

Glomerular Disease

Lec 4

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Glomerular Diseases

- Glomerular diseases are disorders that primarily affect the glomeruli, the filtering units of the kidney.
- They can lead to proteinuria, hematuria, decreased GFR, and may progress to chronic renal failure.

Causes of Glomerular Diseases

- Primary (Intrinsic) causes:
 - Minimal change disease
 - FSGS
 - Membranous nephropathy
 - IgA nephropathy
 - Post-streptococcal GN
- Secondary causes:
 - Diabetes mellitus
 - SLE
 - Amyloidosis
 - Hypertension
 - Vasculitis

Classification by Clinical Presentation

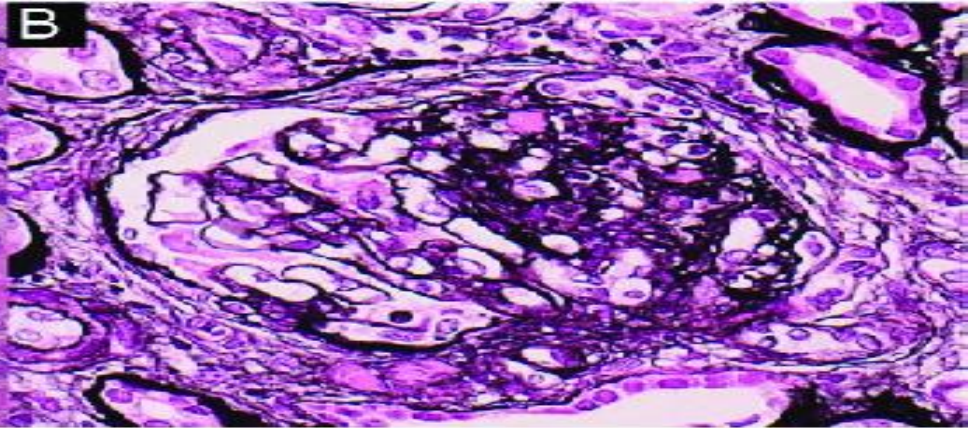
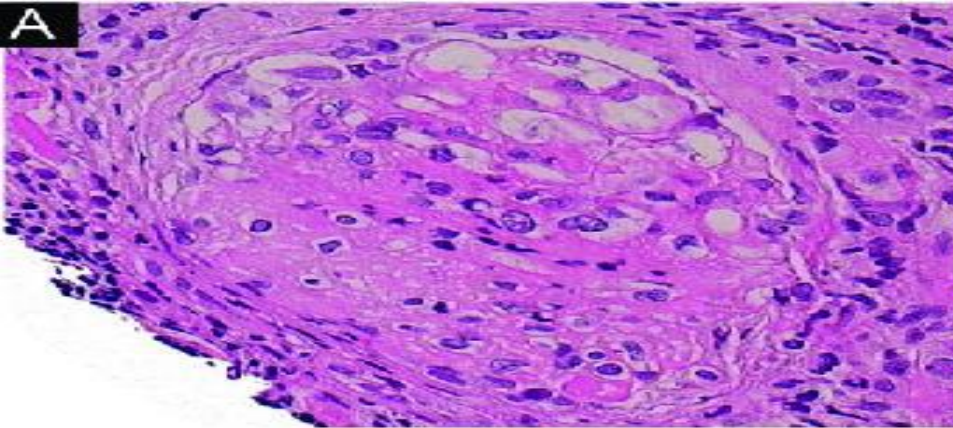
- **Nephritic syndrome:** Hematuria, hypertension, mild proteinuria, ↓GFR
- **Nephrotic syndrome:** Heavy proteinuria, hypoalbuminemia, edema, hyperlipidemia
- **Asymptomatic hematuria/proteinuria**
- **RPGN:** Rapid loss of renal function, crescents
- **Chronic GN:** End-stage fibrosis

Minimal Change Disease (MCD)

