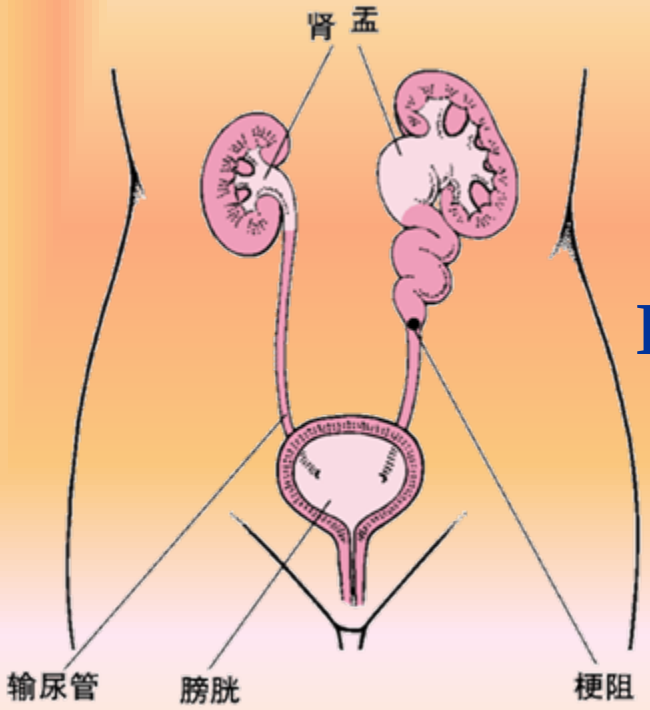
# Diseases of the Urinary System




**Wang Yishu**
**Department of Pathology**
**Basic Medical School**

# Etiology and pathogenesis

**Endogenous antigens**

**Exogenous antigens**

antigen

antbody

*Antibodies to glomerular cells*
*Cell-mediated immunity in glomerulonephritis*
*Activation of alternative complement pathway*

antigen

antbody

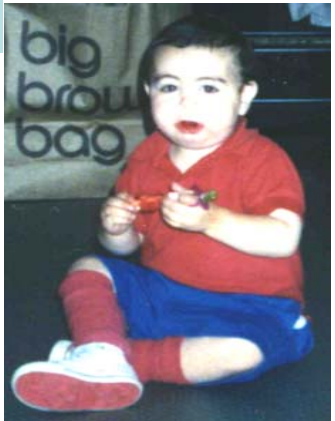
*Circulating immune complex*
*In situ immune complex*

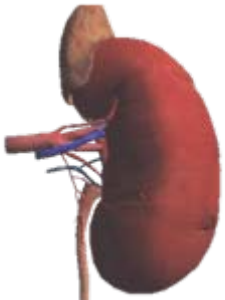
**immune complex**

**Mediators of glomerular injury**

mediators of inflammation

glomerulus damage



# Basic pathological changes

**Glomerulus**

**Proliferation: Parietal epithelial cells, Mesangial cells, Endothelia Cells**
**Infiltration: Neutrophils, LCs ,Monocytes**

- **increase**
- **Hyline change and Sclerosis**
- **Inflammatory exudation and necrosis**

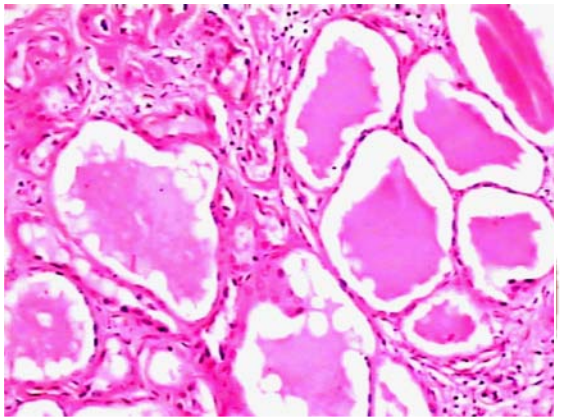
**Tubules**

**Epithelial cell degeneration, Cast, Atrophy, Disappear**

**Renal interstitium**

**Hyperemia, Edema, Inflammatory cell infiltration, fibrosis**

**Blood vessel**



# Basic pathological changes

## Inflammation

**Proliferation**: Epithelial cells, Mesangial cells, Endothelia Cells ;

GBM thickening and Mesangial matrix increase

**Degeneration**: Fibrinoid necrosis, Hyline change and Sclerosis. Epithelial cell of tubulesdegeneration

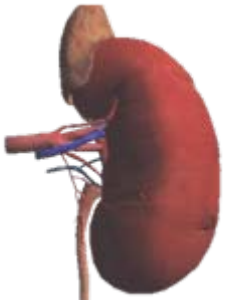
**Exsudation**: Neutrophils, LCs ,Monocytes

## Immunoreaction

*Antigen+Antibody=immune complex*

*Circulating immune complex nephritis*

*In situ immune complex deposition*



# Pathological type

**Acute diffuse proliferative glomerulonephritis (GN)**

**Rapidly progressive GN（RPRN)**

**Crescentic glomerulonephritis(CrGN )**

**Membranous GN（membranous nephropathy）**

**Membranoproliferative GN (MPGN)**

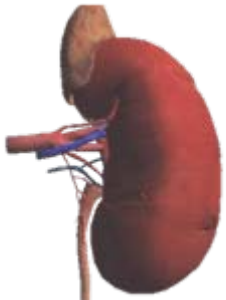
**Mesangial proliferative GN**

**Minimal change GN （lipoid nephrosis ）**

**Focal segmental glomerulosclerosis(FSG )**

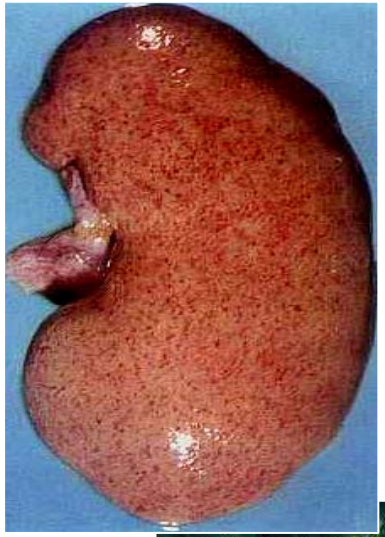
**IgA nephropathy**

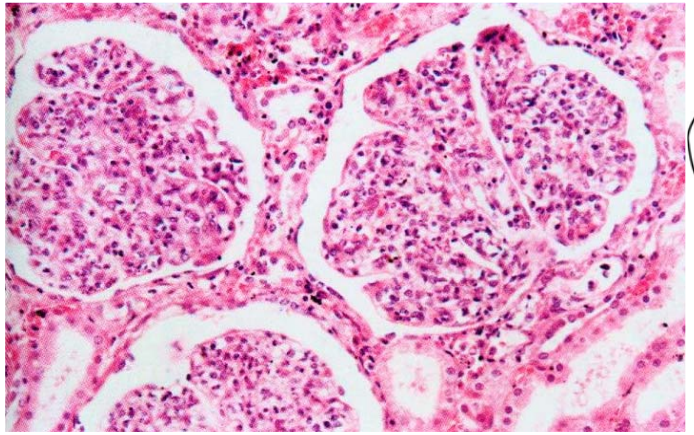
**Chronic GN**

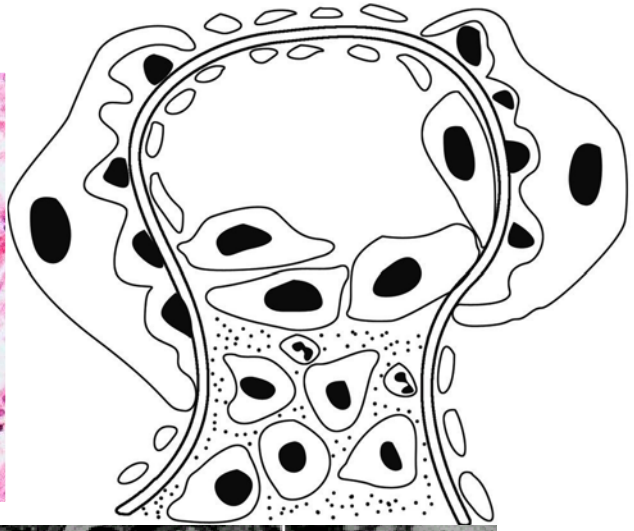


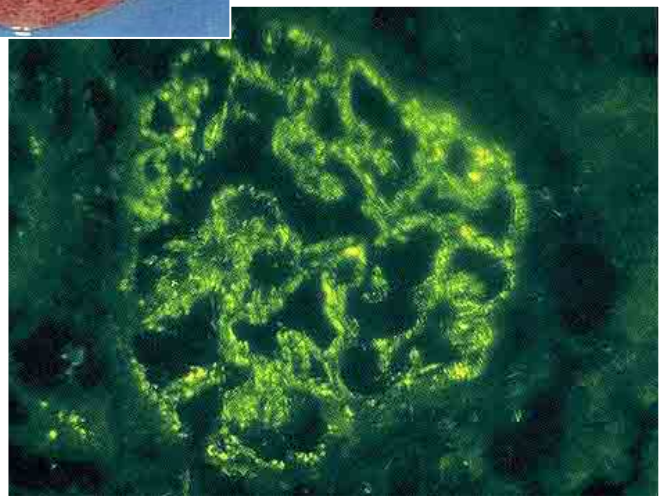
# summary

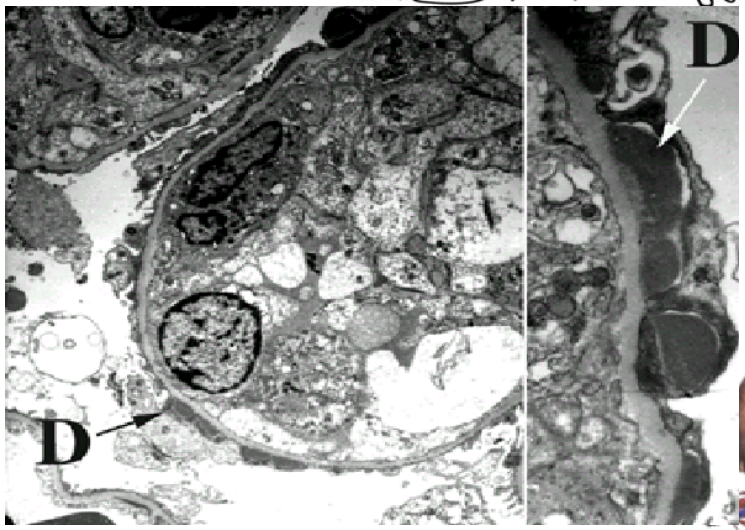
## Acute diffuse proliferative glomerulonephritis, Post-infectious GN

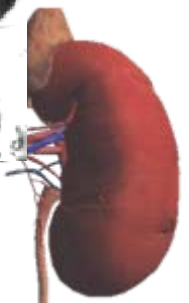






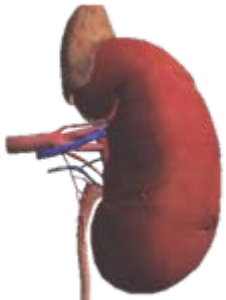




acute nephritic syndrome

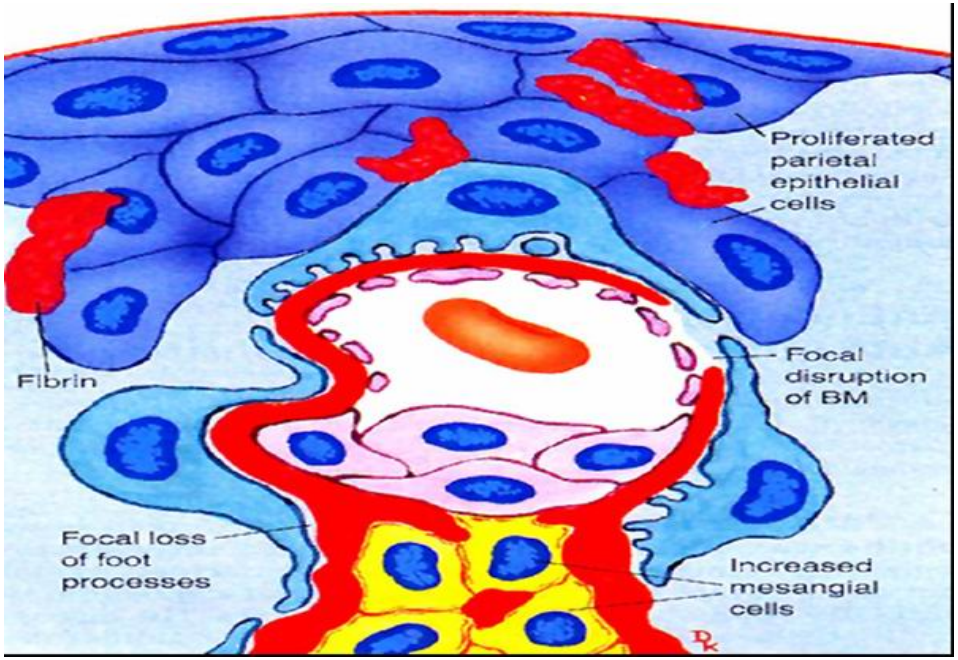
# Rapidly progressive GN，RPGN



# Introduction

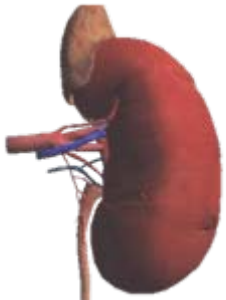
## Pathological character

- Hyperplasia of parietal epithelial cells→ formation of crescent
- Crescentic glomerulonephritis，CrGN




## Clinical manifestation

Rapidly progressive nephritic syndrome



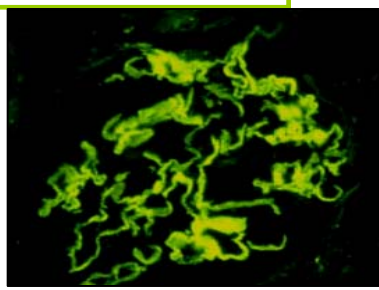
# Classification and pathogenesis

## TypeI RPGN (anti-GBM antibody)

anti-GBM nephritis

**linear immunofluorescence**

linear deposits of IgG, C3
→glomerular and
alveolar BM



## TypeII RPGN (Immune complex)

electron-dense deposits
BM and mesangium

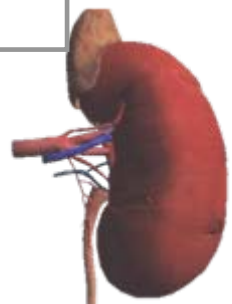
**granular immunofluorescence**

granular (BM, mesangium)

## TypeIII RPGN (pauci-Immune)

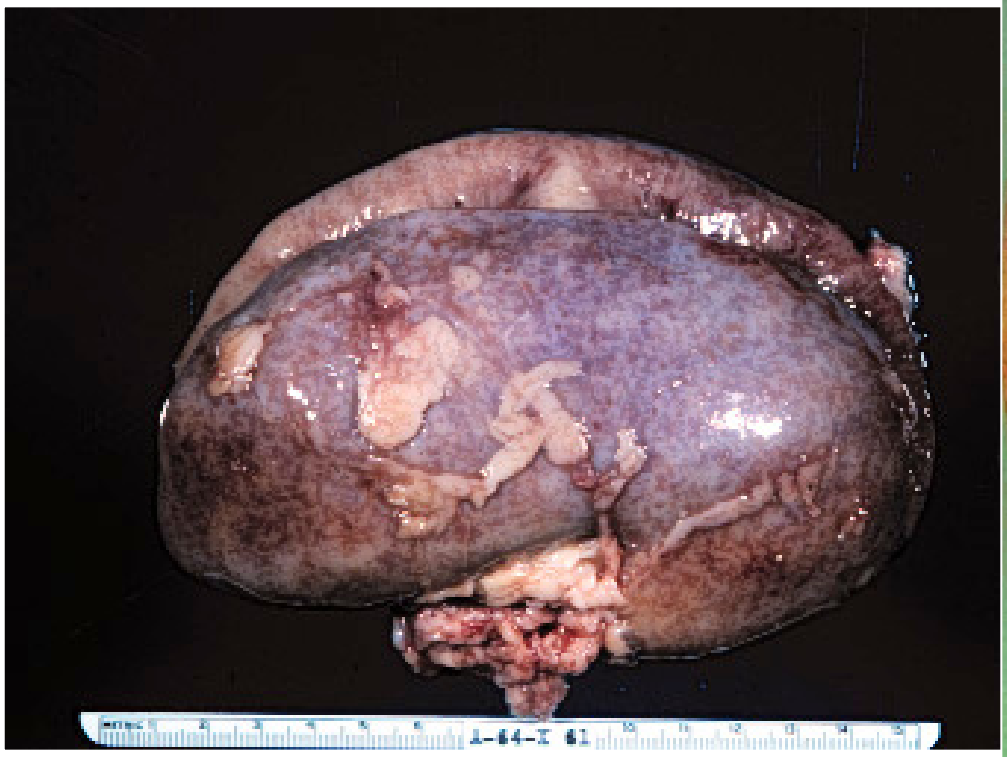
There are minimal immune
deposits or none

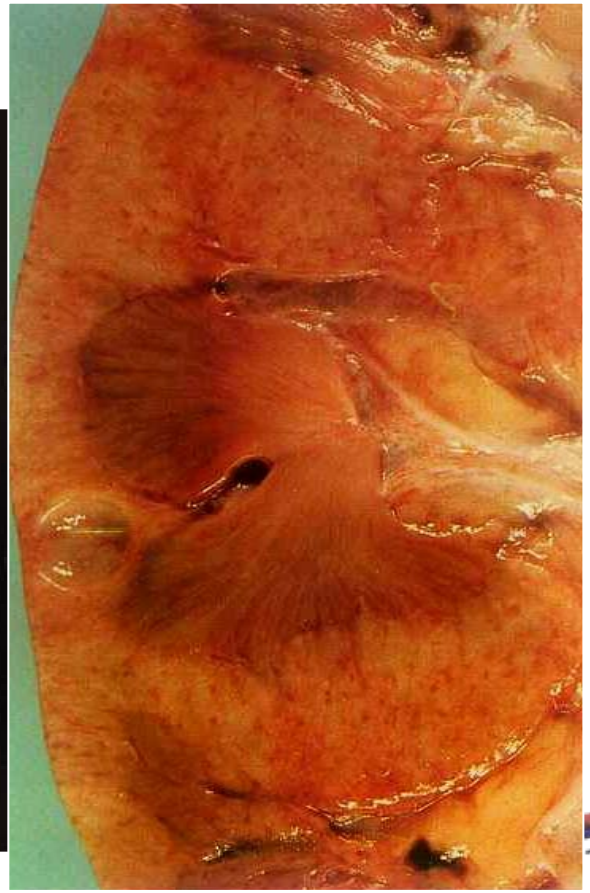
**immunofluorescence (-)**



# Pathological changes

- ✓ **Gross: large pale kidney， the cortex is pale and swollen**





# Pathological changes

**LM:** Crescent formation

**Crescents:** glomeruli(>50%)
proliferation of parietal epithelial cellsin Bowman's space
infiltration of monocytes, macrophages and fibrin

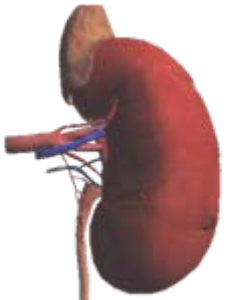
**Process:**

cellular crescent
↓
fibrous-cellular crescent
↓
fibrous crescent

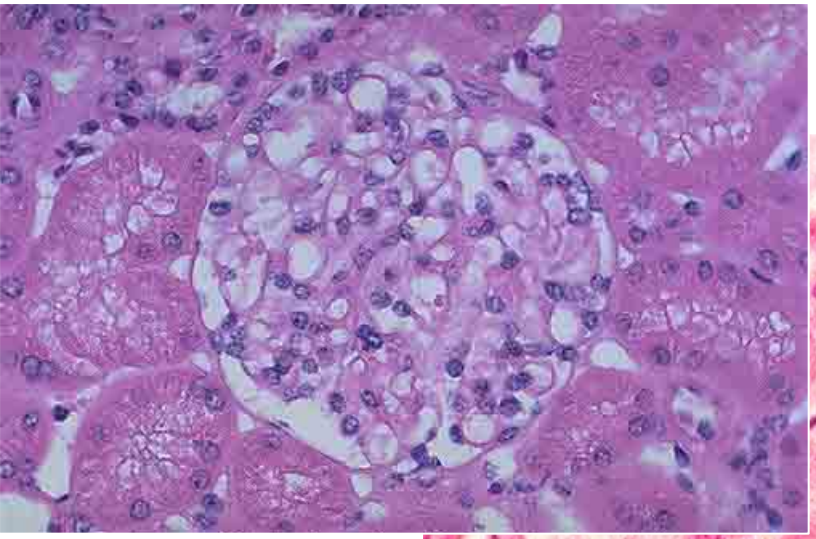
**EM: crescents, focal defect or disruption of BM**

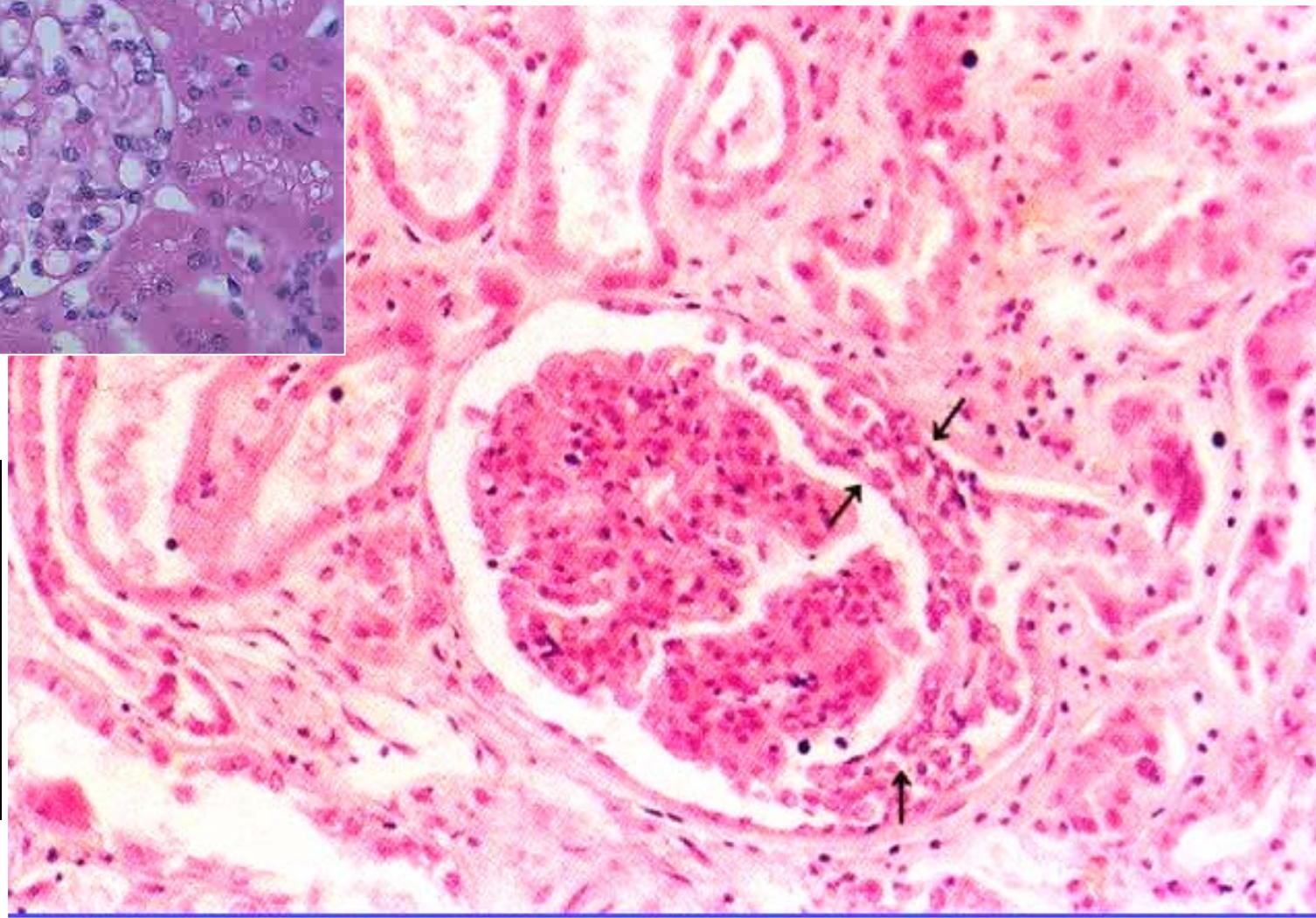
**IF:** Type I (linear immunofluorescence), Type II (granular fluorescence)

Type III (no fluorescence)

