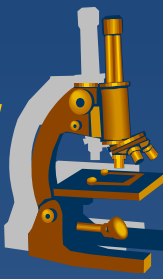
## **x Alport syndrome/ thin basement membrane lesion**

**⇒ mutation in genes for collagen IV, part of basement membranes, (mostly gene COL4A5 encoded on the X. chromosome).**

**⇒ gradual progression of renal failure**

**⇒ in the fully evolved Alport sy – bilateral hearing disorders, ocular abnormalities**

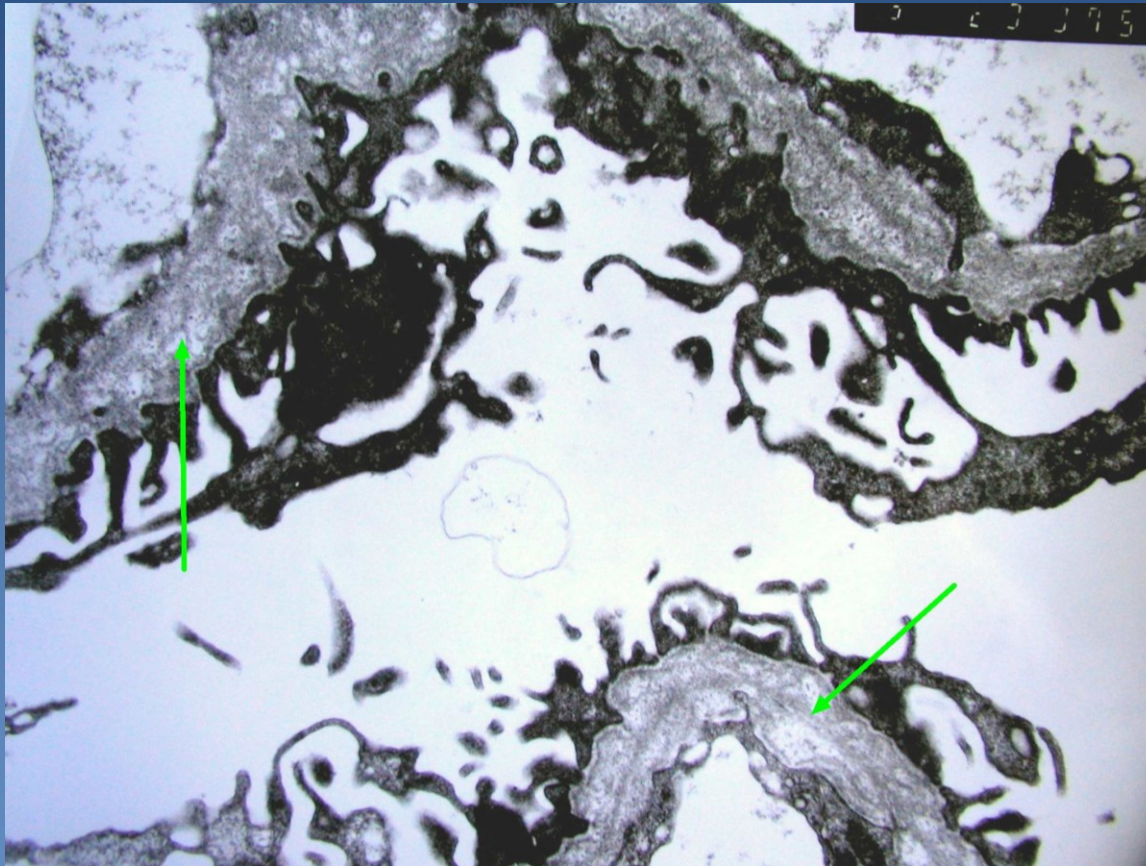
# ***Alport syndrome/ thin basement membrane lesion***



## **x thin basement membrane lesion**

- ⇒ without progression into renal failure, mild clinical signs (benign familial haematuria)***
- ⇒ typical morphology possible in female carriers of X-linked Alport syndrome***

# ***Alport syndrome/ thin basement membrane lesion ELMI***



Characteristic picture of lamellar glomerular basement membrane in hereditary nephropathy.

# ***Glomerulopathies with acute nephritic syndrome***



## **Glomerulopathies with acute nephritic syndrome**

Acute diffuse endocapillary proliferative GN

Membrano-proliferative GN

Rapidly progressive GN (RPGN)

# ***Glomerulopathies with acute nephritic syndrome***



⇒ *usually proliferative glomerulonephritis with increased mesangial and endocapillary cellularity, commonly with crescent formation.*

## **x Acute diffuse endocapillary proliferative GN**

- ⇒ *syn. acute post-infective, acute proliferative, exudative GN*
- ⇒ *immune complex-mediated disorder*

# ***Acute diffuse endocapillary proliferative GN***



- ⇒ usually ***post-infective glomerulonephritis*** ( *beta-hemolytic streptococcus, staph., G-bacteria, viruses, parasites* )
- ⇒ ***systemic disorders*** (*SLE, infective endocarditis, necrotising arteritis*) may be accompanied by this GN
- ⇒ *most commonly children , 1-4 wks. after streptococcal infection*



# *Acute post-infective GN*

---



- ⇒ *haematuria, proteinuria , hypertension, oedemas, renal failure*
- ⇒ *possible asymptomatic course*
- ⇒ *raised **ASLO titre** and drop of C3 ,C4 complement in serum*

# ***Acute post-infective GN***



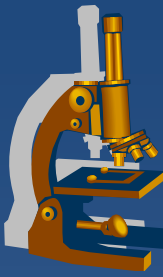
⇒ *benign course in children*

⇒ *protracted course in adults, with hypertension, variable grade of renal failure*

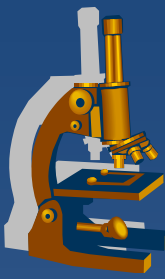
- LM : ↑ endocapillary and mesangial cellularity, capillary lumen compression

# *Acute post-infective GN*

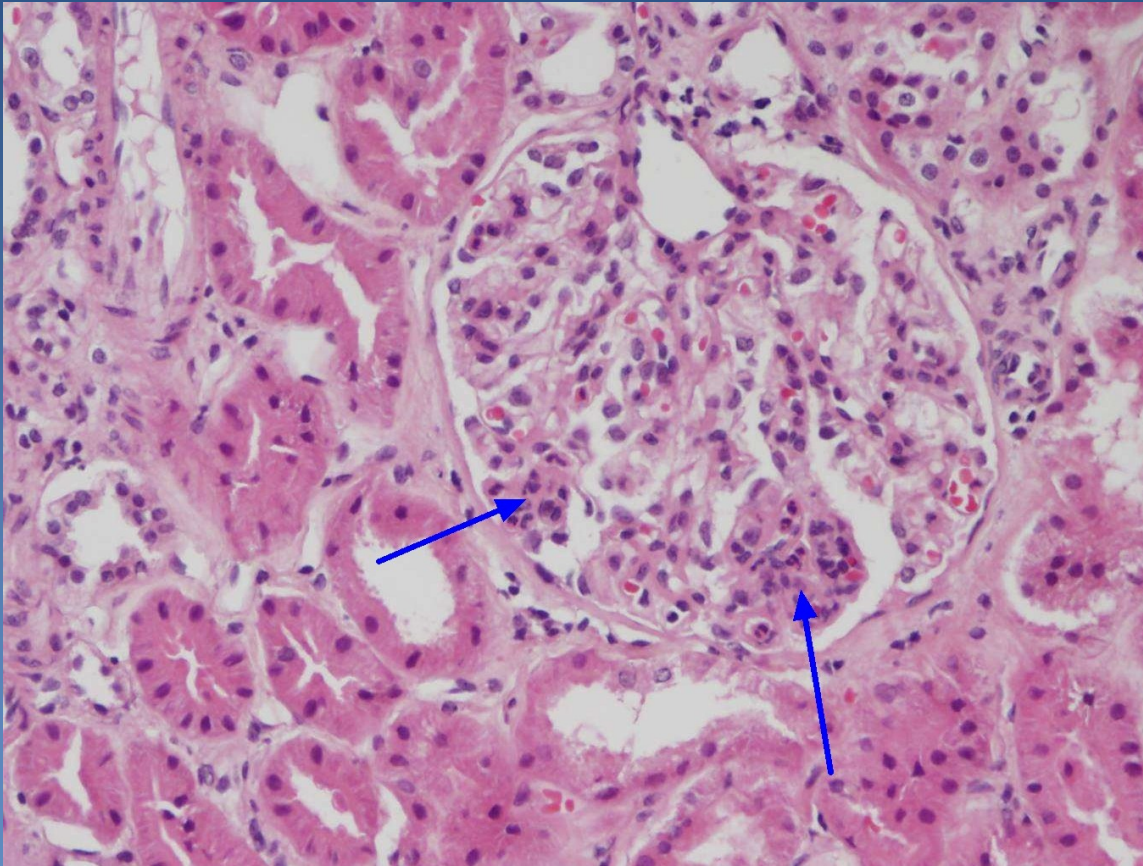
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- IMF: diffuse segmental IgG and C3 granules in capillary loops, in mesangium
- EM: humphs – electron-dense subepithelial immune deposits

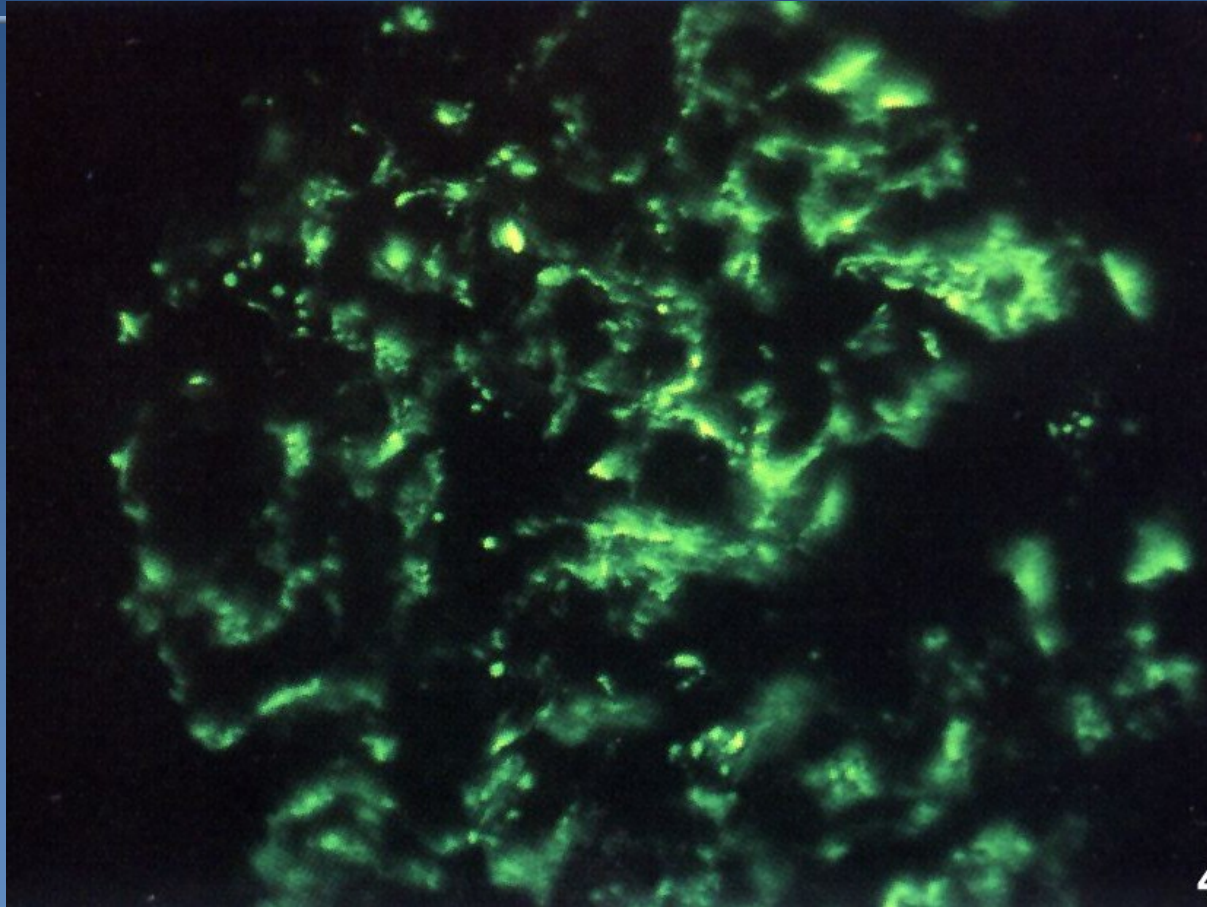


## ***Acute post-infective GN***



hypercellularity, neutrophils

# *Acute post-infective GN (IMF)*

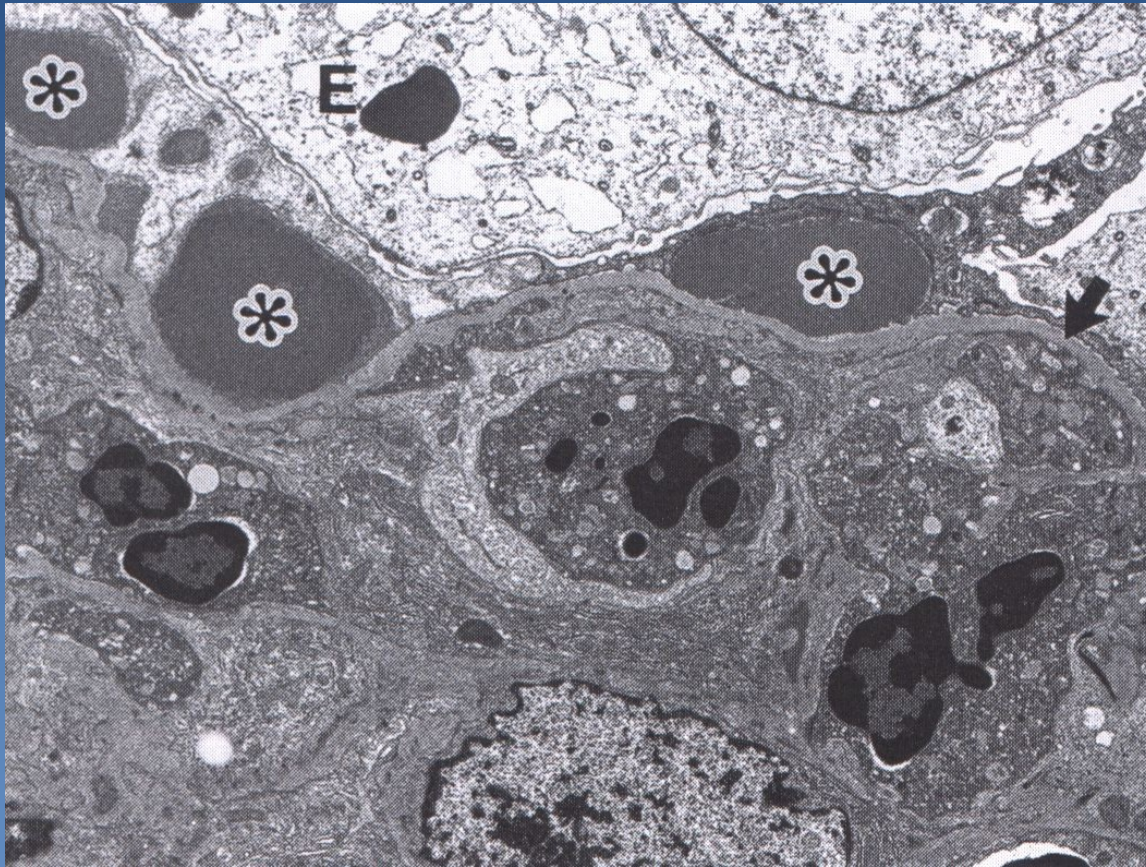


Granular IgG deposits on GBM and in mesangium





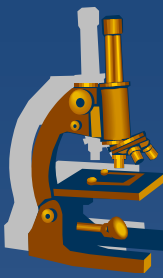
## ***Acute post-infective GN (EM)***



Granular subepithelial deposits



# Glomerulopathies with acute nephritic syndrome



## × Membrano-proliferative GN (mesangio-capillary)

⇒ *Type I.-III. according morphology*

⇒ **Type I.** – *immune complex-mediated, cryoglobulinemia (esp. hepatitis C), other causes - more common in children, teens*

⇒ ↓ *serum complement, nephritic syndrome, nephrotic sy possible.*

- LM: diffuse glomerulopathy, endocapillary and mesangial hypercellularity, accentuation of capillary tuft lobular architecture,

GBM duplication („tram track“) in PAS, silver impregnation.