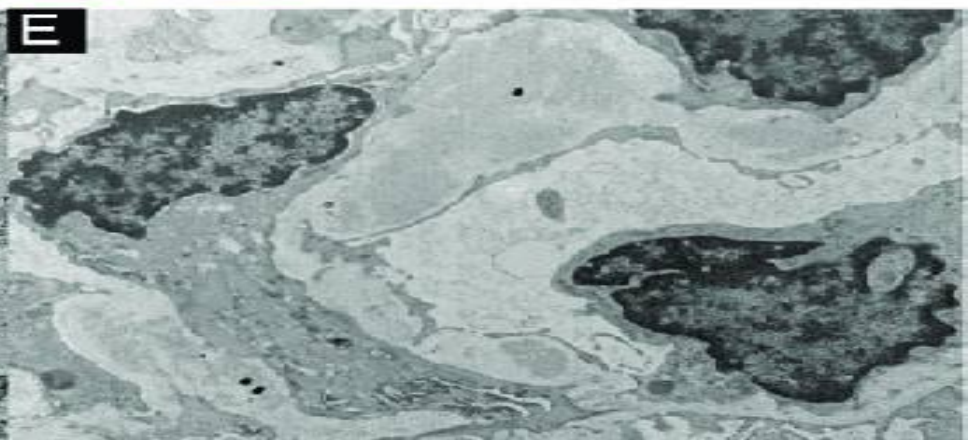
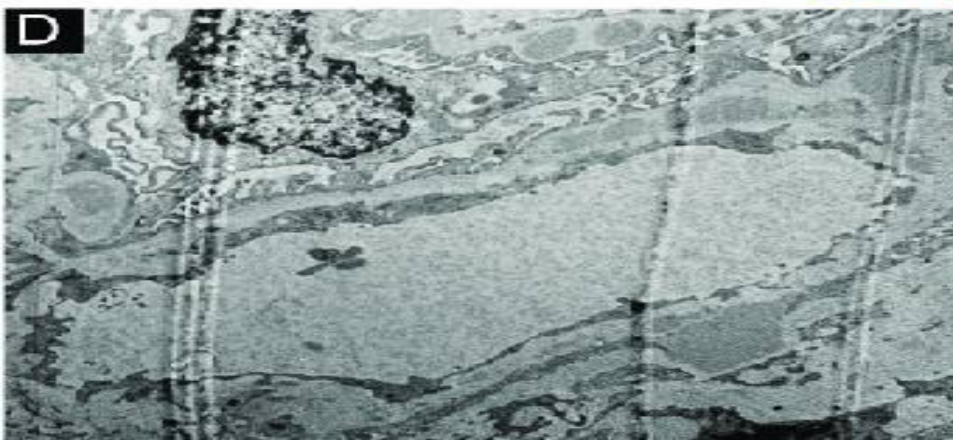
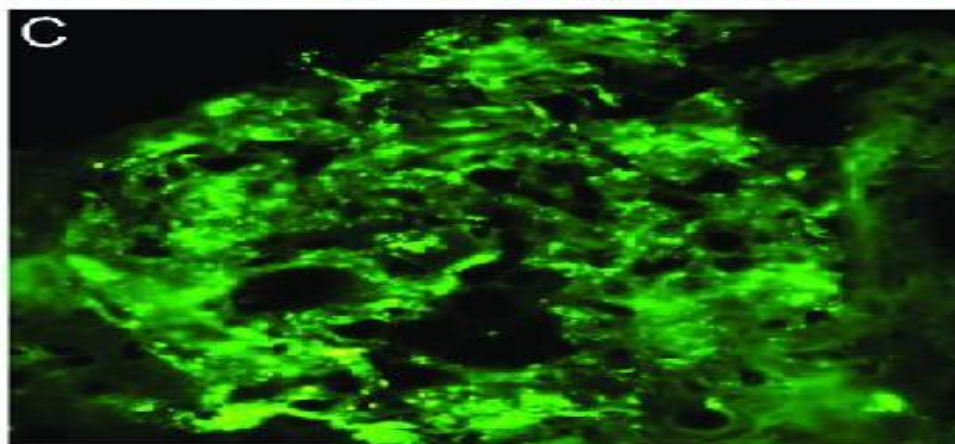
- LM: Normal glomeruli
- EM: Effacement of podocyte foot processes
- No immune deposits
- Common in children, responds to steroids
- **Note(LM:light microscope,EM:Electrone microscope,IF:immunoflorescence)**

Focal Segmental Glomerulosclerosis (FSGS)

- LM: Segmental sclerosis in some glomeruli
- IF: IgM and C₃ trapped in sclerotic areas
- EM: Loss of foot processes
- May lead to chronic renal failure