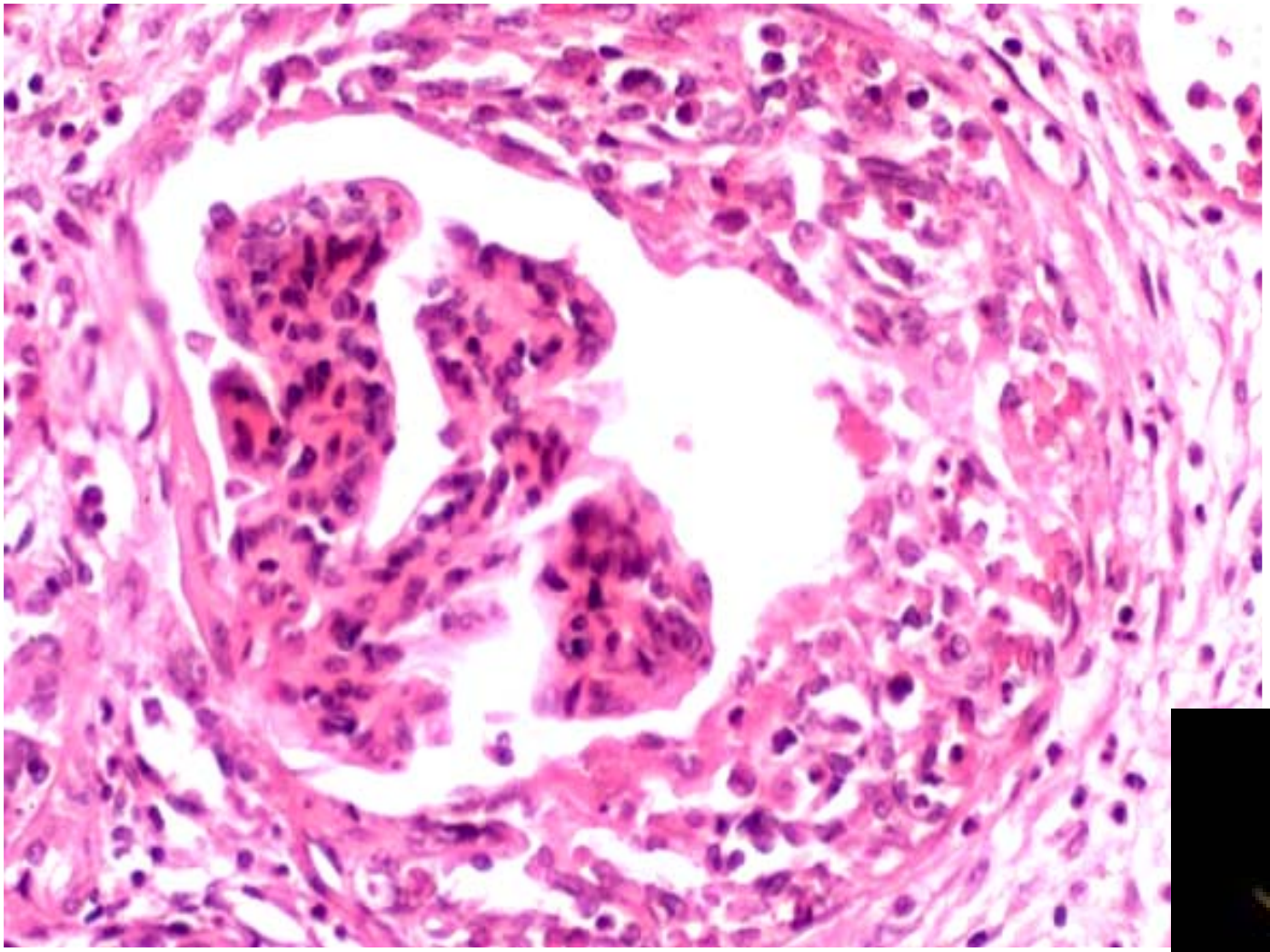


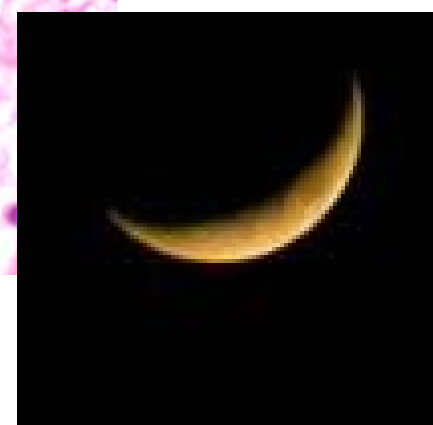
# Pathological changes





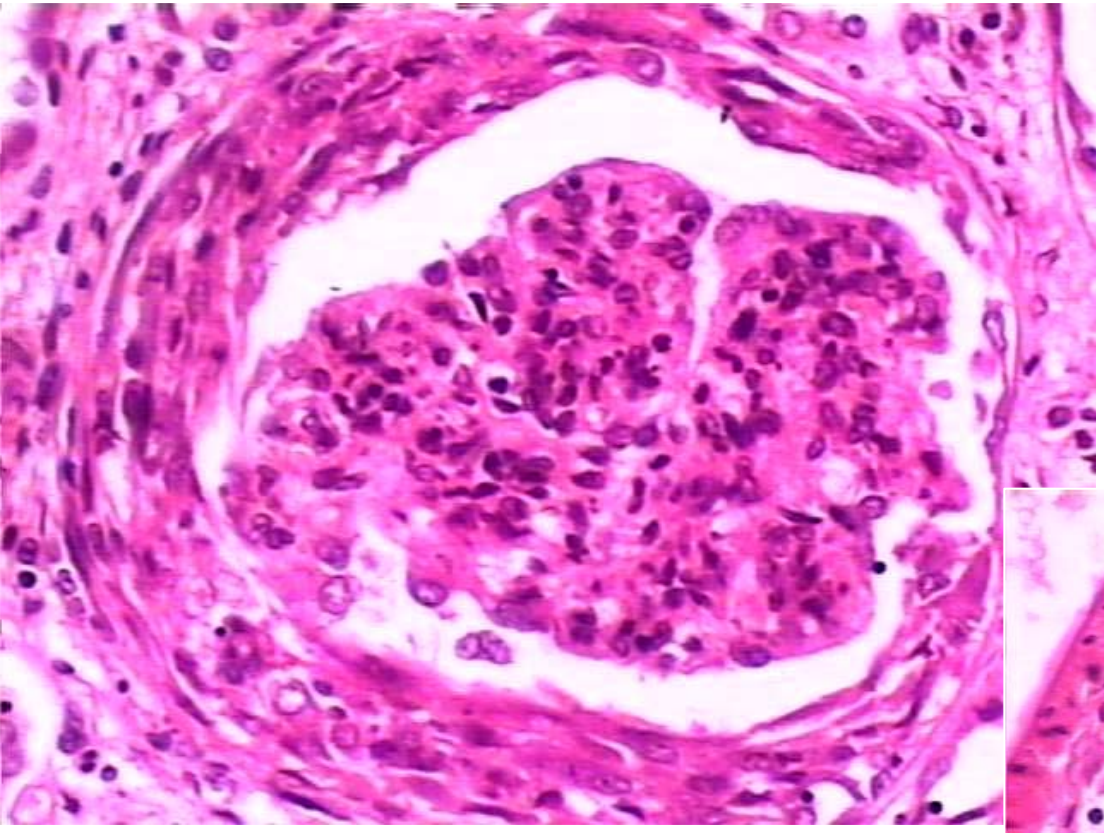
# Pathological changes





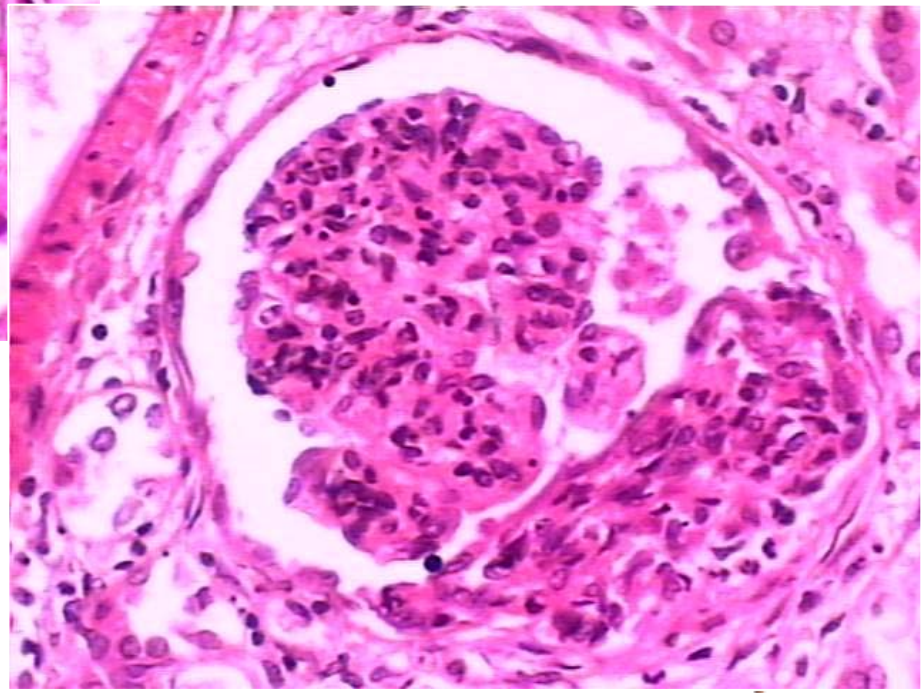
**cellular crescent**

# Pathological changes

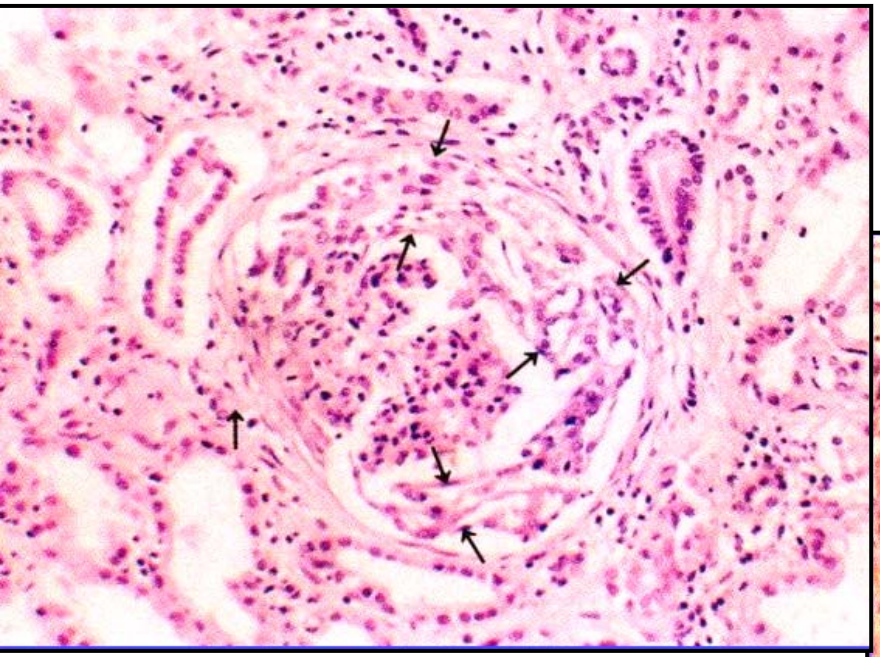


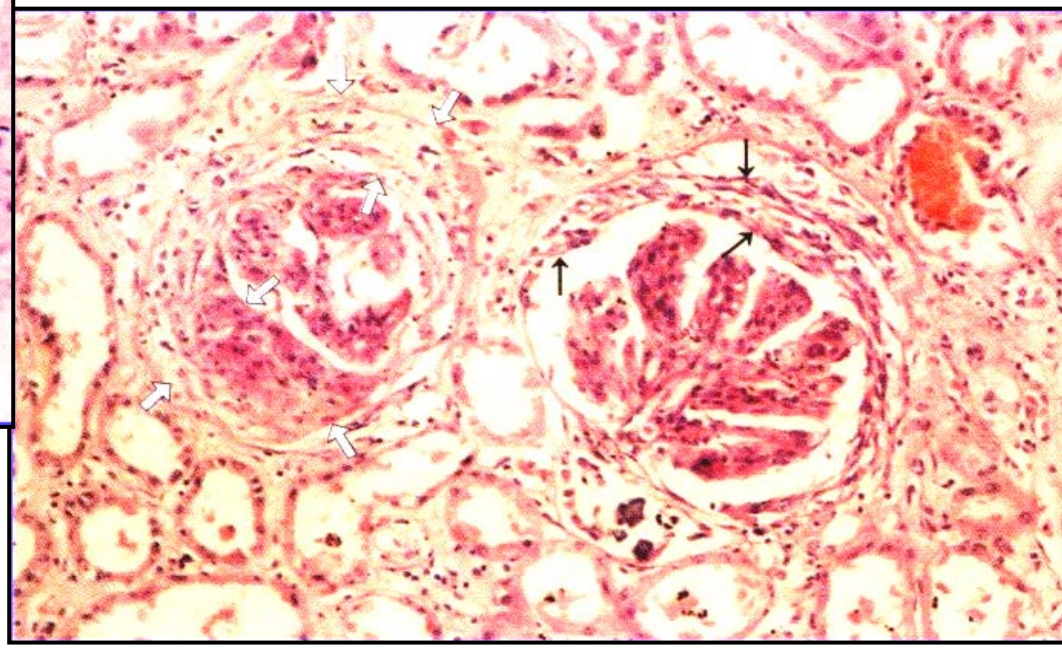
**fibrous-crescent**

**fibrous-cellular crescent**

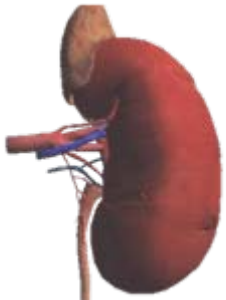


# Pathological changes

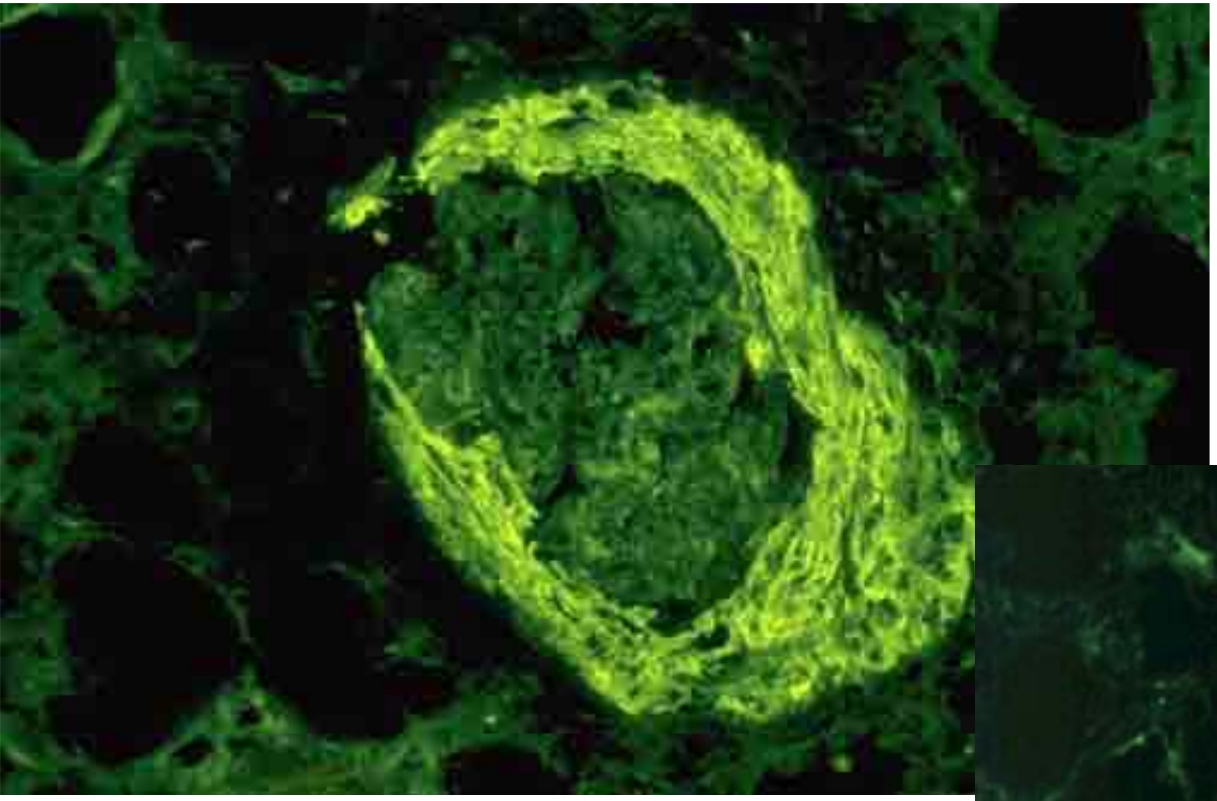


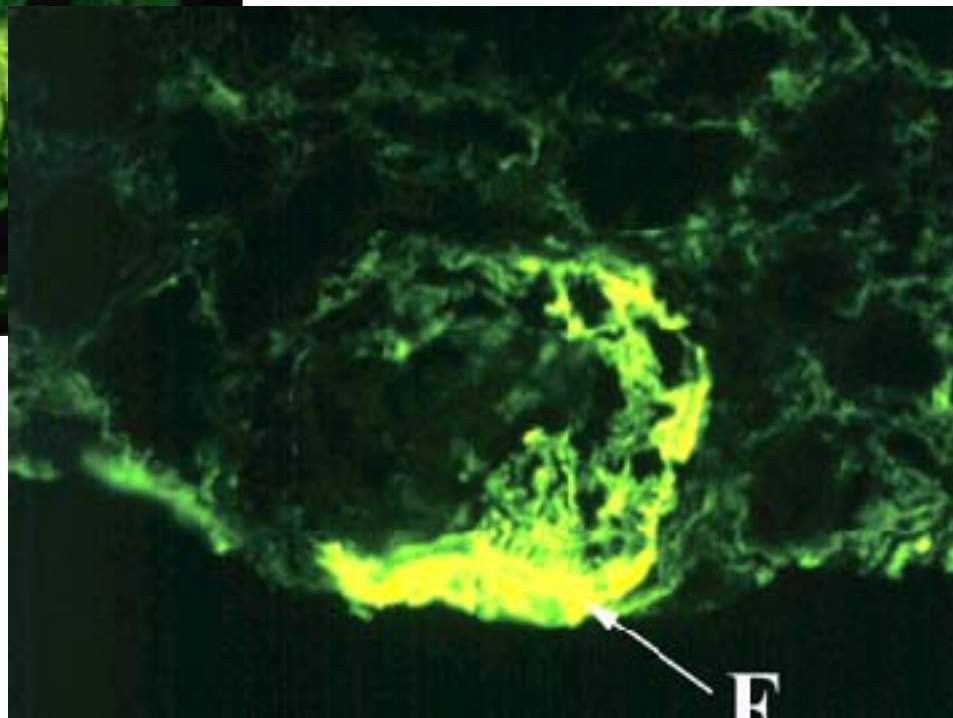


**fibrous-crescent**



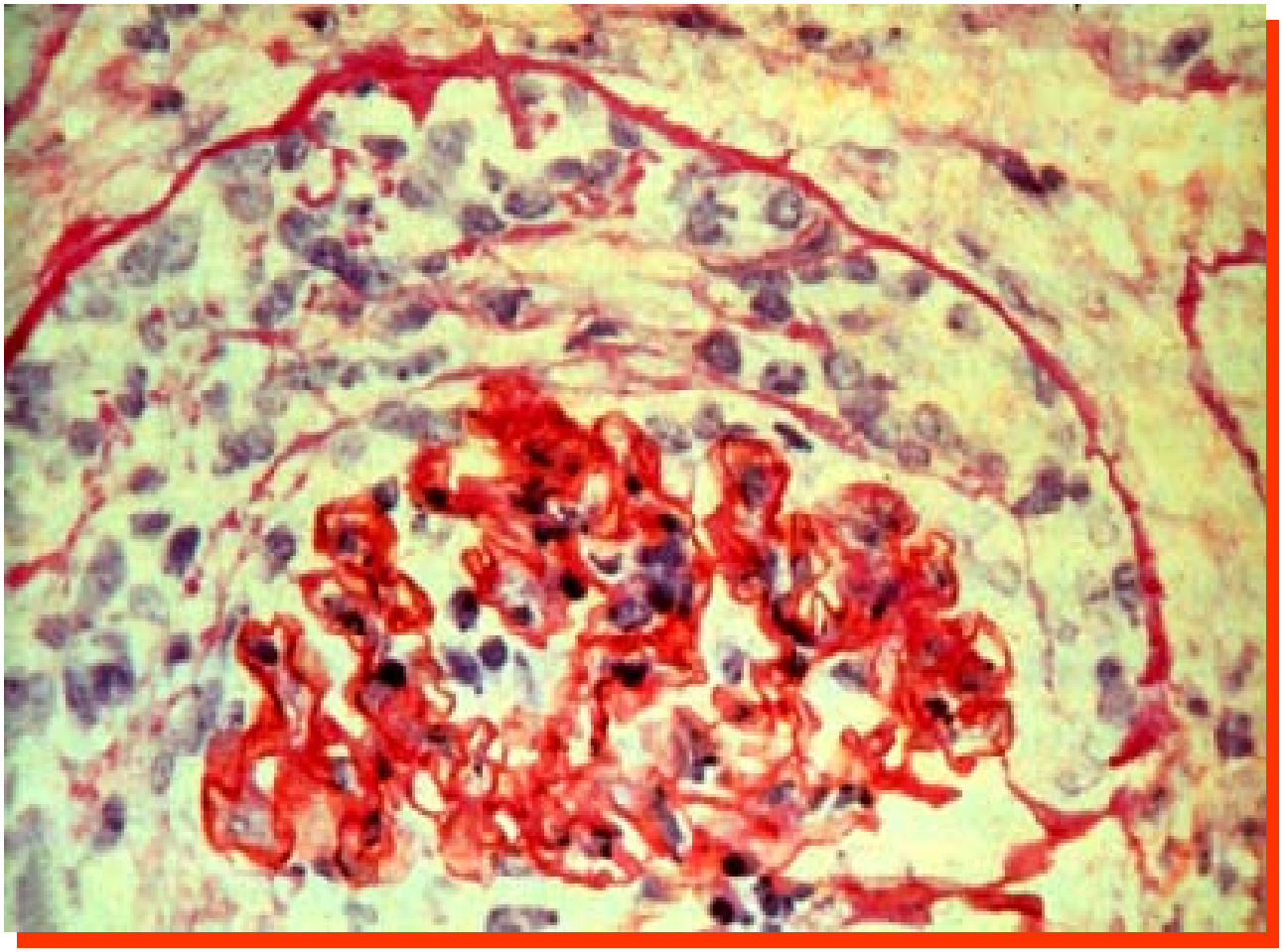
# Pathological changes



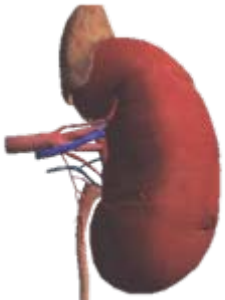



This IF micrograph of a glomerulus demonstrates positivity with antibody to fibrinogen.

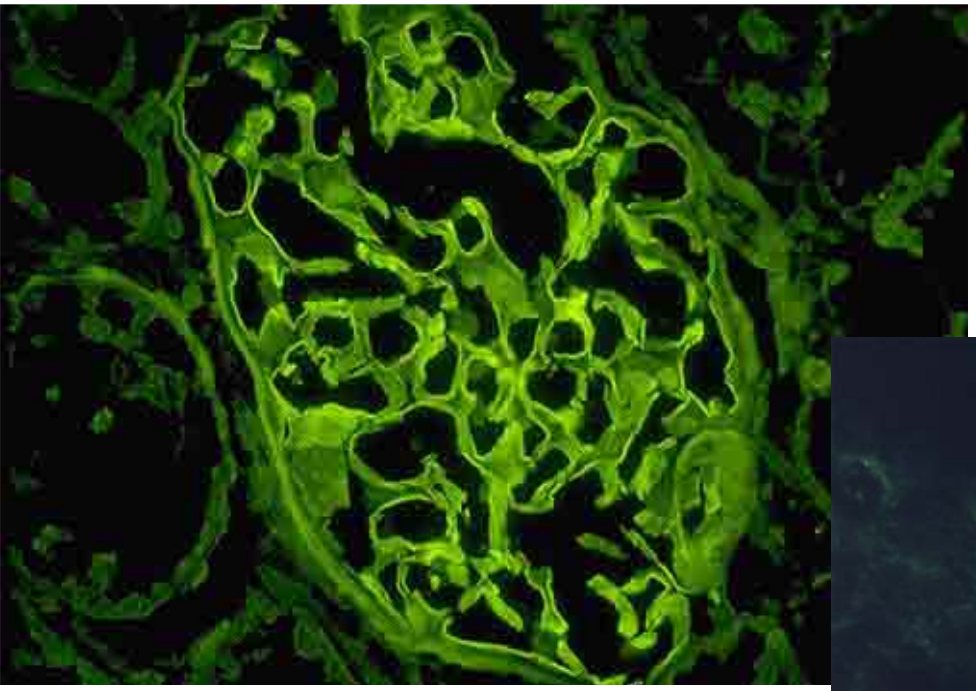
# Pathological changes

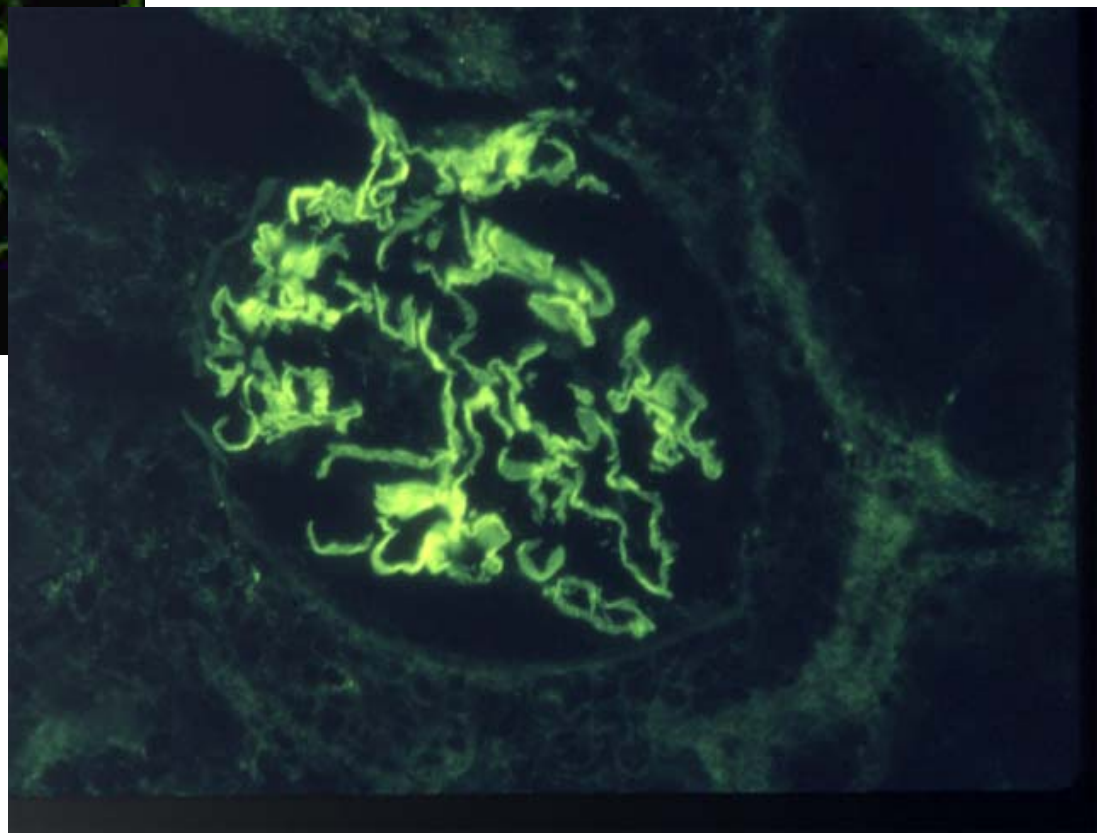


TypeI， linear immunofluorescence of IgG



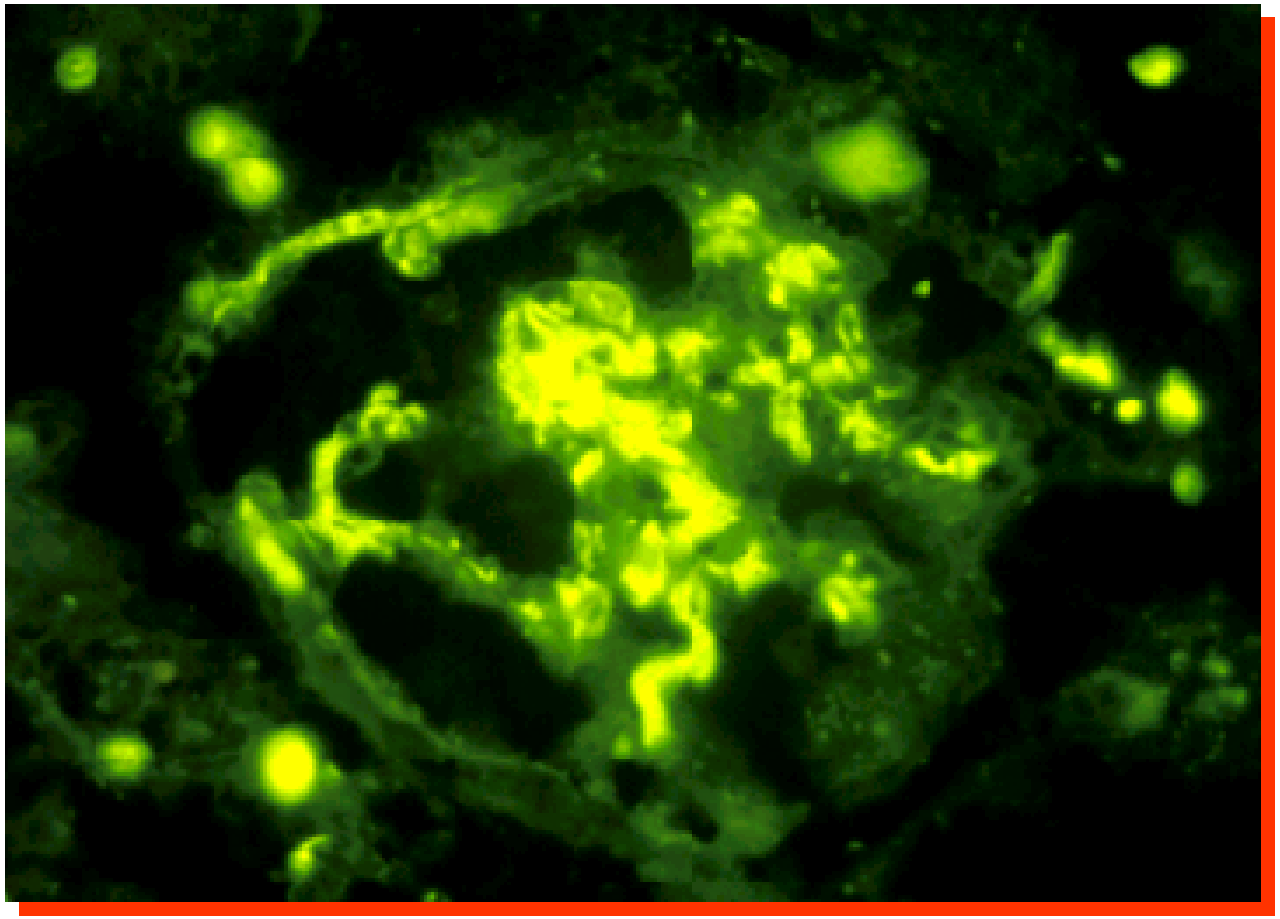
# Pathological changes





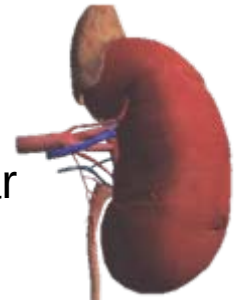
fluorescence in a smooth, diffuse, linear pattern

# Pathological changes

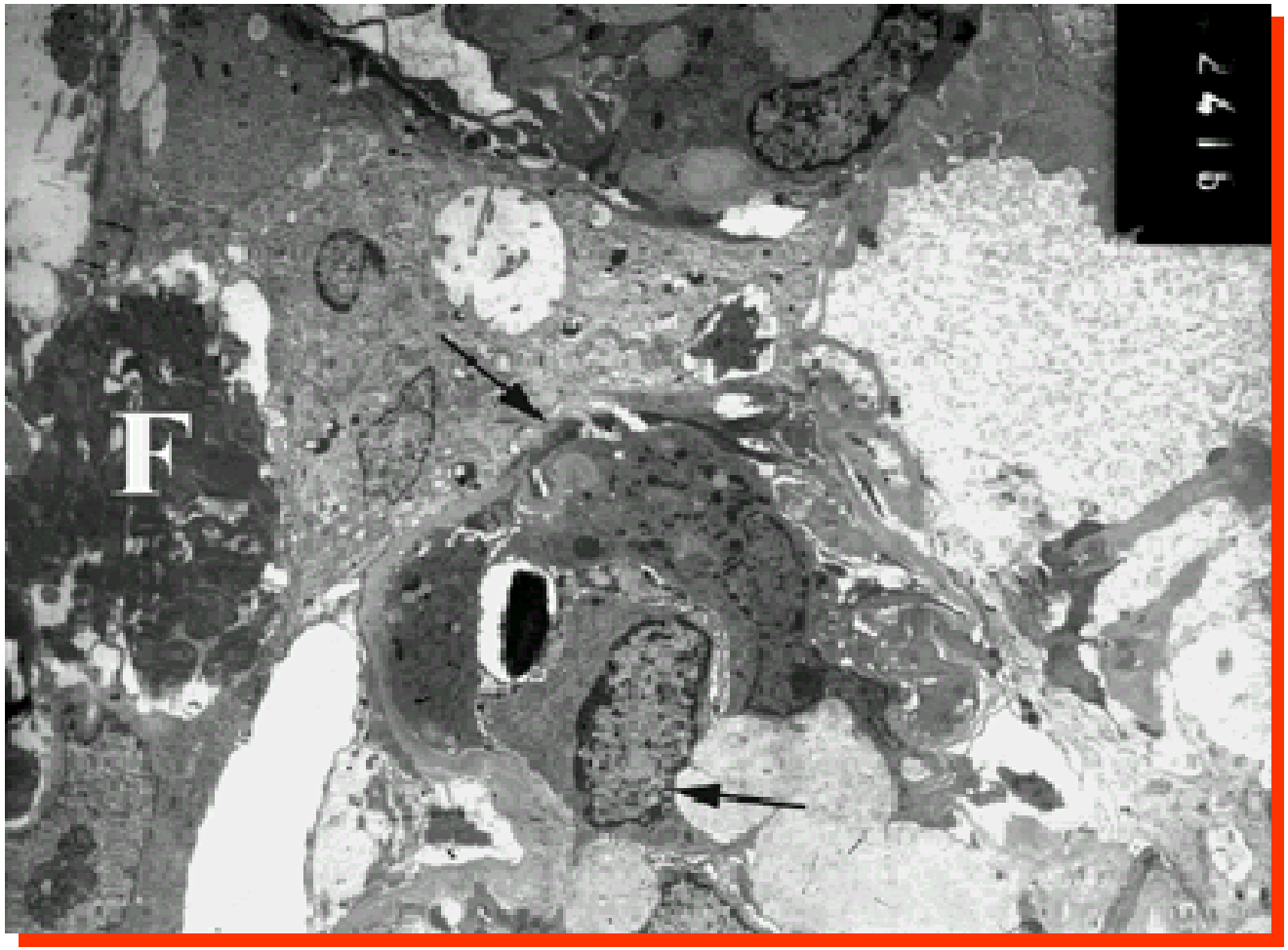


**Crescentic glomerulonephritis, Type** II

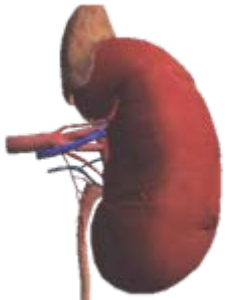
Direct IF shows **mass and granular pattern** staining of the glomerular capillary and Mesangium for IgA.



# Pathological changes




GBM break(↑)，Fibrin deposition (F)。



# Pathological changes

**Renal tubules : hyaline change**

**Intracellular hyaline**

↓

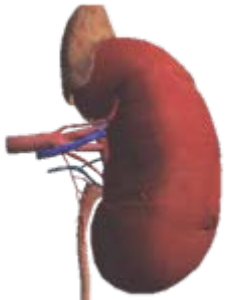
**hyaline droplets**

**Atrophy**

**Interstitium**

**edema**

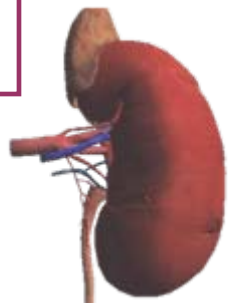
**inflammatory infiltration**



# Clinical features

- Characterized clinically by rapid and progressive loss of renal function associated with severe oliguria and death from renal failure within weeks to months.
- Rapidly progressive nephritic syndrome

**Hematuria, Proteinuria, Oliguria or Anuria, Edema, Azotemia→acute renal failure**

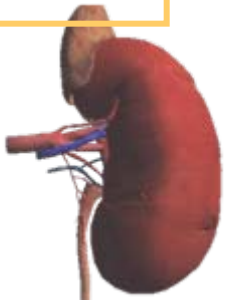


# Rapidly progressive GN (Crescentic GN)

## Pathological changes

- ✓**Gross: large pale kidney**
- ✓**LM: formation of crescents**
- ✓**EM: crescents, focal defect or disruption of BM**
- ✓**IF: Type I (linear immunofluorescence), Type II (granular fluorescence)**

  **Type III (no fluorescence)**



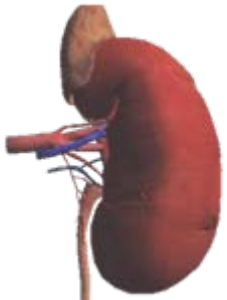
# Case

患者，男性，26岁。因浮肿、血尿、少尿20天，恶心、呕吐3天入院。

体格检查：血压164/100mmHg，面色苍白，颜面部及双下肢浮肿。

实验室检查：24小时尿量150ml，尿色洗肉水样，尿蛋白（++），红细胞（+++），红细胞管型1～3个/HP；肌酐426μmol/L （＜178μmol/L） 。B超检查示：双肾增大。

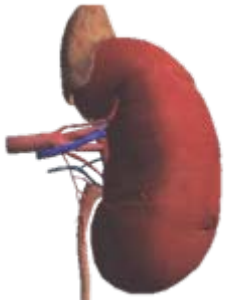
诊断：快速进行性肾小球肾炎



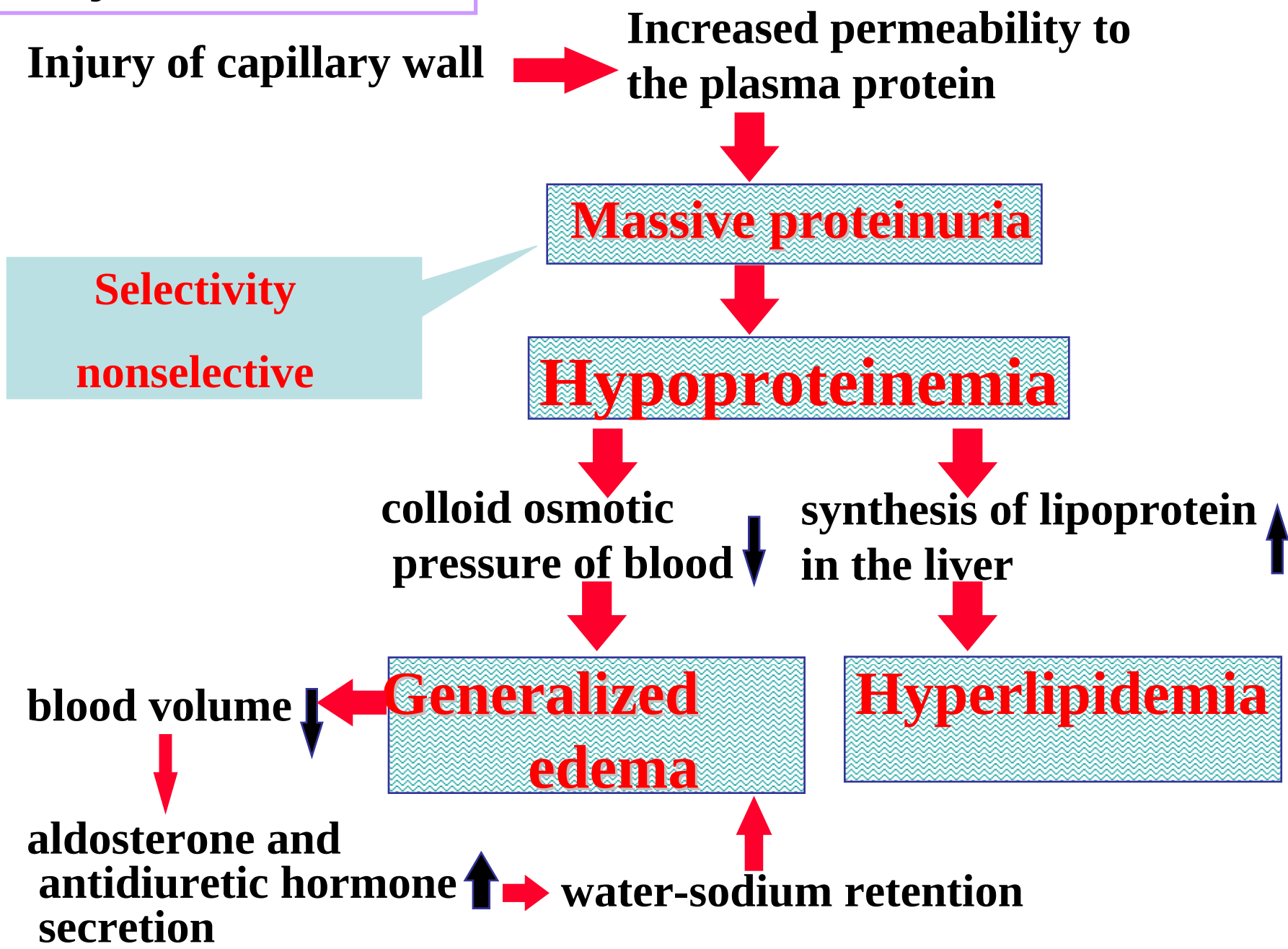
Still remember?