# Membrano-proliferative GN



- EM: subendothelial immune deposits + mesangial interposition (inclusion of mesangium + new layer of BM inbetween the immune deposits and original BM – duplication, „splitting“), subendothelial + mesangial ID.

⇒ **Type II.**

⇒ **> 60% of patients with antibody C3nephritic factor (NeFa) binding to C3 convertase → stabilisation (no enzymatic degradation), → permanent C3 activation of alternative pathway of complement cascade**

# Membrano-proliferative GN

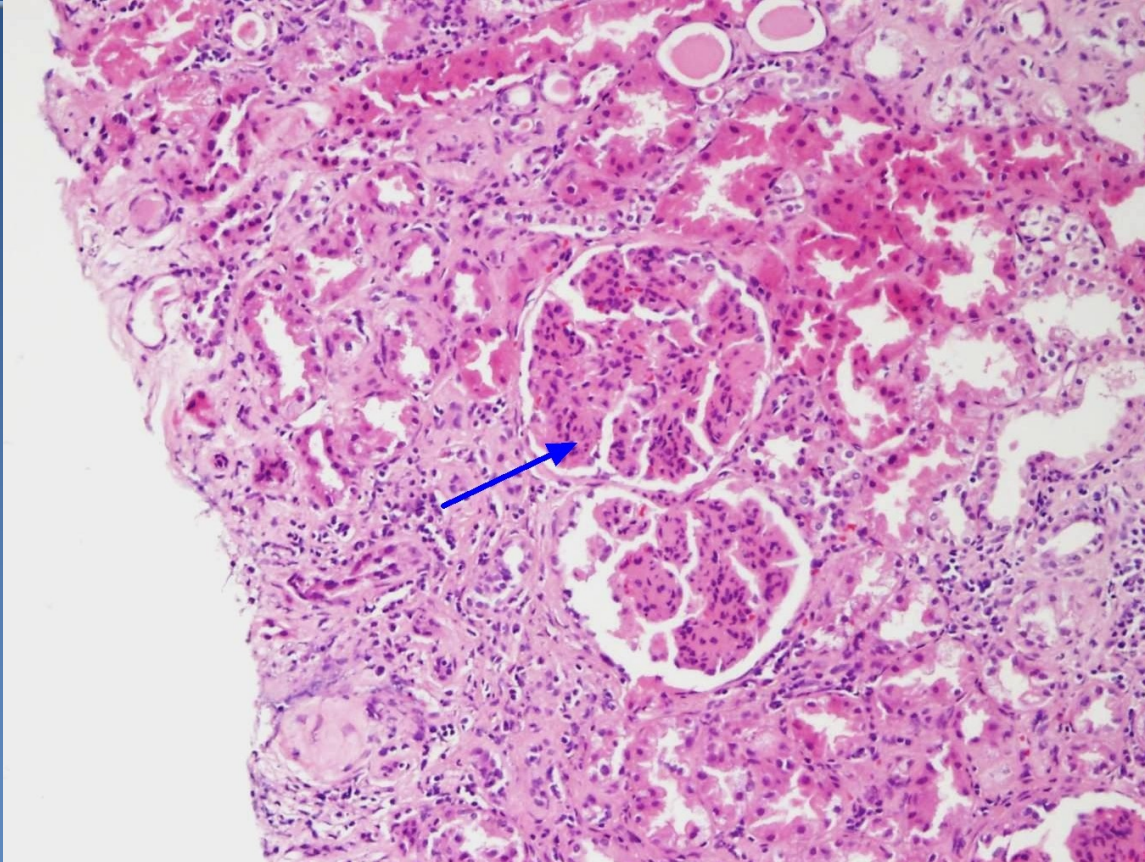


- EM: dense-deposit disease (DDD). Ribbon-like immune deposits in the GBM and mesangium,

⇒ *Typ III. rare*

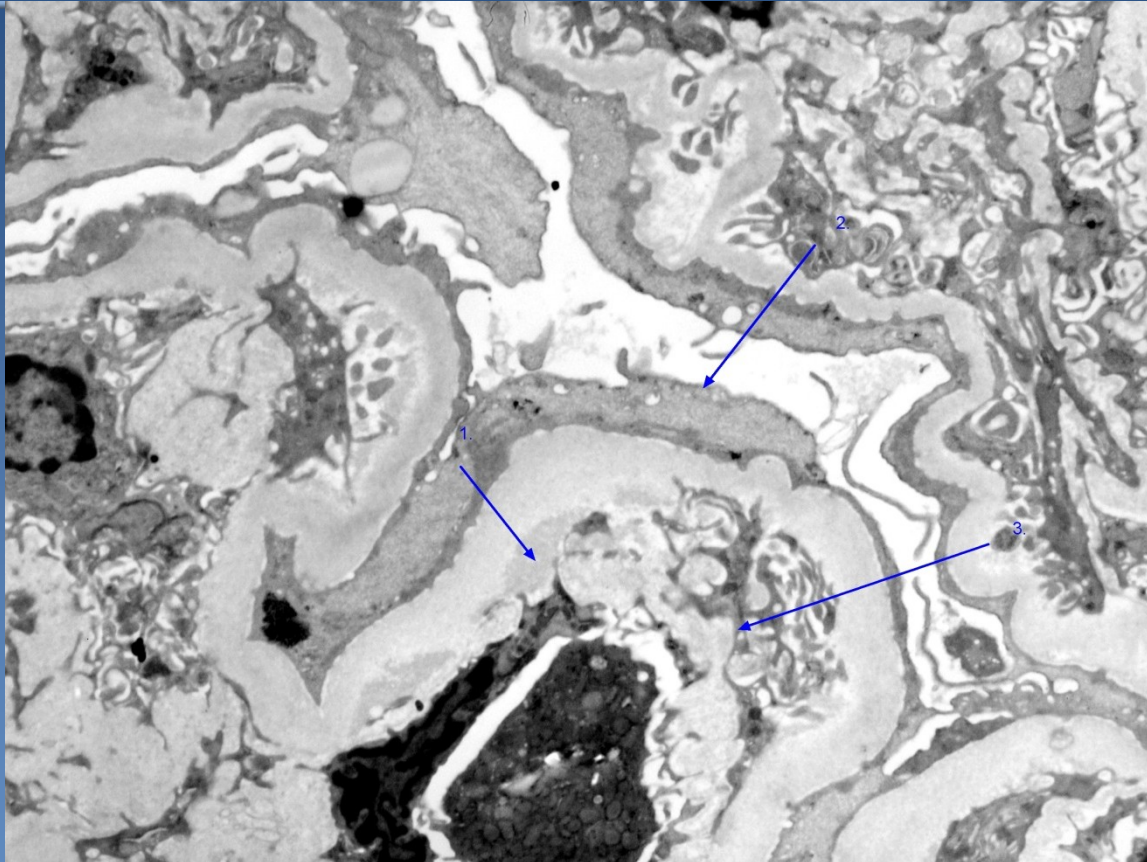
- LM: same findings as in the type I.
- EM: + **subepithelial ID.**

## ***Membrano-proliferative GN***



Lobulisation of the capillary tuft, hypercellularity in mesangium + endocapillary

# Membrano-proliferative GN (EM)



1. Subendothelial immune deposits
2. podocyte foot processes fusion
3. mesangial interposition

# ***Glomerulopathies with acute nephritic syndrome***



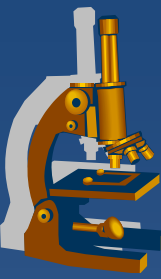
- ✘ Rapidly progressive GN (RPGN), crescentic**
  - ⇒ *Hematuria, proteinuria***
  - ⇒ *Rapid loss of renal functions***
  - ⇒ *Extensive crescentic formation***



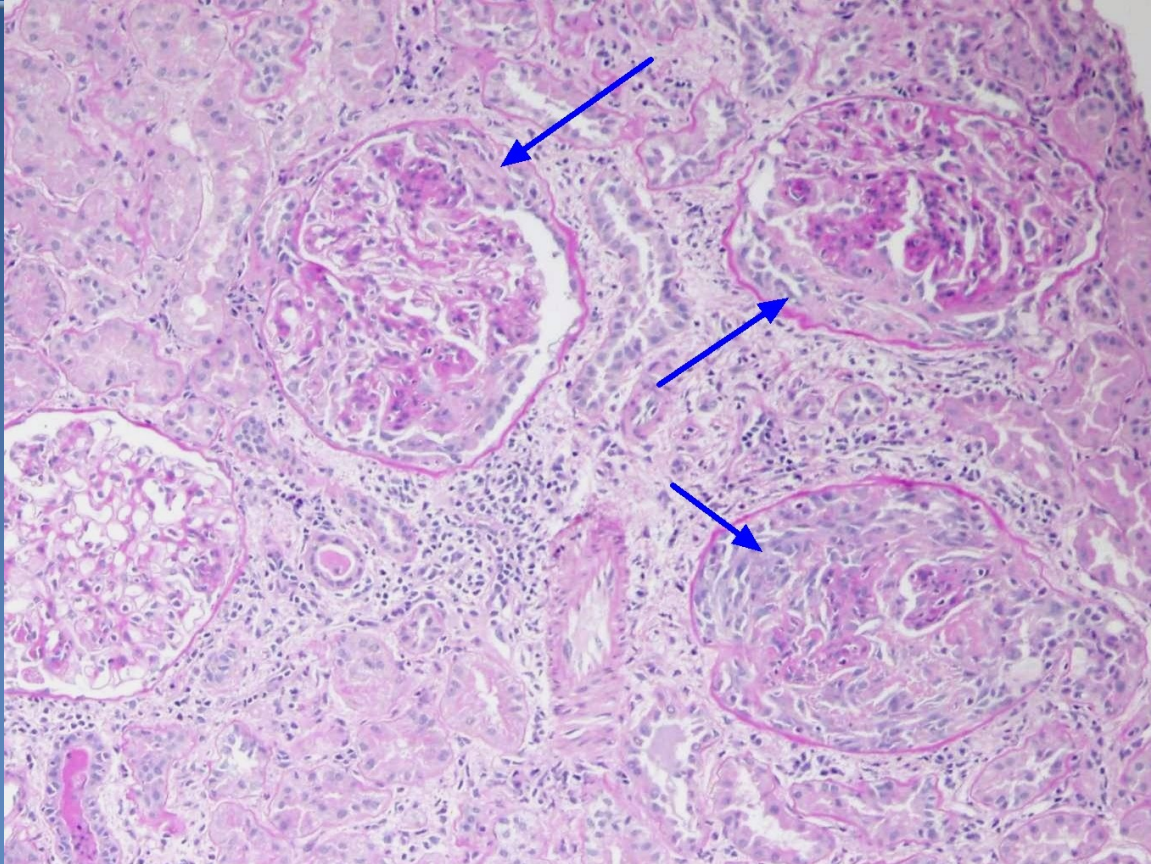
# RPGN



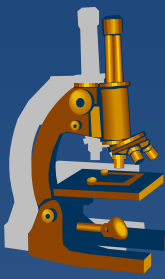
- ⇒ *Variable group of diseases:*
- ⇒ *pauci-immune GN (part of systemic vasculitis, sm. ANCA+)*
- ⇒ *Anti-GBM disease*
- ⇒ *immune-complex mediated GN*  
complication of other GN (IgA, post-infectious GN, SLE)