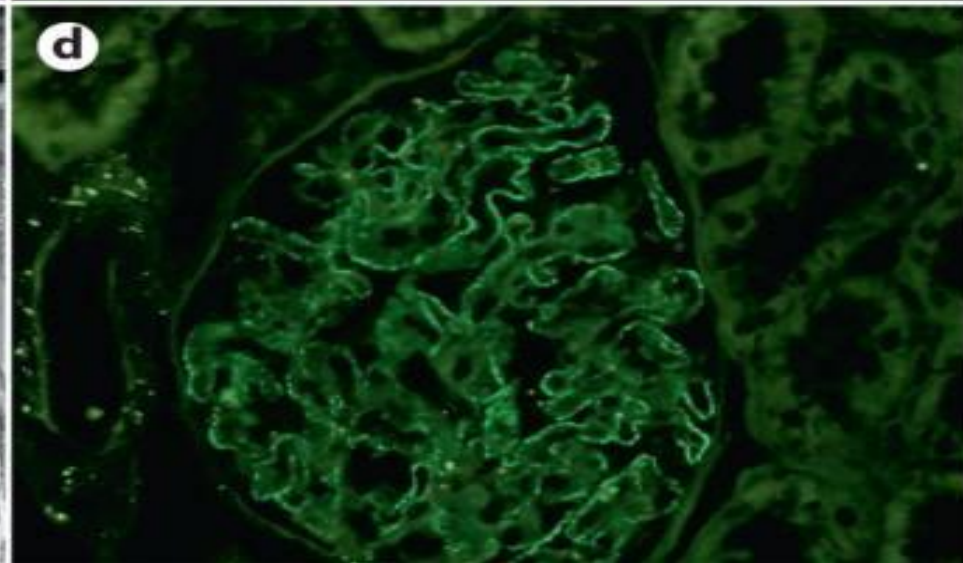
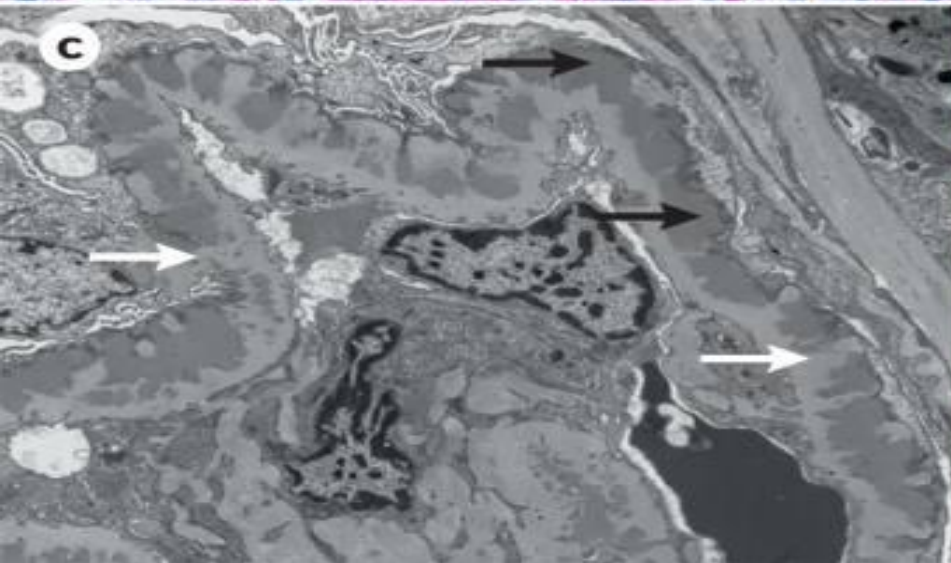