# Nephrotic syndrome(NS)

- **Massive proteinuria**
- **Hypoalbuminemia**
- **Generalized edema**
- **Hyperlipidemia and lipiduria**



# *Do you know?*


Injury of capillary wall
Increased permeability to the plasma protein
Massive proteinuria
Selectivity
nonselective
Hypoproteinemia
colloid osmotic pressure of blood
synthesis of lipoprotein in the liver
blood volume
Generalized edema
Hyperlipidemia
aldosterone and antidiuretic hormone secretion
water-sodium retention

# Causes of Nephrotic Syndrome

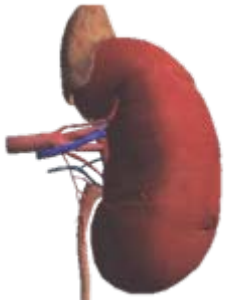
| | Prevalence* (%) Children | Adults | |
|---|---|---|---|
| ***Primary Glomerular Disease*** | | | |
| Membranous glomerulonephritis (GN) | 5 | 40 | |
| Lipoid nephrosis | 65 | 15 | |
| Focal segmental glomerulosclerosis | 10 | 15 | |
| Membranoproliferative GN | 10 | 7 | |
| Other proliferative GN (focal, "pure mesangial," IgA nephropathy) | 10 | 23 | |
| ***Systemic Diseases*** | | | |
| Diabetes mellitus | | | Most common systemic causes |
| Amyloidosis | | | |
| Systemic lupus erythematosus | | | |
| Drugs (gold, penicillamine, "street heroin") | | | |
| Infections (malaria, syphilis hepatitis, B, AIDS) | | | |
| Malignancy (carcinoma, melanoma) | | | |
| Miscellaneous (bee-sting allergy, hereditary nephritis) | | | |

*Approximate prevalence of primary disease is 95% in children, 60% in adults. Approximate prevalence of systemic disease is 5% in children, 40% in adults.

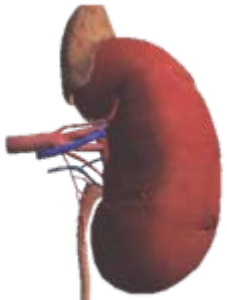
Home Page

# *Pathologic types producing nephrotic syndrome(NS)*

- **Membranous GN（membranous nephropathy）**
- **Minimal change GN （lipoid nephrosis ）**
- **Focal segmental glomerulosclerosis(FSG )**
- **Membranoproliferative GN (MPGN)**
- **Mesangial proliferative GN**



# Membranous GN



# Introduction

- **Features:**
  - Diffuse **thickening of capillary wall**
  - Immune complex depositing along **epithelium (subepithelial) side.**

- **Etiology and pathogenesis**

**anti-glomerular antigen**

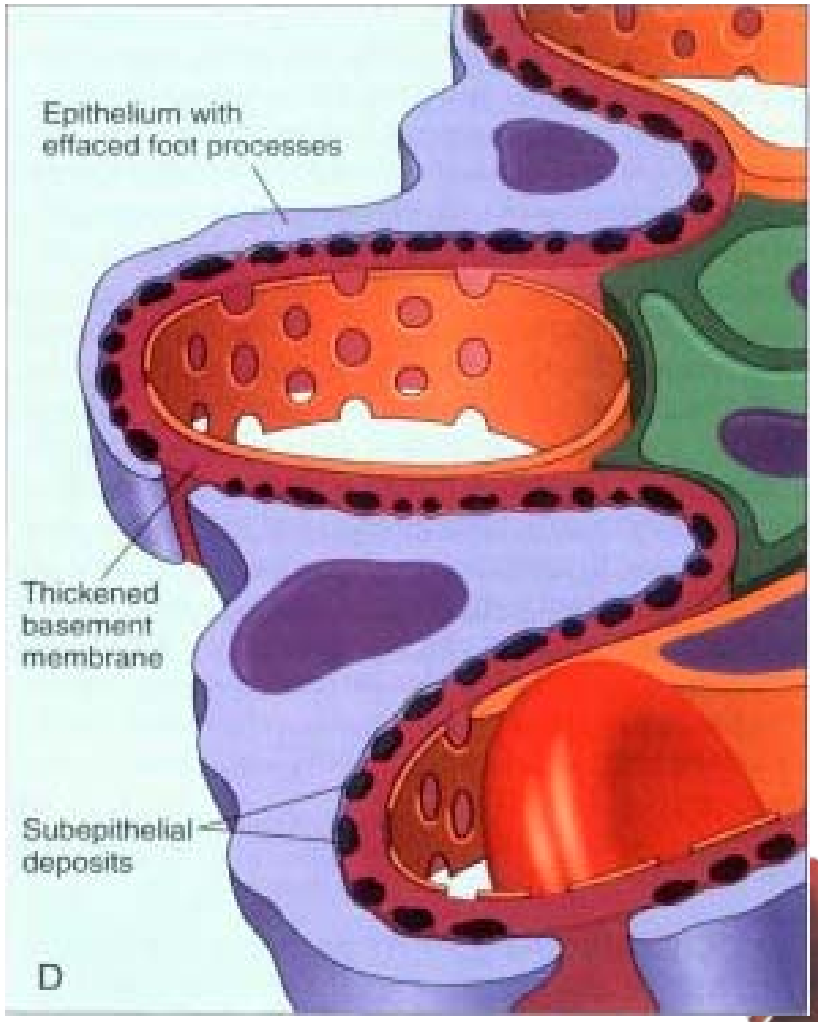
↓

**autoantibody**

↓

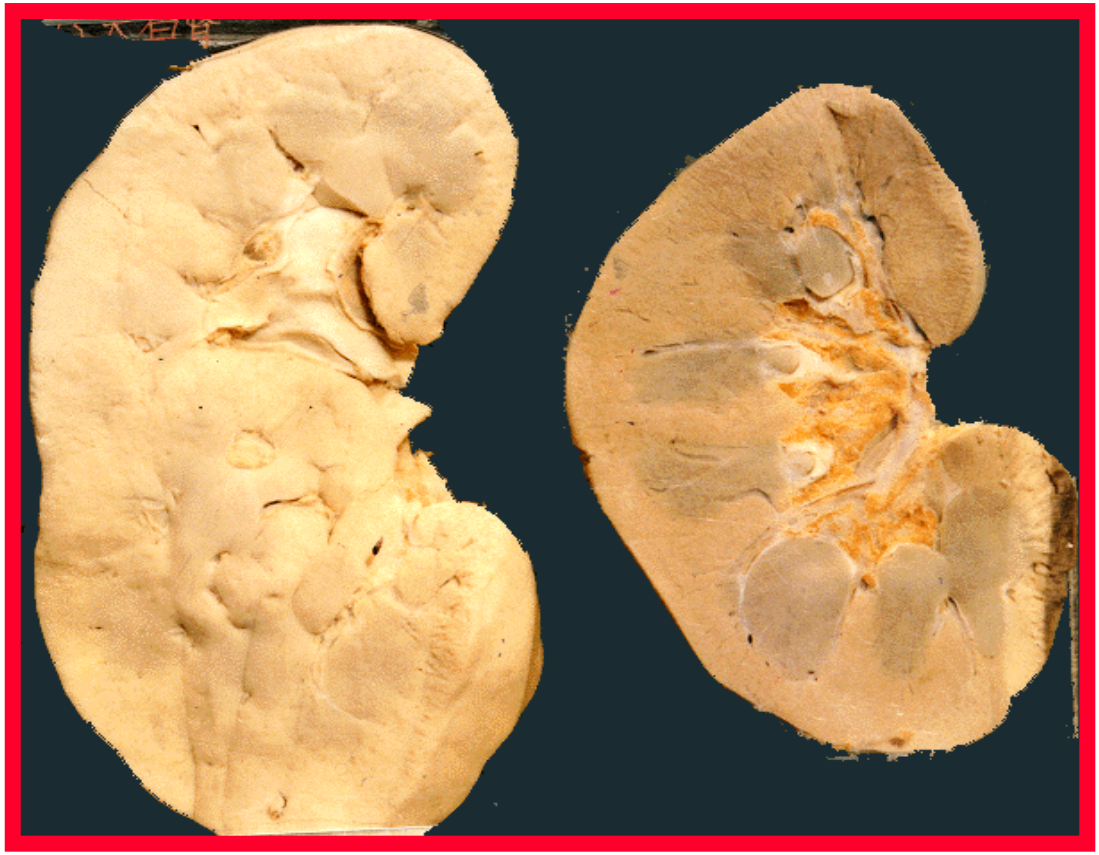
**formation of immune complex +complement**

**in the subepithelial and BM deposits**



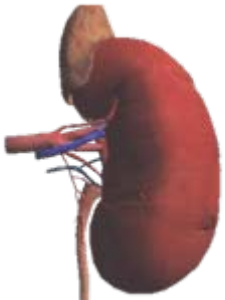
# Pathological changes

**Gross: Enlarged and pale kidney**



**left:**
large white kidney

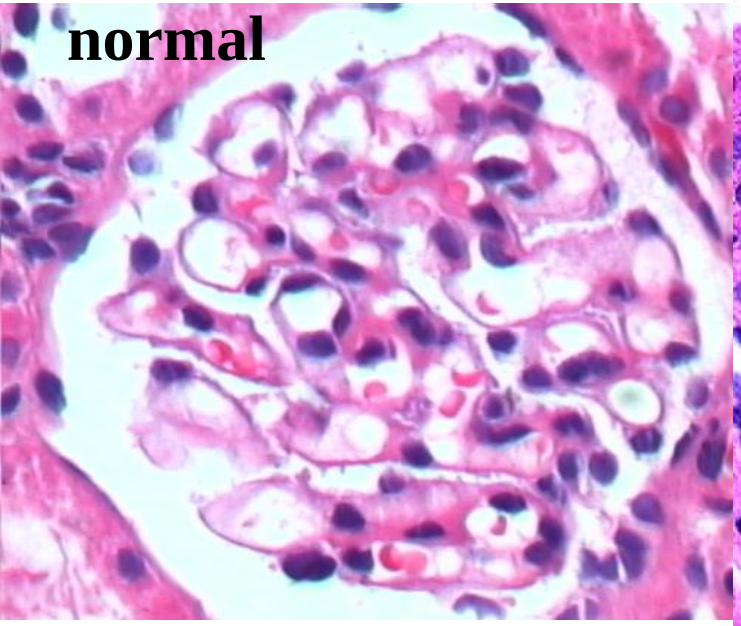
**right:**
Normal kidney



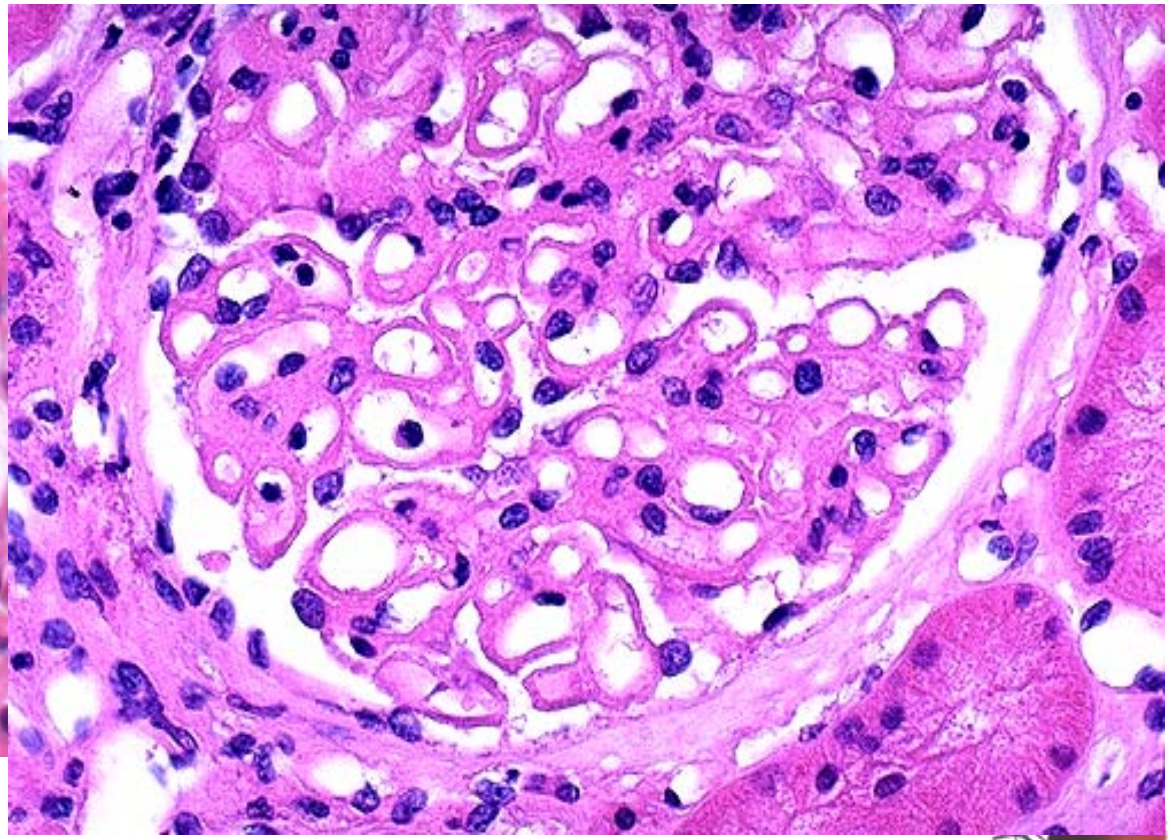
# Pathological changes

**Early stage: glomeruli appear normal.**

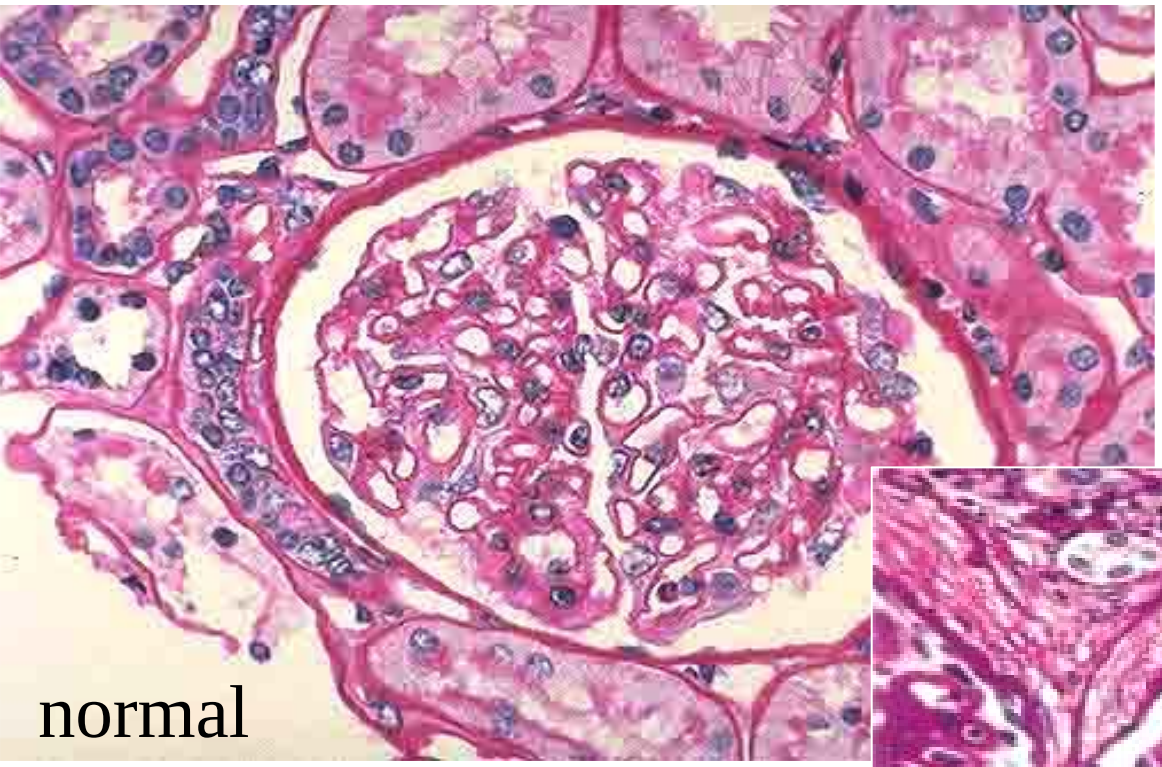
**Later: diffuse thickening of glomerular capillary wall, hyaline change and sclerosis.**



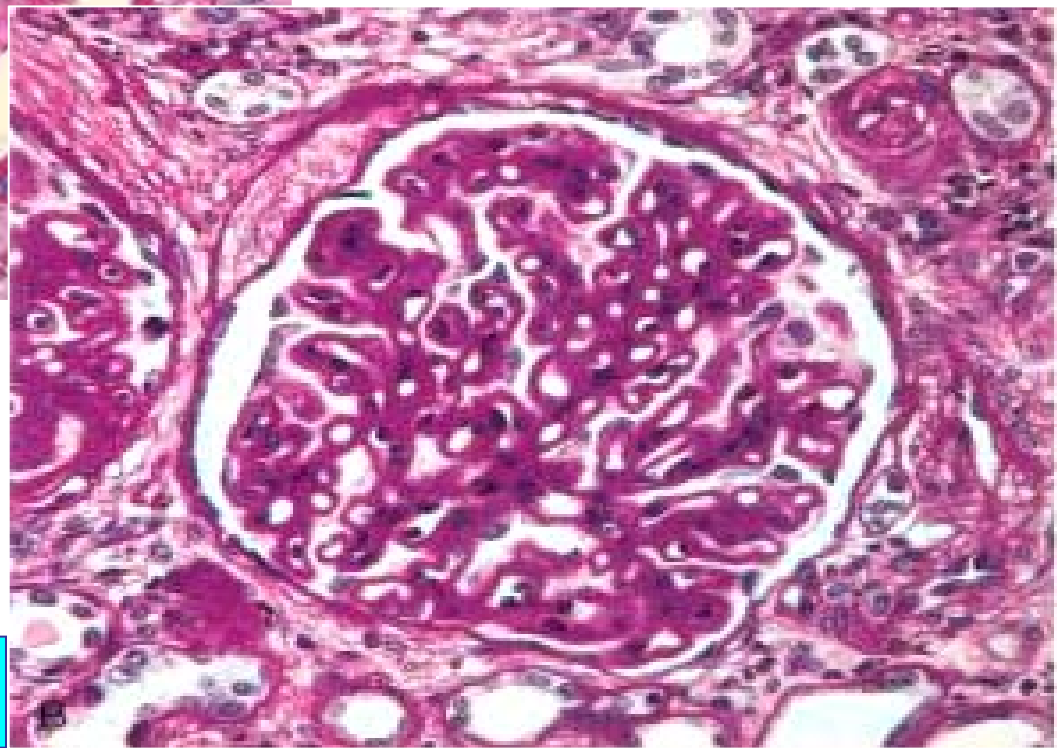
**normal**



# Pathological changes

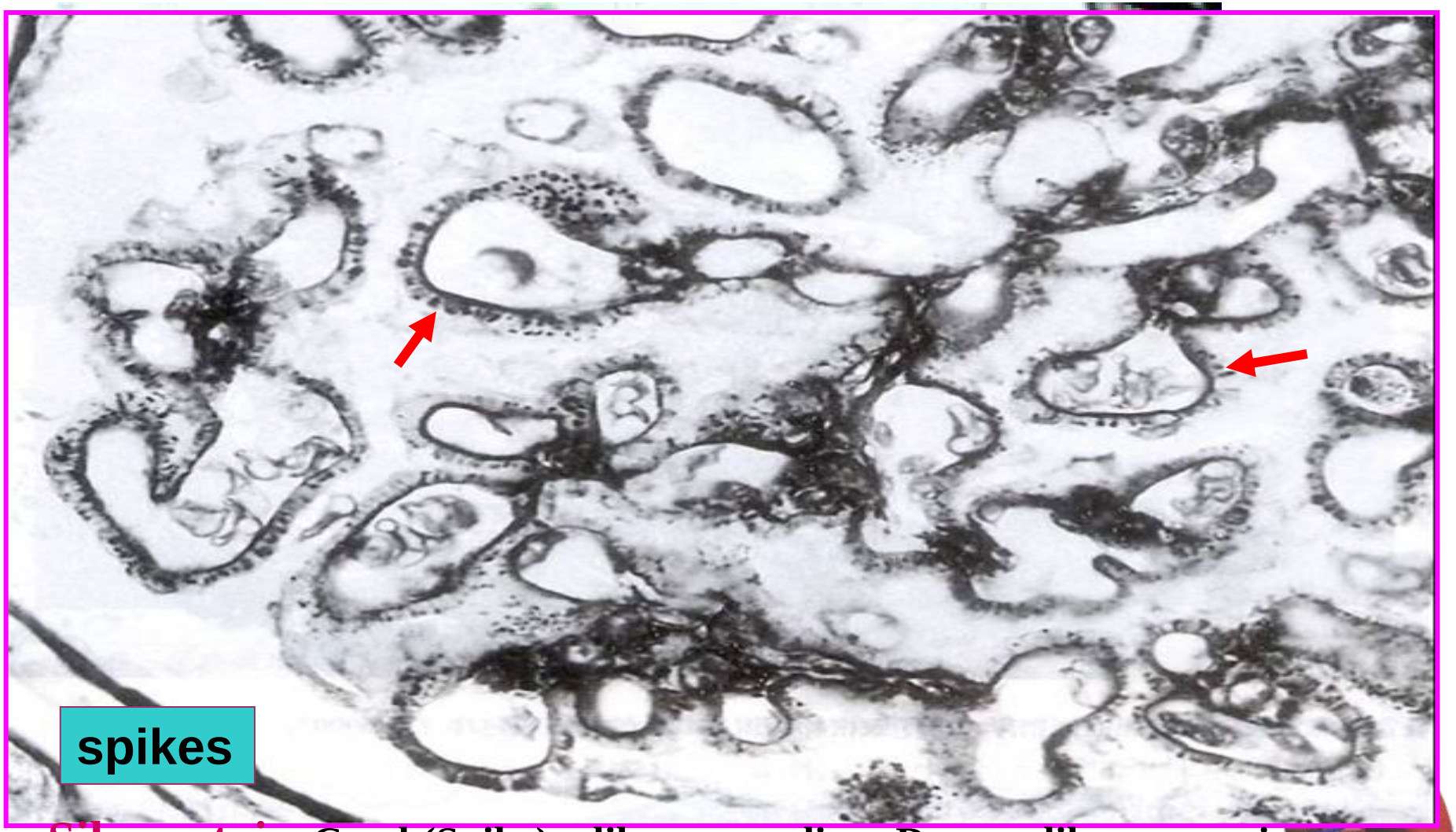


normal



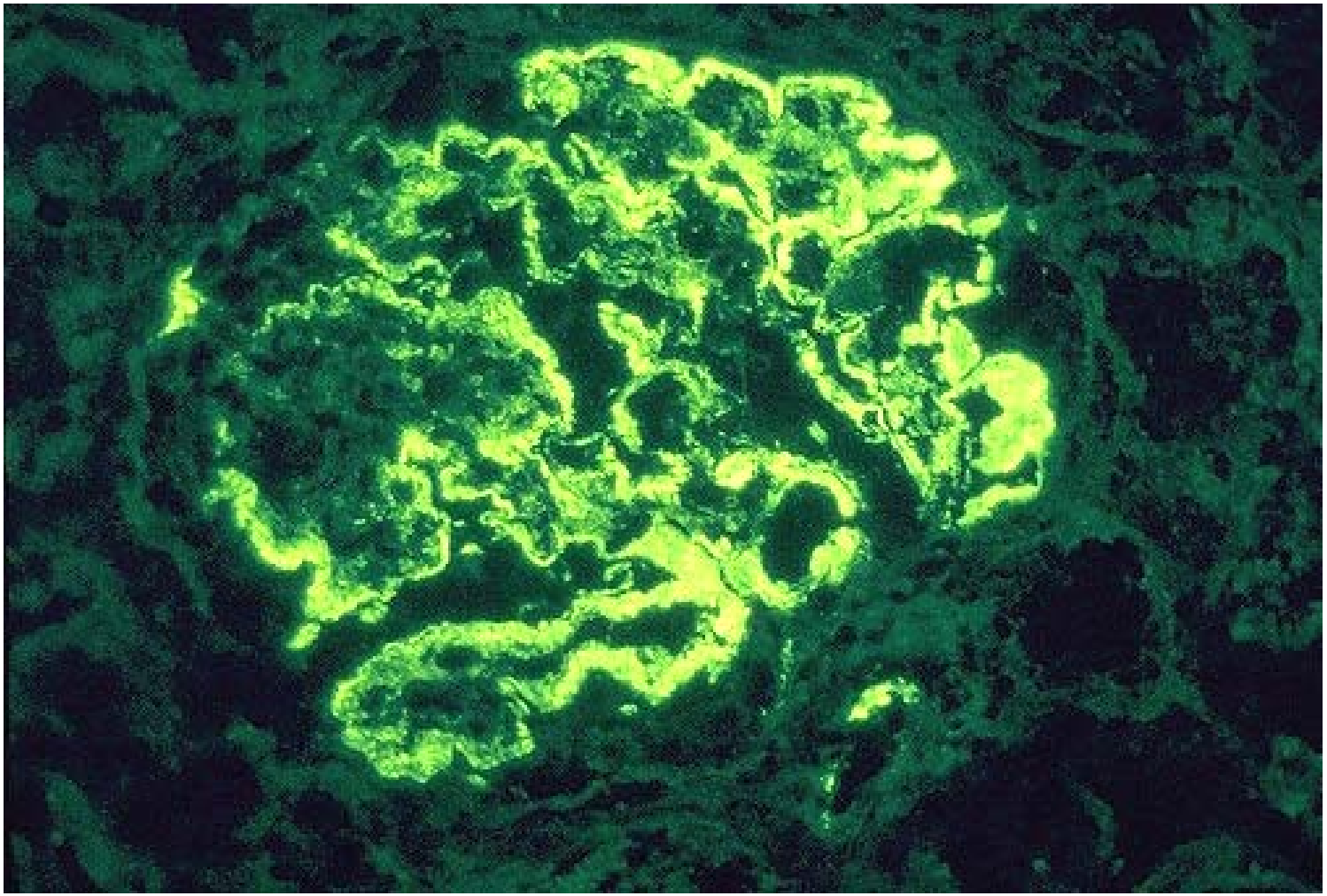
**GBM thickening**

# Pathological changes

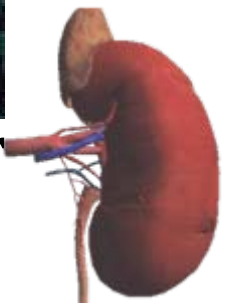



**Silver stain:** Comb(Spike) - like protruding, Dome - like protrusion, Encroach - like space

# Pathological changes



**Immunofluorescence microscopy: -characteristic granular immunofluorescent deposits of IgG & C3 along the GBM**



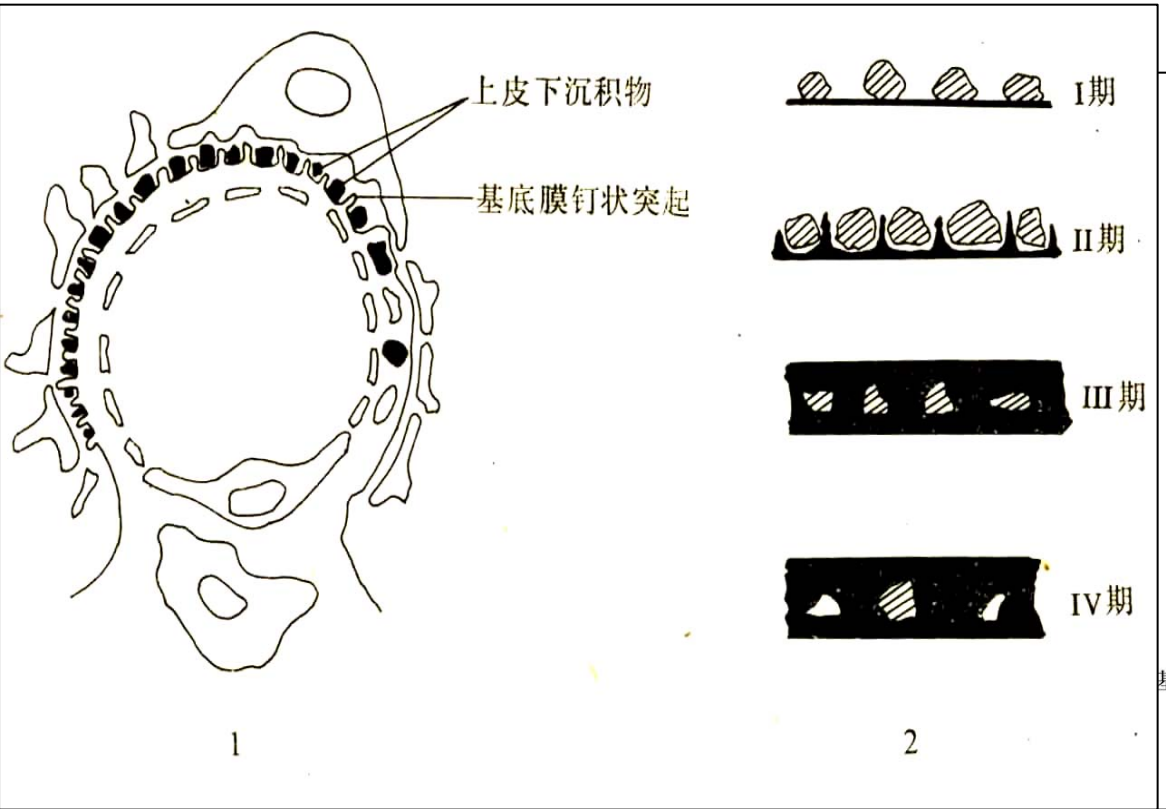
# Pathological changes

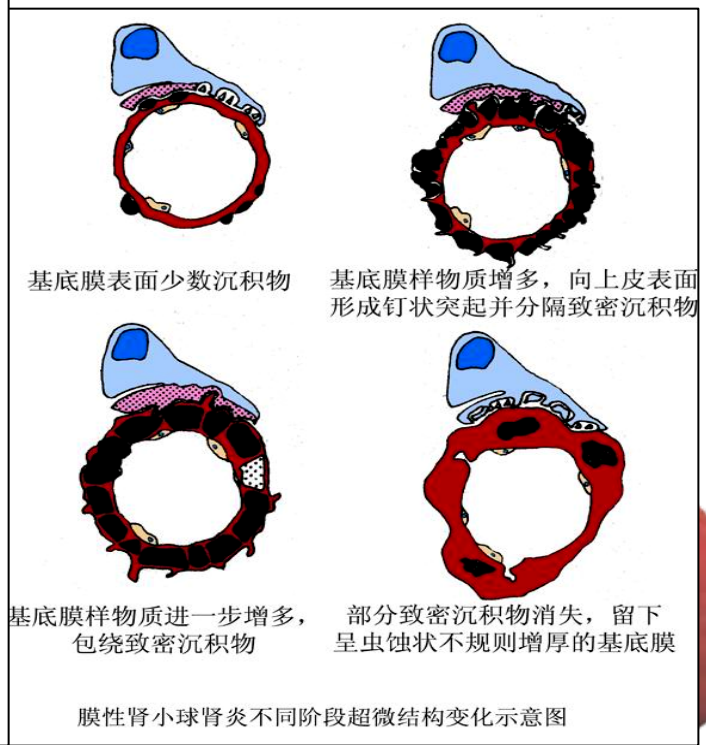
EM: Ⅰ Irregular dense deposits between GBM and the overlying epithelial cells, the latter having effaced foot processes

Ⅱ Spikes protruding from the GBM insert into the deposits.