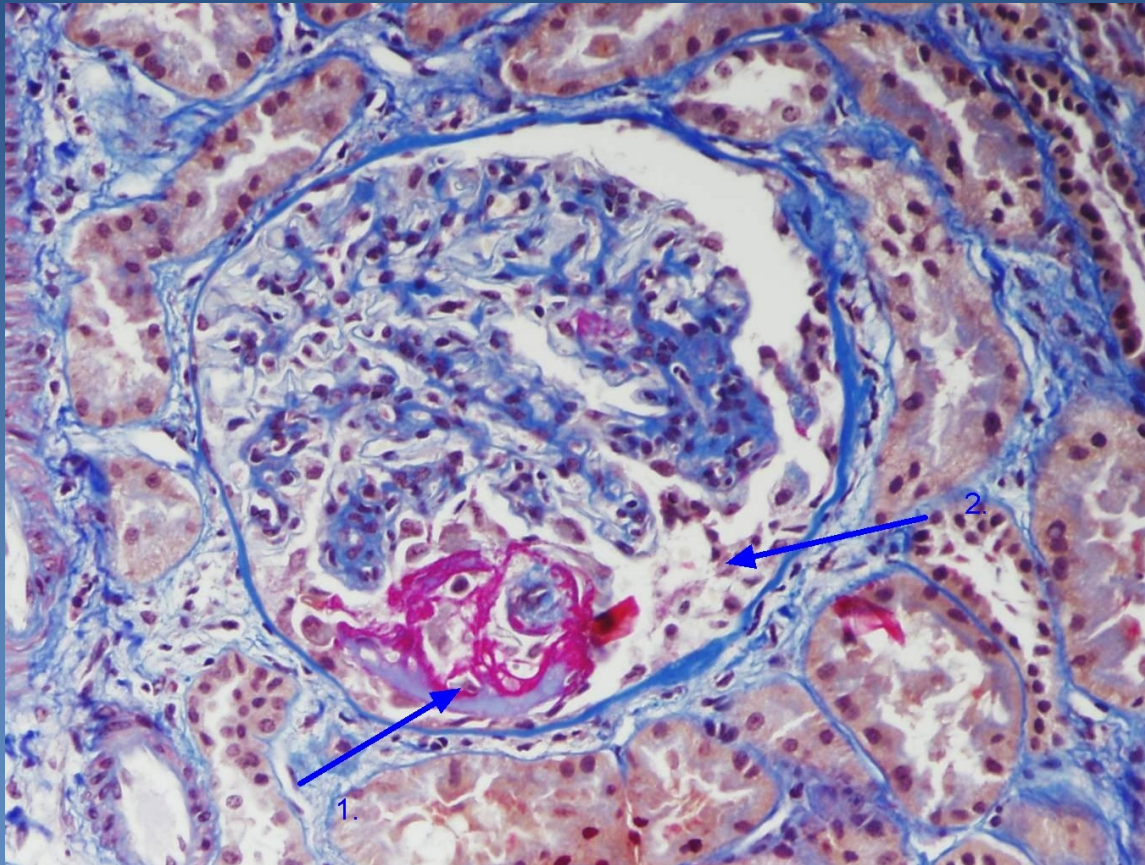
# **RPGN**



Cellular crescents within the Bowman capsule

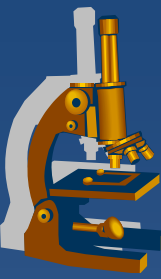


# RPGN

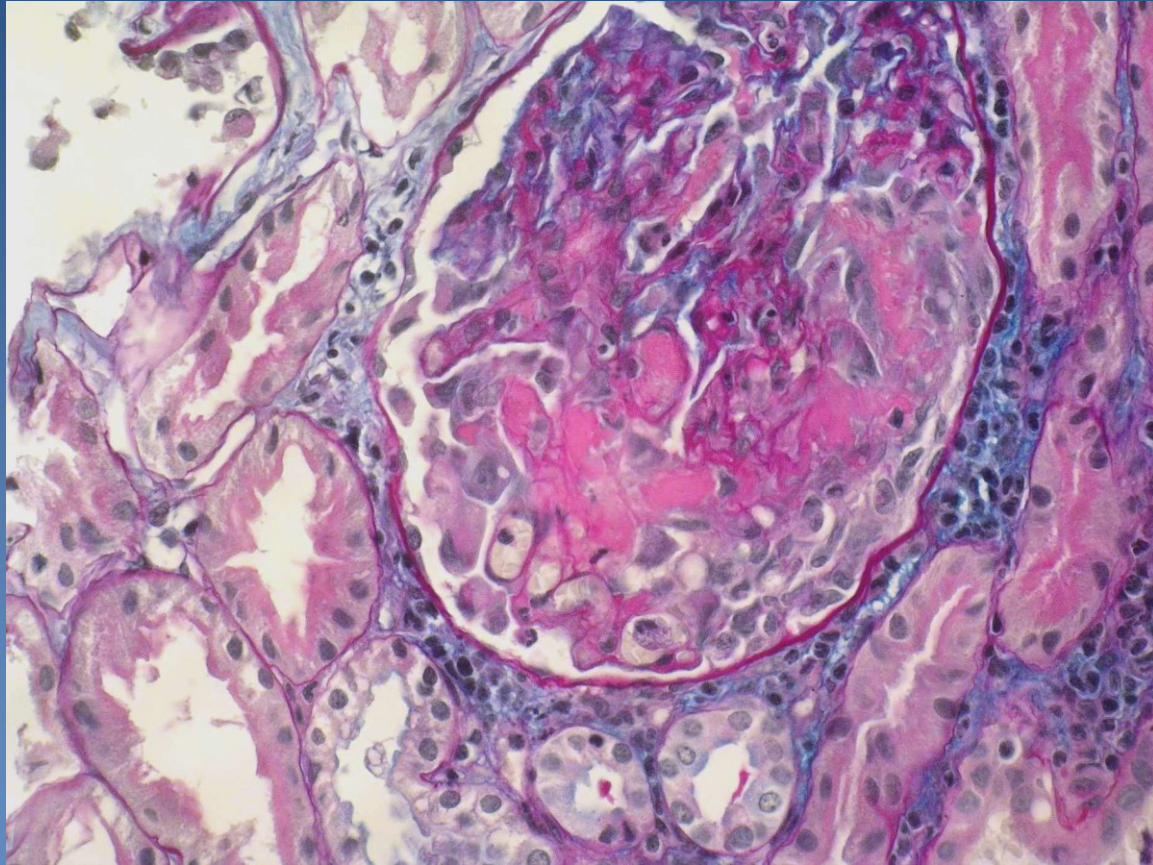


1. Fibrin in the crescent
2. Cellular crescent (incipient)



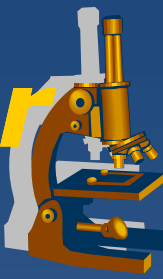


## ***RPGN***



Fibrinoid necrosis of capillaries

# Vascular kidney/glomerular diseases



Systemic vasculitis	anti-GBM vasculitis immune-complex mediated vasculitis ANCA-associated vasculitis
Hypertensive kidney disorders	
Thrombotic microangiopathy	
Others	renal infarction renal artery stenosis

# *Vascular kidney/glomerular diseases*

---



- x *Systemic vasculitis***
- x *Anti-GBM glomerulonephritis***
  - ⇒ *antibodies against Goodpasture antigen (part of noncollagenous portion of the GBM)***



# ***Vascular kidney/glomerular diseases***

---



⇒ ***binding of anti-GBM antibody → complement + proteases activation → GBM destruction***

⇒ ***LM: RPGN appearance***

- IMF: diffuse linear IgG deposits positivity of GBM

## ***Immune complex-mediated vasculitis***

⇒ ***Henoch-Schönlein purpura***

- IgA nephropathy morphology

# *Vascular kidney/glomerular diseases*

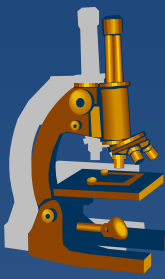


✘ ANCA-associated vasculitis (antineutrophil cytoplasmic antibodies)

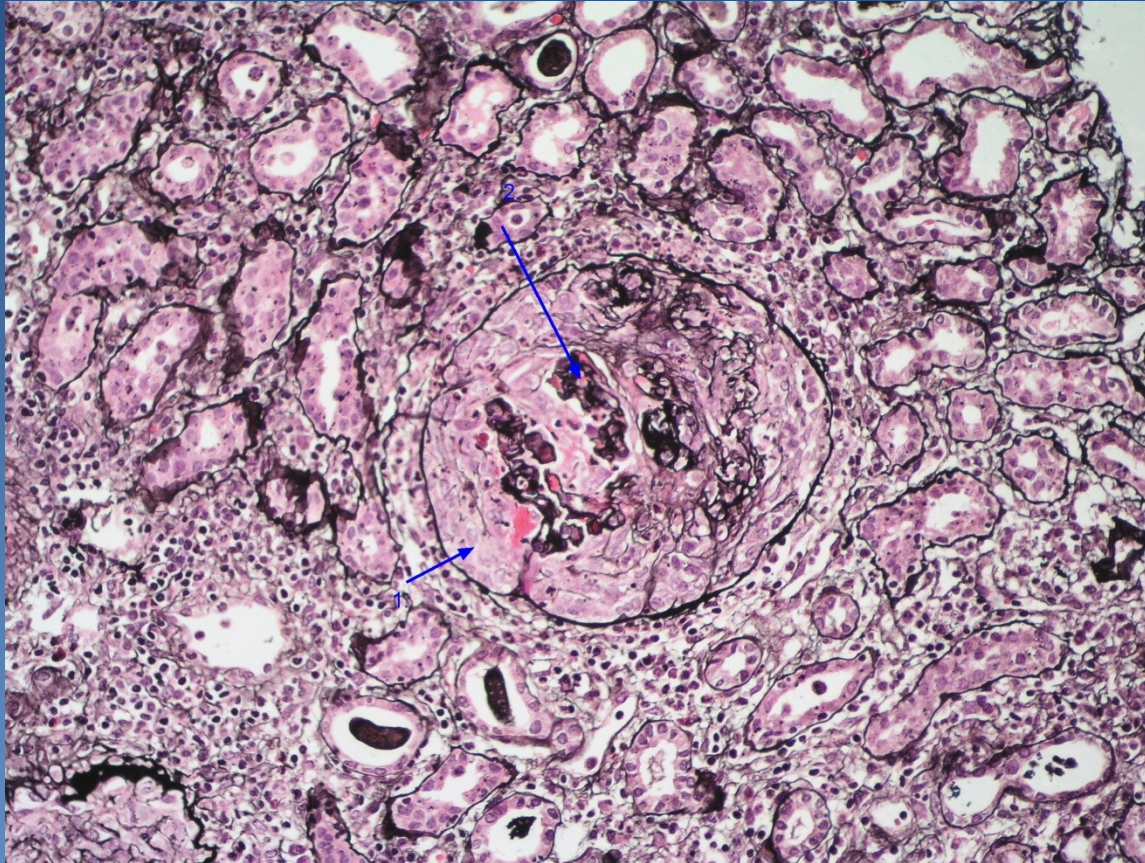
⇒ *Granulomatosis with polyangiitis (Wegener granulomatosis)*

⇒ *Microscopic polyangiitis*

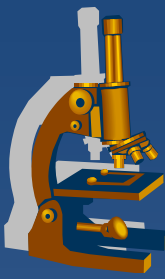
- RPGN morphology



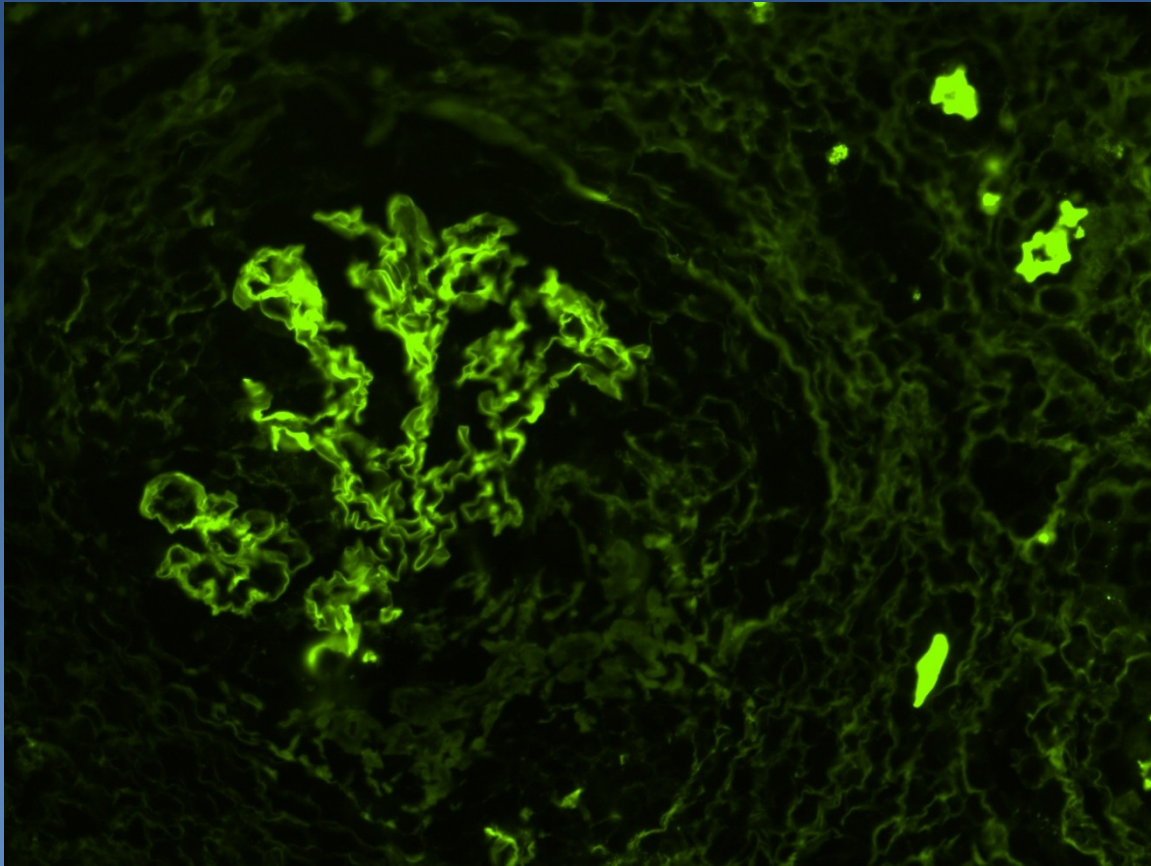
## *Anti - GBM*



1. Cellular compressive crescent
2. Collapsing capillary tuft



## *Anti-GBM (IMF)*



Linear peripheral IgG positivity ( on the GBM)

# ***Vascular kidney/glomerular diseases***



## **x Thrombotic microangiopathy**

⇒ *Haemolytic uremic syndrome, Thrombotic thrombocytopenic purpura – formation of platelet thrombi in small vessels of systemic circulation, platelets consumption, endothelial damage and haemolysis*

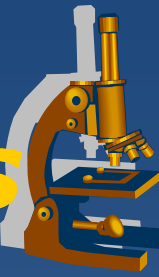
⇒ *Intimal and endothelial oedema, fibrinoid necrosis of the arteriolar wall, fibrin thrombi in capillaries*

⇒ *types:*

- epidemic (E.coli – shiga-like toxin)
- other – drugs, irradiation, infection
- TTP – hereditary/acquired excessive activation of platelets