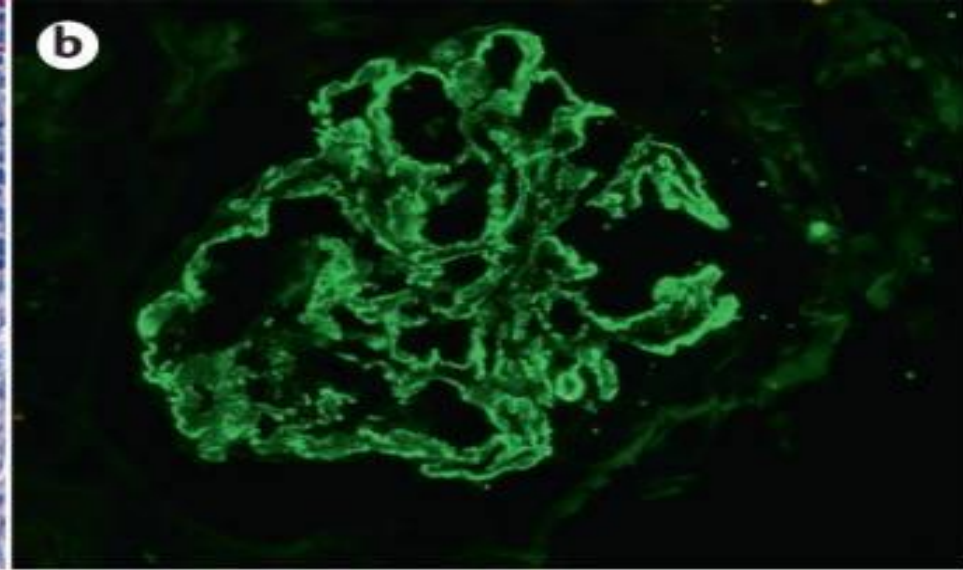
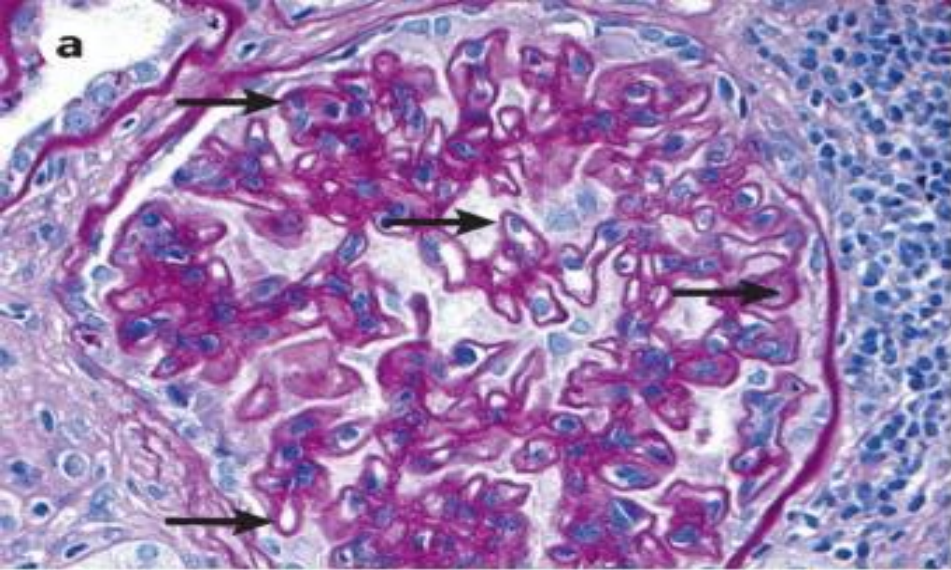