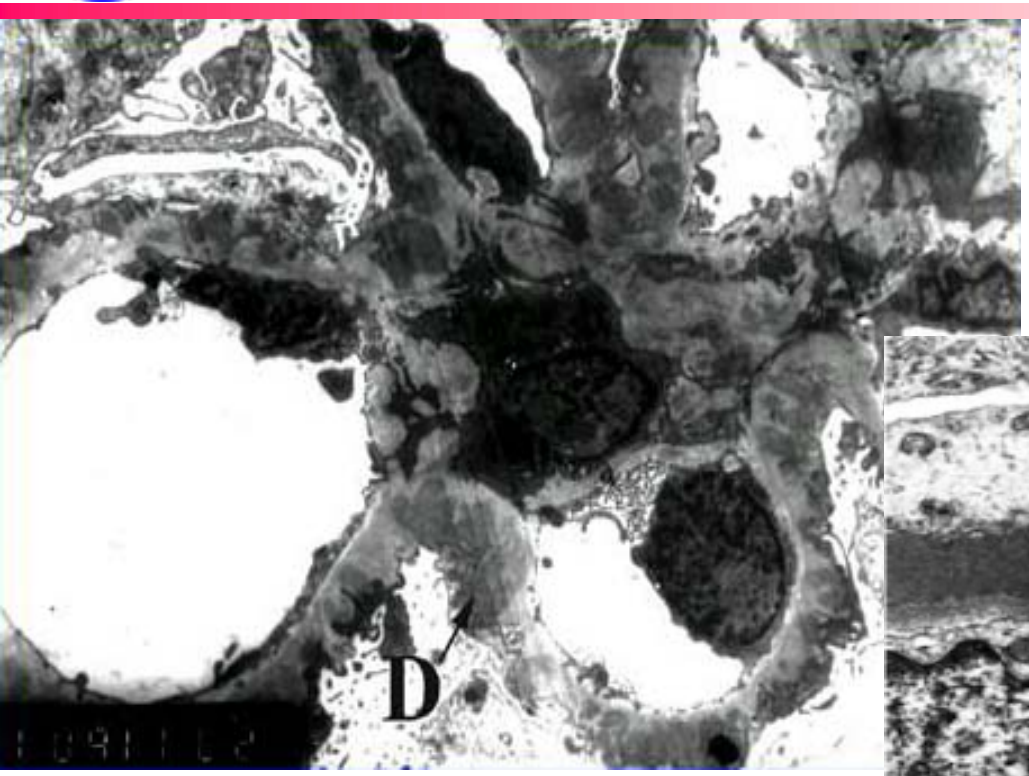
ⅢSpikes thicken to produce dome-like protrusion and close over the immune deposits, burying them within a thickened membrane.

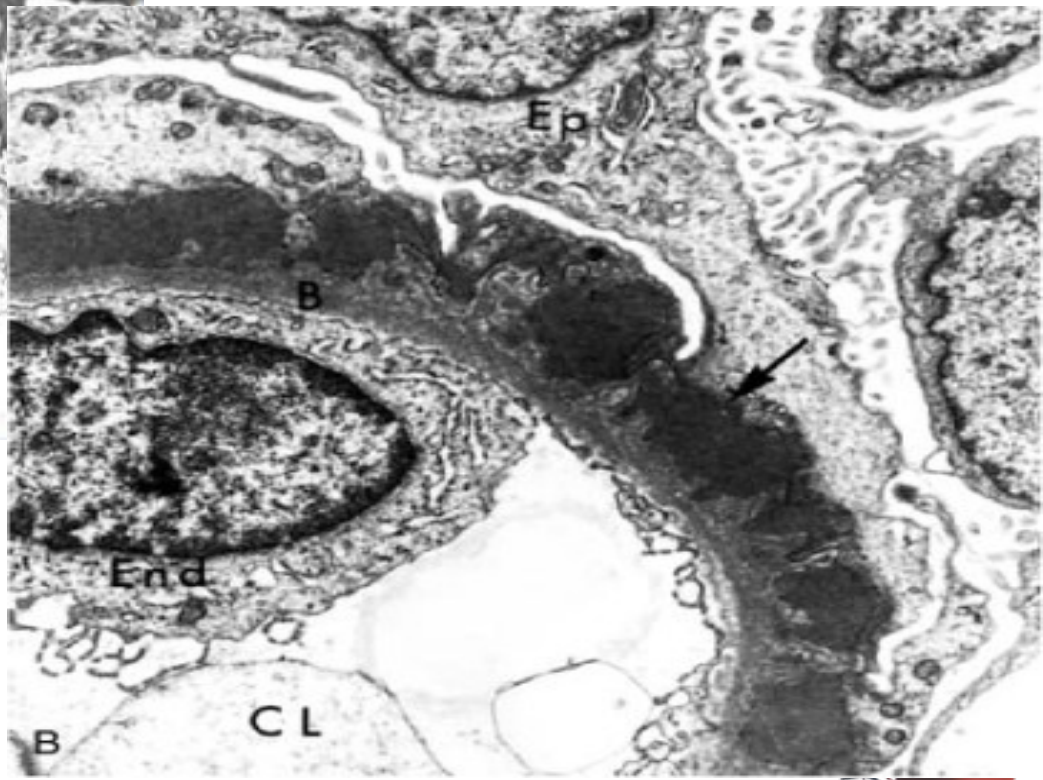
Ⅳ Deposits create irregular spaces in the basement membrane.



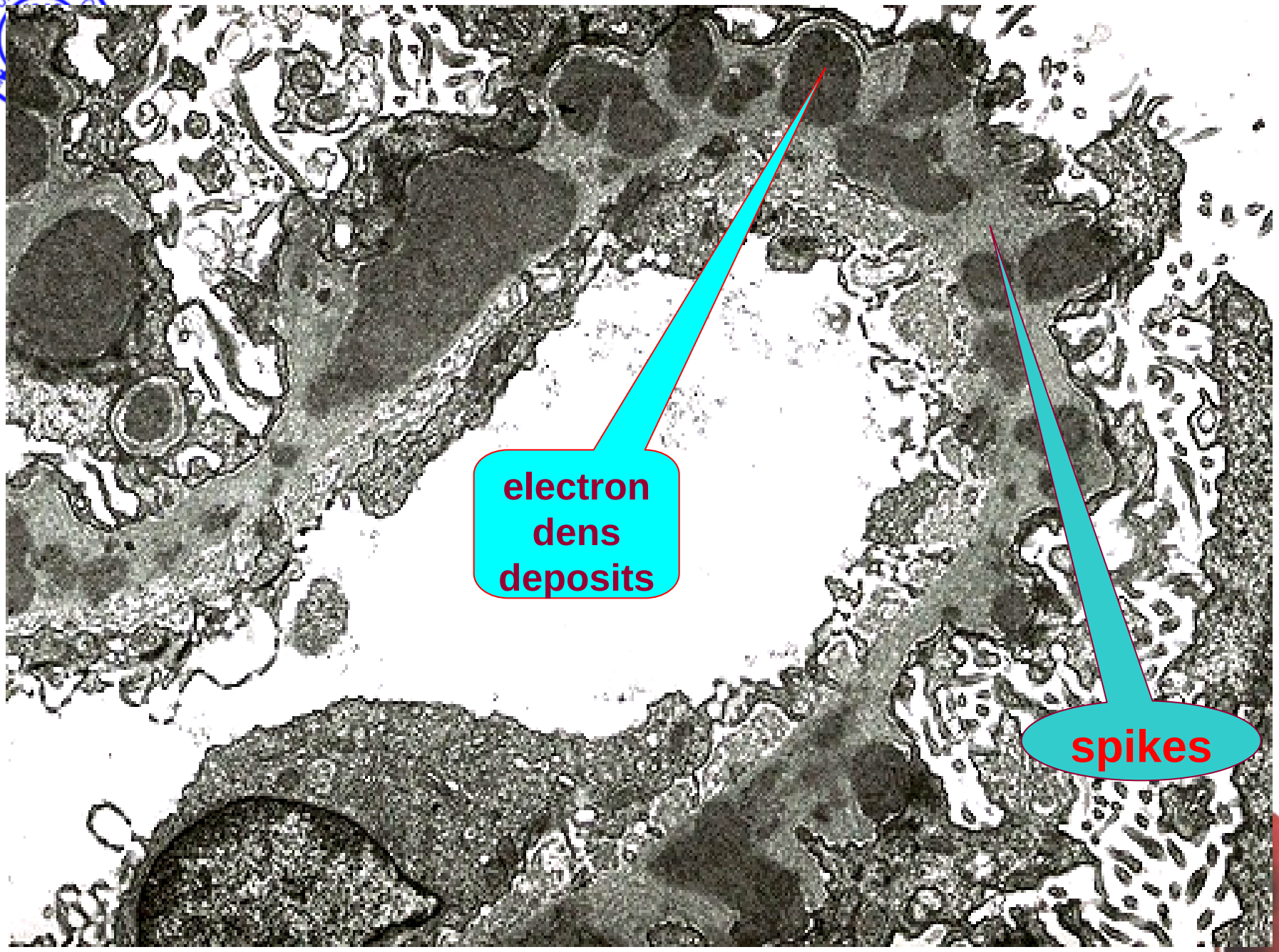



# Pathological changes




EM: **Swelling of podocyte, Loss of foot processes, Dense deposits in the subepithelial cells, Spikes formation.**


electron
dens
deposits
spikes

# Clinical features

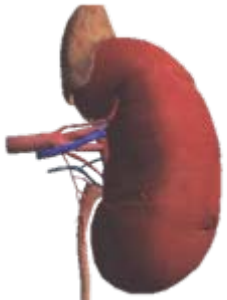
## nephrotic syndrome

- adult→non-selective proteinuria(15%)
- haematuria and mild hypertension

chronic progression→because of increasing sclerosis of glomerulum

10% patients go into renal failure or die within 10 years

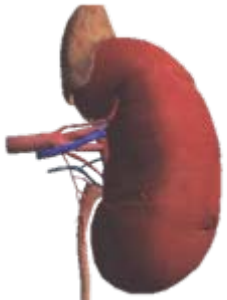
25%-40% patients develop to renal insufficiency



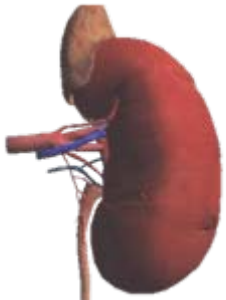
# Membranous GN

- **Gross : Enlarged and pale kidney**
- **LM : Early stage: glomeruli appear normal**

  **Later: diffuse thickening of GBM.**
- **Immunofluorescence: IgG and C3 deposit. Granular**
- **EM : Swelling of podocyte, Loss of foot processes, Dense deposits in the subepithelial cells, Spikes formation.**
- **Silver stain : Thickening of GBM, Comb(Spike) - like protruding**
- **Clinical features :Nephrotic syndrome.**



# Membranoproliferative GN (MPGN)

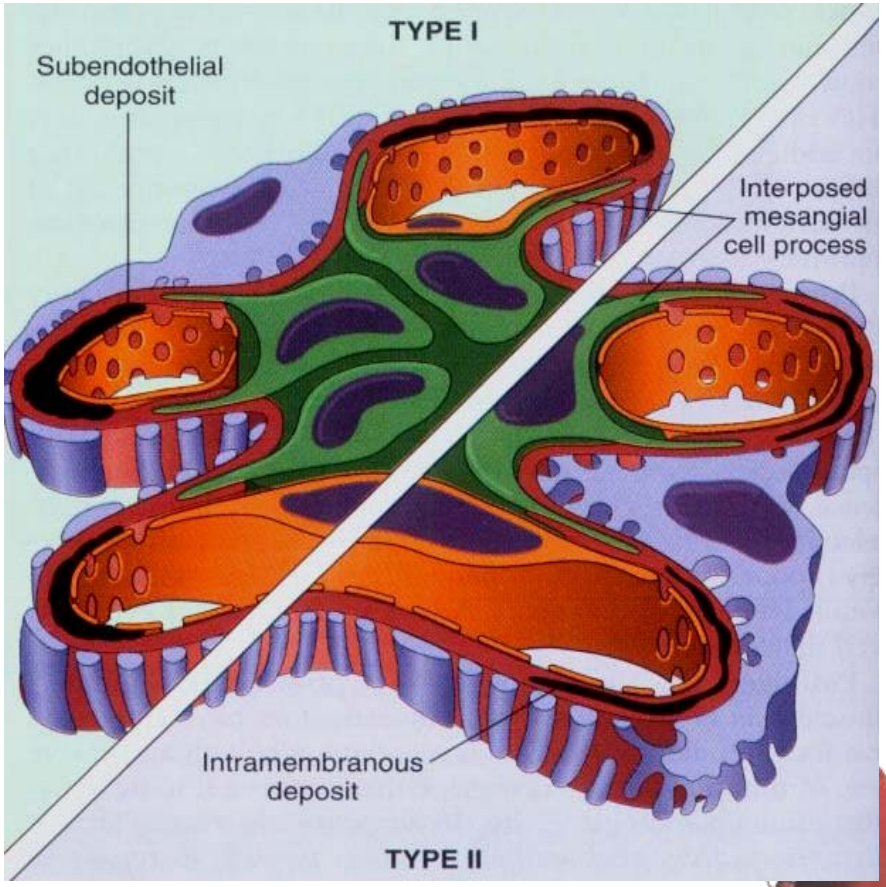


# Introduction

- **Features:**
  - **GBM** Thickening,
  - **Mesangial cell** Proliferation,
  - Increased **mesangial matrix.**

  Mesangiocapillary GN.

**Nephrotic syndrome**

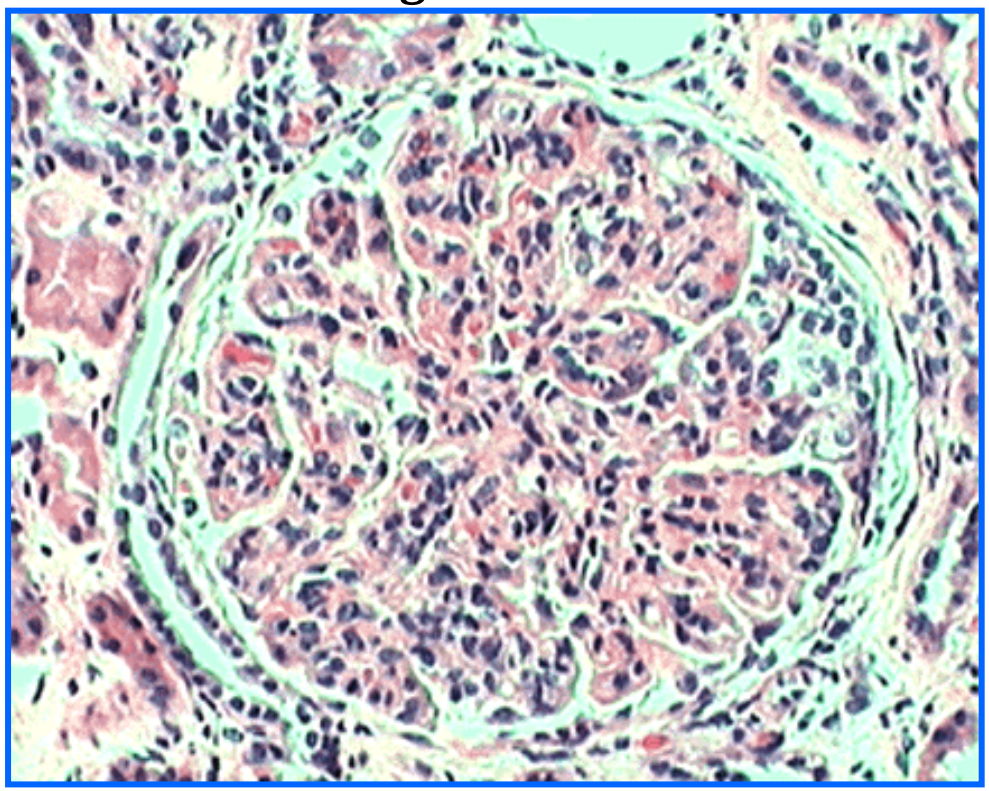


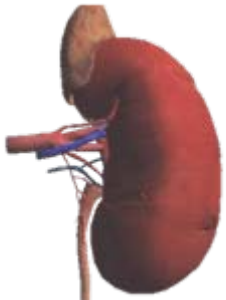

| Types | I | II |
|---|---|---|
| EM | subEC | BM |
| Immunoflu. | C3.IgG.C4 | C3 |
| Pathogenesis | immune complex | alternative complement pathway |

# Pathological changes

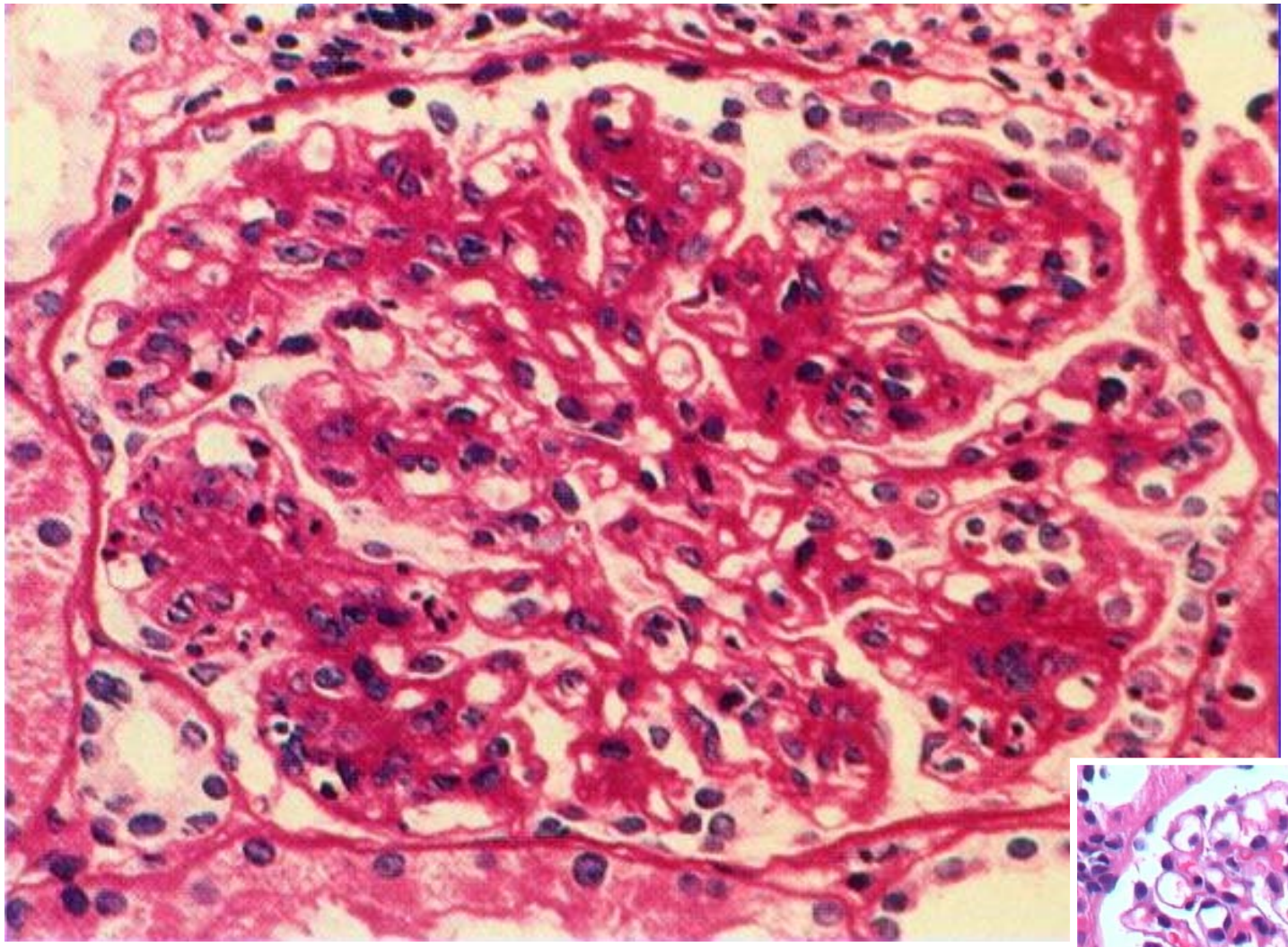
**LM：** **Glomeruli: large , hypercellular**

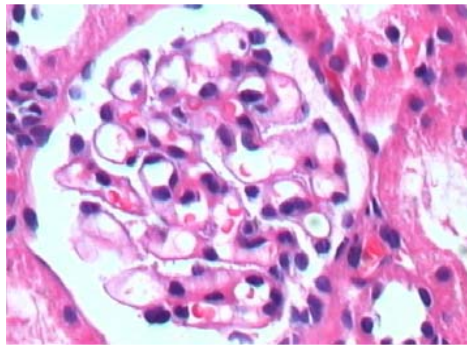
- **Hypercellular**
  - **proliferative mesangial cells and endothelial cells**
  - **infiltrating leukocytes**
- **Glomeruli: “lobular” appearance**
- **GBM thickening**





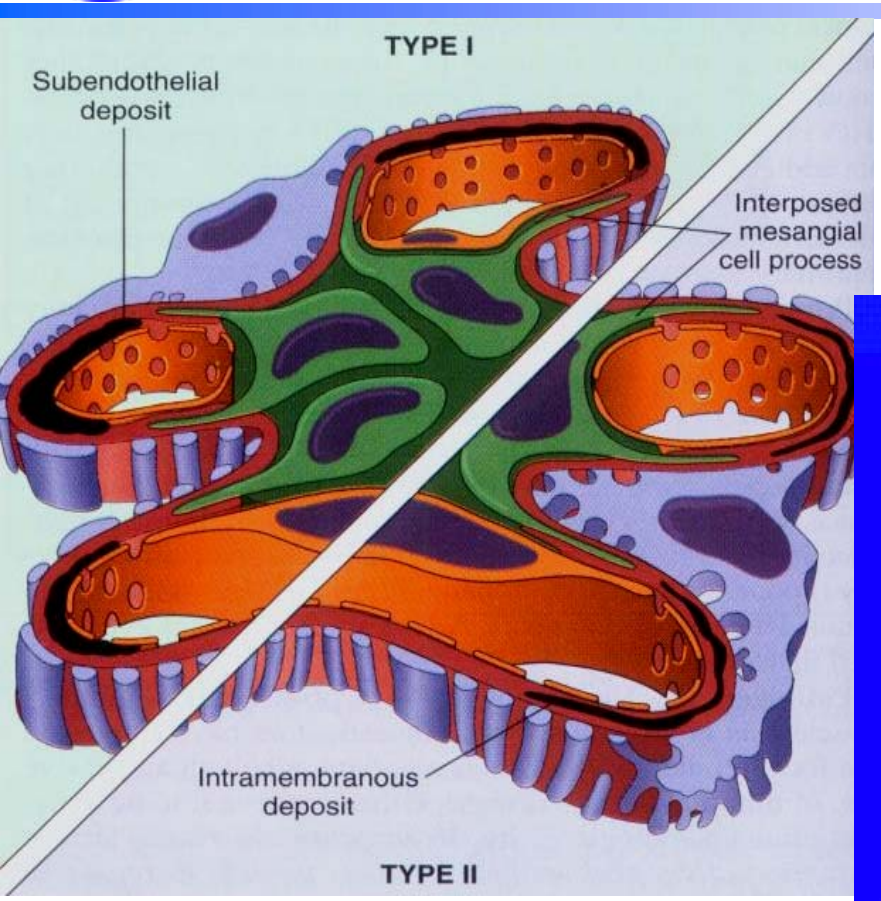
# Pathological changes

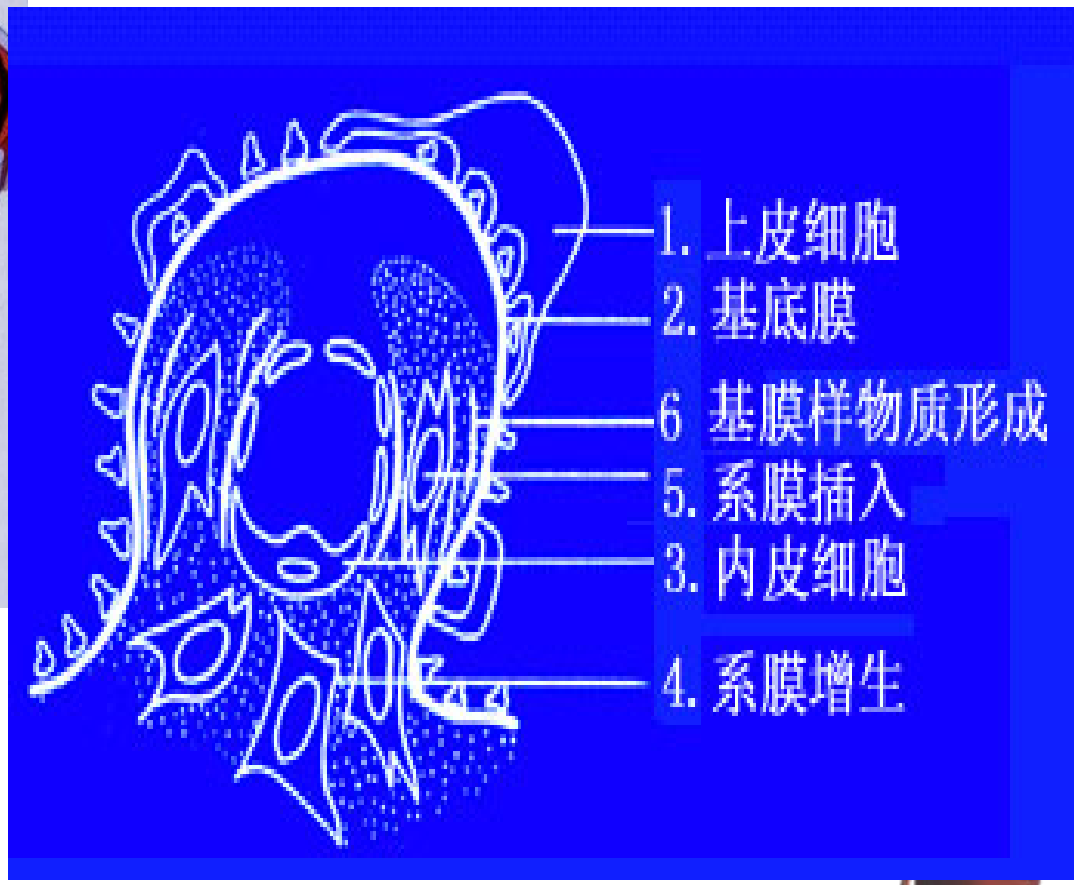




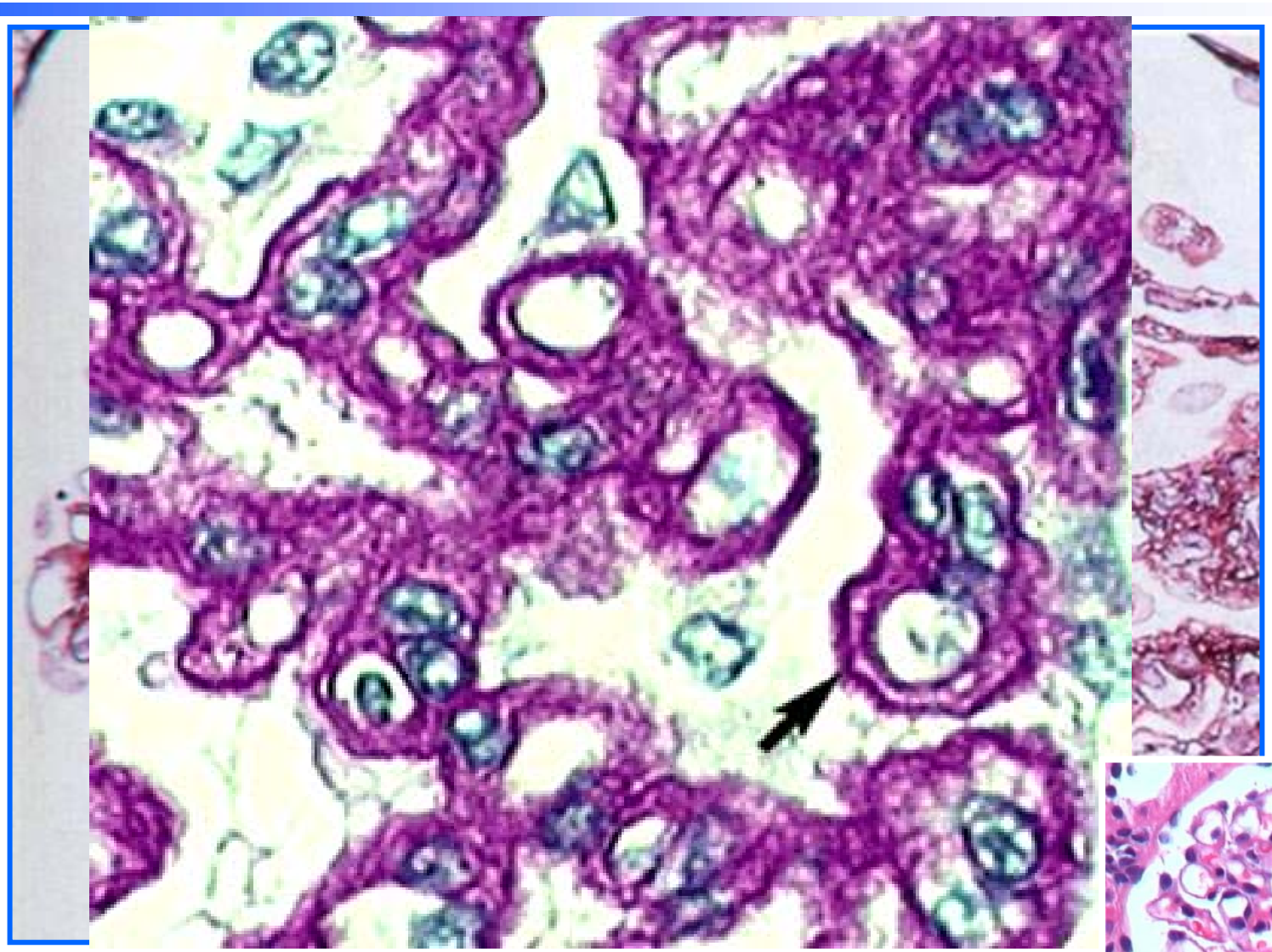
**proliferation of mesangial cells and mesangium**

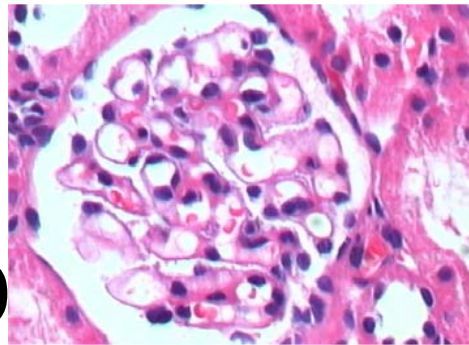
# Pathological changes



# Pathological changes



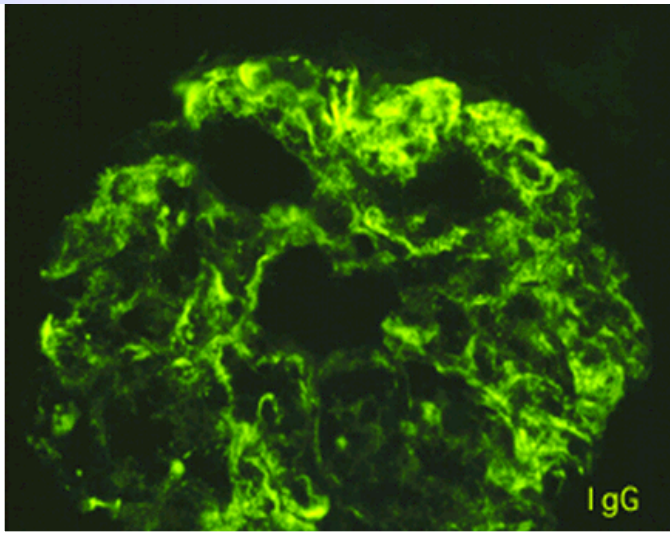


“ Tram-track ” appearance ( PAS )