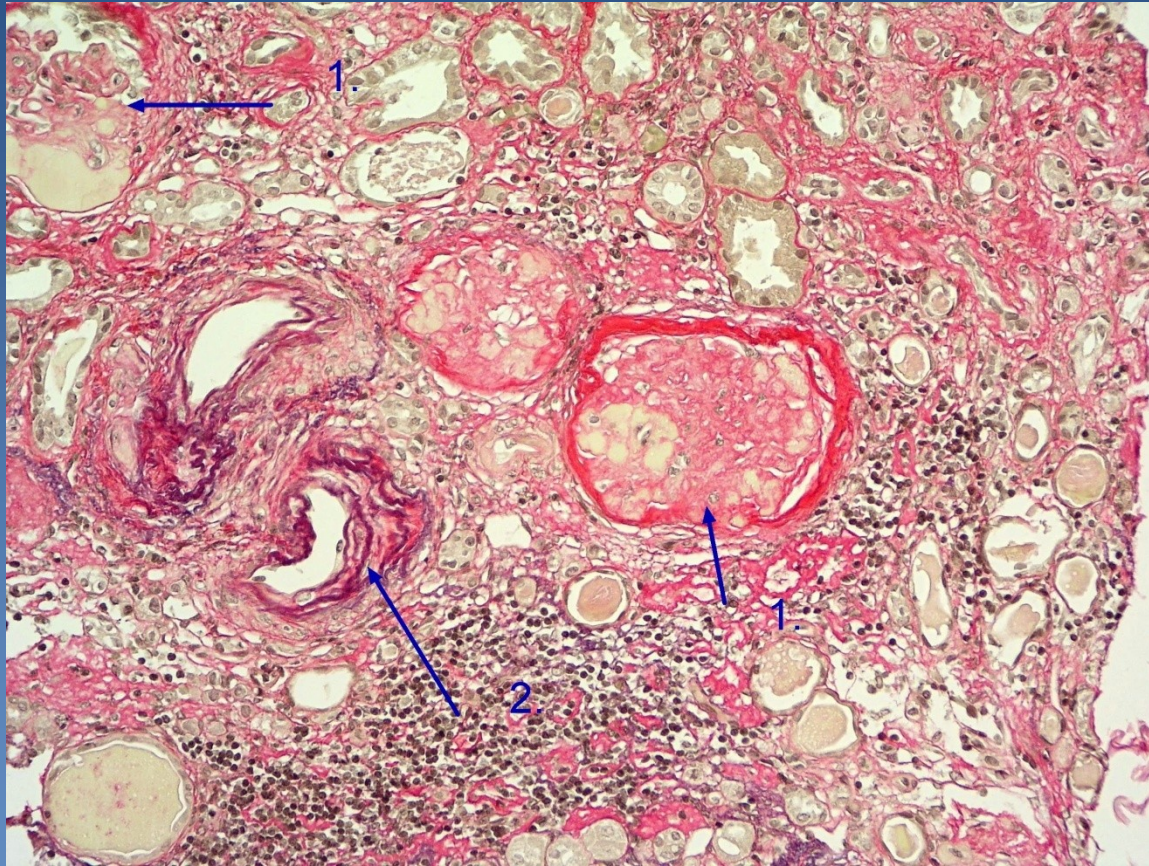
# *Chronic glomerulonephritis*



- ✗ gl. disease in the end-stage (significant renal lesion)
  - gross: kidney contracted, granulated
  - micro: high percentage of globally obliterated glomeruli, interstitial fibrosis, tubular atrophy, vascular changes.



## Chronic glomerulonephritis



1. Obliterated glomeruli
2. Vascular changes

# *Tubulo-interstitial disorders*



⇒ both parts (tubules + interstitium) affected

× Two main categories:

⇒ *Ischemic and toxic lesion (acute tubular necrosis ATN)*

⇒ *Inflammatory (tubulointerstitial nephritis TIN)*

# ***Tubulo-interstitial disorders***



## **x Acute tubular necrosis**

⇒ *etiology: ischemic , toxic*

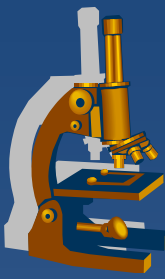
⇒ *acute renal failure with oligouria/anuria, hemodialysis necessary*

- gross: kidney edema, markedly pale cortex

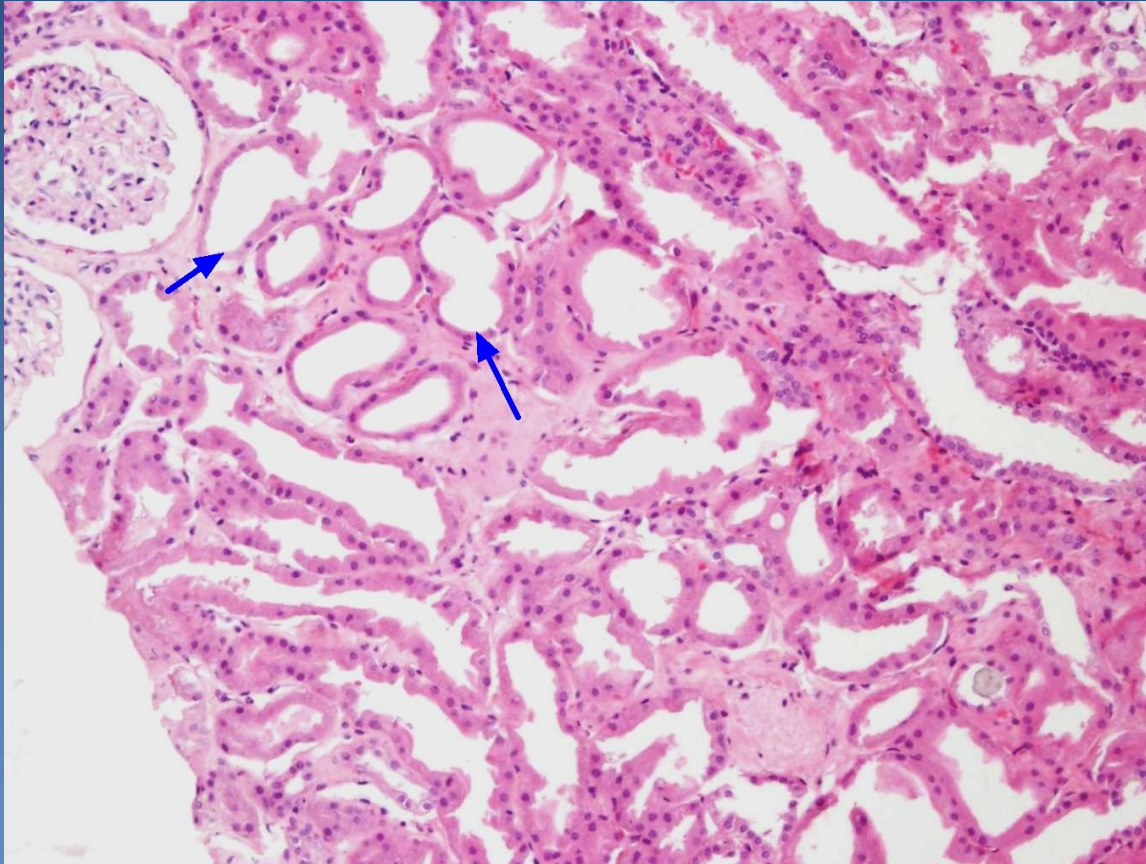
- micro: variable grade of tubular cells injury, from loss of brush border to necrosis.

⇒ *Ischemic – segmental lesions along the whole tubular length*

⇒ *toxic – proximal tubules*



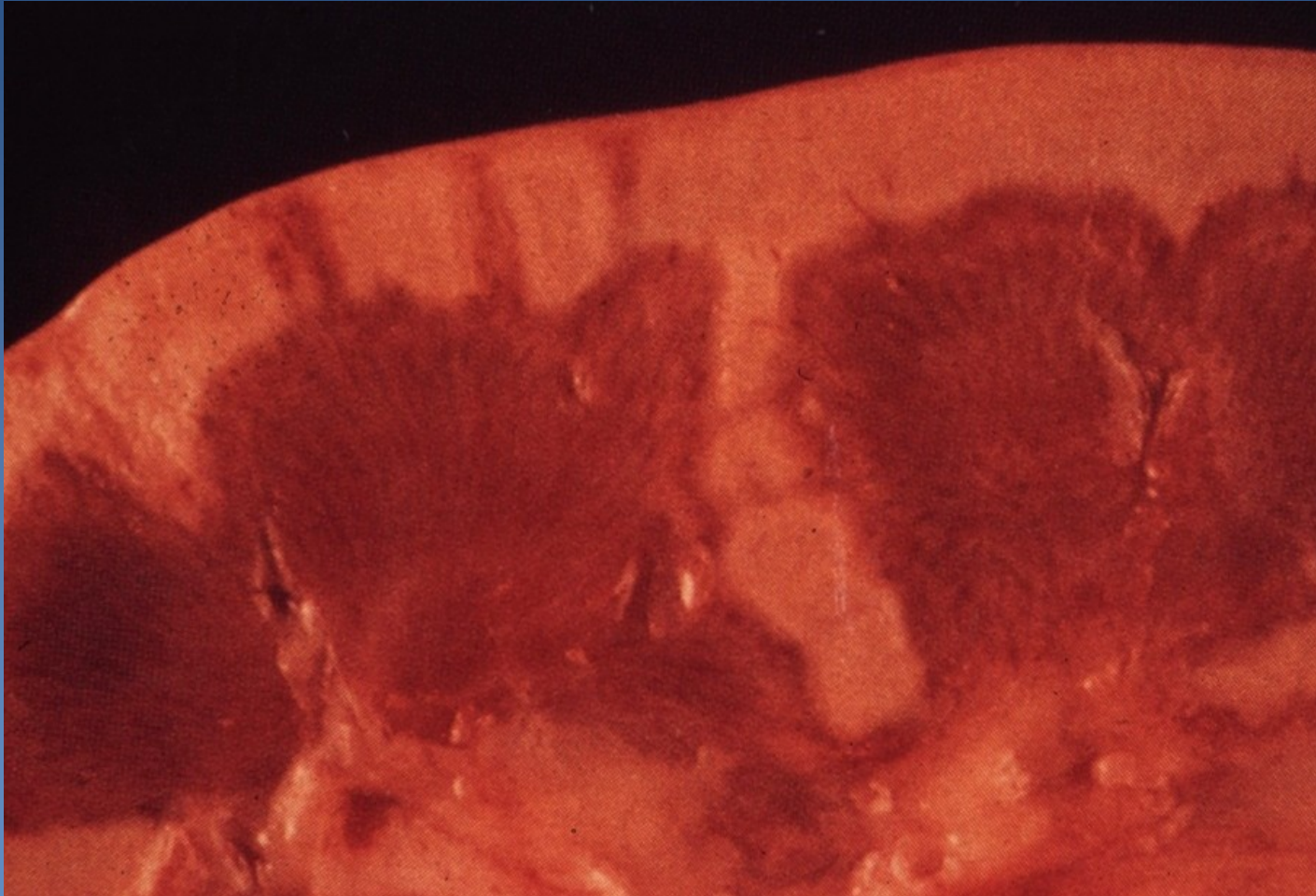
# ***Acute tubular necrosis***



Tubular dilatation, simple flat epithelium



# *Acute tubular necrosis*





# ***Tubulo-interstitial disorders***



## ***x Acute tubulo-interstitial nephritis***

⇒ *Etiology: infectious bacterial (acute pyelonephritis)*

⇒ *toxic drug-induced ( post ATB)*

⇒ *metabolic (diseases with crystal formation)*

⇒ *viral (hantaviruses)*

- micro: interstitial inflammatory infiltrate, variable grade of tubular epithelium injury

# ***Tubulo-interstitial disorders***



## **x Acute pyelonephritis**

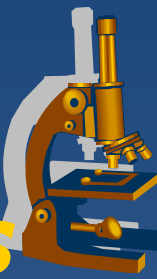
⇒ *acute pelvis + kidney inflammation - mostly ascending - bacterial infection – i.e. E. coli*

⇒ *descending - in sepsis*

⇒ *febrile illness, lumbal pain, dysuria + urging, pyuria with numerous neutrophils*

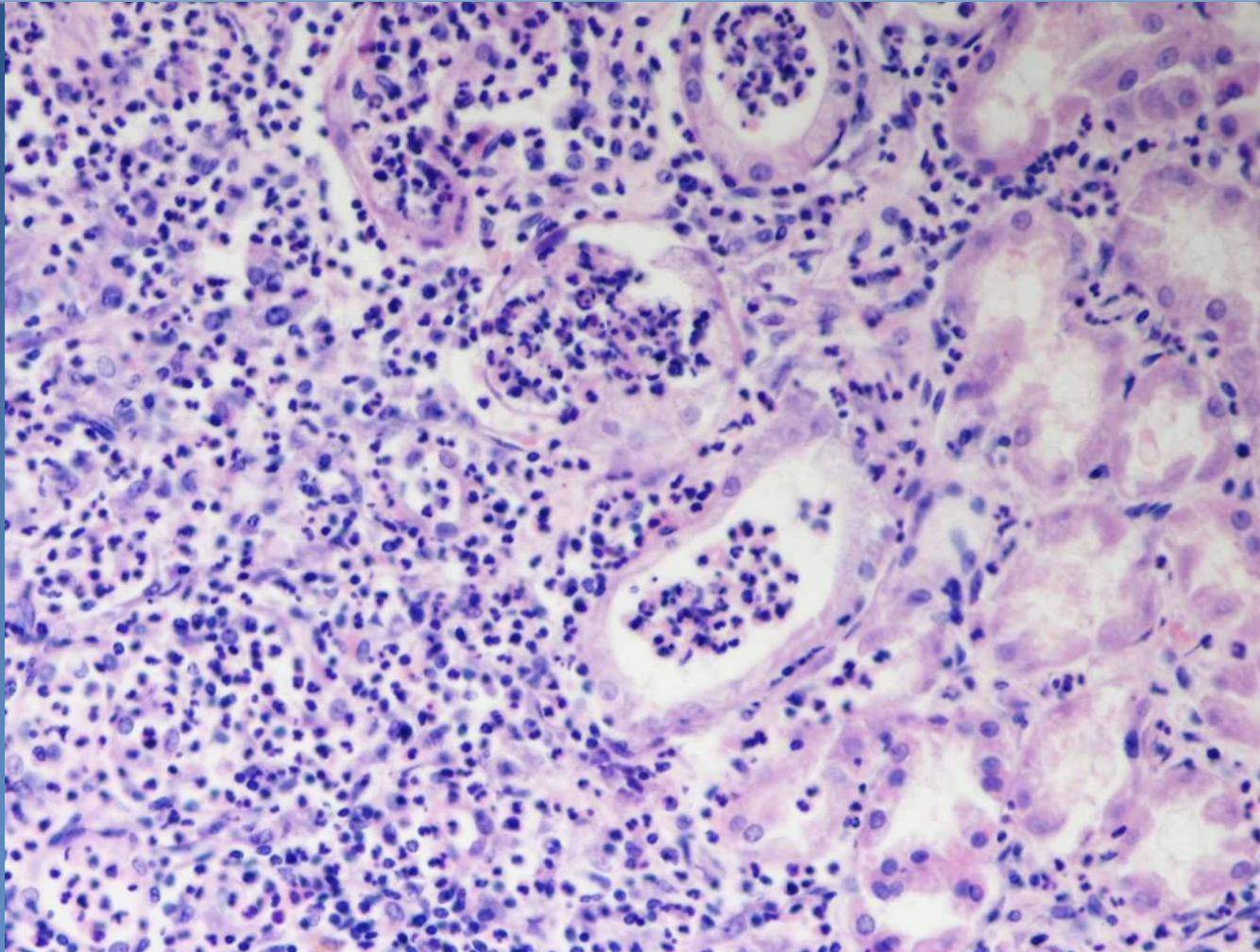
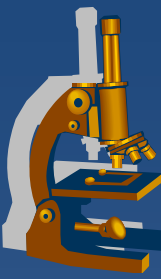
# ***Tubulo-interstitial disorders***