FSGS



Membranous Nephropathy

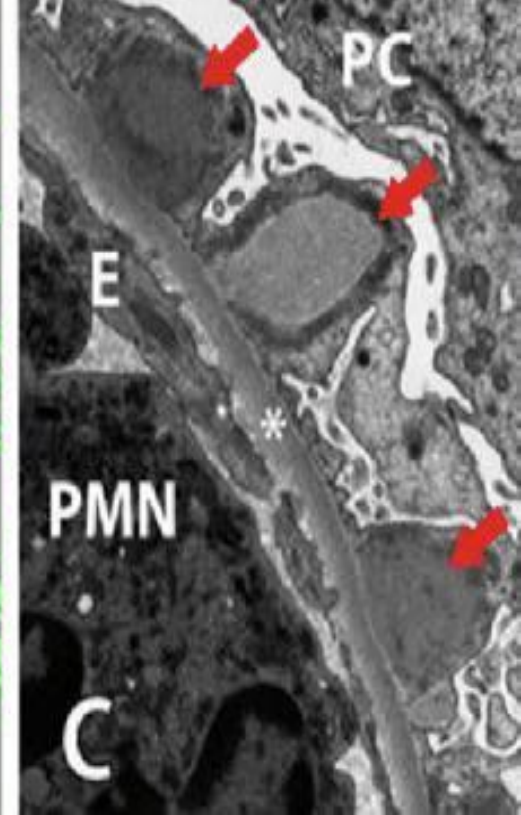
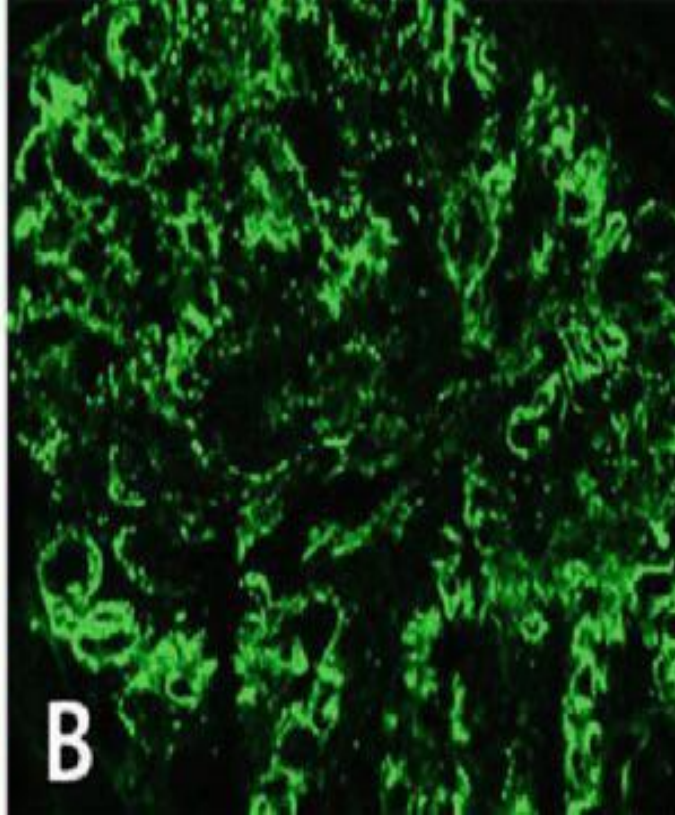
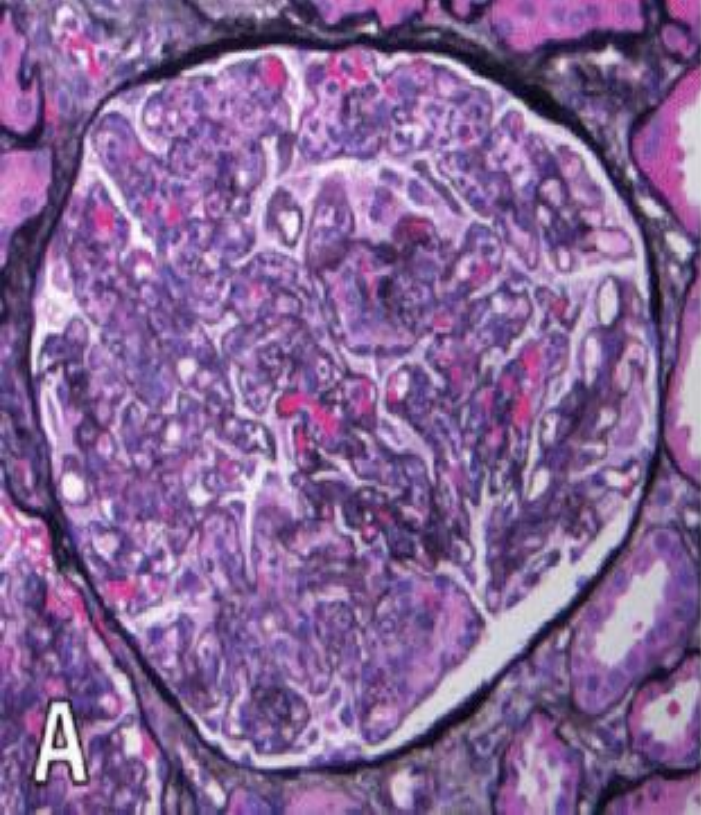
- LM: Diffuse thickening of capillary wall
- IF: Granular IgG and C₃ along GBM
- EM: Subepithelial deposits ('spike and dome')
- Autoimmune (anti-PLA₂R antibodies)
- PLA₂R : Phospholipase A₂ Receptor
- PLA₂R is a receptor located on the surface of podocytes in the glomeruli.