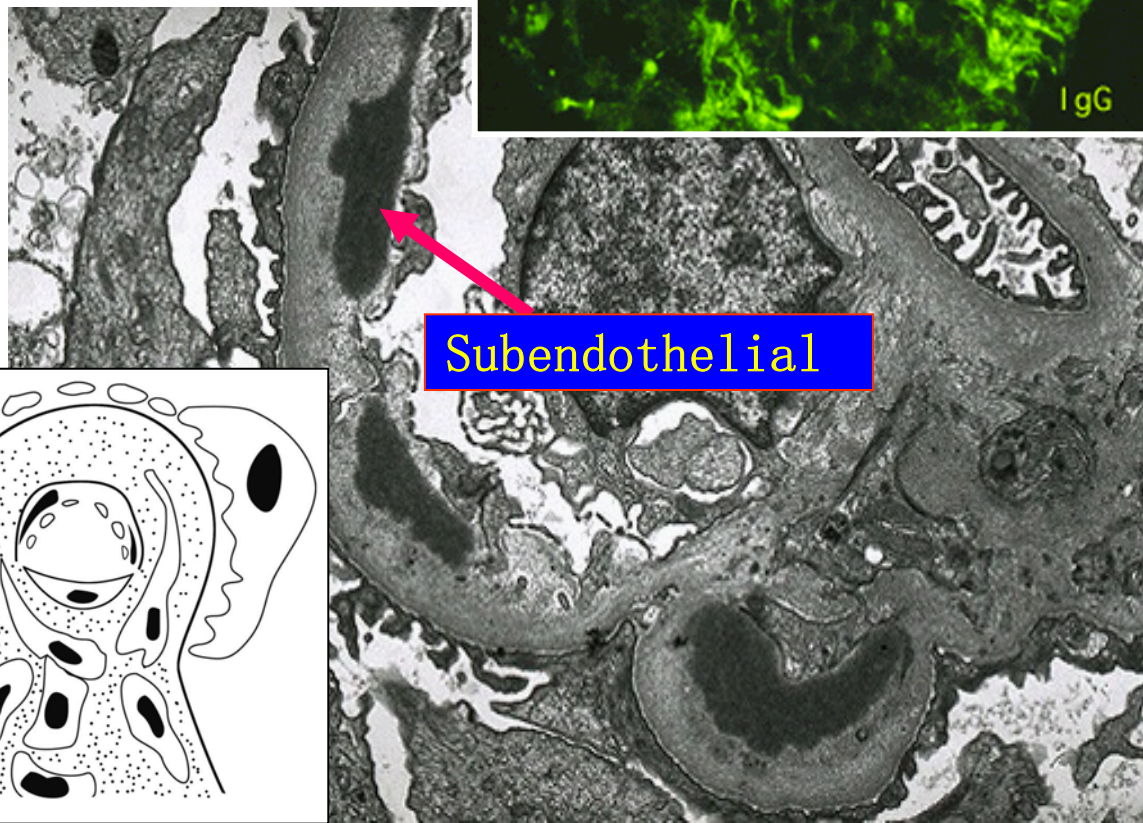
# Pathological changes

## TypeI

**IF--- IgG, C3, C1q and C4 deposition**




**EM--- subendothelial electron dense deposits**

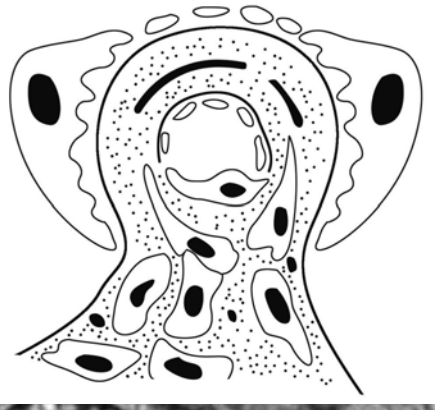



**Pathogenesis--- Circulating immune complex deposition**

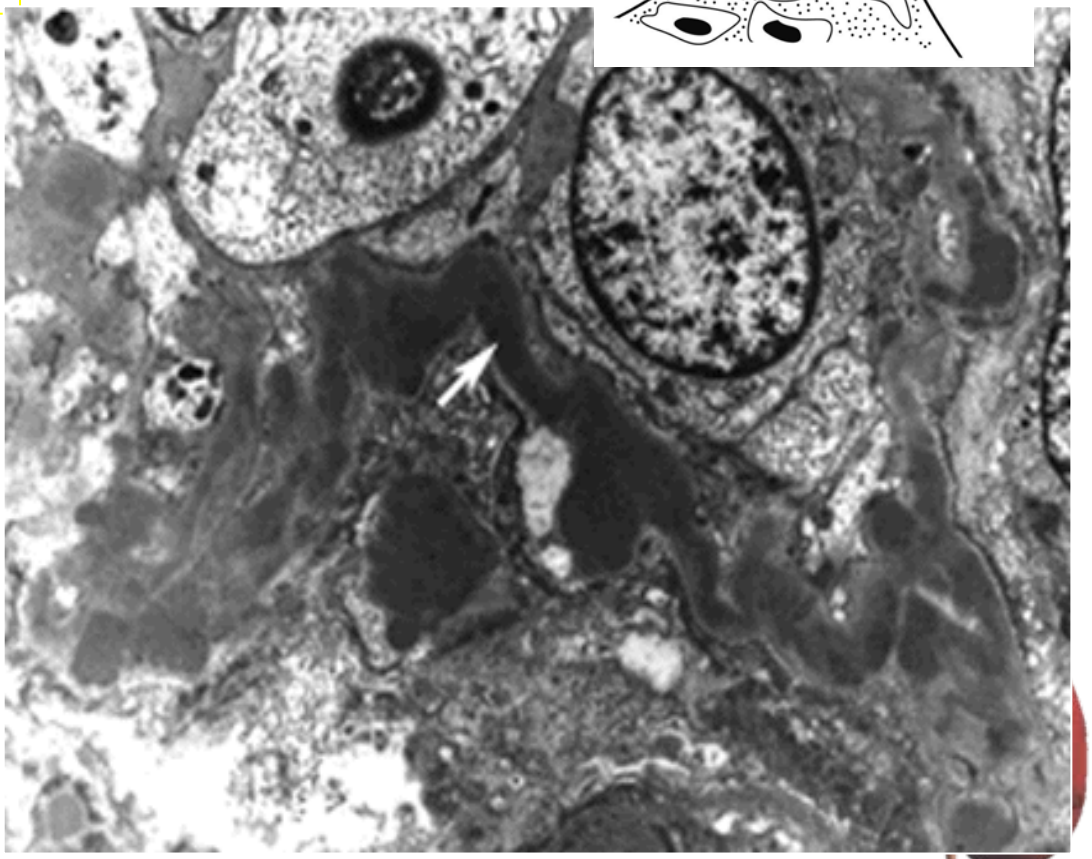
# Pathological changes

## TypeII

**IF---Only C3, No IgG, C1q and C4 deposition**

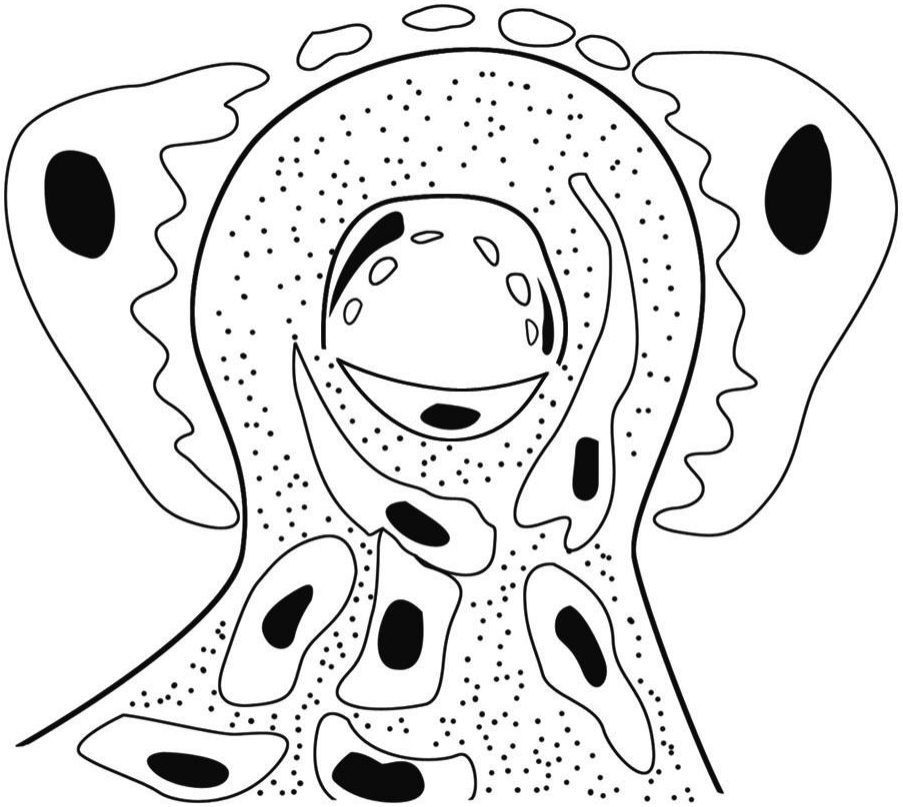


EM--- **Ribbon-like electron-dense deposits in the GBM**

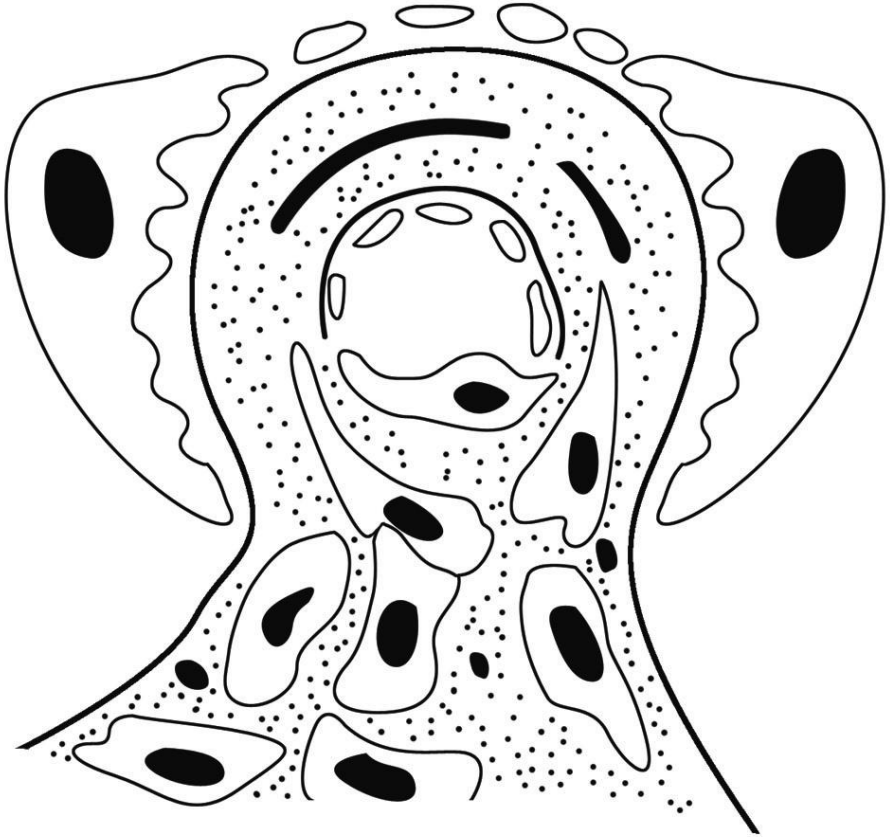


Pathogenesis---
**Activation of alternative complement pathway**

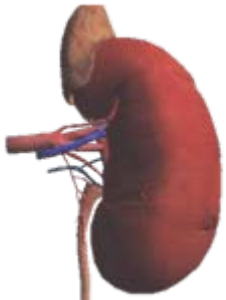
# Pathological changes



Type I



Type II



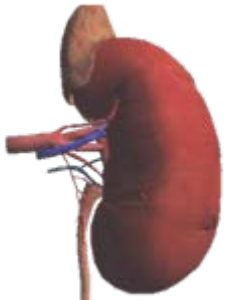
# Clinical features

## nephrotic syndrome

**old children and young adults**

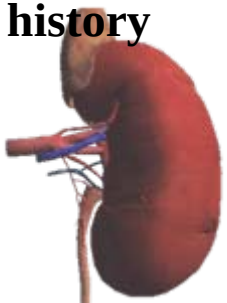
**poor prognosis**

- **50%→chronic renal failure**
- **some cases: rapidly progressive GN**

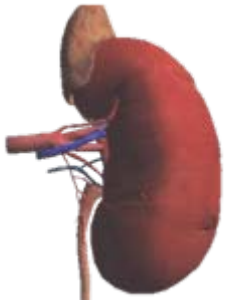


# Membranoproliferative GN (MPGN)

- **LM:**
  - **Proliferation of mesangial cells and mesangium**
    **-- glomeruli enlarged, hypercellular & have a "lobular" appearance**
- **PAS stain or sliver staining:**
  - **GBM thickened with "double-contour" or "tram-track" appearance**
- **IF:**
  - **Type I--Granular pattern of C3, IgG, C1q and C4 deposit**
  - **Type II—positive fluorescence for C3 in a capillary loop pattern**
- **EM:**
  - **Type I： subendothelial electron dense deposits**
  - **Type II： Ribbon-like electron-dense deposits in the GBM**
- **Clinical:**
  - **Nephrotic syndrome in 50%, acute nephritic syndrome in 20%. Recent history of URI in 50%. Hypertension and/or renal insufficiency.**

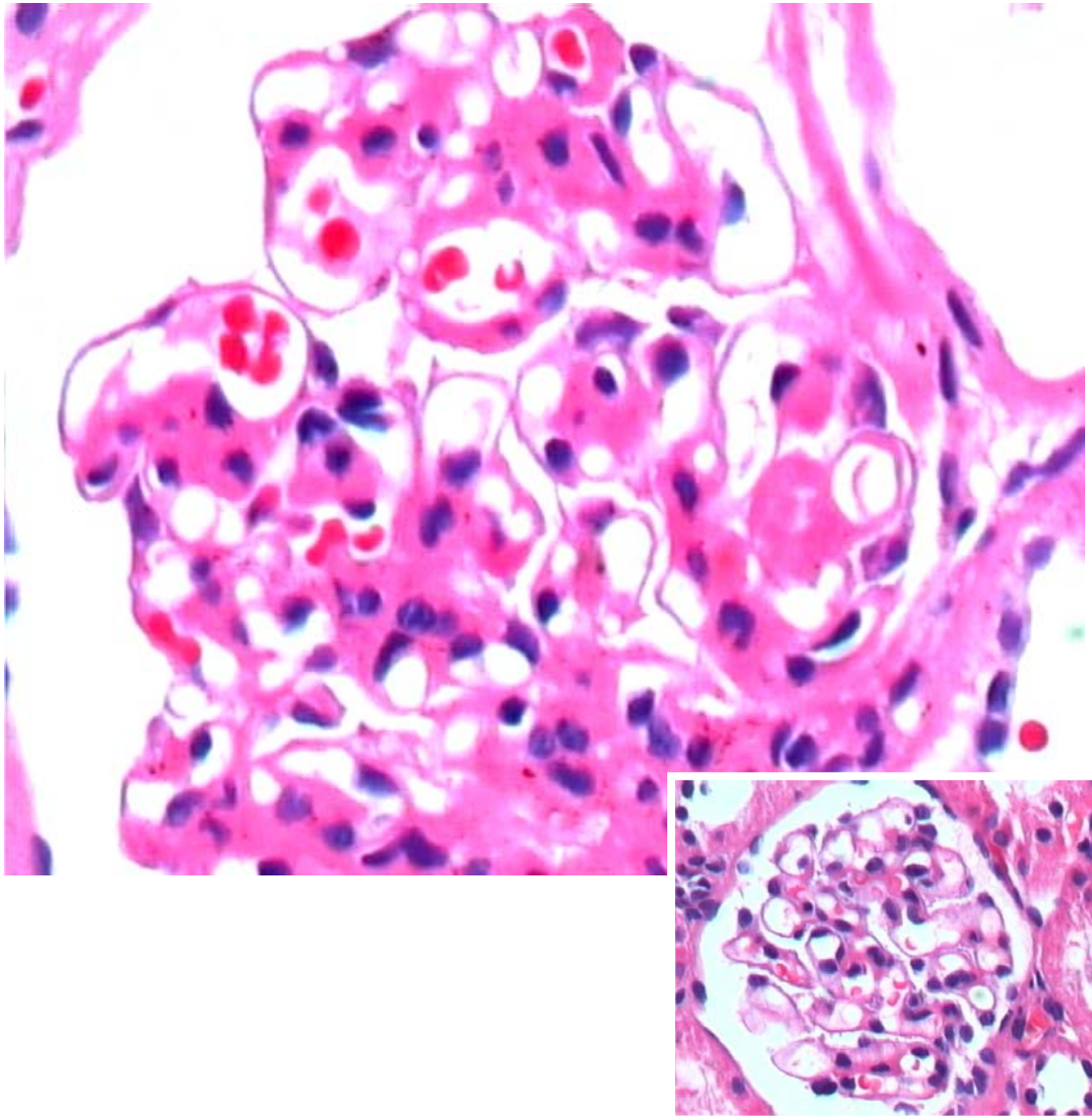


# Mesangial proliferative GN

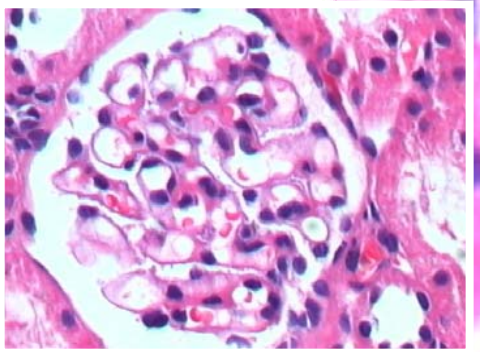


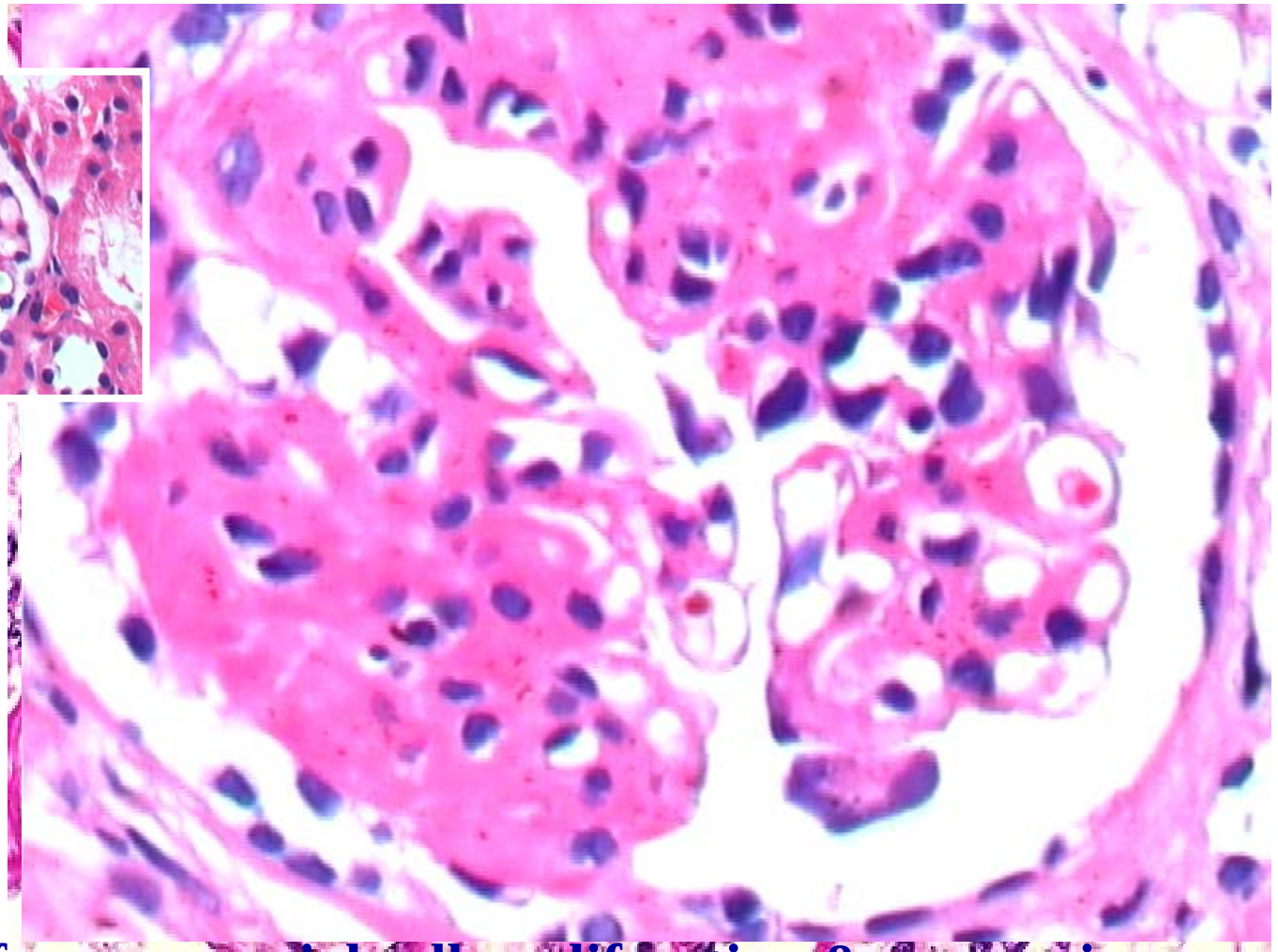
# Introduction

- **Features: Diffuse mesangial cell proliferation & mesangial matrix increase.**
- **Pathogenesis:**
  - **Primary: immune complex**
  - **Secondary: SLE , diabetes**

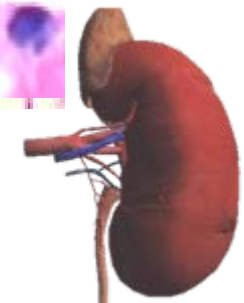


# Pathological changes

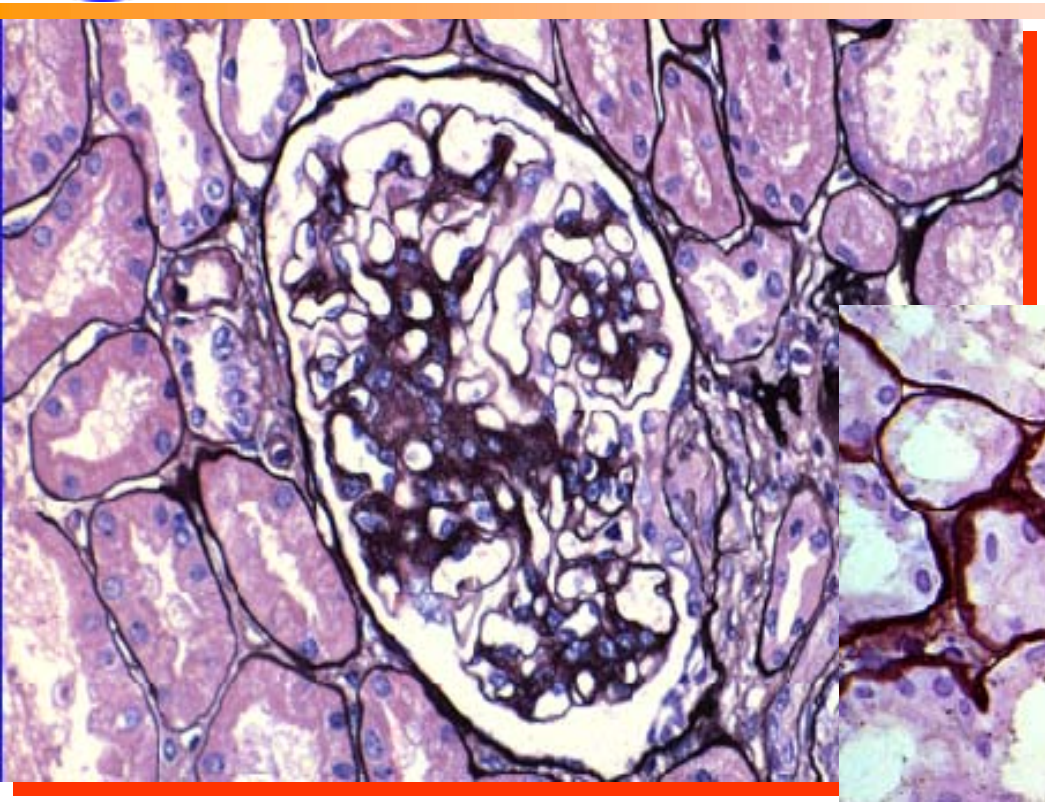




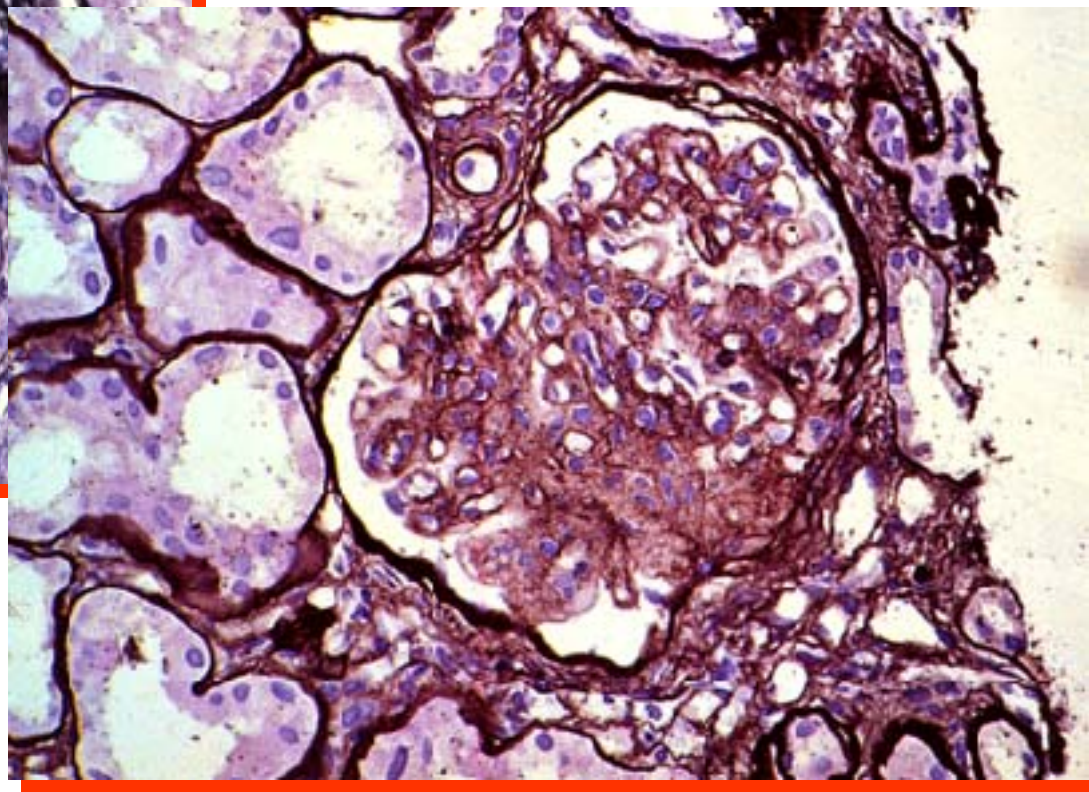
***LM*：Diffuse mesangial cell proliferation & mesangium increase. The capillary loop is normal**



# Pathological changes

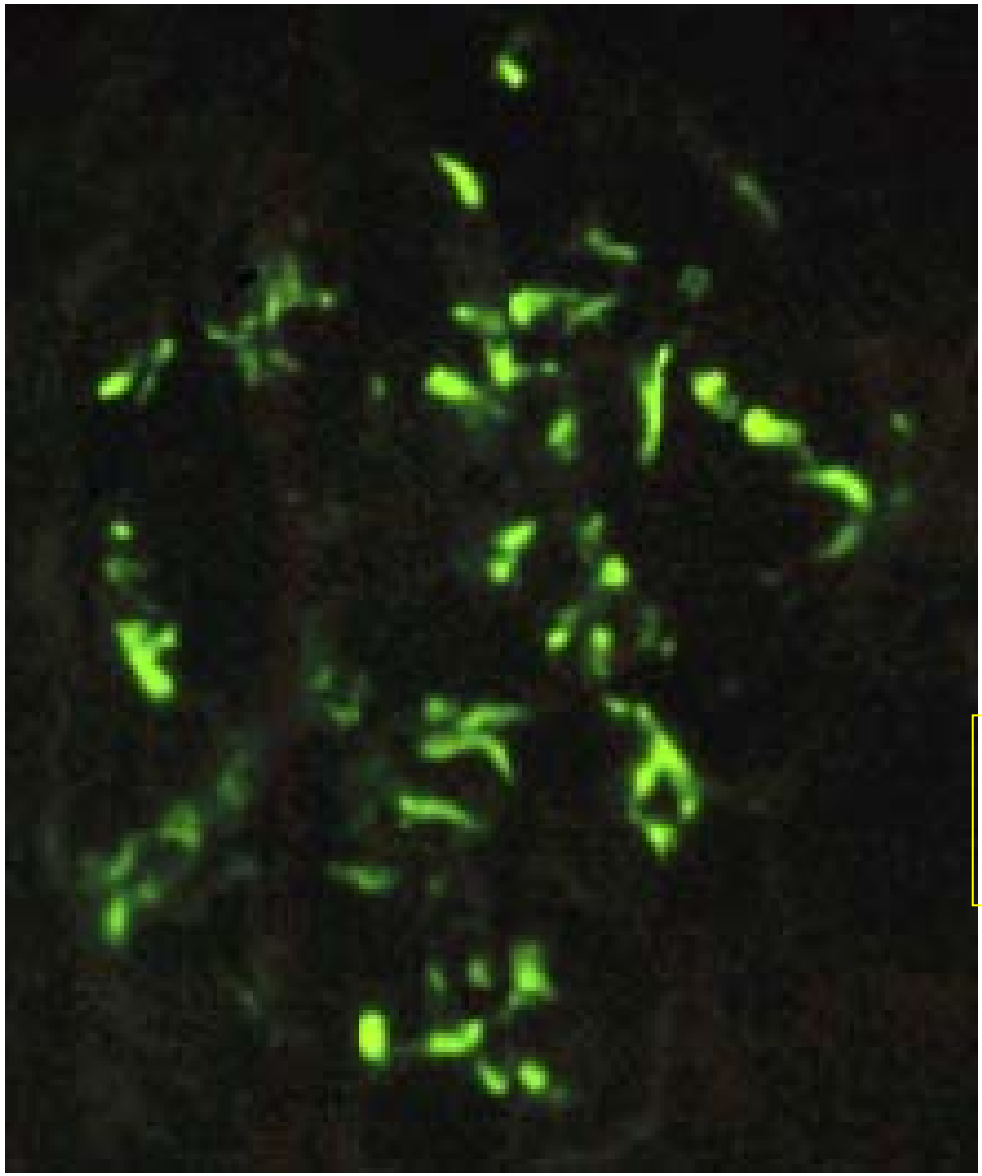


Moderate Mesangial proliferative GN



Severe Mesangial proliferative GN

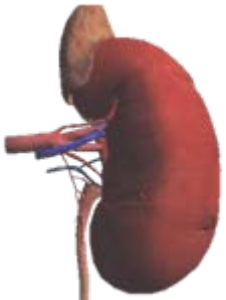
# Pathological changes



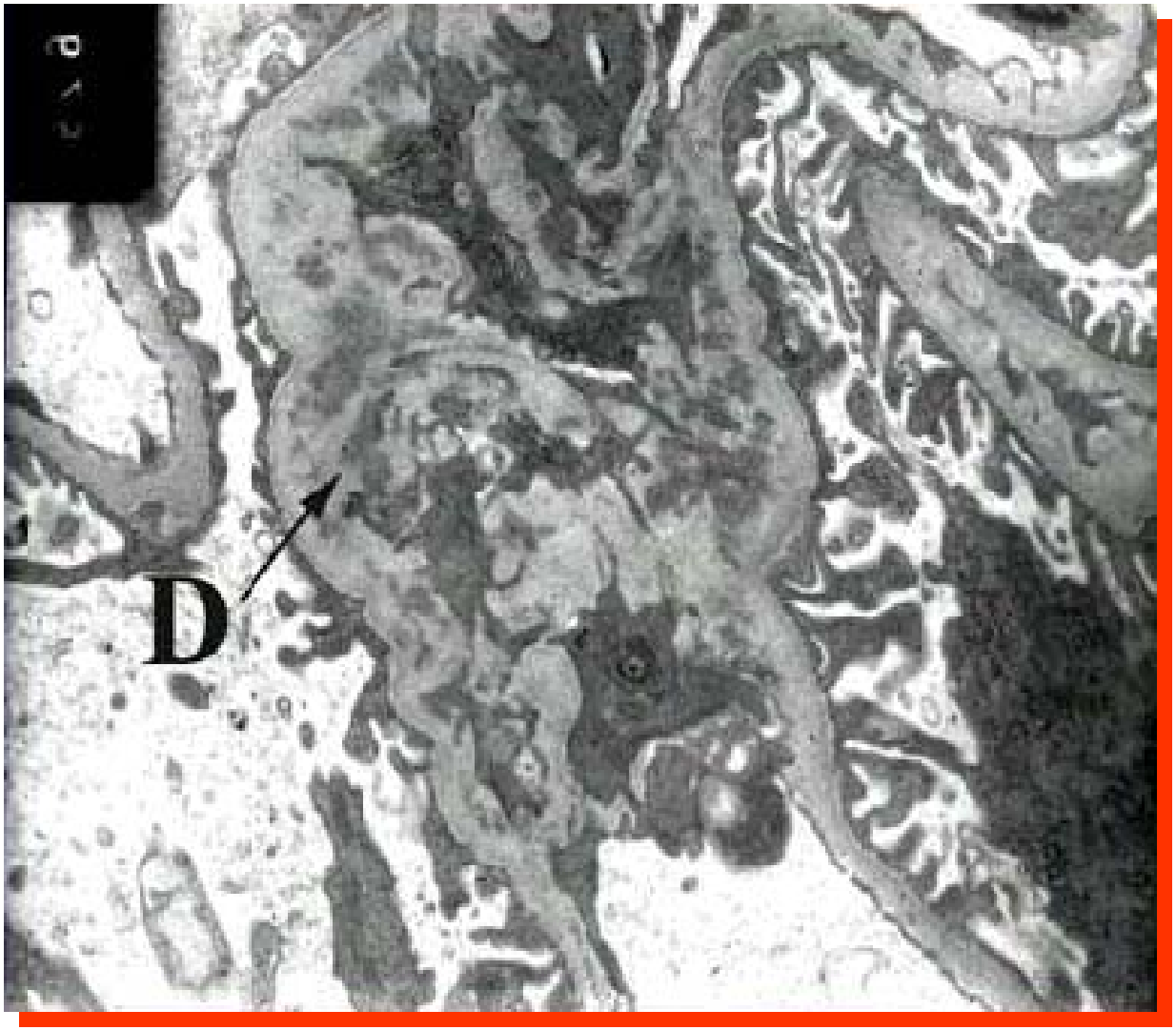
**IF: Granular IgG & C3 deposit in mesangium**

