

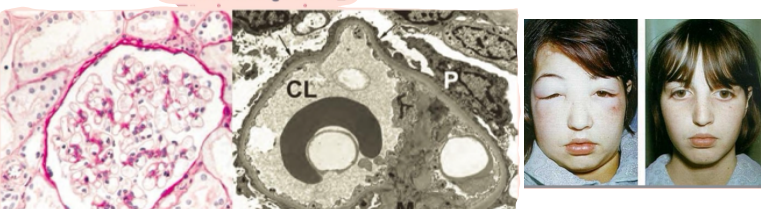
## 1. Minimal Change Disease (MCD)

- \* Most common cause of nephrotic syndrome in children
- \* Caused by T-cell dysfunction → podocyte injury
- \* LM: normal glomeruli
- \* IF: negative
- \* EM: diffuse foot process effacement
- \* Selective albuminuria
- \* Usually no hematuria or hypertension
- \* Excellent response to corticosteroids
- \* Very good prognosis

Most important point

Child with sudden nephrotic syndrome + normal LM + foot process effacement = MCD

Minimal change disease

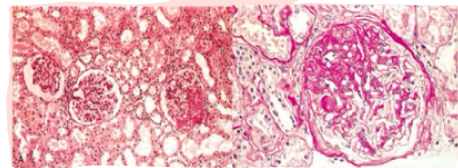


primary kidney diseases w/ Nephrotic Syndrome

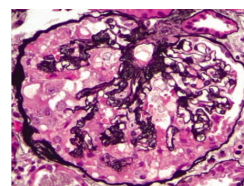
## 2. Focal Segmental Glomerulosclerosis (FSGS)

- \* Most common nephrotic syndrome in adults
- \* Focal: some glomeruli affected
- \* Segmental: only part of glomerulus affected
- \* Primary (idiopathic) or secondary
- \* Secondary causes:
  - \* HIV
  - \* heroin abuse
  - \* nephron loss
  - \* IgA nephropathy
- \* Podocyte injury is key mechanism
- \* LM: focal segmental sclerosis
- \* EM: foot process effacement
- \* Poor steroid response
- \* High risk of progression to renal failure

FSGS - Morphology



Collapsing Glomerulopathy



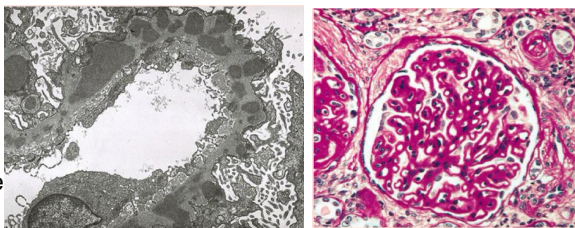
- \* Severe variant of FSGS
- \* Associated with HIV
- \* Collapse of glomerular tuft + epithelial proliferation
- \* Worse prognosis

Most important point

FSGS = nephrotic syndrome with hematuria + hypertension + poor steroid response

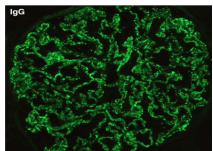
## 3. Membranous Nephropathy

- \* Common nephrotic syndrome in older adults
- \* Chronic immune complex disease
- \* Most primary cases due to antibodies against PLA2R
- \* Secondary causes:
  - \* HBV
  - \* SLE
  - \* malignancy
  - \* NSAIDs
  - \* gold
  - \* penicillamine



Morphology

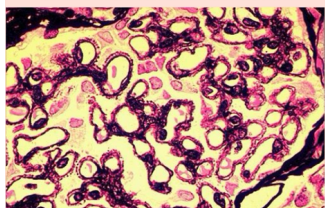
- \* LM: diffuse GBM thickening
- \* EM: subepithelial deposits with spike and dome
- \* IF: granular IgG and C3



A silver stain (black) of the GBM → appears with characteristics spikes (projections in capillary loops)

Clinical

- \* Nonselective proteinuria
- \* Poor steroid response
- \* Many progress to renal failure



Most important point

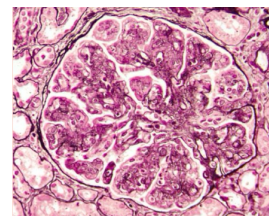
Membranous nephropathy = nephrotic syndrome + spike and dome subepithelial deposits

## 4. Membranoproliferative Glomerulonephritis (MPGN)

- \* Immune-mediated glomerular injury
- \* Mesangial proliferation + GBM alteration

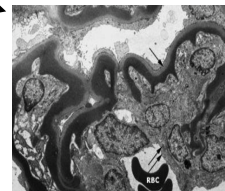
Type I MPGN

- \* Immune complex mediated
- \* Associated with HBV and HCV



Dense Deposit Disease (Type II)

- \* Due to uncontrolled complement activation
- \* C3 nephritic factor stabilizes C3 convertase