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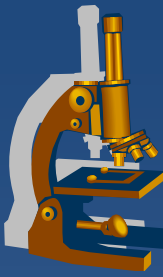
- *gross* : swollen kidney, yellow subcapsular abscesses.
- edematous, hyperemic pelvis, sm. with pus, progression of purulent inflammation to the adjacent tissues - paranephritic abscess
- *micro*: interstitial + tubular neutrophils

# *Acute pyelonephritis*



# ***Tubulo-interstitial disorders***

---



- x** Chronic pyelonephritis

  - ⇒ *one of the most common causes of renal failure*

  - ⇒ *possible insidious start, manifestation due to hypertension, commonly after multiple attacks of acute pyelonephritis.*



# *Tubulo-interstitial disorders*

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- ✘ gross: irregular shrunken kidney, flat scars, commonly + nephrolithiasis, progressive atrophy - end-stage kidney
- ✘ micro: interstitial fibrosis, tubular atrophy, dilatation + casts (follicular colloid-like), glomerular hyalinisation

# *Tubulo-interstitial disorders*

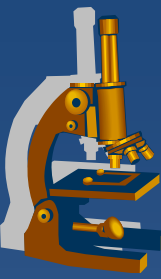
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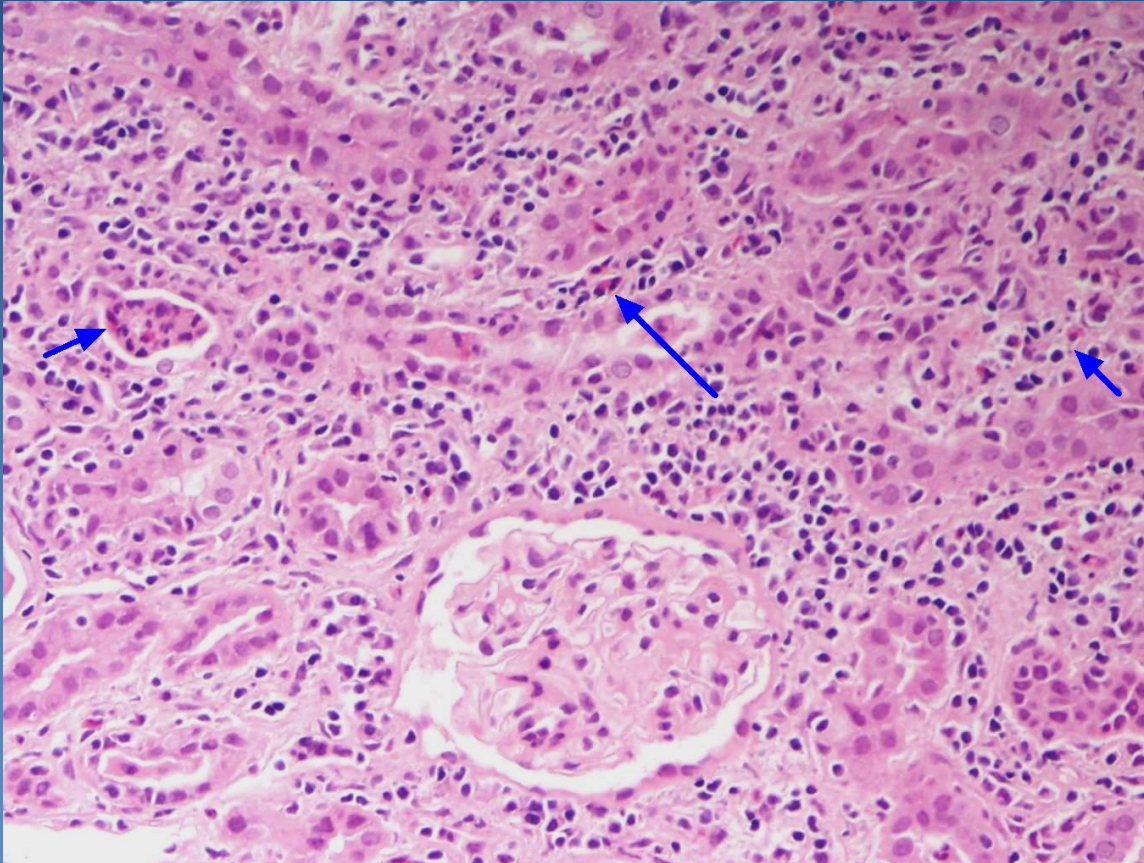
## × Drug-induced TIN

⇒ *Antibiotics, NSAIDs*

- micro: interstitial oedema, mixed interstitial inflammatory infiltrate with eosinophils



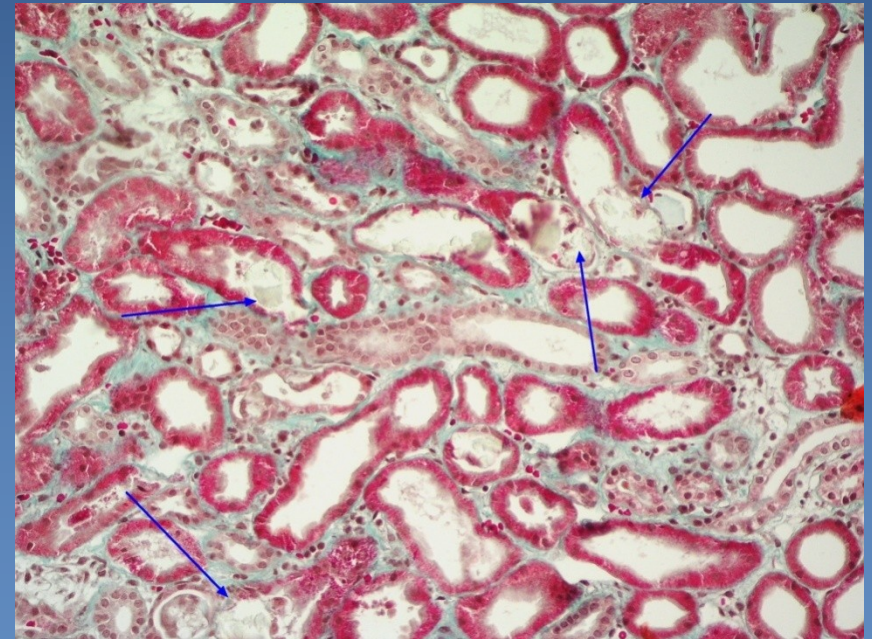
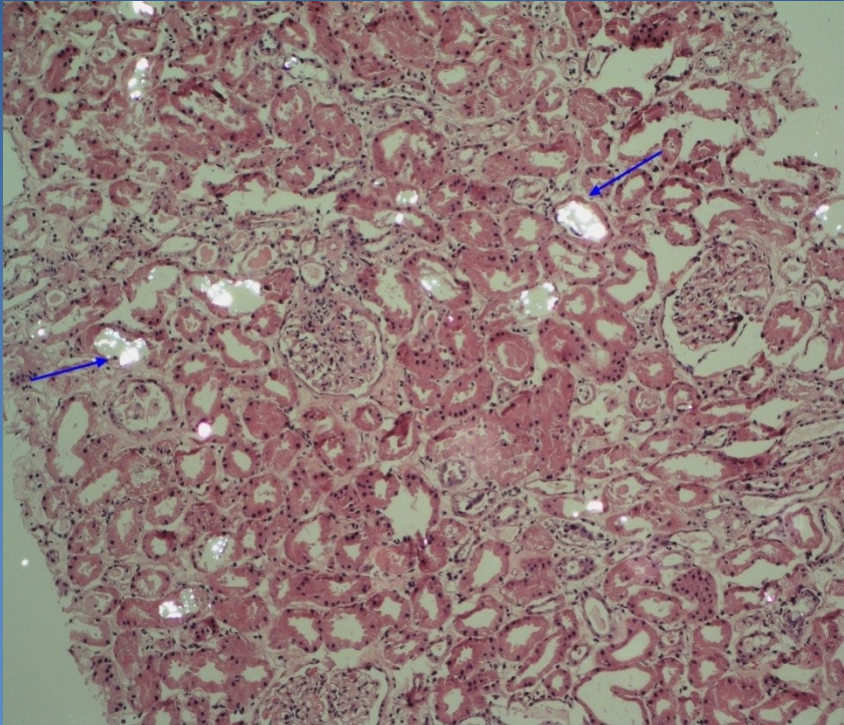
## **TIN**



Eosinophils in inflammatory infiltrate



# *Oxalate nephropaty*



**Oxalate crystals/deposits in tubules**

# *Tubulo-interstitial disorders*



## *x Myeloma nephropathy*

*⇒ renal damage due to myeloma*

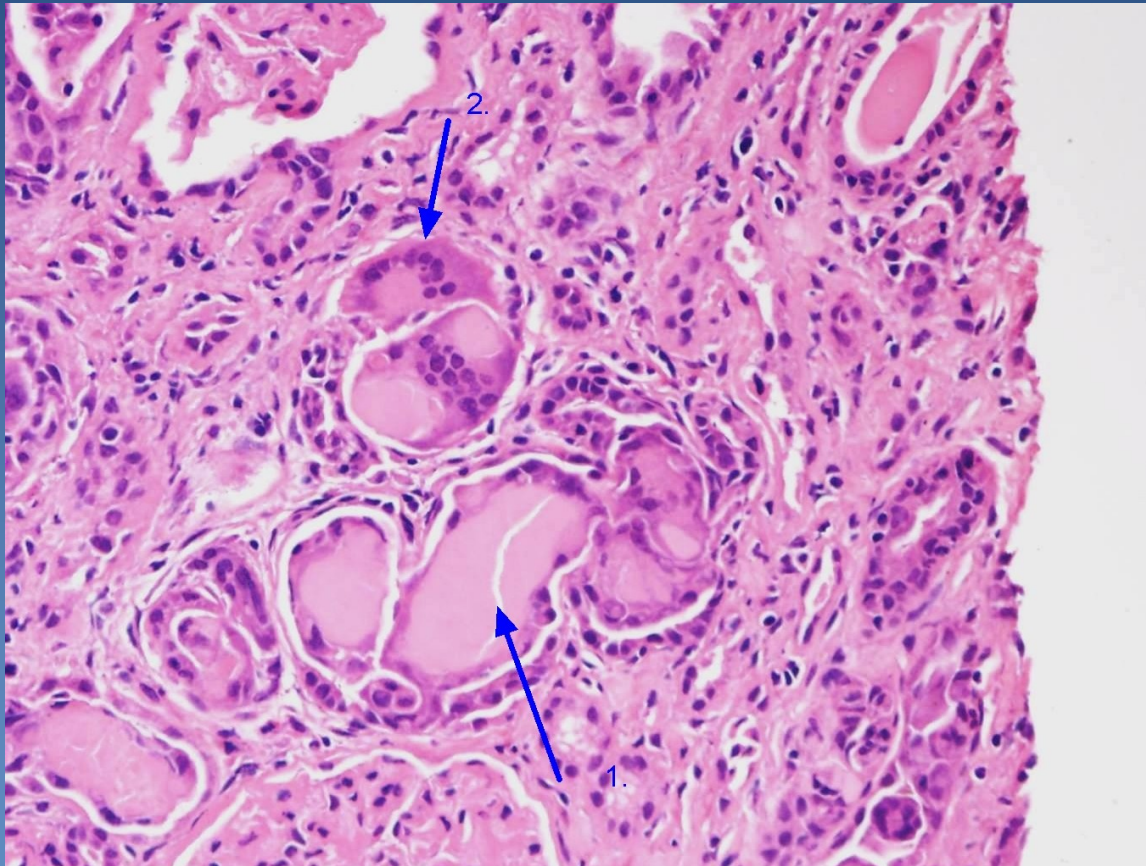
*⇒ excretion of light chains (**BJ protein**) into primary urine, toxic to epithelia*

*⇒ + casts formation → **nephrohydrosis**, blockage of urine outflow within renal parenchyme.*

*⇒ tubular epithelial damage, multinucleated macrophages*



# *Myeloma nephropathy*



1. Protein casts
2. Giant multinucleated macrophages

# Renal tumors



× Benign x malignant

× **Benign**

⇒ **angiomyolipoma**

- Mesenchymal (perivascular epithelioid cell – PEComa)  
more common in patients with tuberous sclerosis

⇒ **cortical adenoma**

- **micro:** papillary structure
- **gross:** ochre colour, size < 5mm
- accidental finding