**IgG and C3---China**

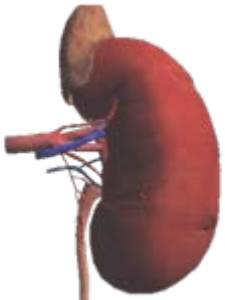
**IgM and C3---other country**



# Pathological changes




**EM: Eletron-dense deposits in mesangium ( IgG, C3, IgM)**(D)

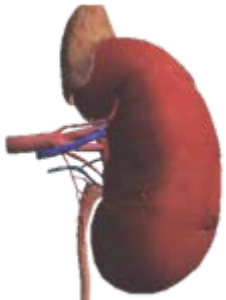


# Clinical manifestation

## nephrotic syndrome

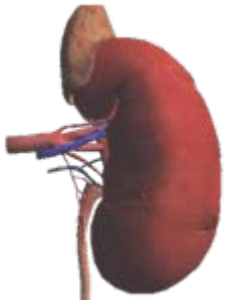
asymptomatic { proteinuria
hematuria

chronic nephrotic syndrome

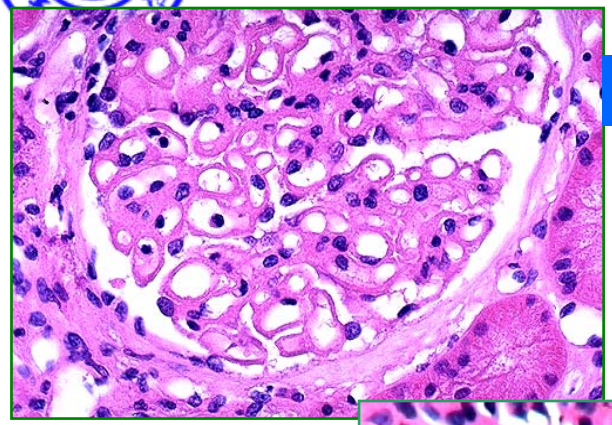


# Mesangial proliferative GN

- **LM：Diffuse mesangial cell proliferation & mesangium increase. The capillary loop is normal**
- **EM：Mesangial cell proliferation + Eletron-dense deposits .**
- **IF：IgG and C3 deposits or IgM and C3 deposits**
- **Clinical manifestation ： Diversity.**
  - **nephrotic syndrome**
  - **asymptomatic proteinuria or/and hematuria**

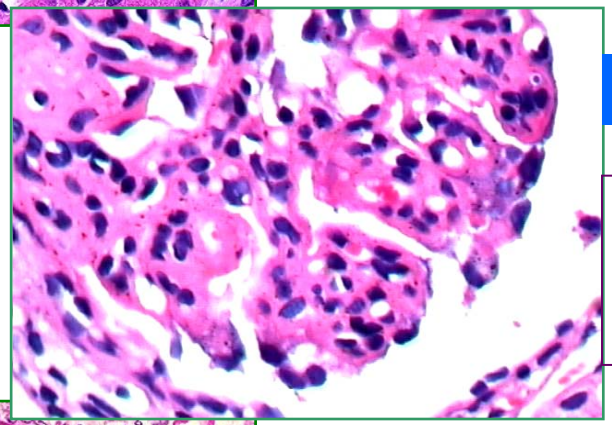


# Comparison between three “membrane”- related glomerulonephritis types



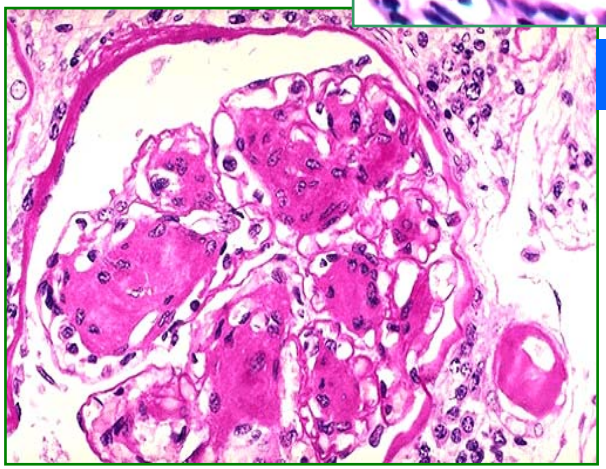
**membranous GN**

- ✓diffuse thickening of GBM
- ✓spike-like protrusions from the GBM



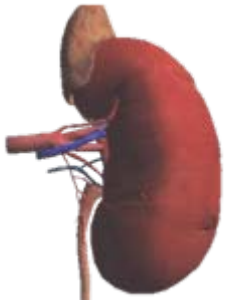
**membranoproliferative GN**

- GBM thickening with “tram-track” appearance
- proliferation of mesangial cells and mesangium
- glomeruli show “lobular” appearance



**mesangial proliferative GN**

- proliferation of mesangial cells & mesangium
- GBM is normal

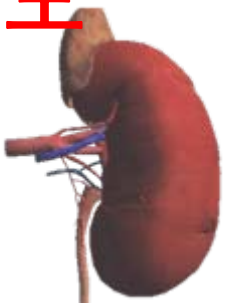


# 三种带膜字肾炎的区别：

膜性肾小球肾炎：膜指基底膜增厚

膜增生性肾小球肾炎：膜指系膜增生和基底膜增厚

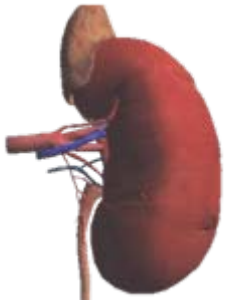
系膜增生性肾小球肾炎：膜仅指系膜增生



# 系膜增生性、膜性增生性、膜性肾小球肾炎三者的比较

| 病　名 | 光　镜 | 毛细血管壁 | 特殊染色 | 临　床 |
| --- | --- | --- | --- | --- |
| 系膜增生性 | 系膜细胞增生，基质增多，系膜区增宽 | 无改变 | 系膜区宽，基底膜正常 | 复发性蛋白尿、血尿，肾病综合征 |
| 膜性增生性 | 同上，肾小球呈分叶状 | 增　厚 | 血管壁示“双轨”征 | 肾病综合症，血尿、蛋白尿、慢性 |
| 膜　性 | 无上述病变 | 增　厚 | 基底膜显示“梳齿” | 肾病综合征 |

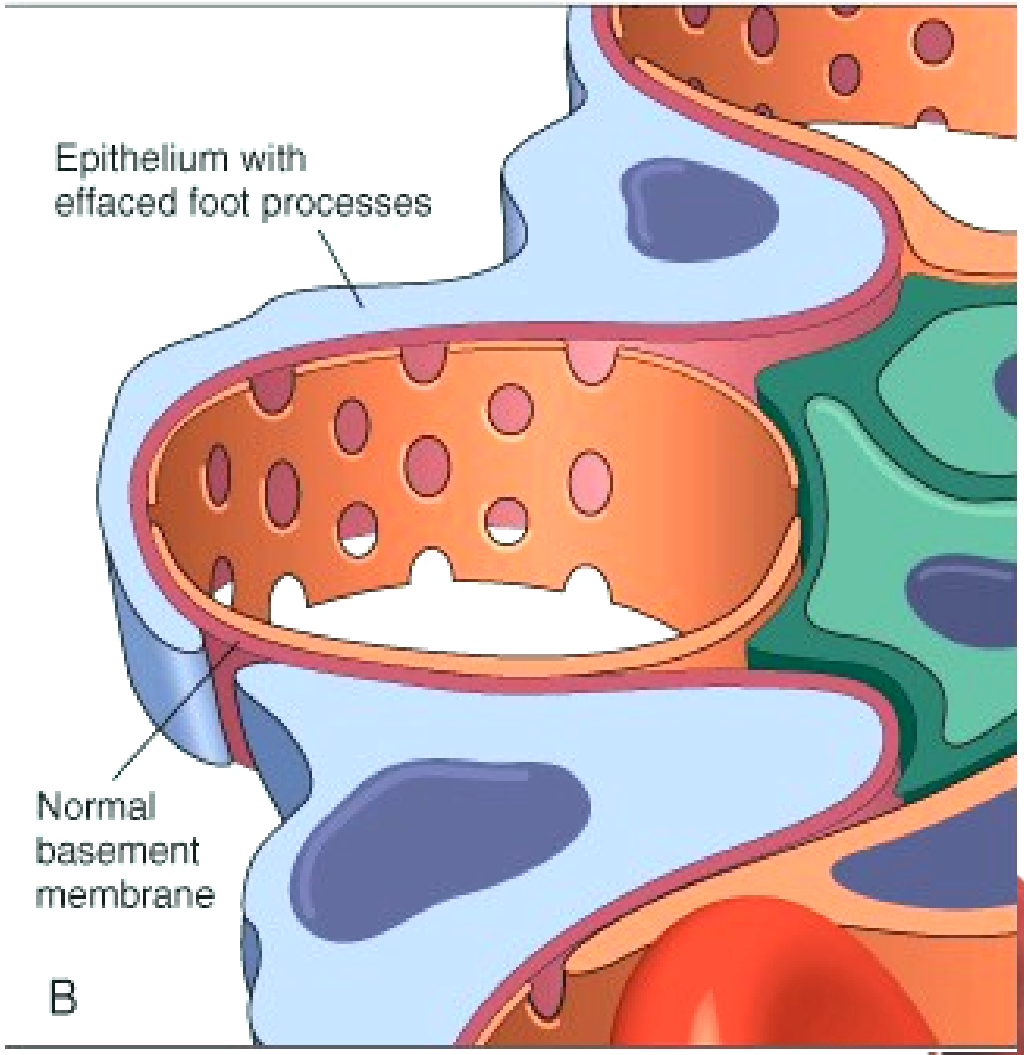
# Minimal change GN
# Lipoid nephrosis



# Introduction

**Features: Diffuse loss of foot processes of podocytes .**

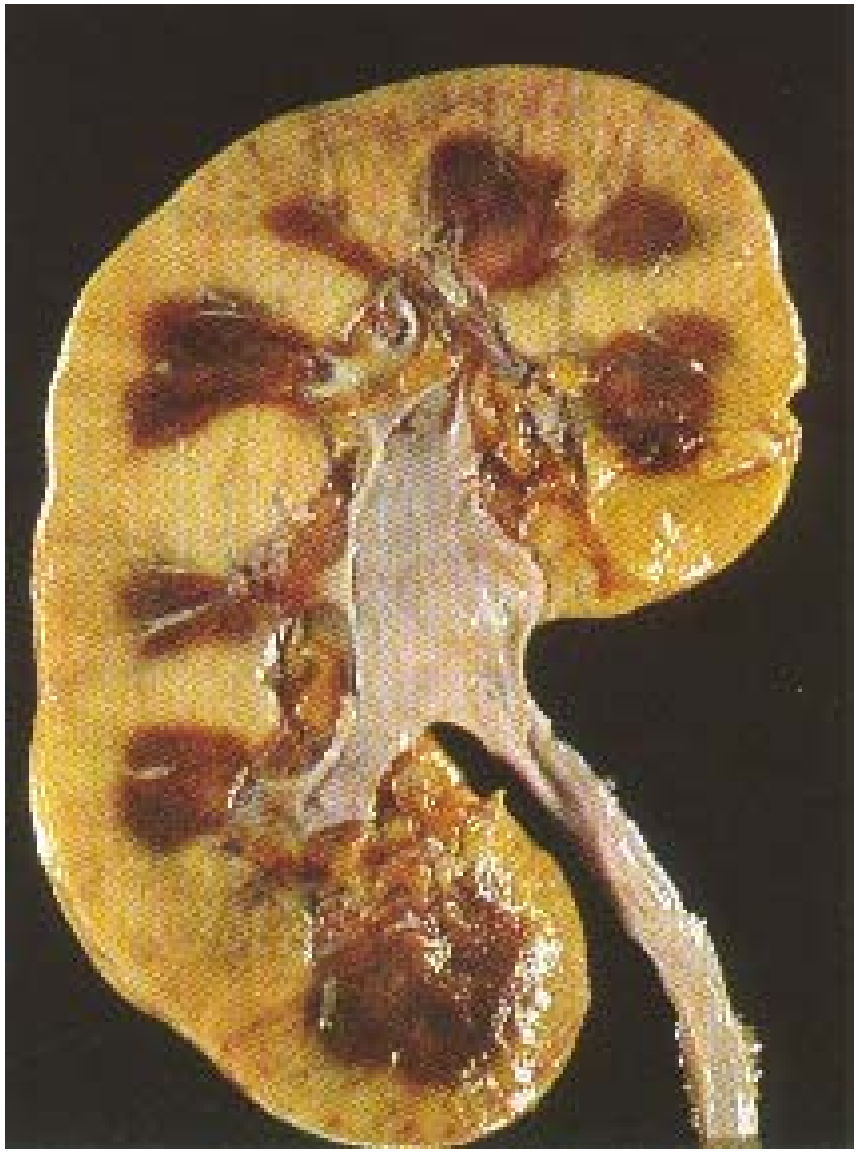
**Pathogenesis :**
**Unclear, relate to T cells mediated immunity.**

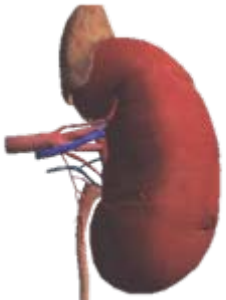


# Pathological changes

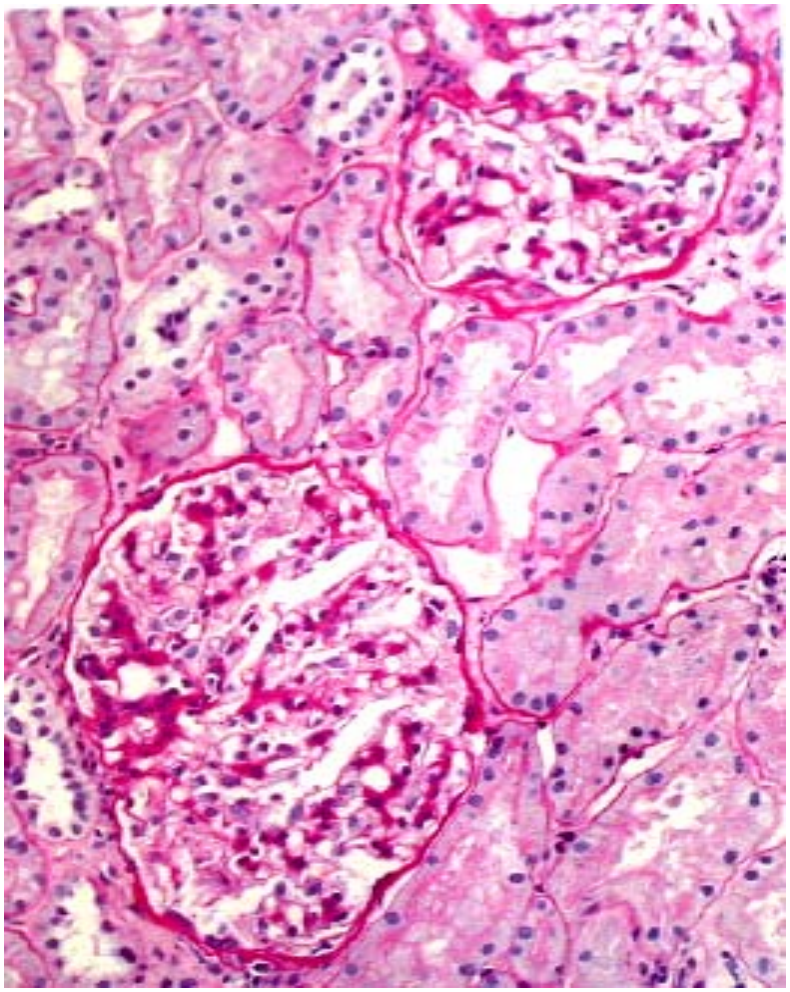
**Gross:**

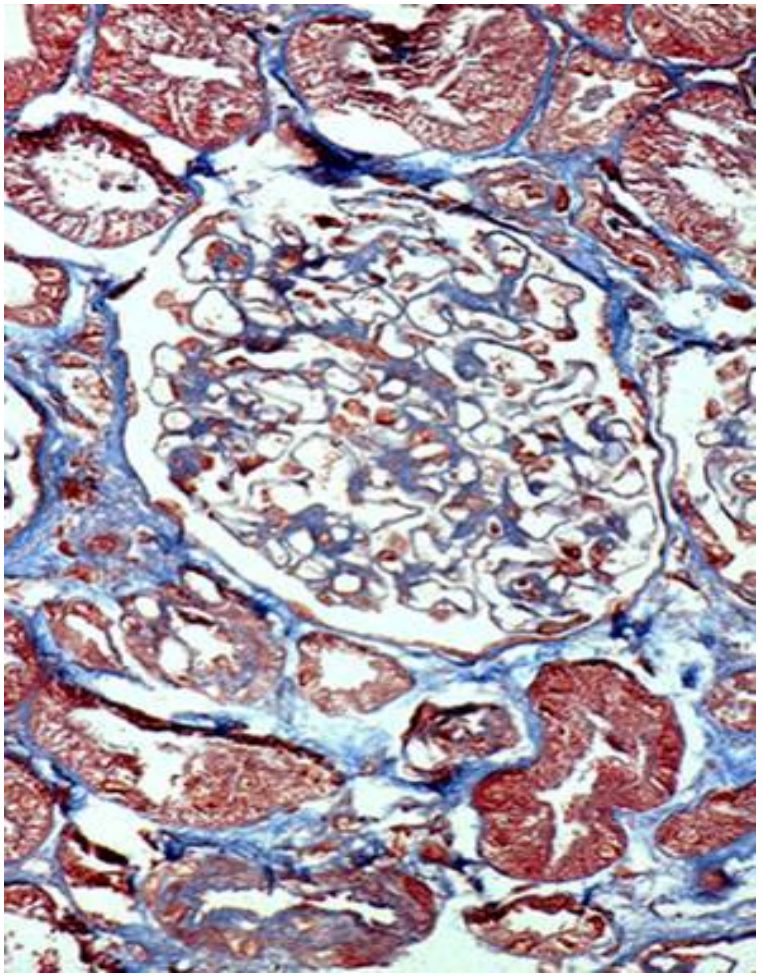
**Enlarged and pale**



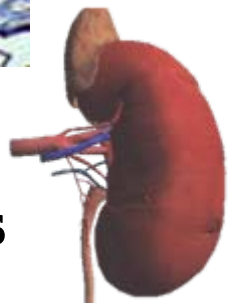


# Pathological changes

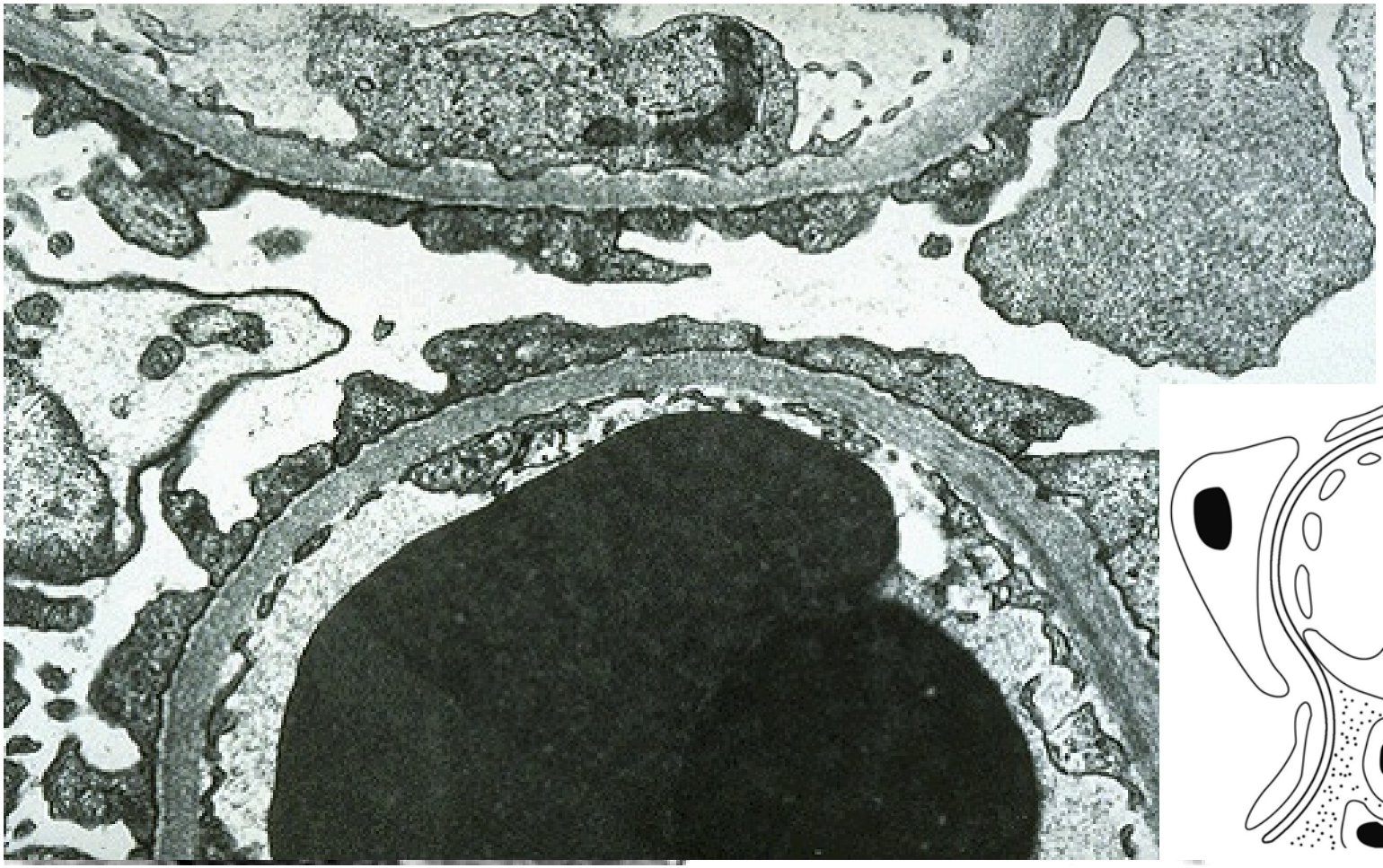


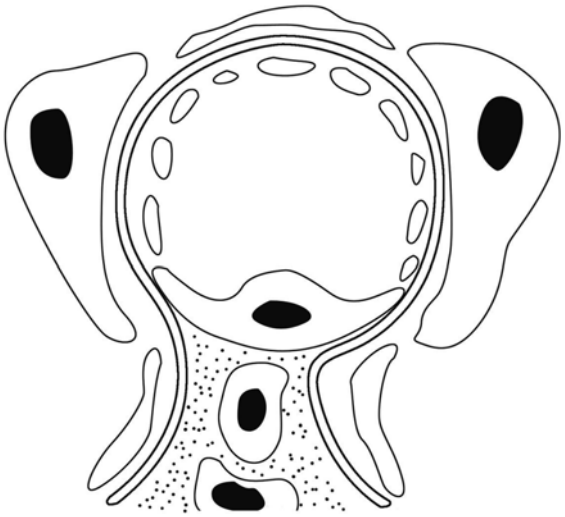


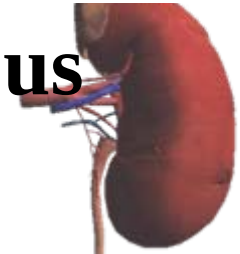
**LM: glomerulous: appear normally,**
**renal tubule: lipid or protein droplets →proximal tubules**



# Pathological changes

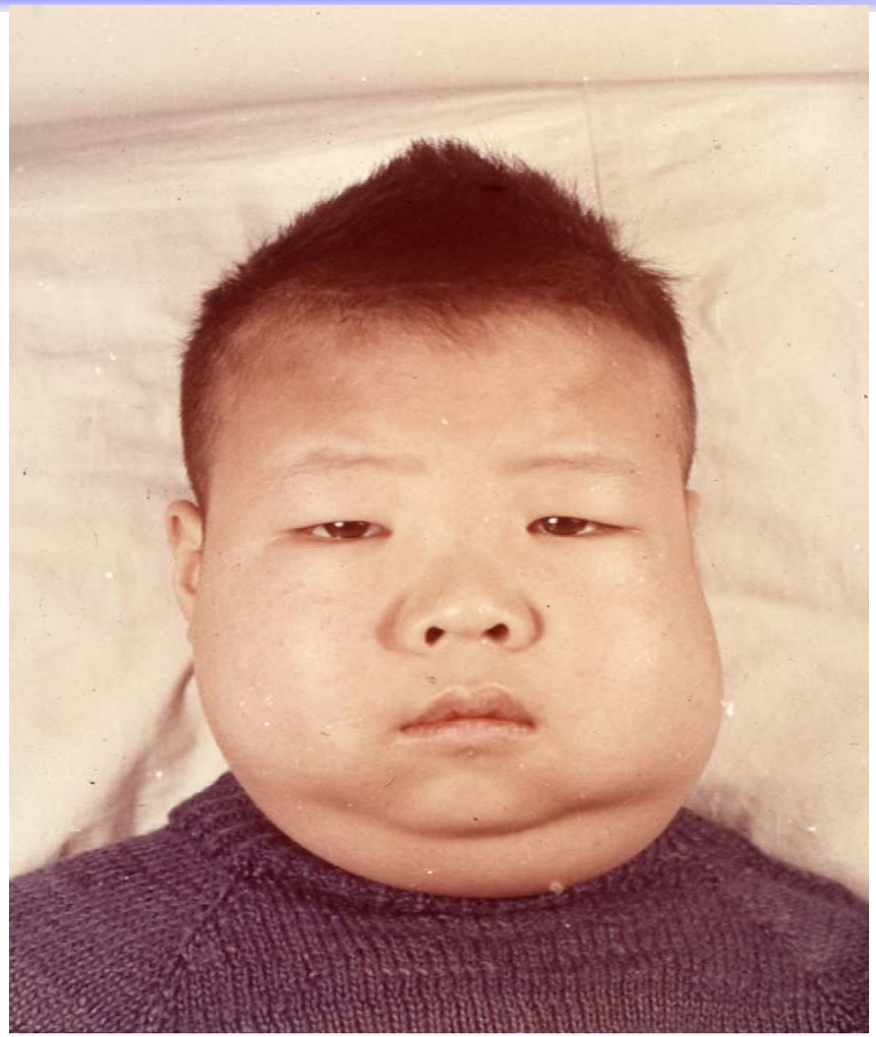




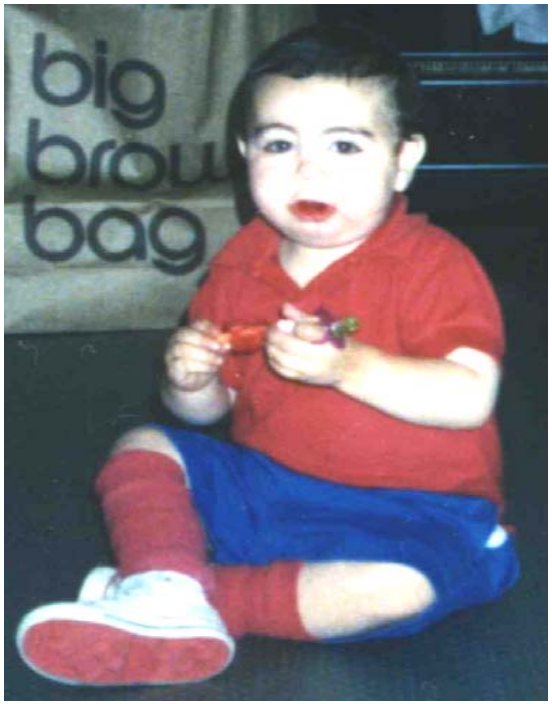


**foot processes disappear and pseudovillous transformation**

# Clinical features



355 肾炎—激素治疗后
Glomerulonephritis-after hormonic treatment




**The most frequent cause of nephrotic syndrome in children.**

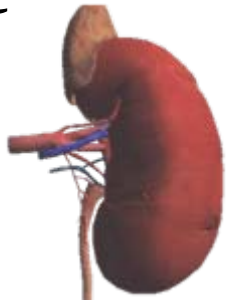
**2-8 years of age**

**90%: reversible after corticosteroid therapy**

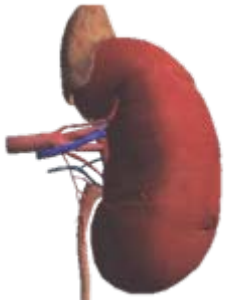
**5%→renal failure**

# Minimal change GN--- lipoid nephrosis

- **Features: Diffuse loss of foot processes of podocytes .**
- **Gross: Enlarged and pale.**
- **LM: glomerulous: appear normally, renal tubule: lipid or protein droplets →proximal tubules**
- **IF and EM: foot processes disappear**
- **Clinical features: the most frequent cause of nephrotic syndrome in children.**



# Focal segmental glomerulosclerosis （FSG)

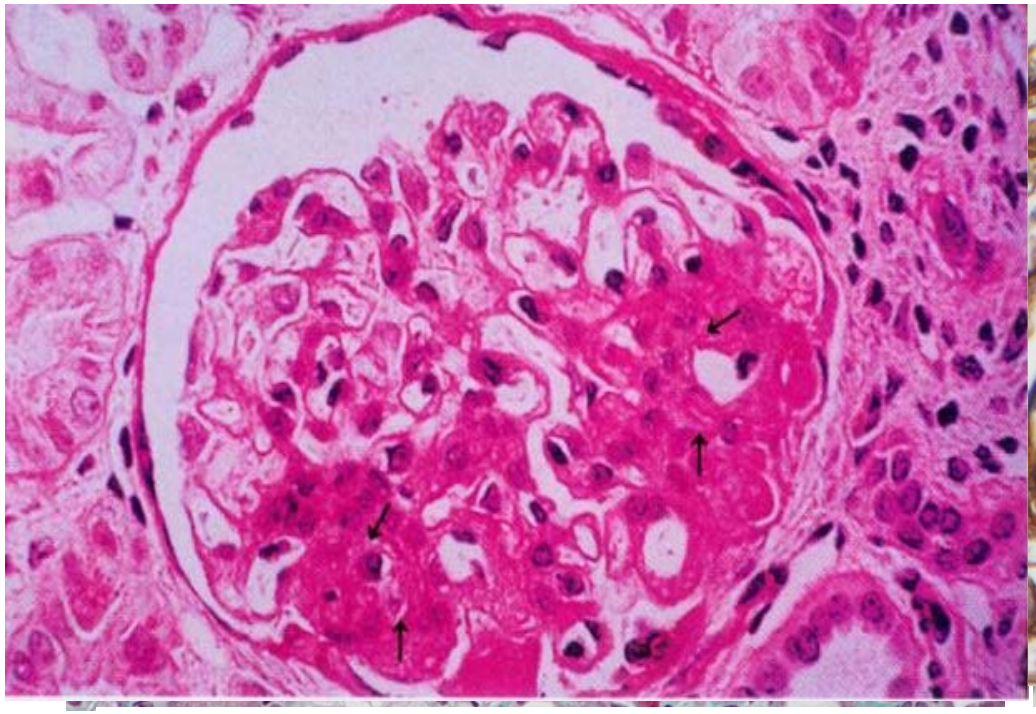


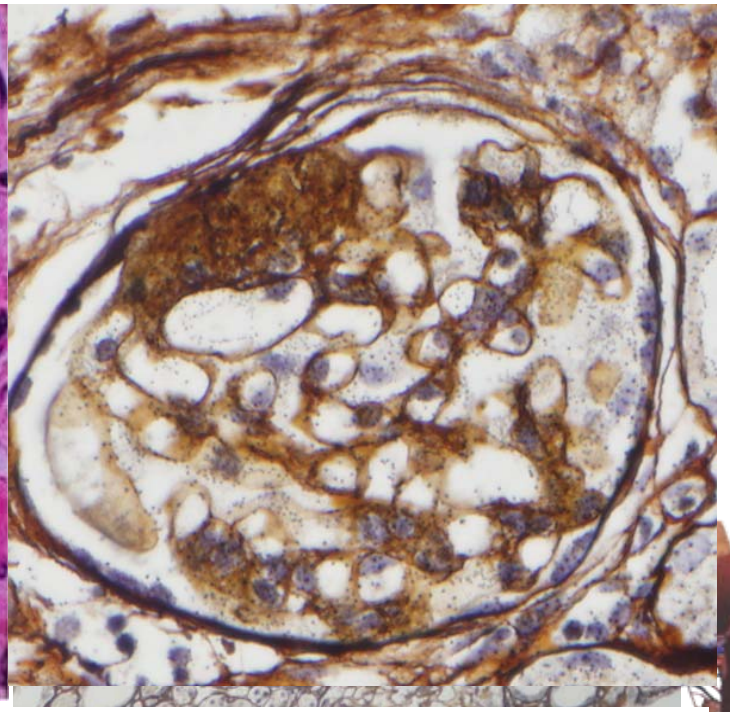
# Pathological changes

**Features:** Sclerosis of { portion of glomeruli
portion of capillary tuft(<50%)

**LM:**

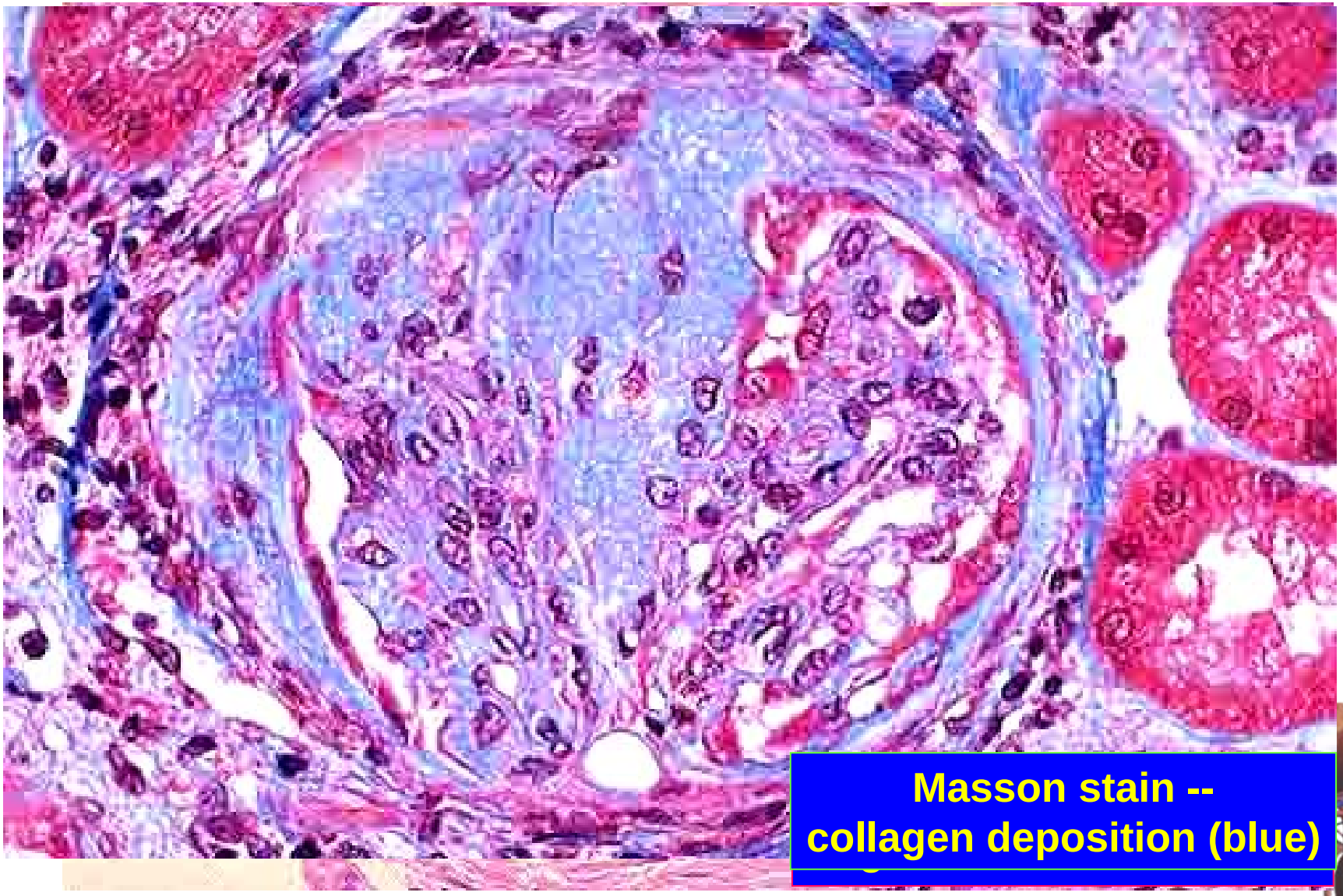
local distribution; increased in mesangial matrix; collapse of BM; deposition of hyaline masses





