Diseases of the Glomeruli

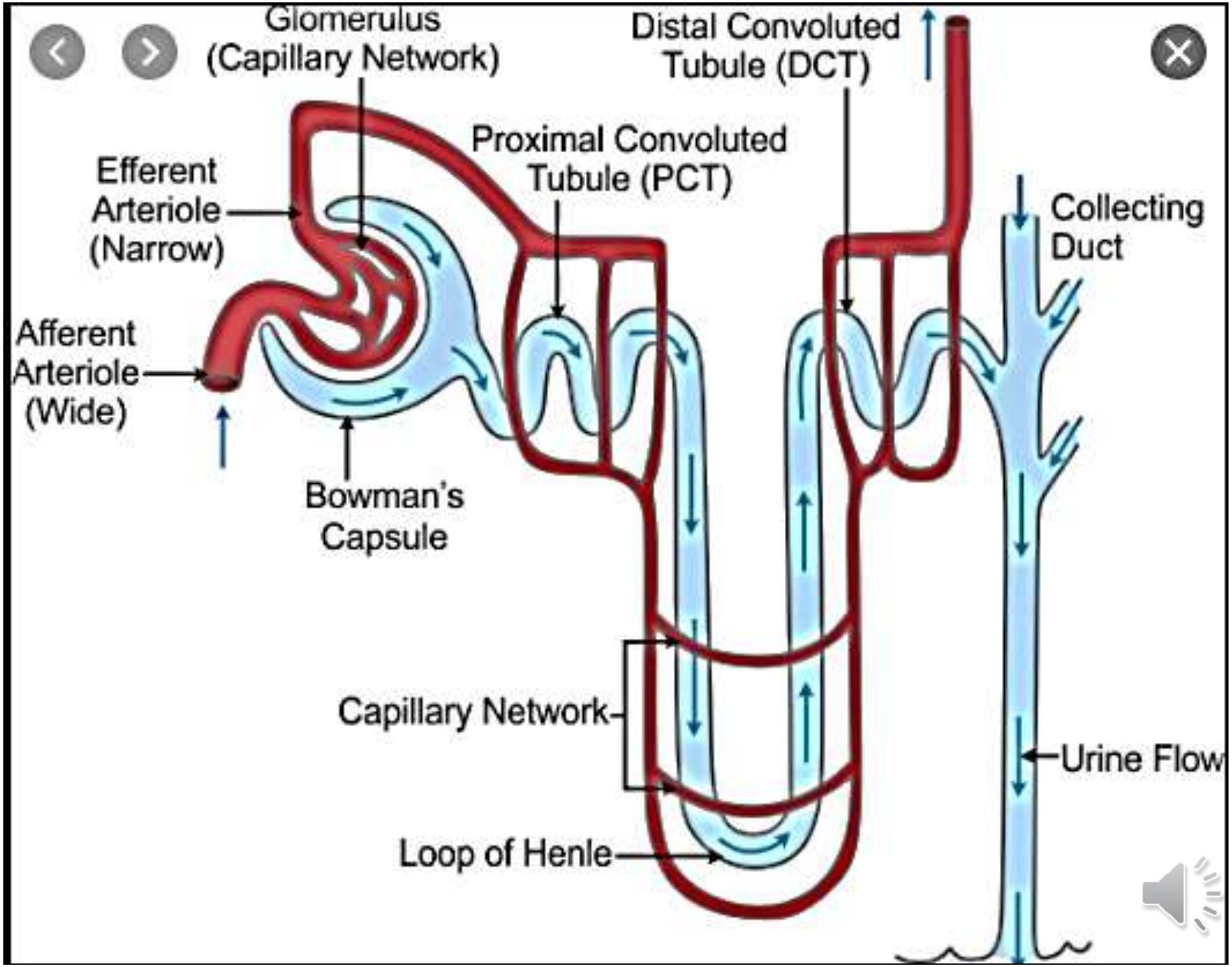
By

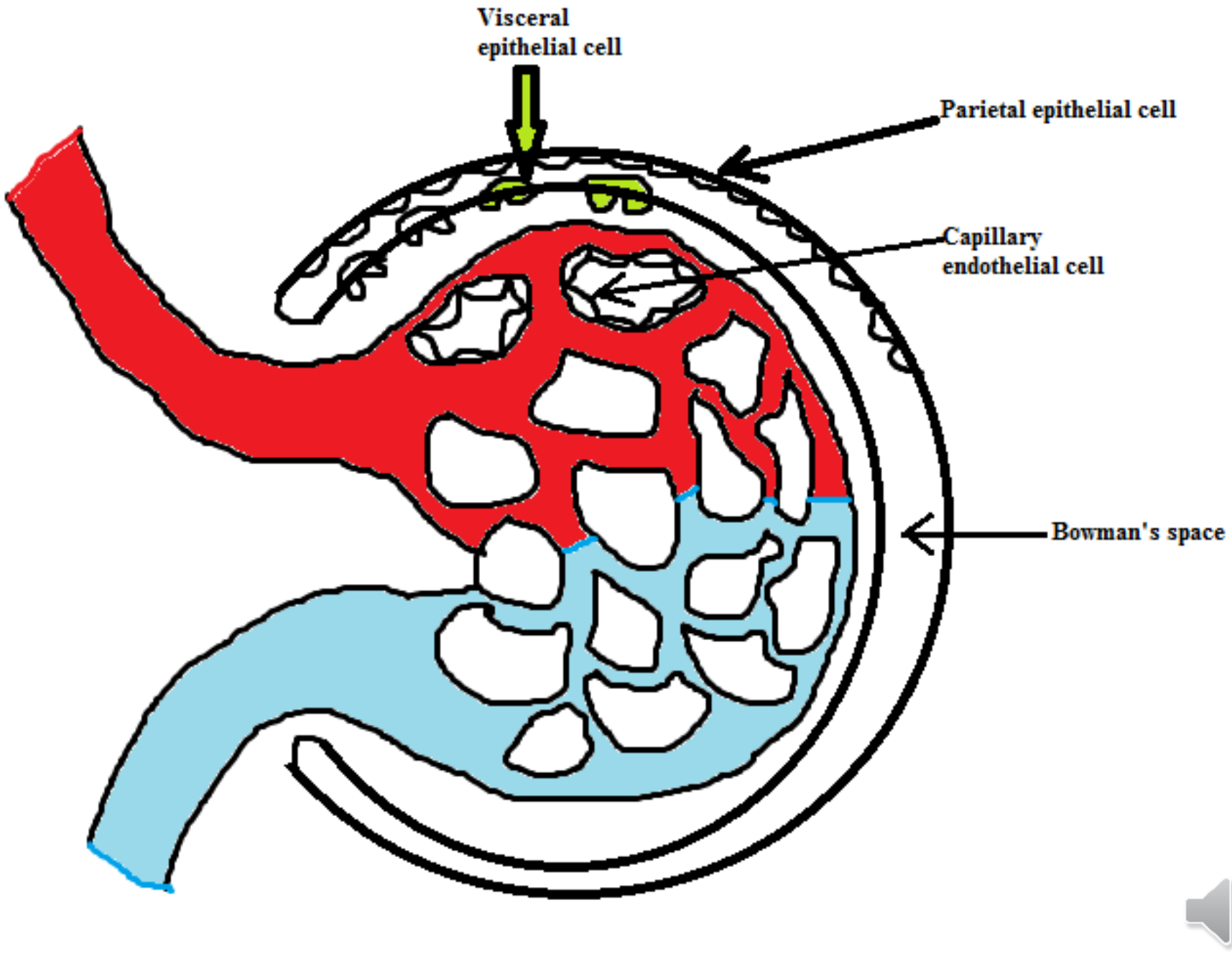
Dr/ Maisa Hashem

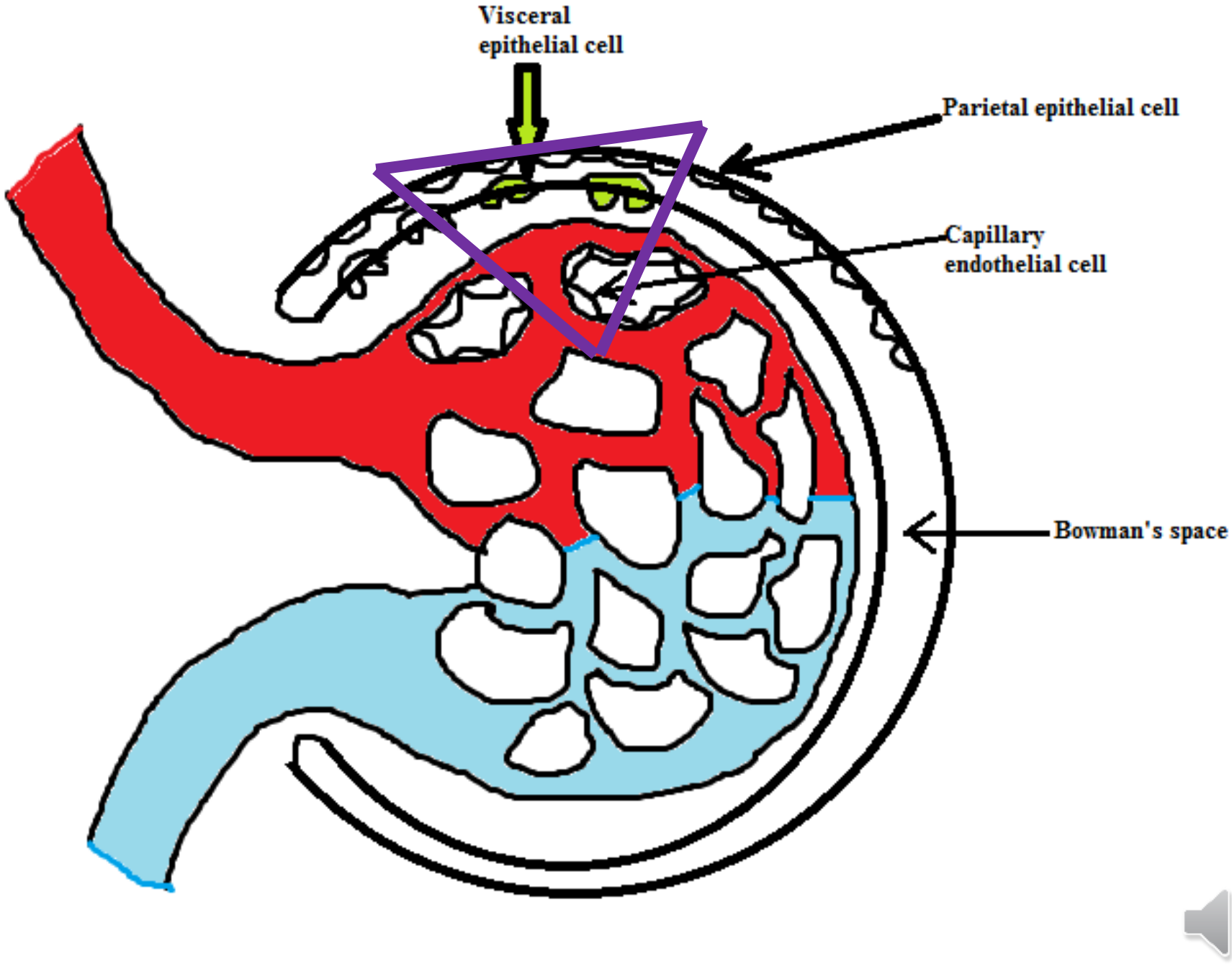
A Lecturer of Pathology

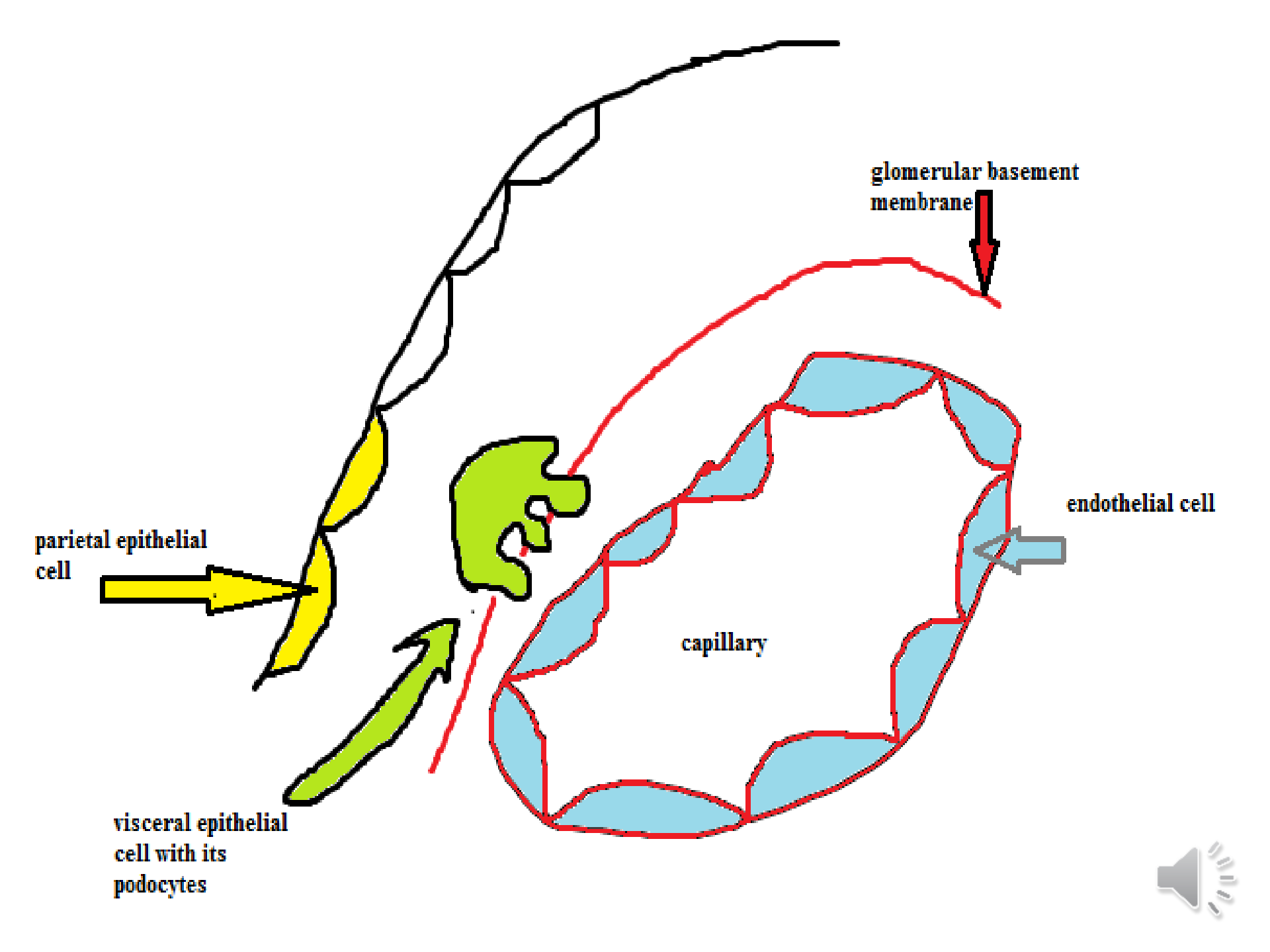
Aim of This Lecture

- Mentioning clinicopathological features of common types of Glomerulonephritis.


Glomerulus
(Capillary Network)
Distal Convoluted
Tubule (DCT)
Proximal Convoluted
Tubule (PCT)
Efferent
Arteriole
(Narrow)
Collecting
Duct
Afferent
Arteriole
(Wide)