# Benign tumors

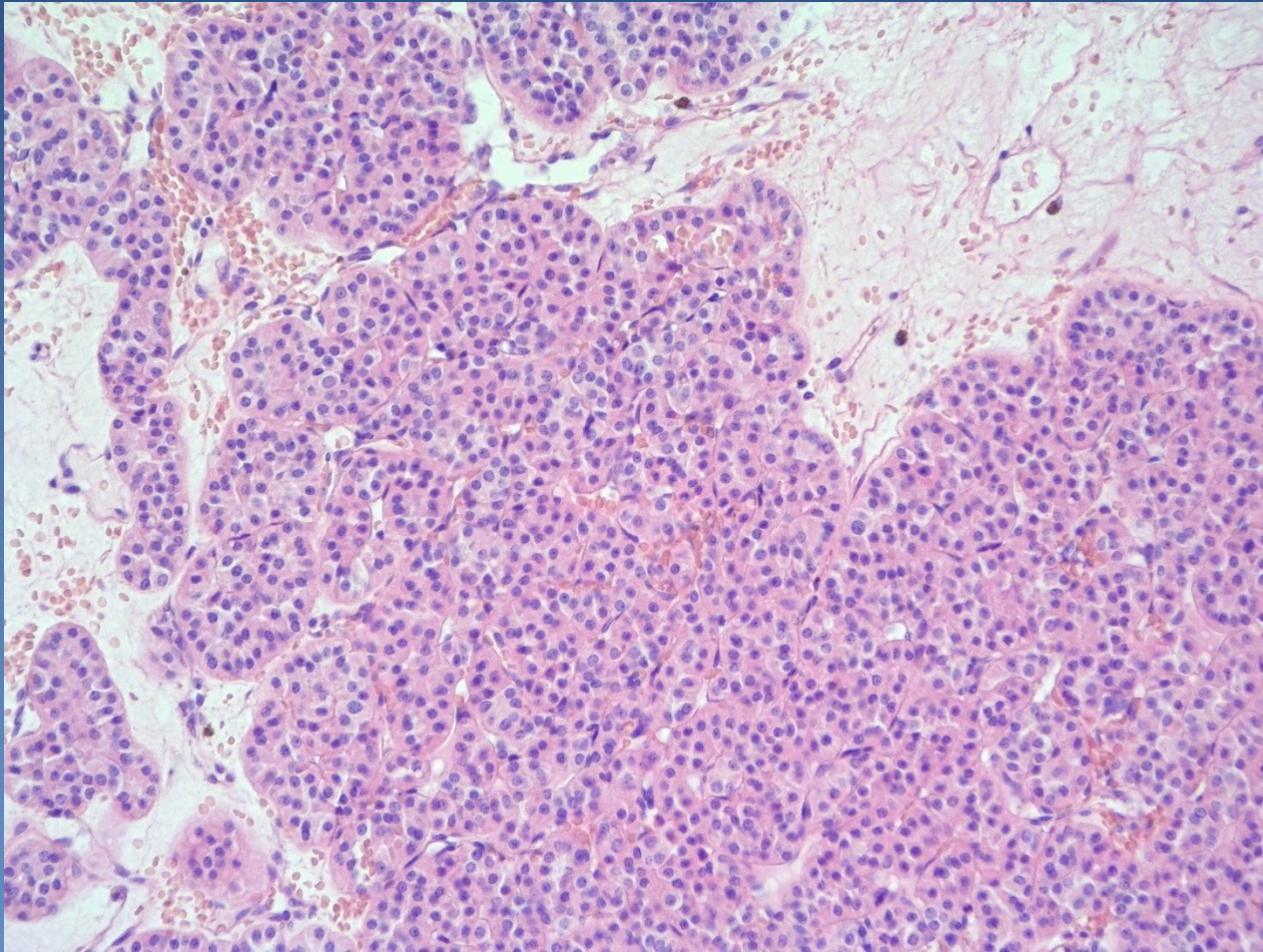
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## ⇒ *renal oncocytoma*

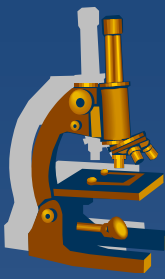
- **gross:** demarcated tumor of red-brown colour, variable size  
central scar
- **micro:** eosinophilic, granular cytoplasm, cells in acinar,  
tubular, solid nests; central hyaline scar

# *Renal oncocytoma*



# *Renal cell carcinoma (RCC)*

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- ✘ More common in males; middle-older age
- ✘ Smoking as major risk factor
- ✘ mostly sporadic tumors, 4% part of hereditary syndromes



# Clear cell RCC



⇒ **70-80% of all RCC**

- *gross* : demarcated tumor, yellowish colour commonly with haemorrhagic, necrotic, fibrotic foci
- angioinvasive tendency – direct grow into renal vein, vena cava;
- invasion into pelvis - haematuria

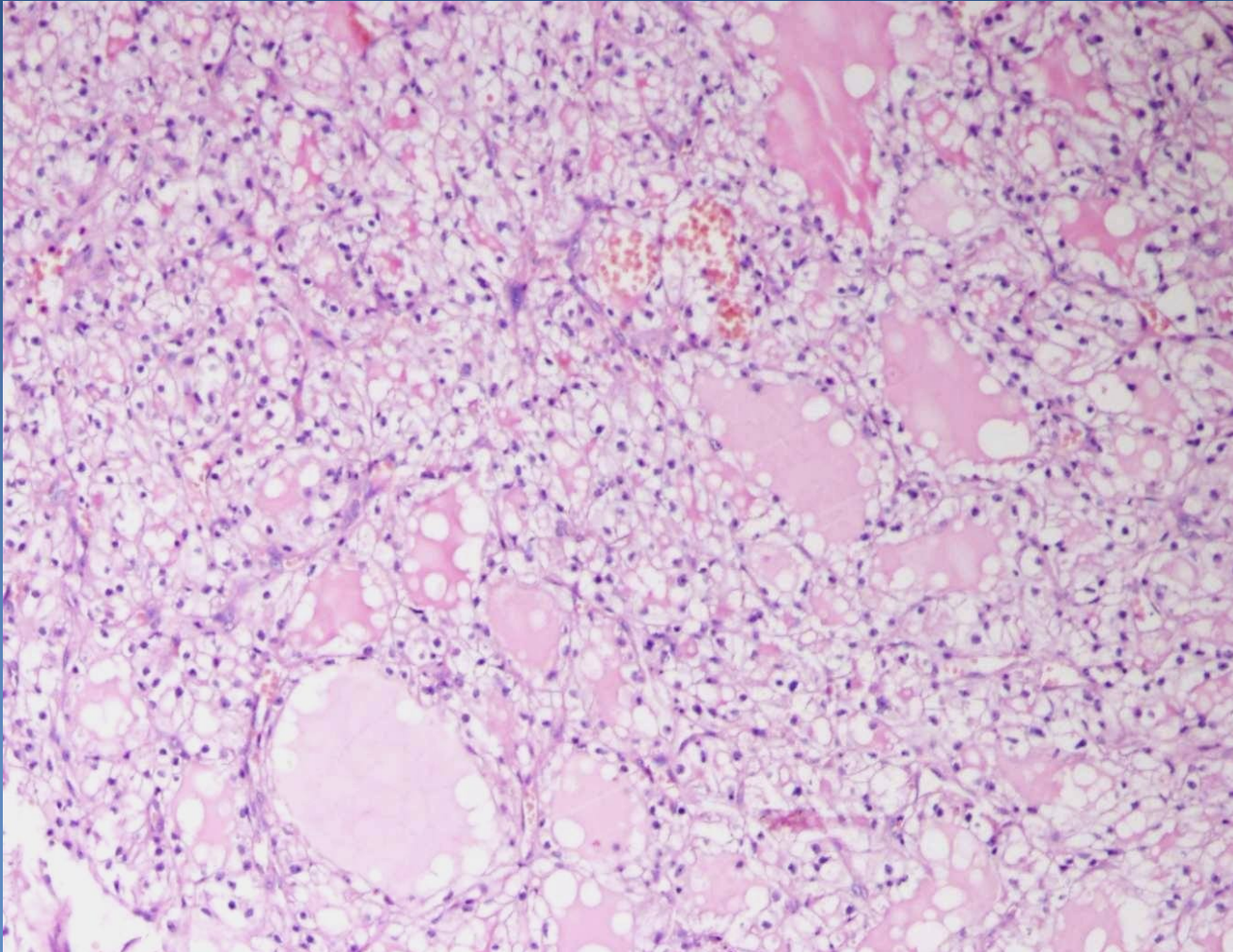
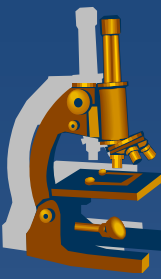
# *Clear cell RCC*

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- Metastases via blood mostly (lungs, bones, brain)
- **micro** : large cells with clear granular cytoplasm (glycogen + lipids)

# *Clear cell RCC*



# ***Clear cell RCC***

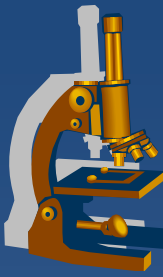
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- ⇒ *clinical : local symptoms late, haematuria. Fever, paraneoplastic syndromes*
- ⇒ *prognosis according to the tumor size/stage*
- ⇒ *ca < 3 cm quite good*

# *Papillary RCC*

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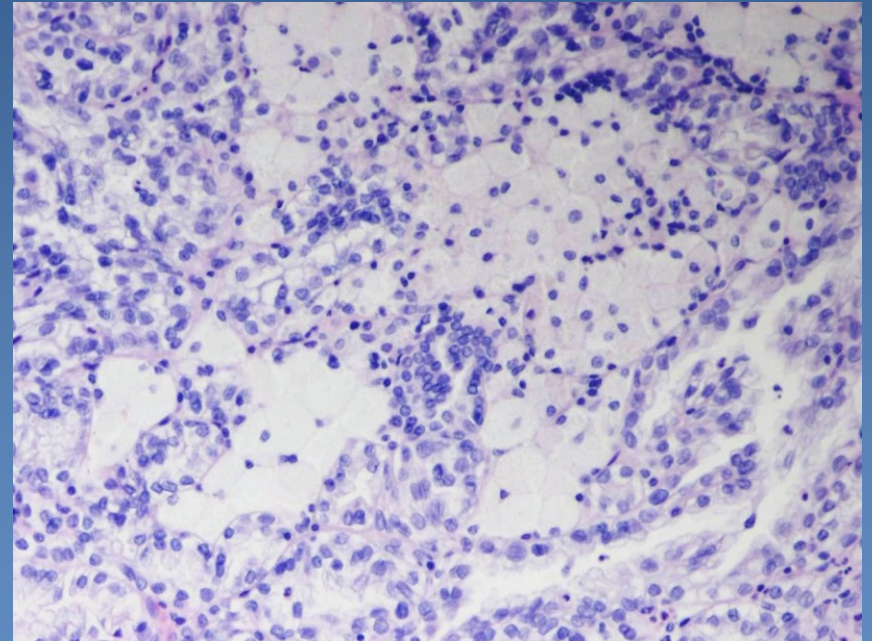
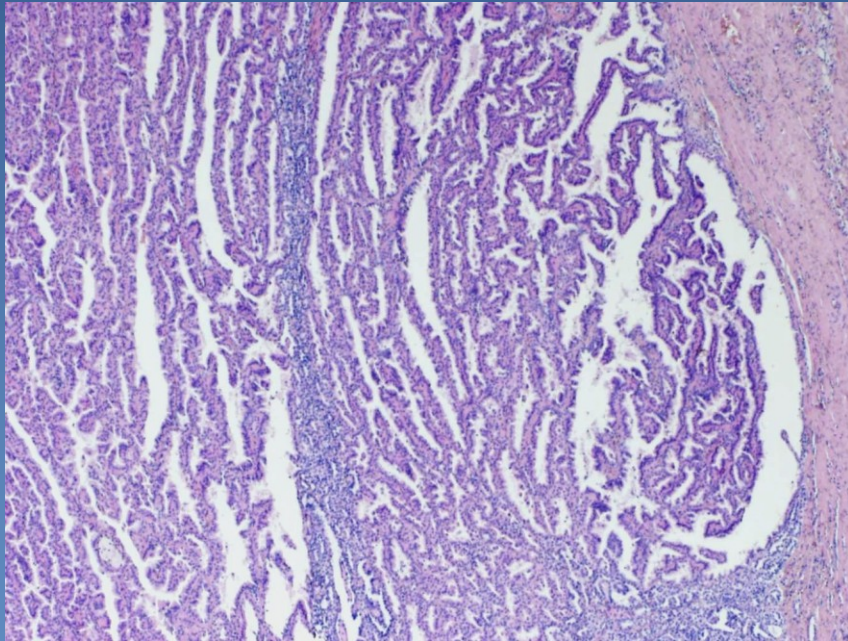


⇒ *15% of all RCC*

- **gross:** well-demarcated, regressive changes, commonly multifocal and bilateral
- **micro:** malignant epithelial cells covering stromal papillae, with stromal foam macrophages



# *Papillary RCC*



# *Chromophobe RCC*

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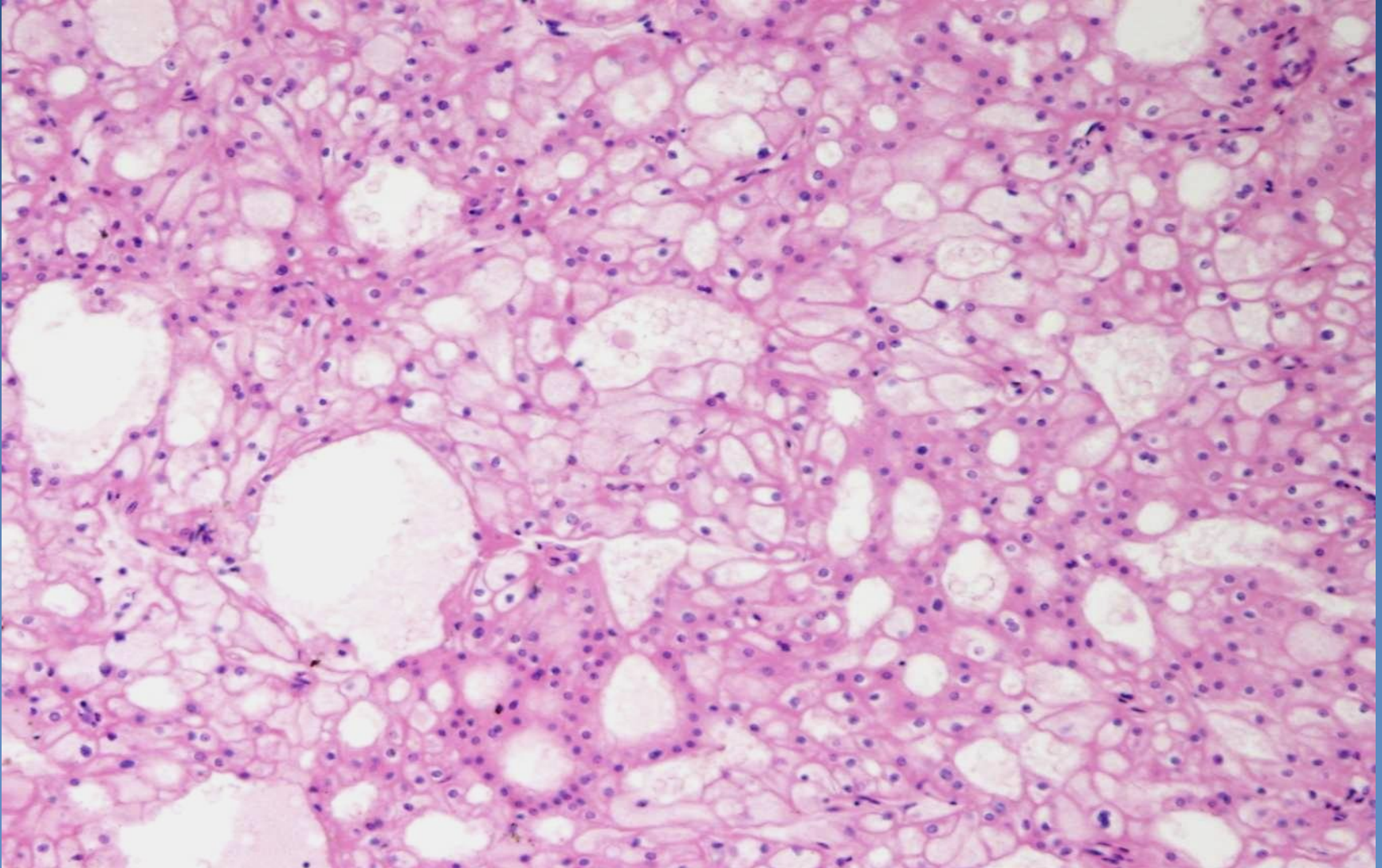