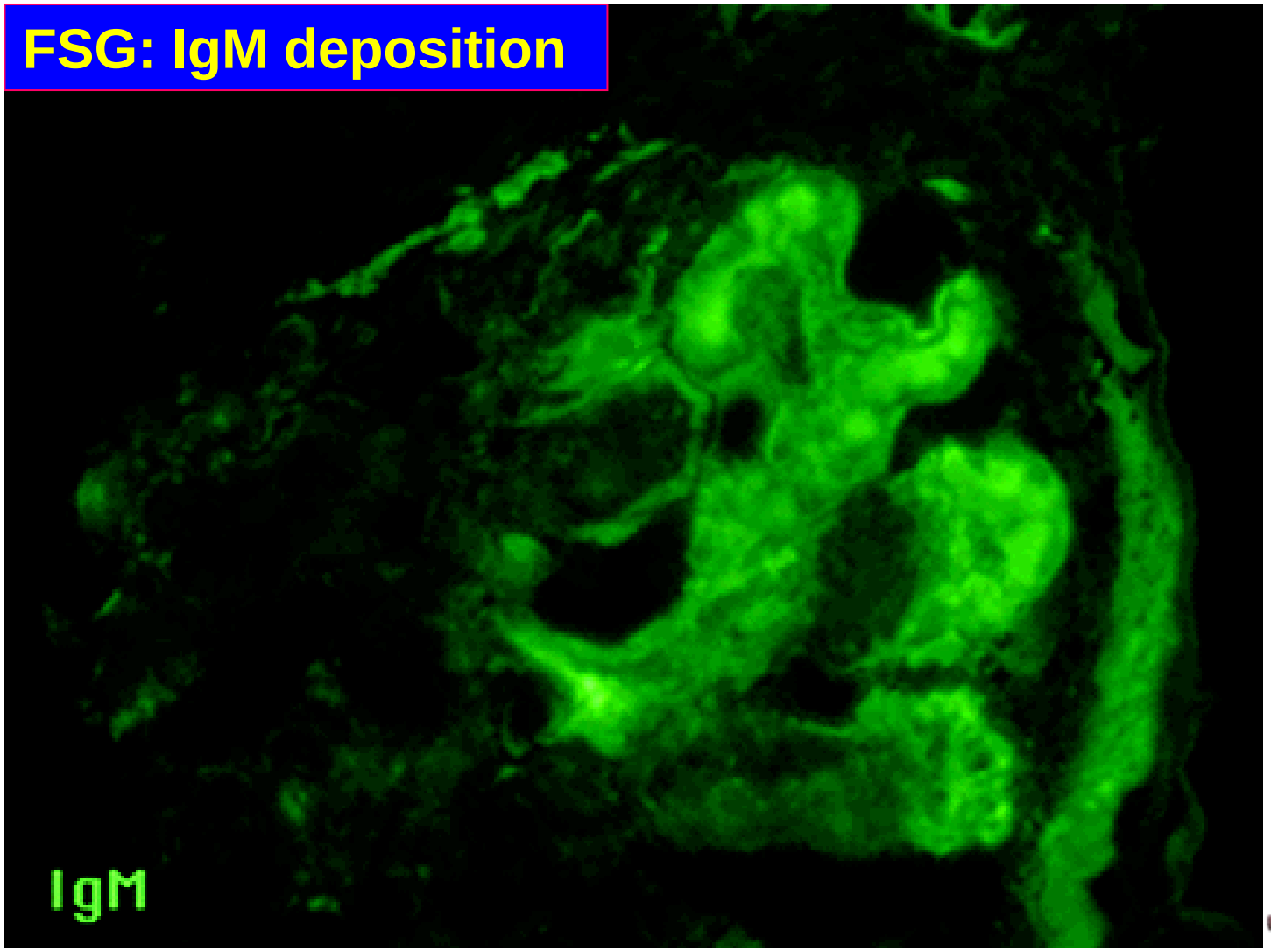
collagenous sclerosis area

# Pathological changes

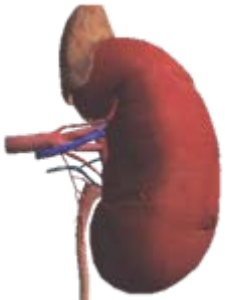


Masson stain --
collagen deposition (blue)

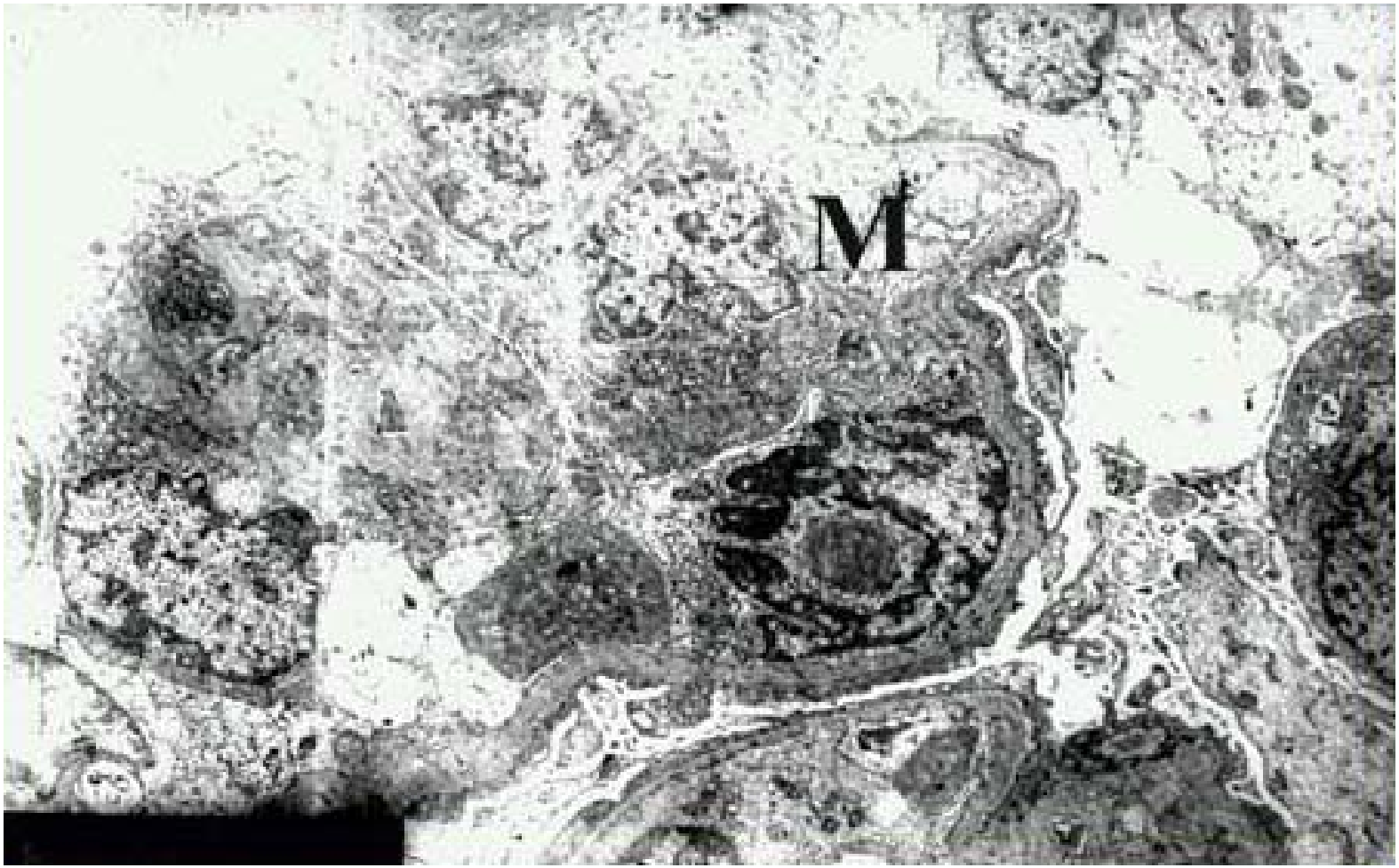
# Pathological changes




deposition of IgM , C3 in the sclerotic region

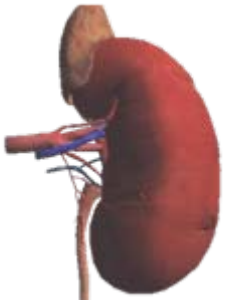


# Pathological changes




**EM:** **diffuse loss of foot processes of visceral epithelial cells**

**focal detachment of visceral epithelial cells from GBM**



# Clinical features

## Clinical features

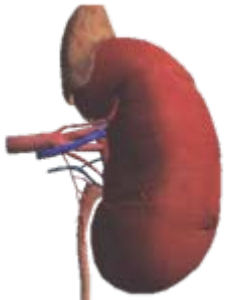
**nephrotic syndrome or proteinuria**

① **High incidence of hematuria and hypertension**
② **Nonselective proteinuria**
③ **Poor response to corticosteroid therapy**
④ **Deposition of IgM , C3 in the sclerotic region**

**Prognosis**

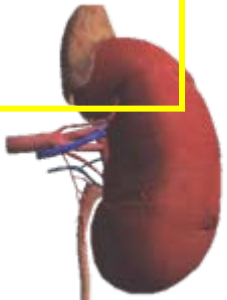
**children: good**

**adult: most→chronic GN (<10years)**



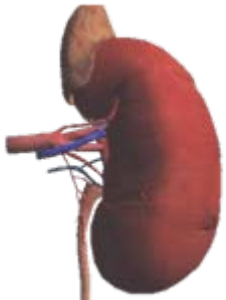
# Focal segmental glomerulosclerosis

- LM: normal and segmental sclerotic glomeruli
- IF: IgM and C3 in the affected sclerotic segments
- EM: diffuse effacement of foot process, loss of epithelial cell, collapsed basement membranes, excess mesangial matrix material
- Gross
  - no gross abnormalities in the early phases of the disease.
  - the kidneys small and shrunken



# *Pathologic types producing nephrotic syndrome(NS)*

- **Membranous GN（membranous nephropathy）**
- **Minimal change GN （lipoid nephrosis ）**
- **Focal segmental glomerulosclerosis(FSG )**
- **Membranoproliferative GN (MPGN)**
- **Mesangial proliferative GN**

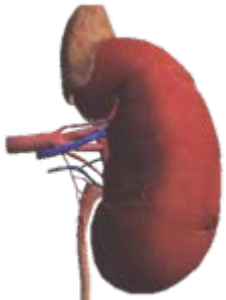


- **Membranous Glomerulopathy (MGN) = thickening of GBM**

  **MCC of nephrotic syndrome in ADULTS**
- **Minimal change disease (MCD) = MCC of Nephrotic syndrome in children**
- **Both clinically present with Nephrotic Syndrome (NS)**

| Feature | Adult NS | Childhood NS |
|---|---|---|
| MCC of NS | Membranous GN | Minimal Change disease |
| Proteinuria | Non- selective | Highly selective (mainly albumin) |
| light microscopy | Thick GBM | No change |
| Electron Microscopy | GBM thick | Effacement of foot process |
| Prognosis | Not good | Excellent (steroid Rx) |

# IgA Nephropathy
# (Berger Disease)

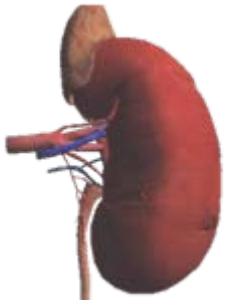


# Introduction

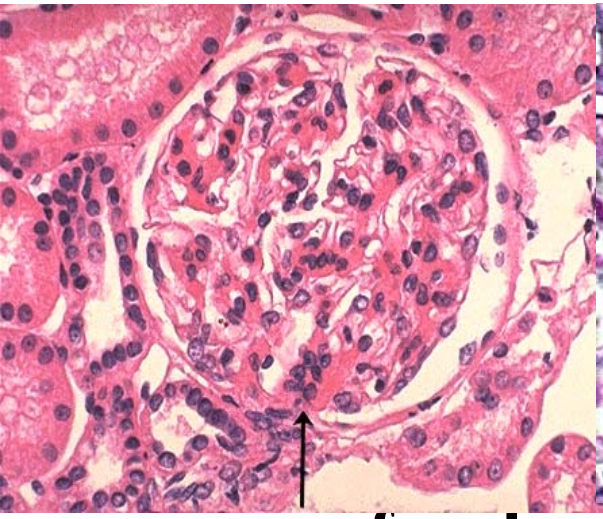
**Feature**: **Immunofluorescence—prominent IgA deposits in the mesangial regions .**

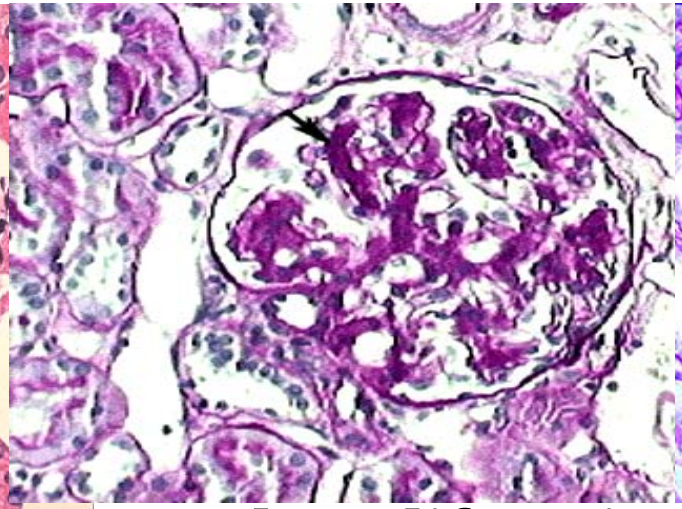
**Recurrent macroscopic or microscopic hematuria**

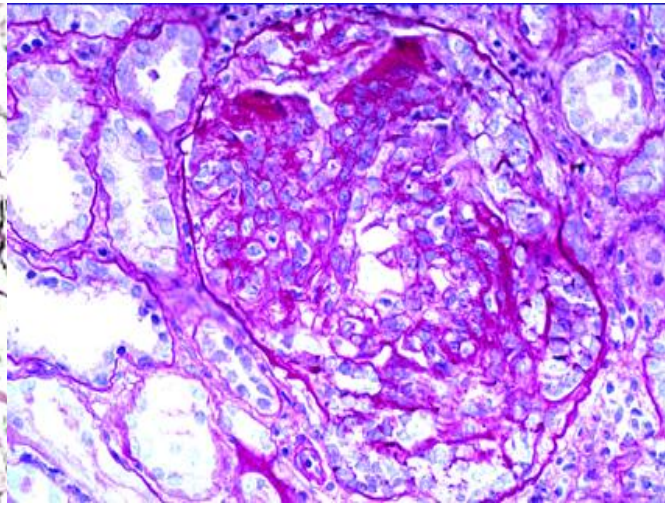
**The most common type of GN worldwide**



# Pathological changes







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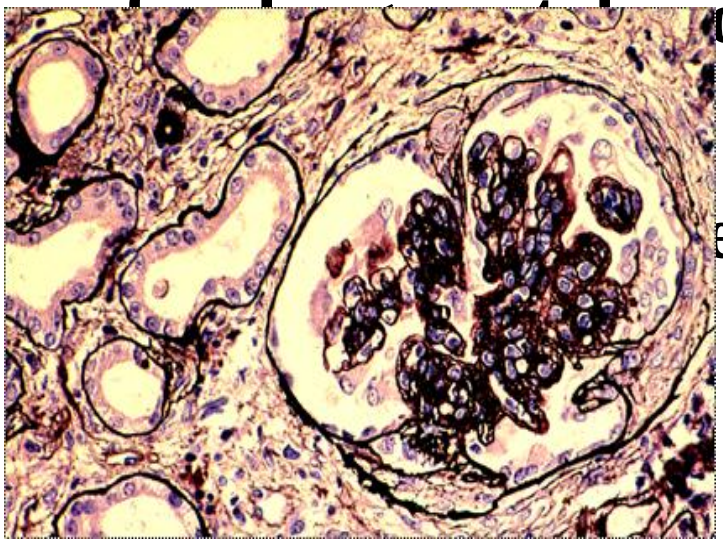
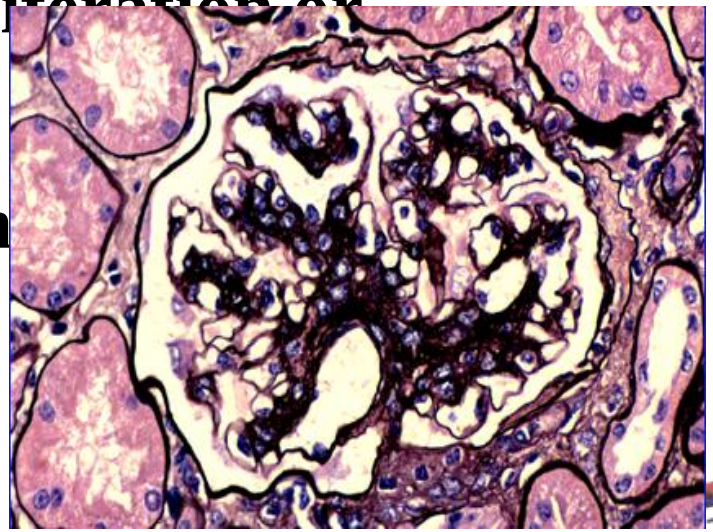
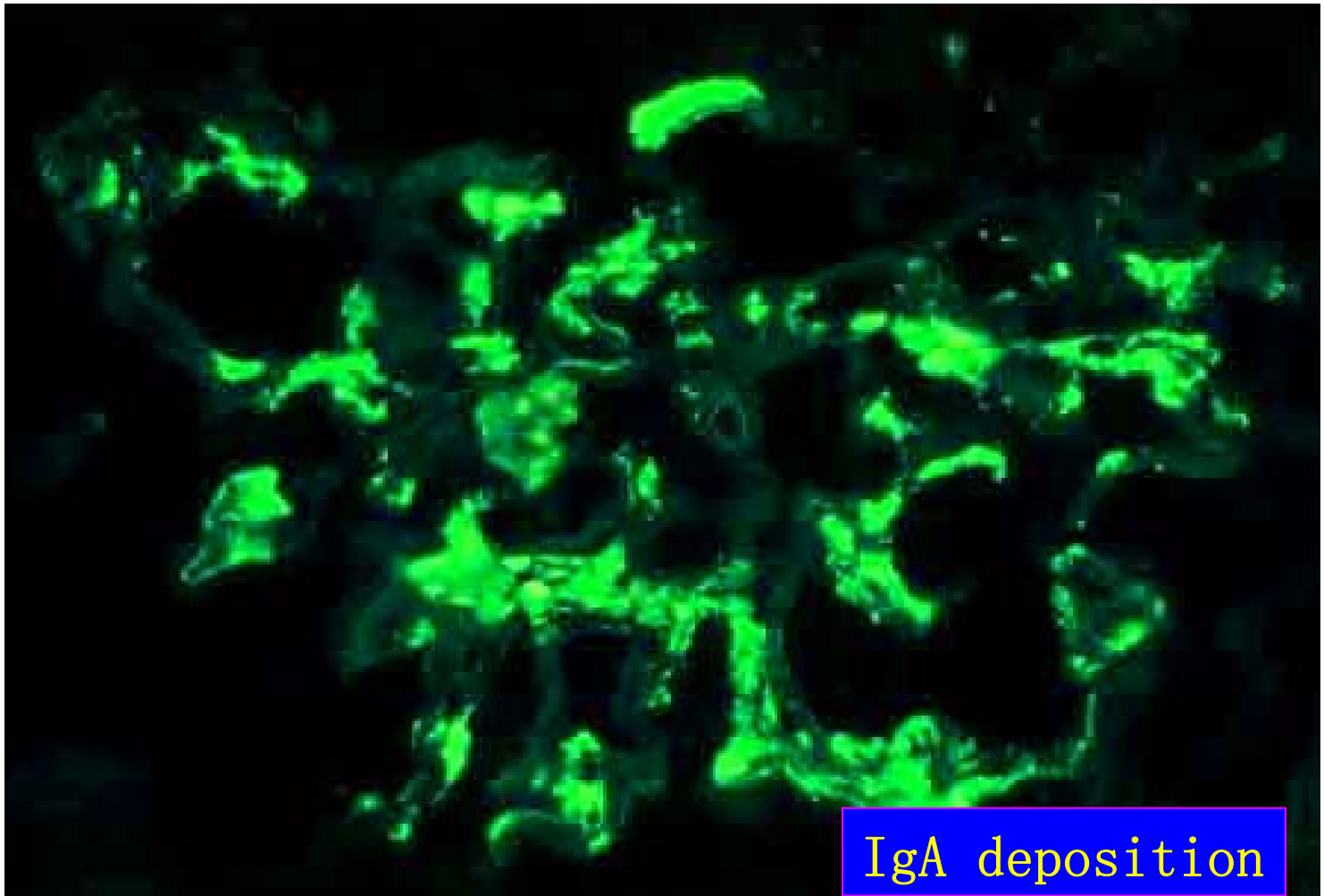
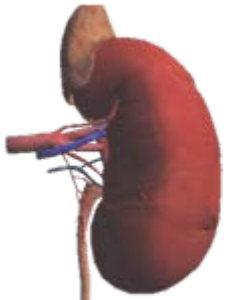




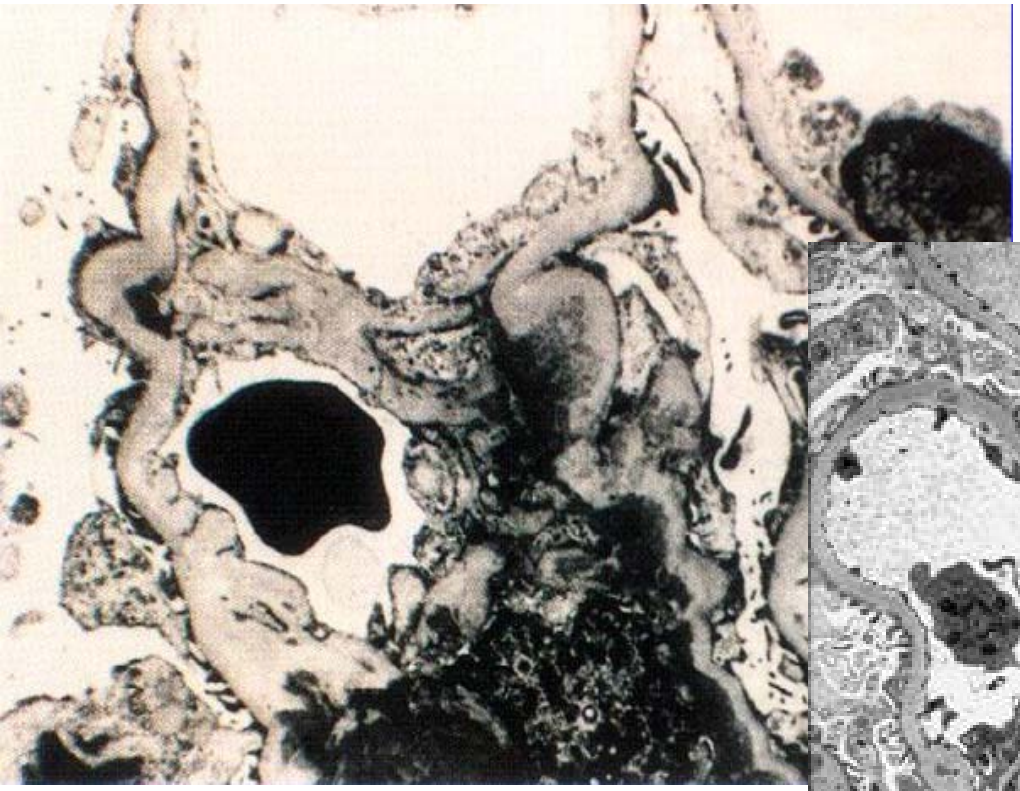
# Pathological changes

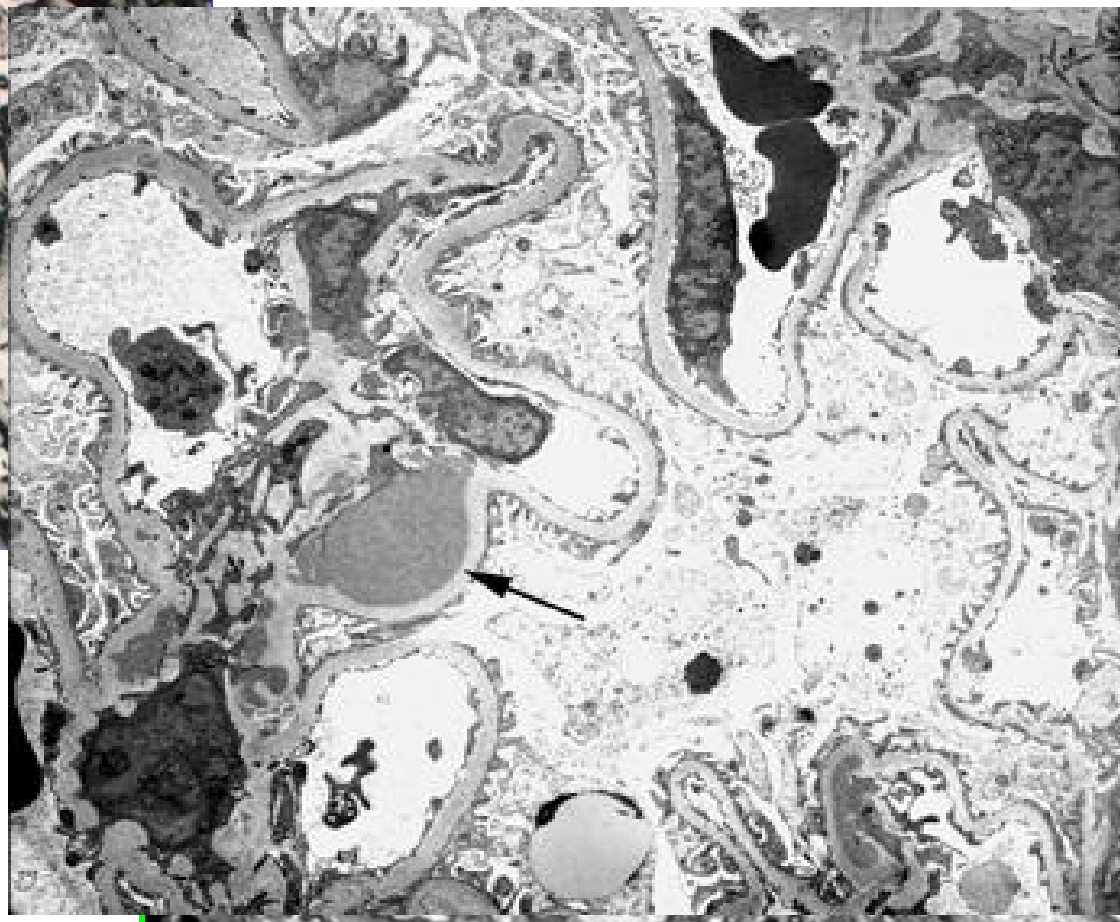
IF： **Characteristic deposition of IgA,** principally in mesangial regions, detected by immunofluorescence.