- Membranous Nephropathy

Post-Streptococcal GN

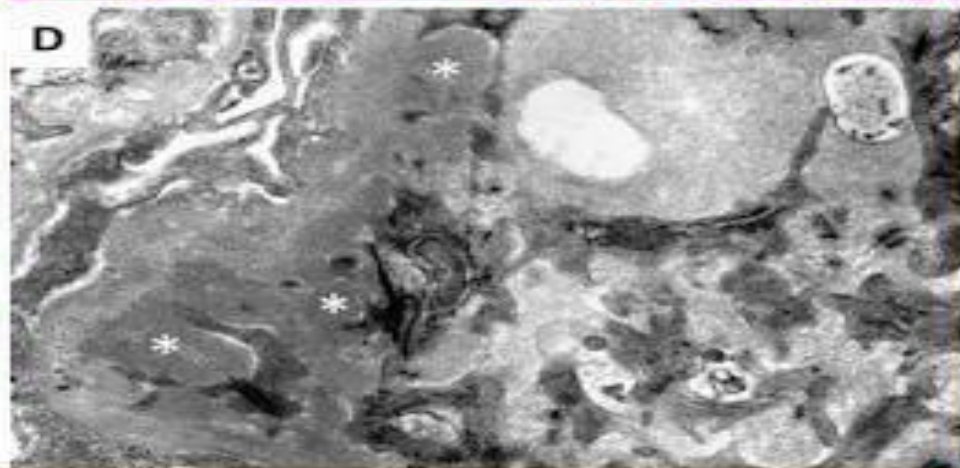
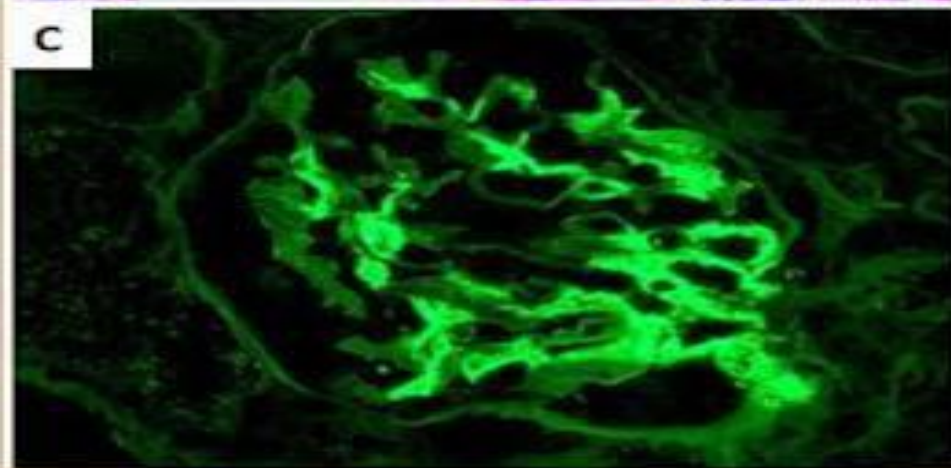
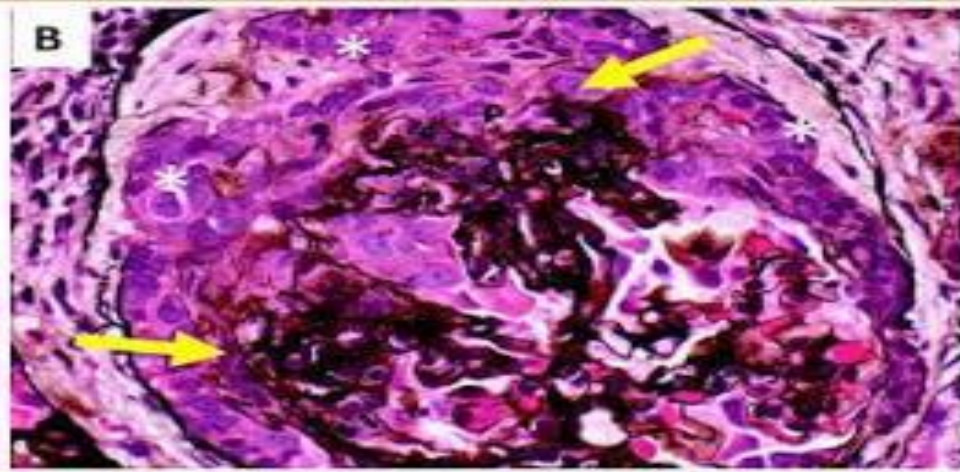
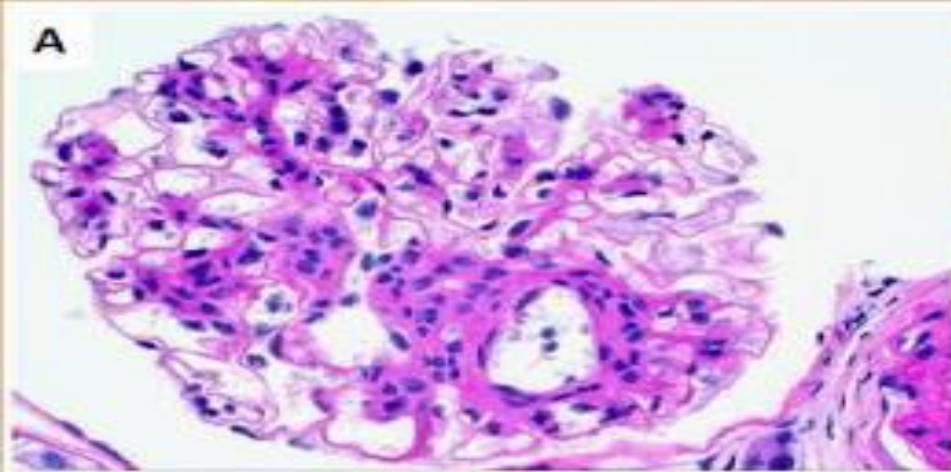
- LM: Hypercellular glomeruli (neutrophils, proliferation)
- IF: Granular IgG, IgM, C₃ ('starry sky')
- EM: Subepithelial 'humps'
- Follows streptococcal infection