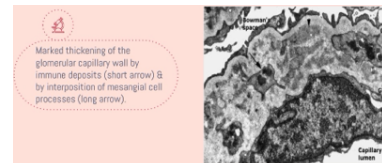


Morphology

- \* LM: tram-track GBM splitting
- \* Lobulated glomeruli
- \* Hypercellularity

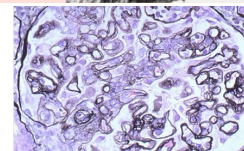
IF

- \* Type I: IgG + C3
- \* Dense deposit disease: only C3



EM

- \* Dense ribbon-like deposits within GBM in dense deposit disease

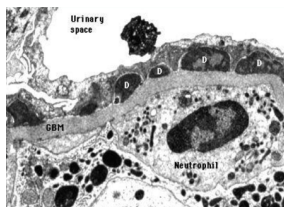
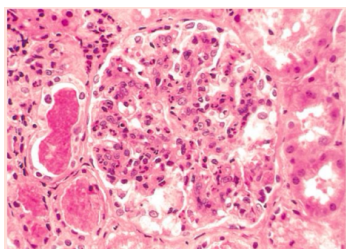


Most important point

MPGN = tram-track appearance

## 5. Acute Poststreptococcal (Postinfectious) Glomerulonephritis

- \* Occurs 1–4 weeks after streptococcal infection
- \* Usually in children
- \* Immune complex disease



### Clinical

- \* Acute nephritic syndrome
- \* Hematuria
- \* Mild proteinuria
- \* Low complement
- \* Elevated ASO titer

### Morphology

- \* LM: diffuse hypercellular glomeruli with neutrophils
- \* IF: granular IgG and C3
- \* EM: subepithelial humps

### Most important point

Child after sore throat + cola-colored urine + subepithelial humps = PSGN

## 6. IgA Nephropathy (Berger Disease)

- \* One of the most common causes of recurrent hematuria
- \* Usually affects children and young adults
- \* Triggered by URI or GI infection
- \* Increased IgA production → mesangial deposition

### Clinical

- \* Recurrent episodic hematuria
- \* Occurs within 1–2 days of URI

### Morphology

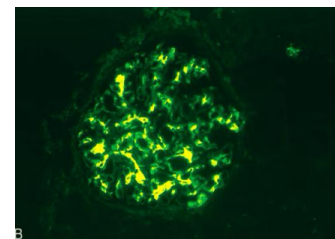
- \* IF: mesangial IgA and C3 deposition

### Association

- \* Henoch-Schönlein purpura

### Most important point

Hematuria immediately after URI = IgA nephropathy



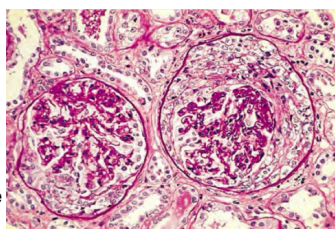
Different LM findings but whatever the histologic lesions, the pathognomonic feature by IF is the deposition of IgA and C3, in the mesangial region. (diagnostic)

## 7. Rapidly Progressive (Crescentic) Glomerulonephritis (RPGN)

- \* Rapid loss of renal function within weeks to months
- \* Severe nephritic syndrome
- \* Characterized by crescents

### Crescent formation

- \* Proliferation of parietal epithelial cells
- \* Macrophage infiltration into Bowman space



### Types

#### 1. Anti-GBM disease (Goodpasture)

- \* Linear IgG deposition
- \* Pulmonary hemorrhage + renal failure
- \* Anti-GBM antibodies in serum

#### 2. Immune complex RPGN

- \* Complication of other GN diseases

#### 3. Pauci-immune RPGN

- \* Associated with ANCA

### Morphology

- \* Crescents compress glomerular tuft

### Most important point

RPGN = crescents + rapidly progressive renal failure

## 8. Goodpasture Syndrome

- \* Anti-GBM antibody disease
- \* Antibodies attack:
  - \* glomerular basement membrane
  - \* alveolar basement membrane

### Clinical

- \* Hematuria
- \* Renal failure
- \* Pulmonary hemorrhage

### IF

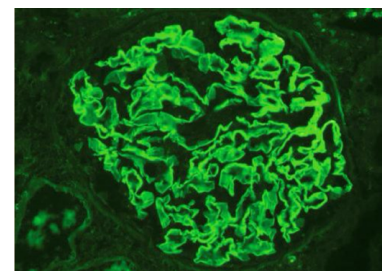
- \* Linear IgG deposition

### Treatment importance

- \* Plasmapheresis removes antibodies

### Most important point

Hemoptysis + nephritic syndrome + linear IF = Goodpasture syndrome



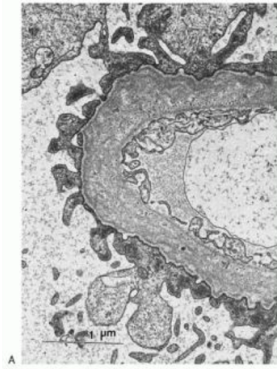
## 9. Alport Syndrome

- \* Hereditary nephritis due to type IV collagen mutation
- \* Usually X-linked

Clinical triad

- \* Nephritis
- \* Sensorineural deafness
- \* Eye abnormalities

EM



- \* Basket-weave GBM appearance
- \* Splitting and lamellation of GBM

Most important point

Nephritis + deafness + eye defects = Alport syndrome