# Pathological changes





EM---many discrete electron dense deposits located in the mesangium

# Clinical features

**Children and young adults were often affected**

**Accompanied with mucosal infections of**

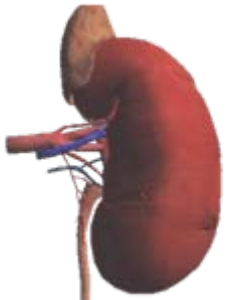
- **respiratory tract**
- **gastrointestinal tract**
- **urinary tract**

**most common**

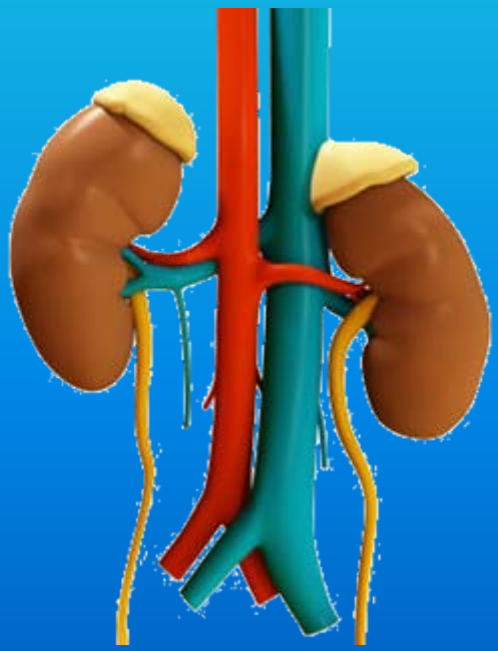
- **gross hematuria**
- **microscopic hematuria: less**

**nephrotic syndrome or acute nephritic syndrome (5%-10%)**

**15-40%→chronic renal failure**



# Glomerulonephritis, GN



# Pathological type

**Acute diffuse proliferative glomerulonephritis (GN)**

**Rapidly progressive GN（RPRN)**

**Crescentic glomerulonephritis(CrGN )**

**Membranous GN（membranous nephropathy）**

**Membranoproliferative GN (MPGN)**

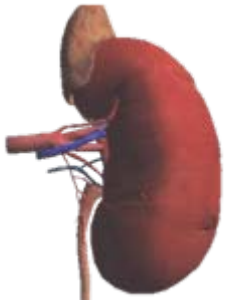
**Mesangial proliferative GN**

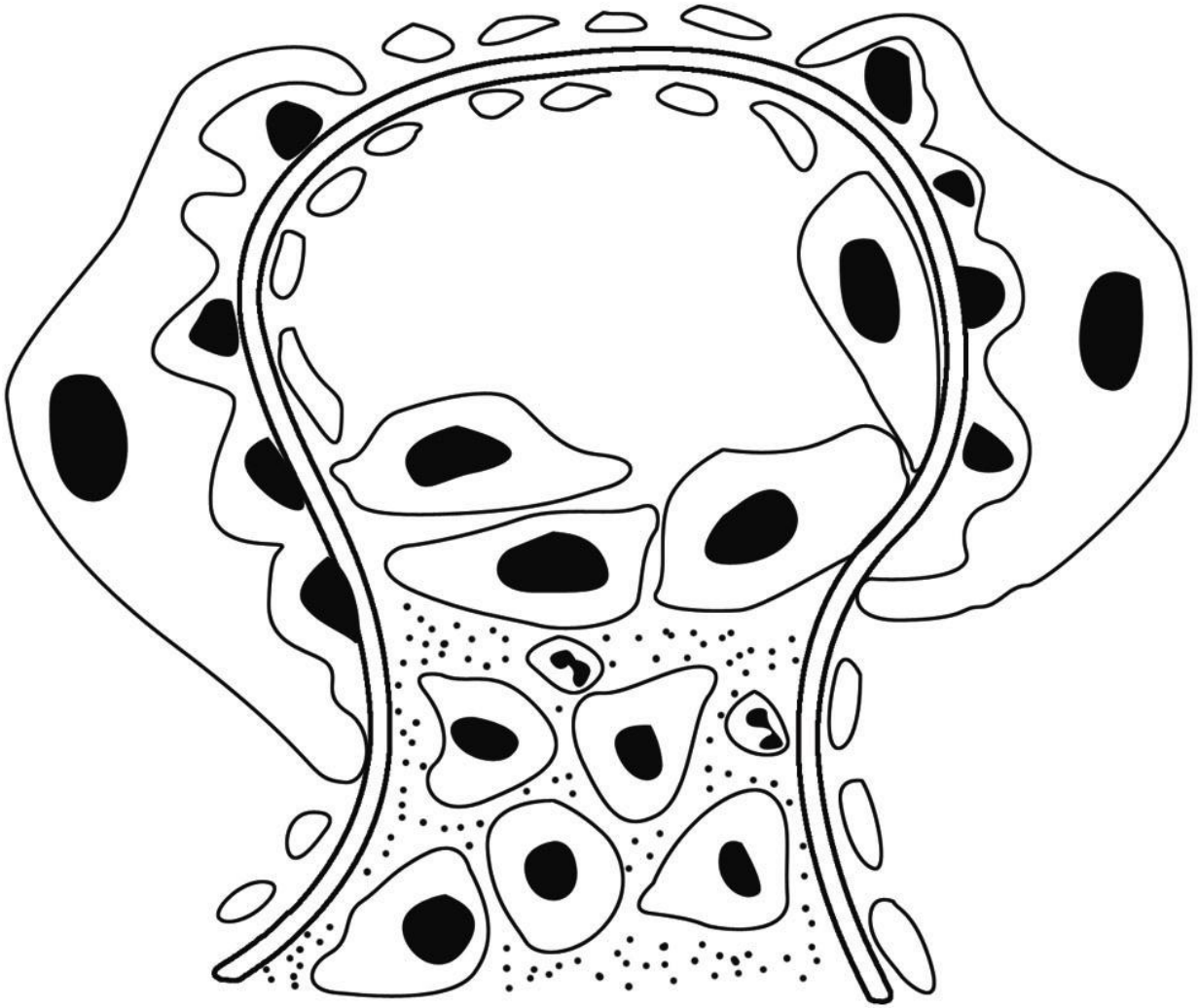
**Minimal change GN （lipoid nephrosis ）**

**Focal segmental glomerulosclerosis(FSG )**

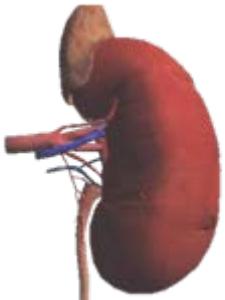
**IgA nephropathy**

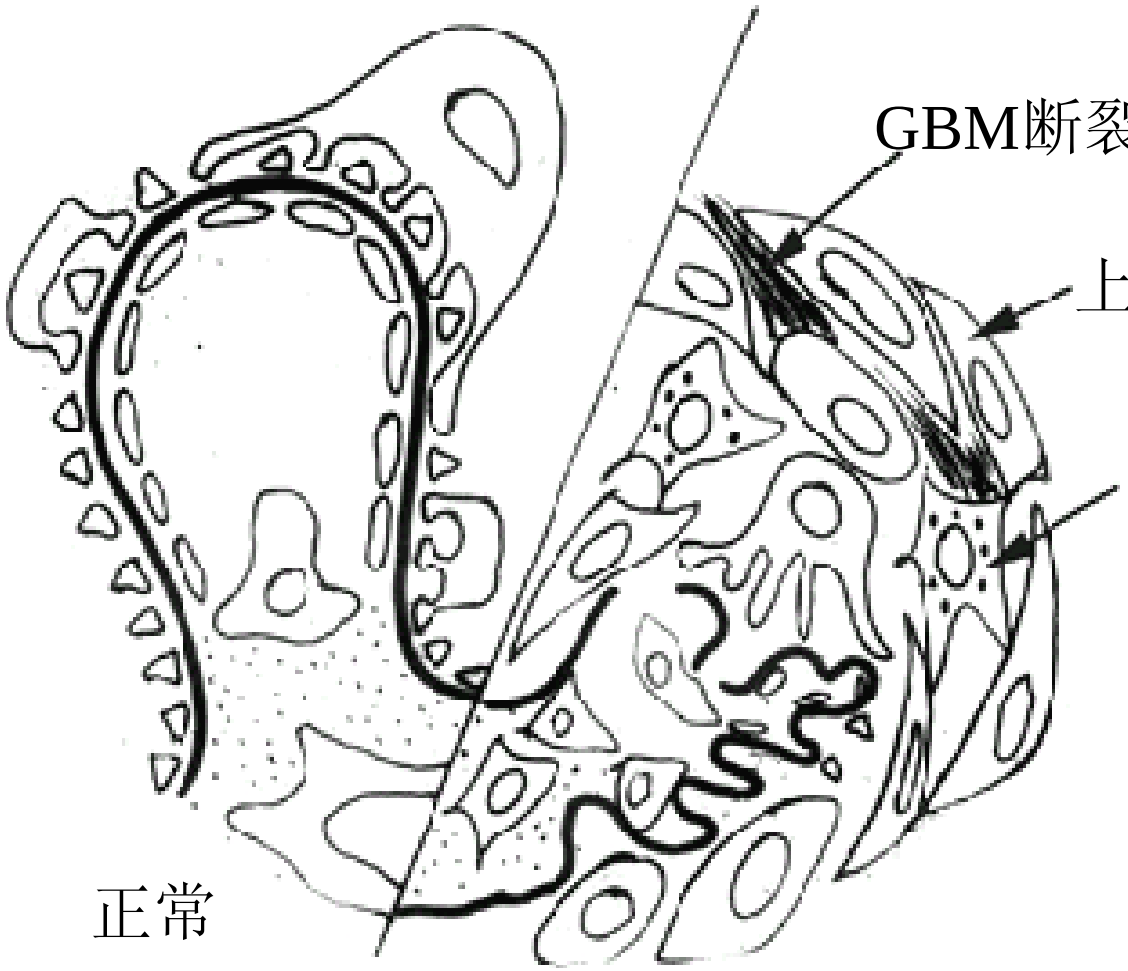
**Chronic GN**



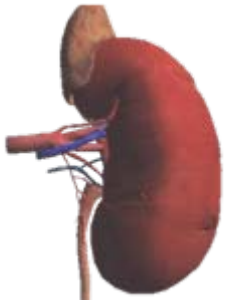


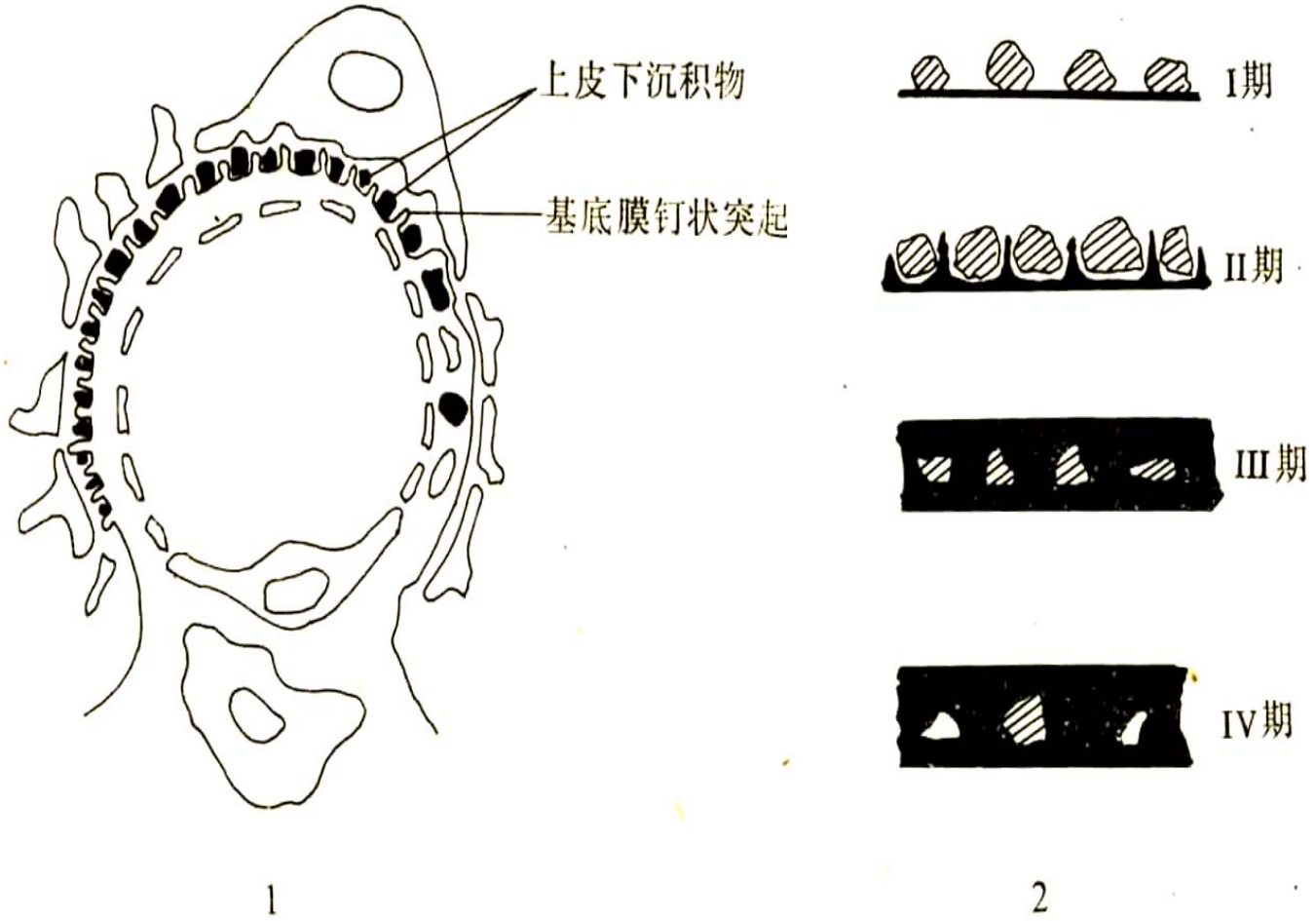
**Endocapillary proliferative GN**



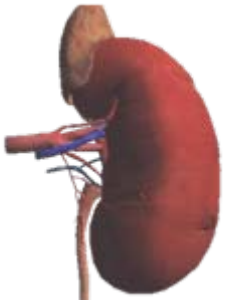


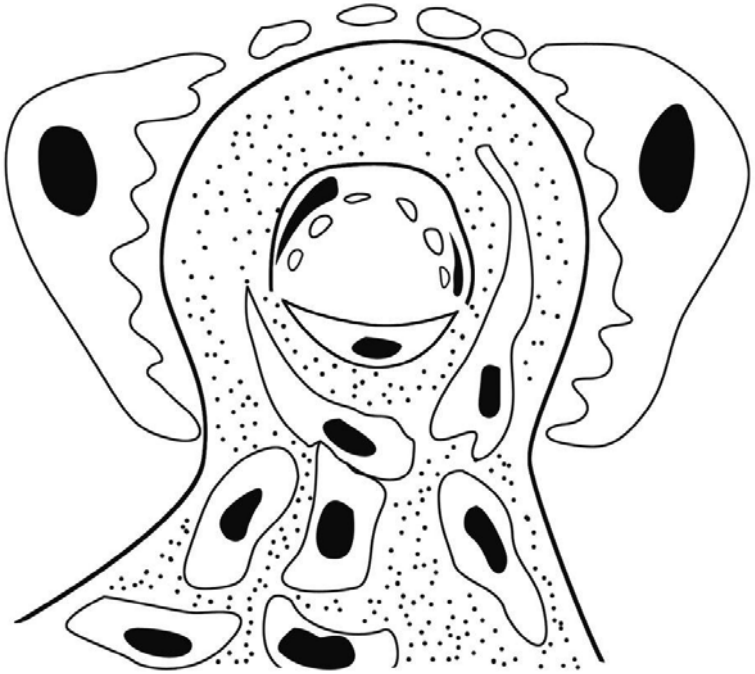

**Crescentic GN**



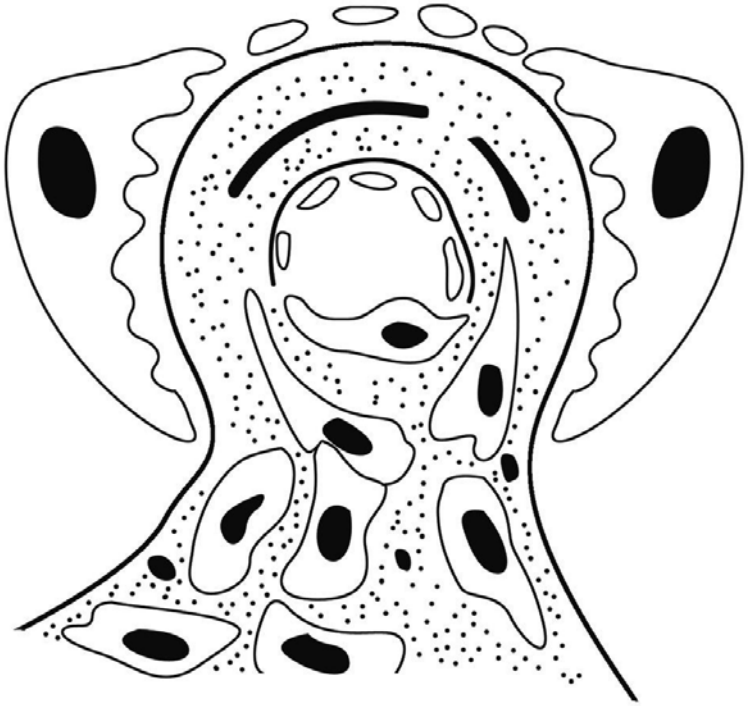



# Membranous GN



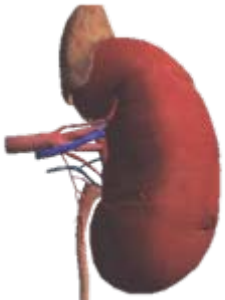


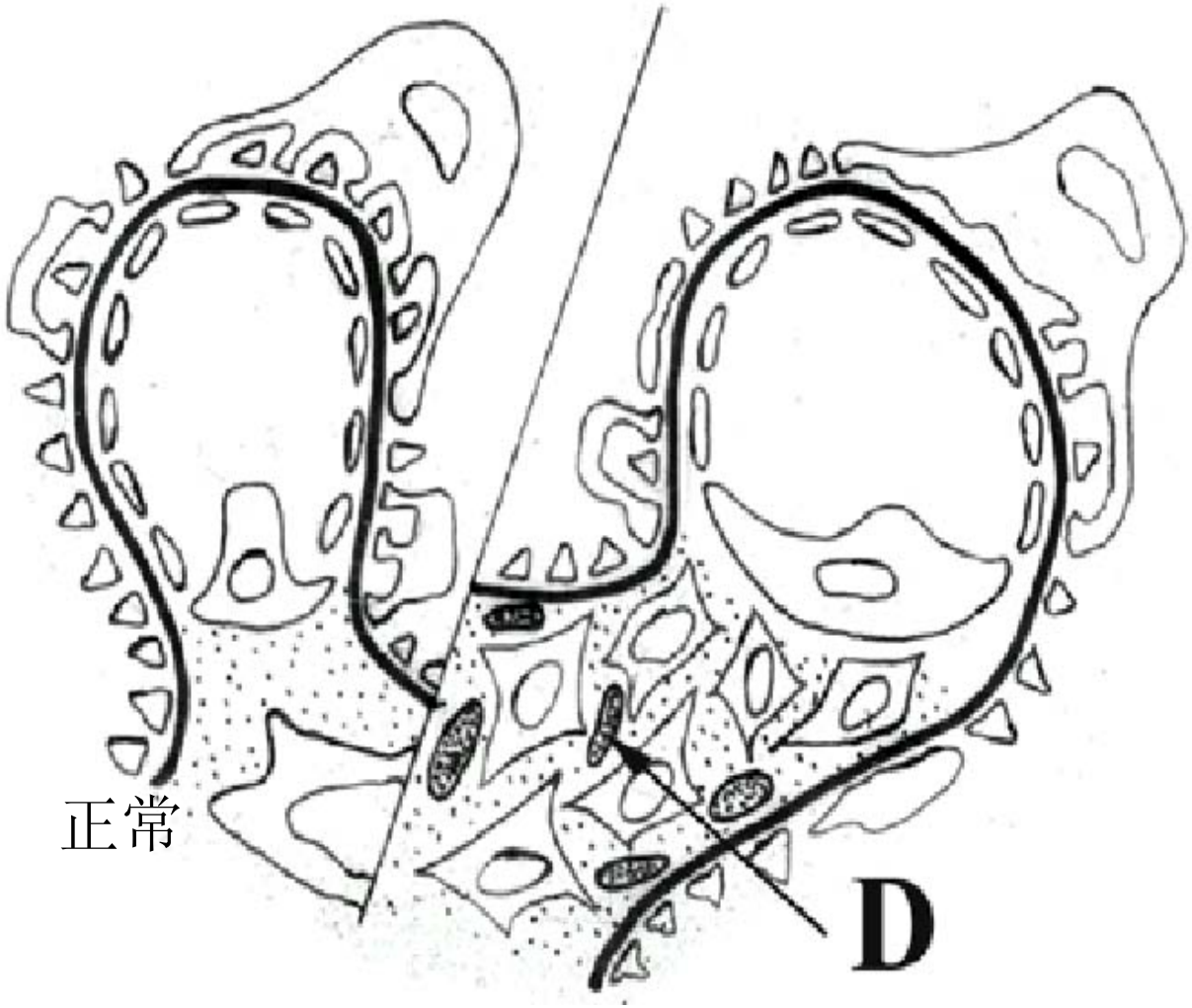
Type I



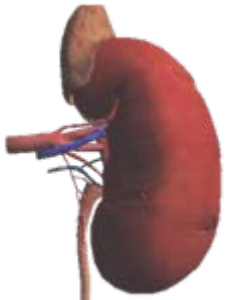
Type II

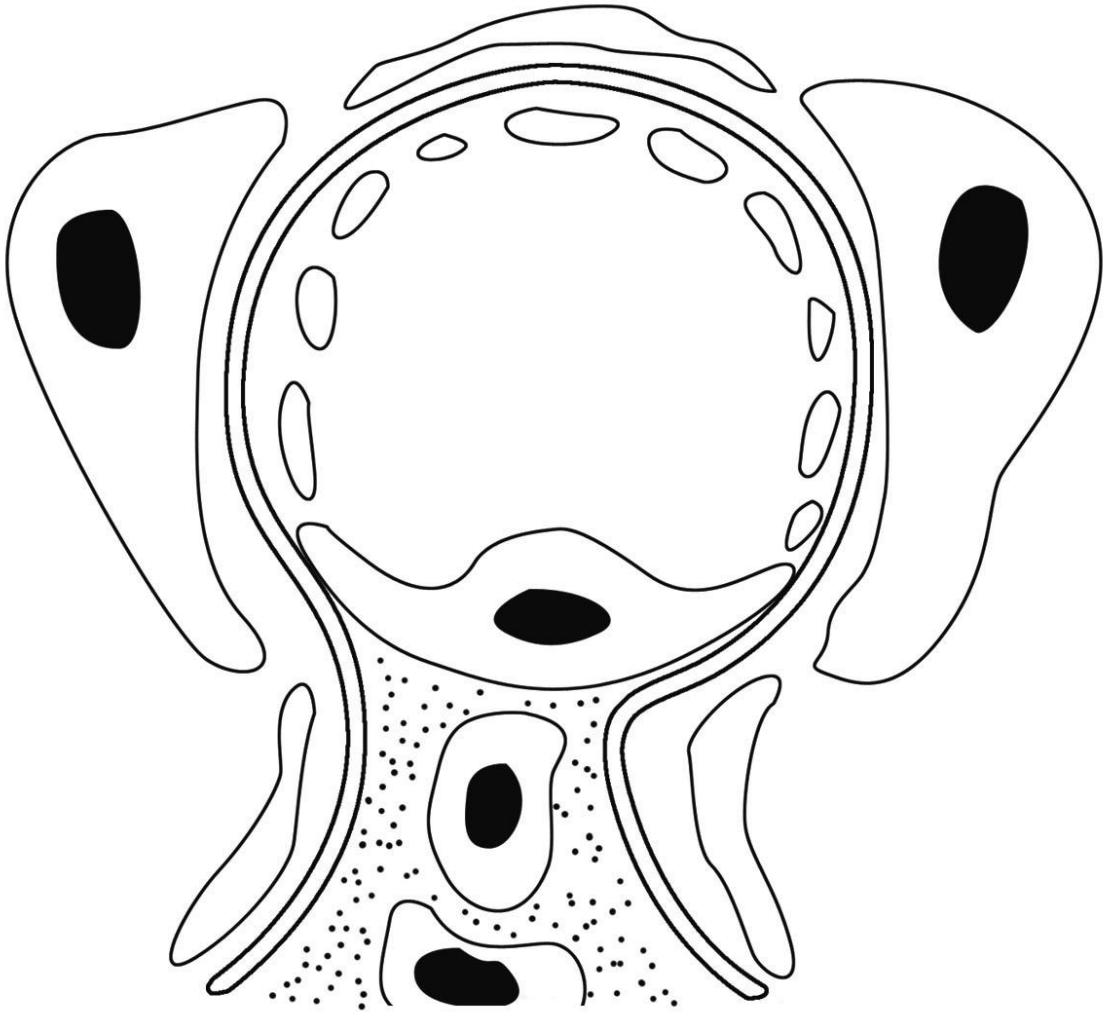
- **Membranoproliferative GN**



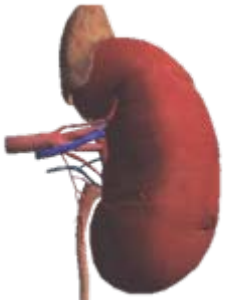


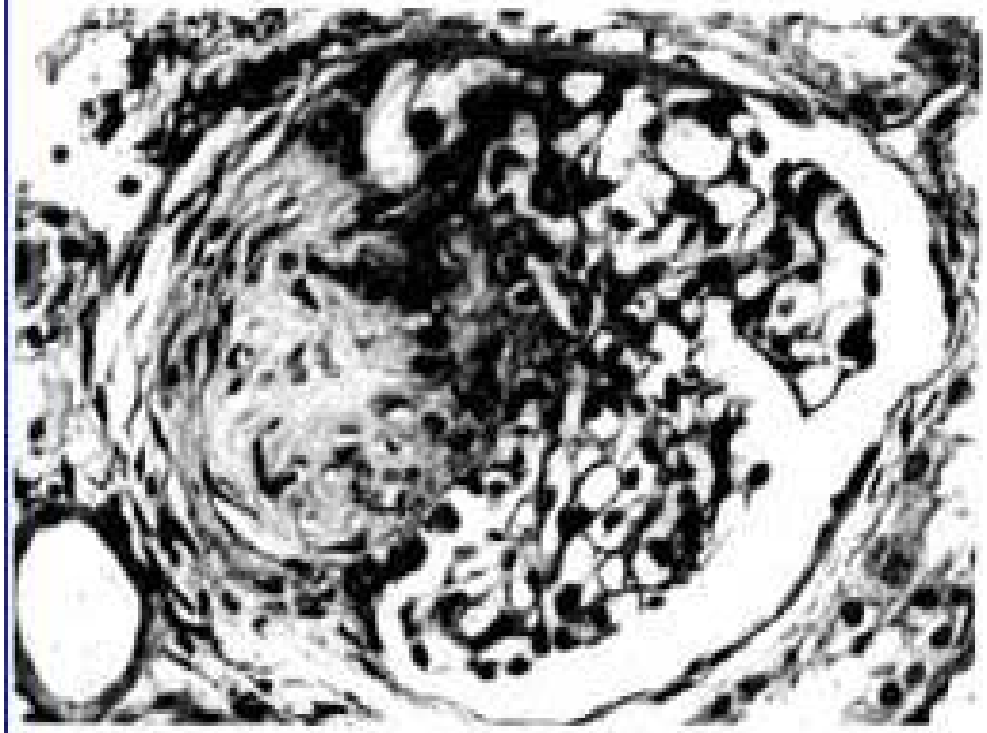

**Mesangioproliferative GN**

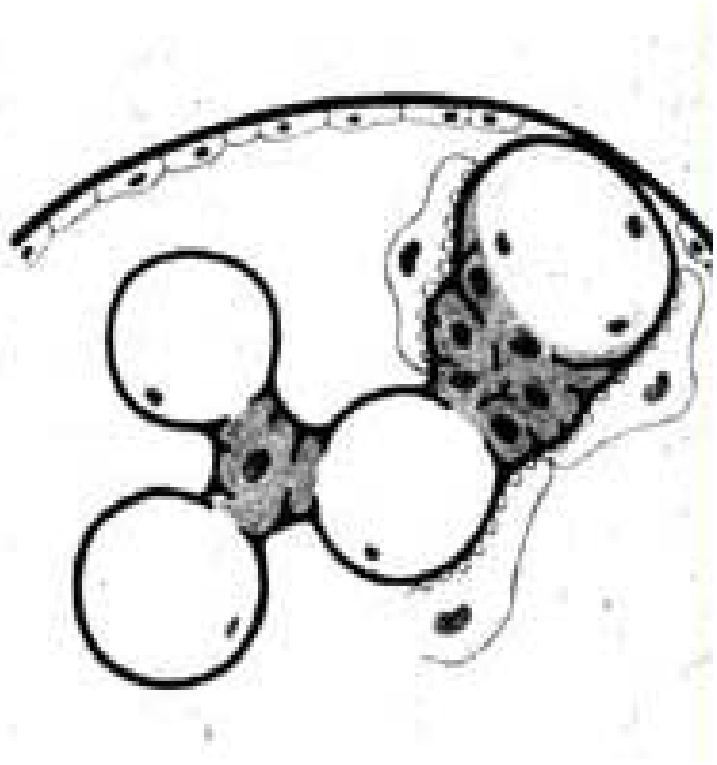




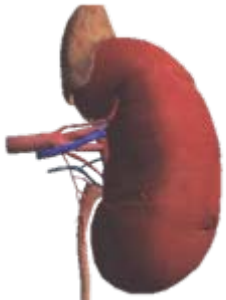
**Minimal change GN**

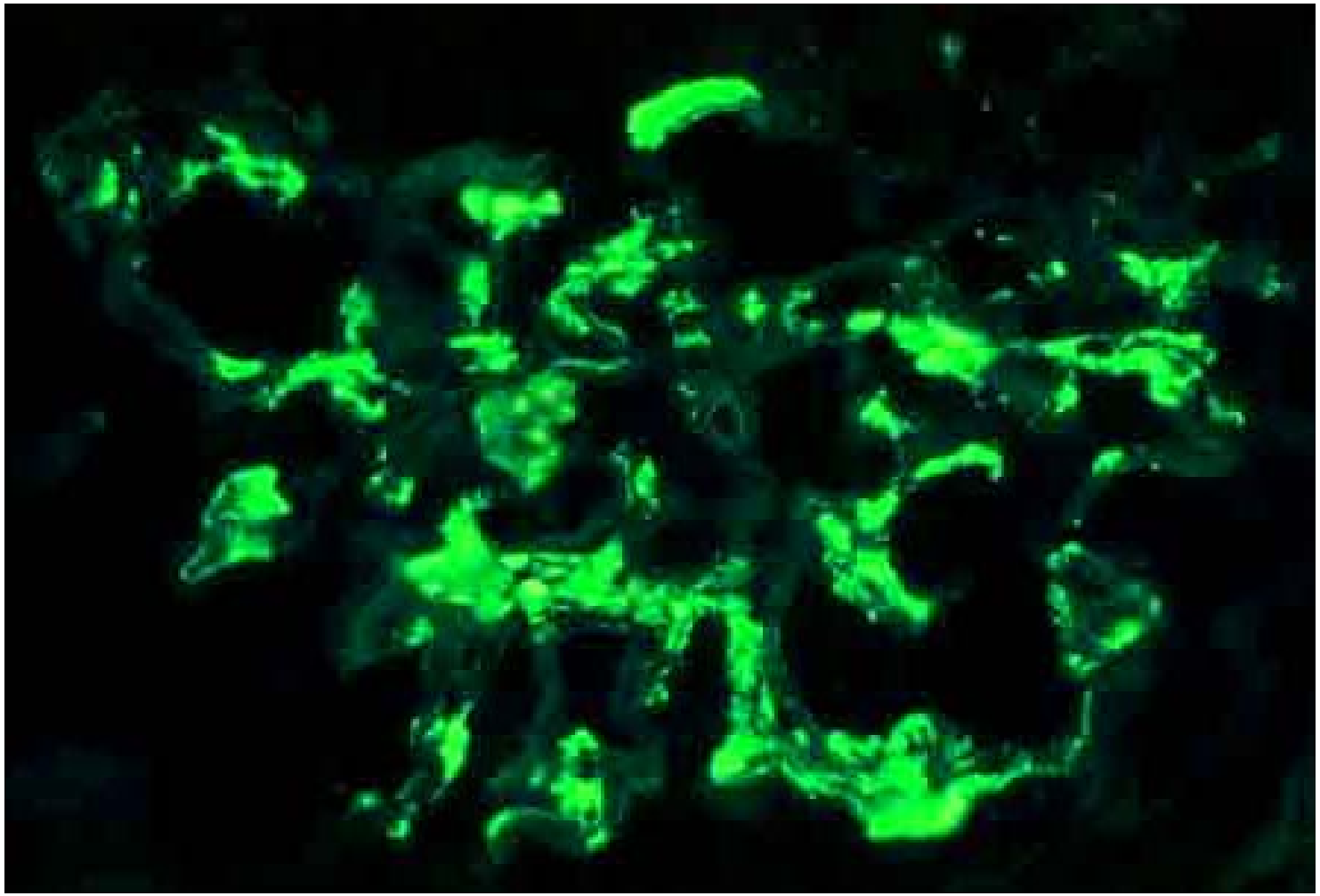




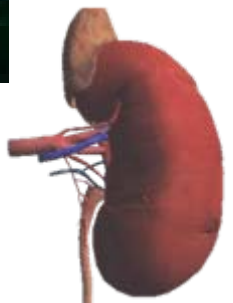


## Focal GN





**IgA nephropathy**



| 临床类型 | 临床表现 | 病理类型 | 病理特点 | | | 发病机制 |
|---|---|---|---|---|---|---|
| | | | 光镜 | 电镜 | 免疫荧光 | |
| 急性肾炎综合征 | 起病急，明显的血尿、蛋白尿、管型尿，水肿和高血压 | 急性弥漫性增生性肾炎 | 系膜细胞和内皮细胞弥漫性增生 | 脏层上皮细胞下驼峰状沉积物 | IgG和$C_3$沿基底膜和系膜区内呈颗粒状沉积 | 常与感染有关，免疫复合物沉积 |
| 快速进行性肾炎综合征 | 出现水肿、血尿和蛋白尿后，迅速发生少尿或无尿、氮质血症及急性肾功能衰竭 | 快速进行性肾炎 | 肾小球新月体形成 | 无沉积物<br>毛细血管袢沉积物<br>无沉积物 | IgG和$C_3$沿肾小球毛细血管壁呈线状沉积<br>IgG和$C_3$沿肾小球毛细血管壁呈颗粒状沉积<br>阴性或极弱 | 抗基底膜型免疫复合物型<br>免疫反应不明显 |
| 肾病综合征 | 大量蛋白尿、 明显水肿、高脂血症、脂尿、低蛋白血症 | 轻微病变性肾炎 | 肾小球正常<br>肾小管脂质沉积 | 无沉积物，脏层上皮细胞足突消失 | 阴性 | 不明，T细胞功能异常 |
| | | 局灶性节段性肾小球硬化 | 局灶性节段性肾小球玻璃样变和硬化 | 脏层上皮细胞足突消失及细胞剥脱 | 局灶性IgM和C3 | 不清楚 |
| | | 膜性肾炎 | 弥漫性基底膜增厚，钉状突起形成，系膜无增生 | 脏层上皮细胞下沉积物，基底膜增厚 | IgG和$C_3$沿肾小球毛细血管壁呈弥漫性颗粒状沉积 | 自身抗体与抗原原位反应 |
| | | 膜性增生性肾炎 | 系膜增生，基底膜呈双轨状 | （I）内皮细胞下沉积物<br>（II）肾小球基底膜致密层的条带状沉积物 | I 型内皮细胞下IgG和$C_3$、C1或C4沉积<br>II型基底膜内$C_3$颗粒状荧光 | I 型免疫复合物<br>II型自身抗体，补体替代途径激活 |
| | | 系膜增生性肾炎 | 弥漫性系膜增生，基底膜无变化 | 系膜区沉积物 | 系膜区IgG（IgM）和C3沉积 | 不明 |
| 无症状性血尿或蛋白尿 | 持续或复发性肉眼或镜下血尿，或轻度蛋白尿 | IgA肾病 | 弥漫性或局灶性节段性系膜增生 | 系膜区沉积物 | 系膜区IgA和C3沉积 | 不明 |