- Post-Streptococcal GN

IgA Nephropathy (Berger's Disease)

- LM: Mesangial proliferation
- IF: Mesangial IgA deposition
- EM: Electron-dense mesangial deposits
- Presents after upper respiratory infection



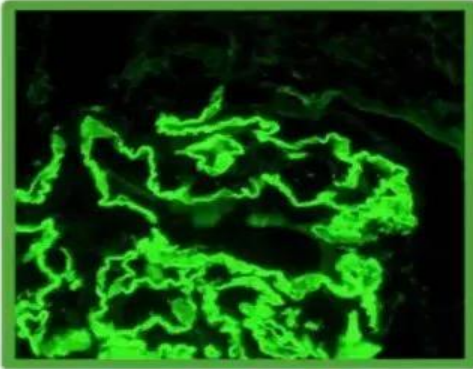
- IgA Nephropathy (Berger's Disease)

Crescentic GN (RPGN)

- LM: Crescents in Bowman's space
- IF/EM varies:
 - Linear (Anti-GBM)
 - Granular (Immune complex)
 - Negative (Pauci-immune)

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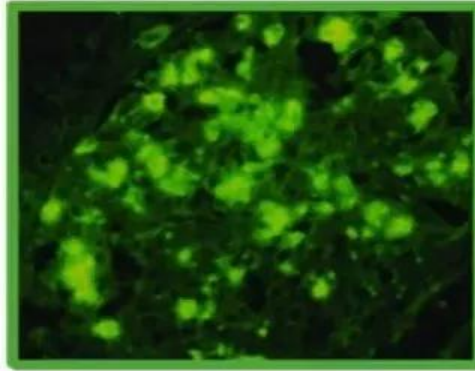
TYPE I



"LINEAR"

* ANTI-GBM binds to
COLLAGEN of GBM

TYPE II



"GRANULAR"

* Immune complex
deposition in
subendothelium

TYPE III



* **NEGATIVE** *

- Crescentic GN (RPGN)

Pathogenesis Overview

- Immune complex deposition → SLE, post-strep GN
- Anti-GBM antibodies → Goodpasture
- Cell-mediated injury → FSGS
- Metabolic/hemodynamic → Diabetes, hypertension

Outcome

- Acute: May resolve completely (post-strep GN)
- Chronic: Glomerulosclerosis, renal failure
- Early recognition and treatment slow progression