⇒ *5% of RCC.*

- **gross:** well demarcated, partial lobulisation, brown colour
- **micro:** eosinophilic granular cytoplasm, distinctive cell membranes, shrunken („raisin“) nucleus

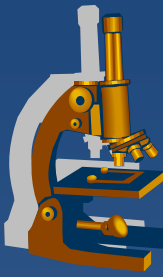


# *Chromophobe RCC*



# *Nephroblastoma*

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- ⇒ *3rd most common malignant pediatric tumor*
  - ⇒ *Diagnosed mostly in the 3rd-4th year of age*
  - ⇒ *Sporadic, or part of some syndromes*
- 
- *gross*: large, well demarcated tumor, greyish colour, regressive changes

# *Nephroblastoma*



- **micro**: structures attempting to recapitulate variable stages of **nephrogenesis**
  - Triphasic combination of blastemal, stromal and epithelial cell types in variable percentage
  - Highly cellular foci resembling embryonal blastema divided by strands of immature mesenchyme



# ***Nephroblastoma***

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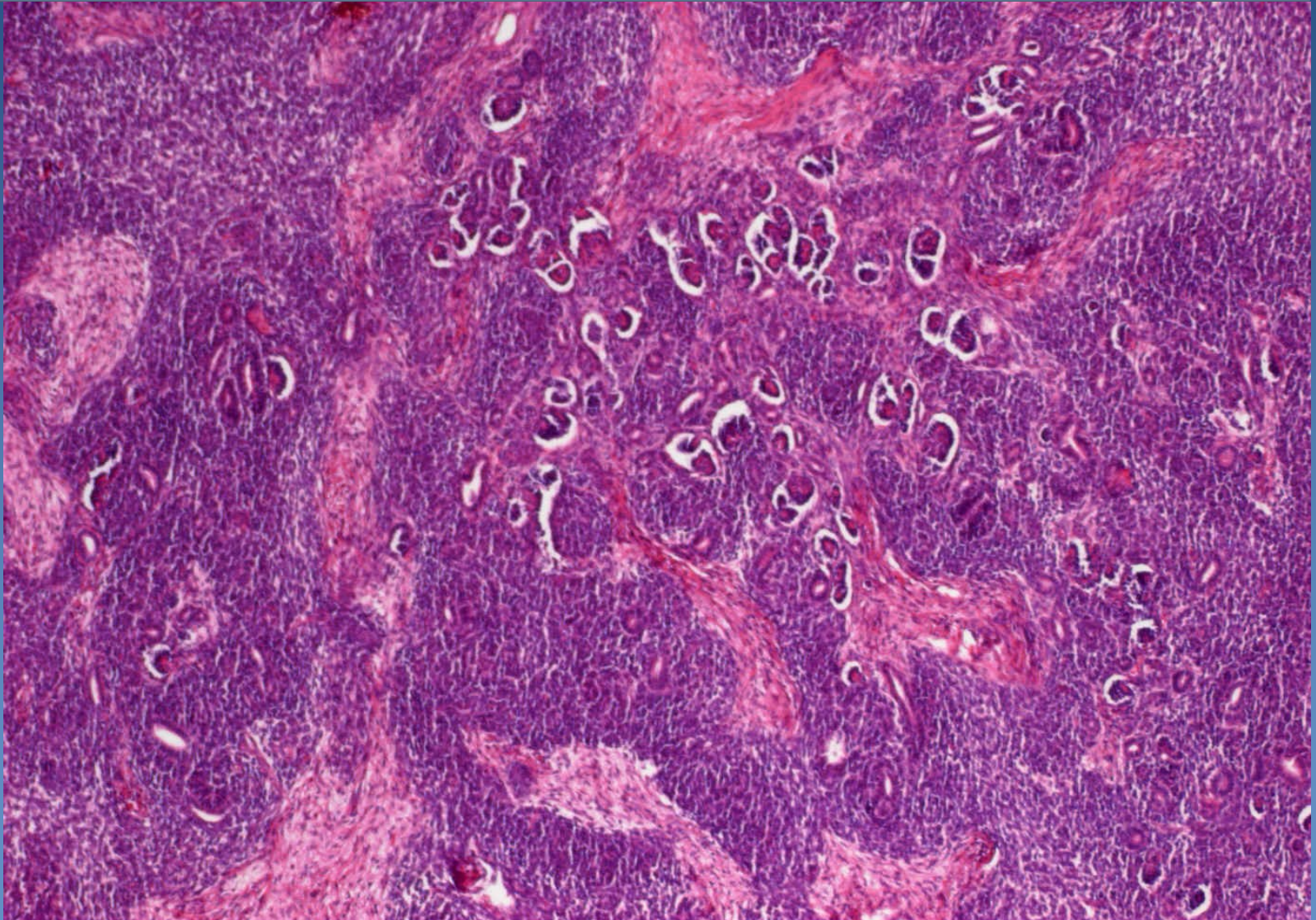
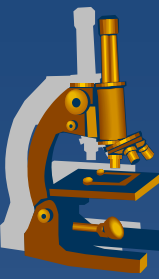


- ⇒ *clinical: large tumor, palpable, complications due to compression of adjacent organs, hematuria*
- ⇒ *prognosis: good, CHT (RT carefully, second malignancies possible)*

# ***Nephroblastoma***

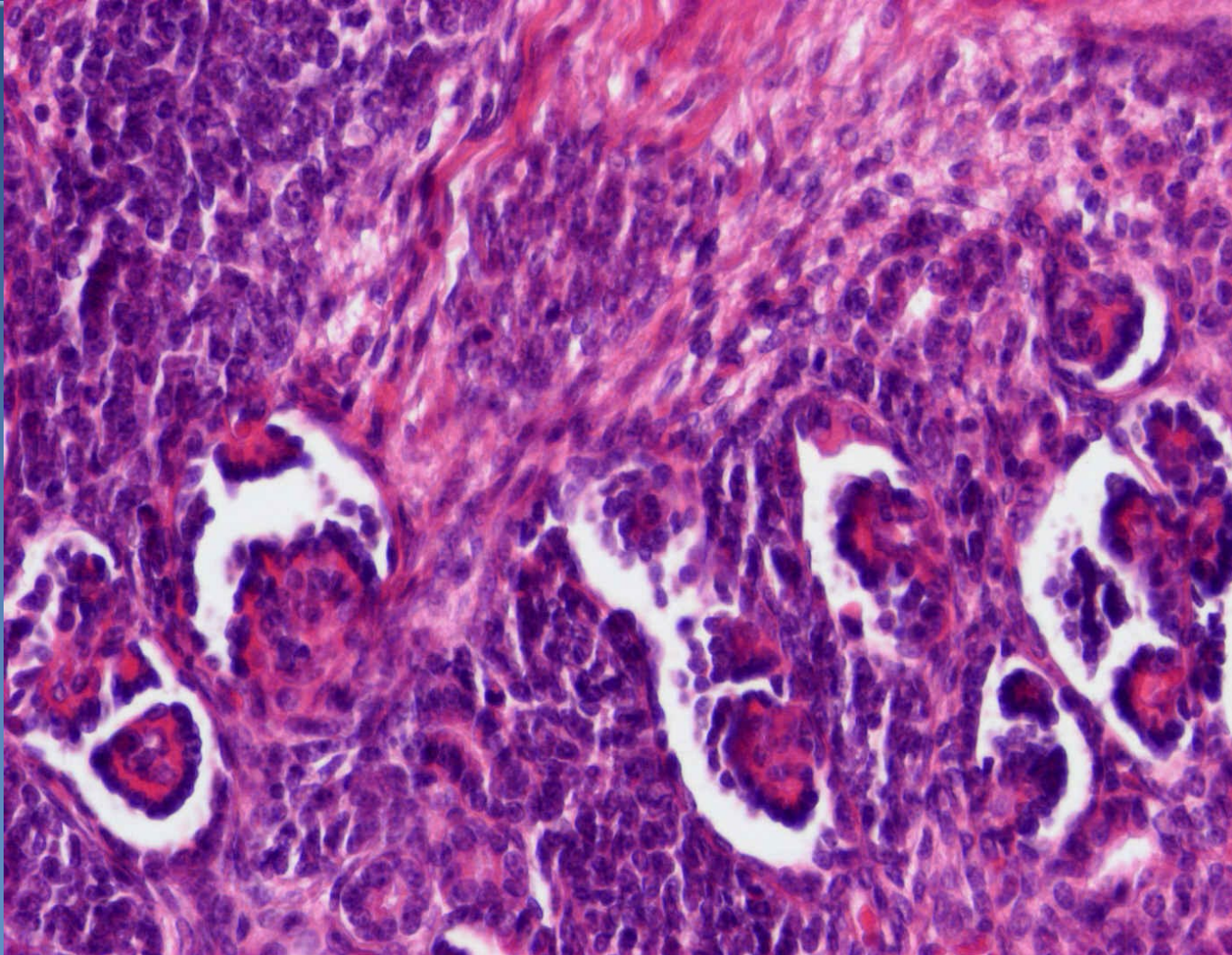


# ***Nephroblastoma***

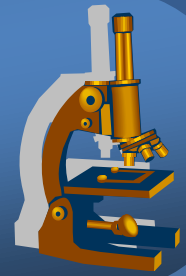




# ***Nephroblastoma***



# *Urinary tract disorders*





# *Urinary tract*

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⇒ *Calices*

⇒ *Pelvis*

⇒ *Ureters*

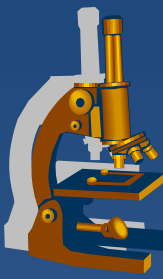
⇒ *Urinary bladder*

⇒ *Urethra*

# Inflammations



- ⇒ Mostly ascending infection
  - ⇒ *urethritis*
  - ⇒ *urocystitis*
  - ⇒ *possible progression into kidney*
  
- ⇒ *etiology: E.coli, Proteus, Klebsiella, Enterococcus, Neisseria gonorrhoeae, etc.*
- ⇒ *Candida, Schistosoma,*



# Inflammations

- ⇒ *dysuria, polakisuria (urging), raised temperature*
  - gross: haematuria, pyuria
    - Hypaemic mucosa, possible pseudomembrane, ulceration
  
- ⇒ *complications : progression of inflammation into adjacent structures: glands, interstitium – phlegmona, periurethral abscess*



# Inflammations



- micro:

- **acute inflammation** with prevalence of neutrophils, regressive changes of transitional cell epithelium

- **chronic inflammations** - reactive changes of transitional cell epithelium, squamous/glandular metaplasia. Brunns nests – cystitis cystica

⇒ *urethra – caruncula urethrae – pseudotumorous hyperplastic polyp in the region of urethral orifice.*

# *Hydronephrosis*



## ✘ Pathological dilatation of the renal pelvis and calyces

- Causes:
  - Impacted stone, ...
  - Tumors
  - External compression (pregnancy, prostatic hyperplasia, ...)



# Tumors



× benign × low malignant potential ×  
frankly malignant

× flat × papillary lesions

⇒ *Mostly urothelial*

***Precursor lesions:***

⇒ *Urothelial dysplasia*

⇒ *risk factors:*

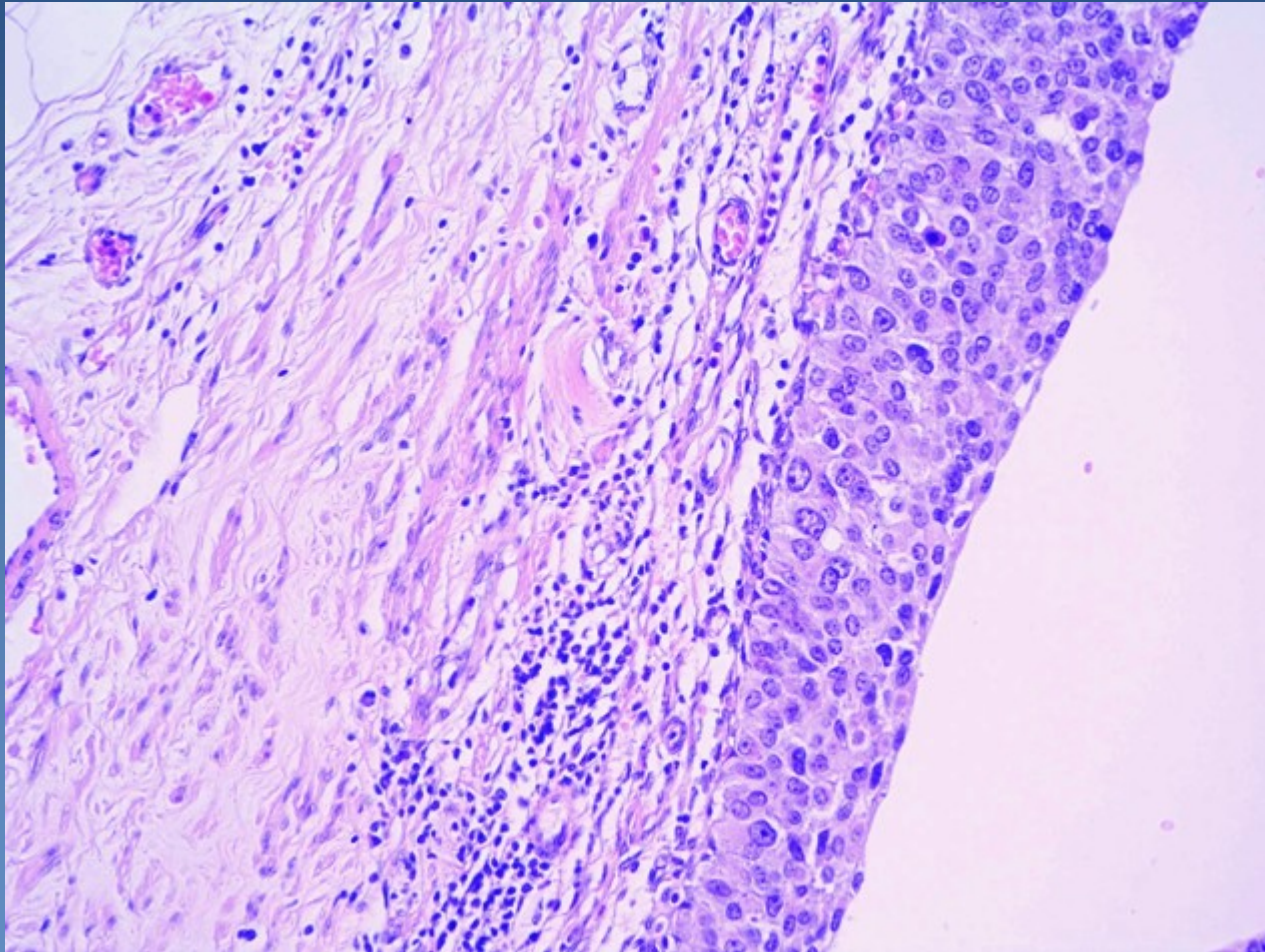
- M:F 3:1
- smoking
- professional exposure (aromatic amines, etc.)

# *Urothelial dysplasia*



- Micro: flat lesion, cytologic atypia with **loss of cell polarity**, **↑ mitotic activity** in upper layers of urothelium, **↑ N/C ratio**, coarse chromatin
- **x LG (low grade) IUN (intraurothelial neoplasia) x HG IUN (CIS)**

# ***Urothelial ca in situ***



# *Papillary urothelial neoplasm*

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## **x** urothelial papilloma

- Solitary papillary lesion covered by normal urothelium without cytological or architectonic atypias.

# ***Papillary urothelial neoplasm***

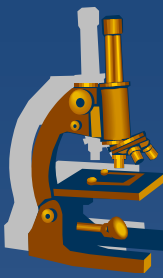


⇒ ***papillary urothelial neoplasm of low malignant potential (PUNLMP)***

- recurrent tumor
- papillae covered by hyperplastic urothelium with preserved stratification, minimal cytonuclear atypia, sporadic mitoses.



# *Papillary urothelial neoplasm*



⇒ *non-invasive papillary urothelial carcinoma*

- low grade
- high grade

⇒ *Papillary neoplasia without signs of invasion into stroma (suburothelial mesenchymal tissue)*

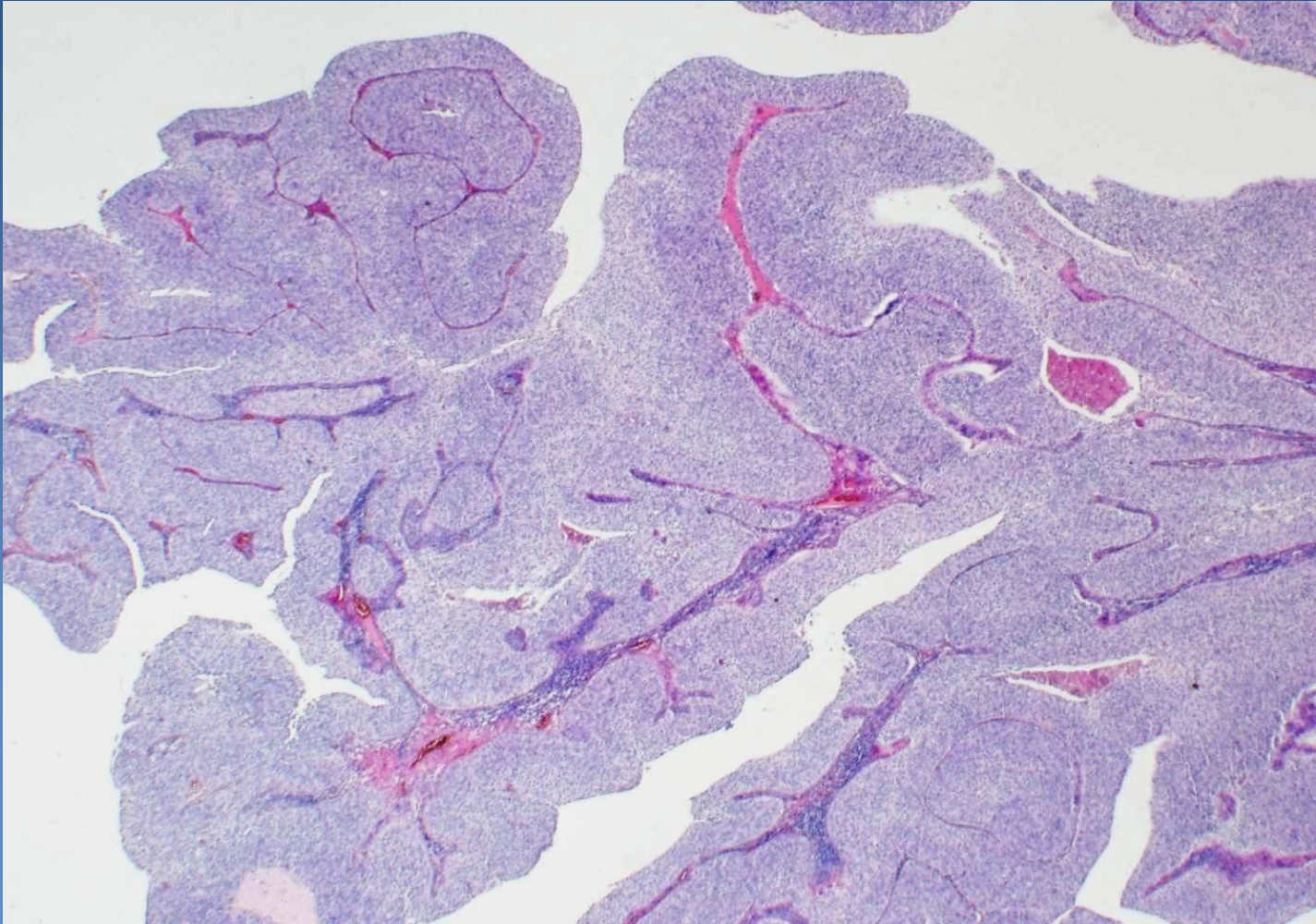
⇒ **LG**

⇒ *altered papillary architectonics,*

⇒ *mild cytonuclear atypia*

⇒ *basal layer mitoses*

# ***Low grade non-invasive papillary urothelial carcinoma***



# *Non-invasive papillary urothelial carcinoma*

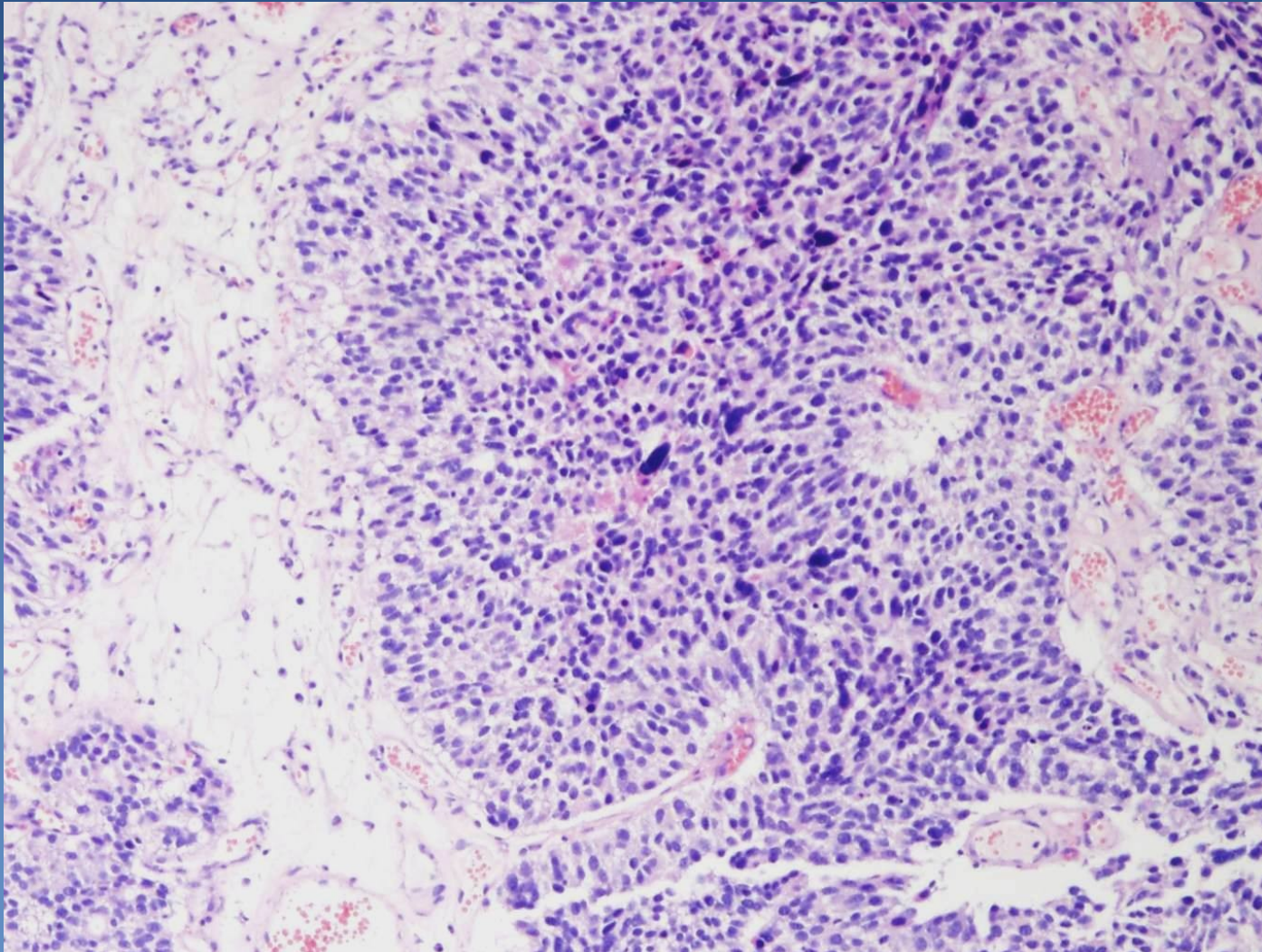
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- ⇒ **HG**
- ⇒ *papillary fusion, solid foci*
- ⇒ *loss of cell polarity*
- ⇒ *moderate – high grade of anisocytosis and anisokaryosis*
- ⇒ *atypical mitoses in upper layers of neoplastic epithelium*



# ***High grade urothelial carcinoma***



# *Bladder carcinoma*





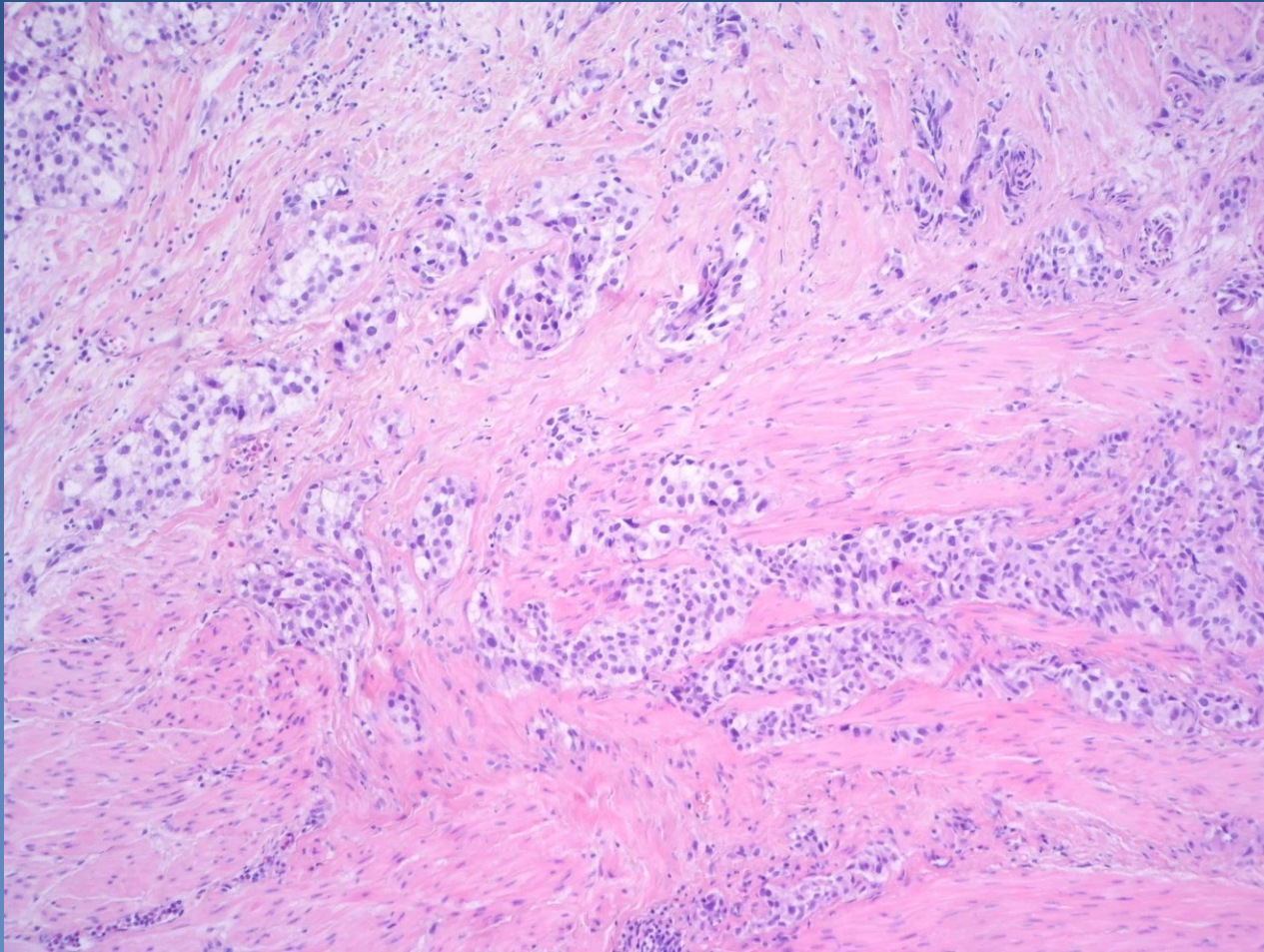
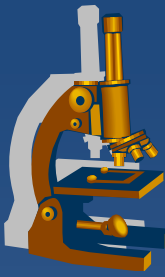
# ***Invasive (infiltrating) urothelial carcinoma***



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⇒ ***ca invasion into sub-urothelial fibrotic tissue or deeper (muscle, ...)***

# *Invasive urothelial carcinoma*



# ***Bladder carcinoma***

---



## ***x Less common carcinomas***

⇒ *squamous cell carcinoma (schistosomiasis)*

⇒ *adenocarcinoma*

⇒ *neuroendocrine carcinoma*



# *Mucinous adenocarcinoma*