Bowman's
Capsule
Capillary Network
Urine Flow
Loop of Henle


Visceral
epithelial cell
Parietal epithelial cell
Capillary
endothelial cell
Bowman's space


Visceral
epithelial cell
Parietal epithelial cell
Capillary
endothelial cell
Bowman's space


glomerular basement membrane
endothelial cell
parietal epithelial cell
capillary
visceral epithelial cell with its podocytes

Glomerular diseases

Glomerular diseases constitute some of major problems in nephrology. Chronic glomerulonephritis is one of the most common causes of chronic renal failure. Glomeruli may be injured by a variety of factors and in the course of number of systemic diseases. Here, the glomerular injury is called secondary glomerulonephritis. In other conditions, the kidney is the only or the main organ involved. The latter constitutes various types of primary glomerulonephritis.

Glomerulopathy is a glomerular disease that don't have a cellular inflammatory component.

Glomerular diseases (Glomerulonephritis/ G.N.)

I- Primary glomerulonephritis:

- Acute diffuse proliferative G.N.
- Rapidly progressive G.N.
- Membranous G.N.
- Membrano-proliferative G.N.
- Minimal change G.N.
- Chronic G.N.

II- Secondary glomerulonephritis

- Immunological as SLE.
- Vascular as hypertension.
- Metabolic as D.M.

Clinical Manifestations of Glomerulonephritis

- Asymptomatic proteinuria, asymptomatic haematuria.
- Nephrotic syndrome.
- Nephritic syndrome.
- Rapidly progressive glomerulonephritis causing acute renal failure.
- Chronic renal failure.

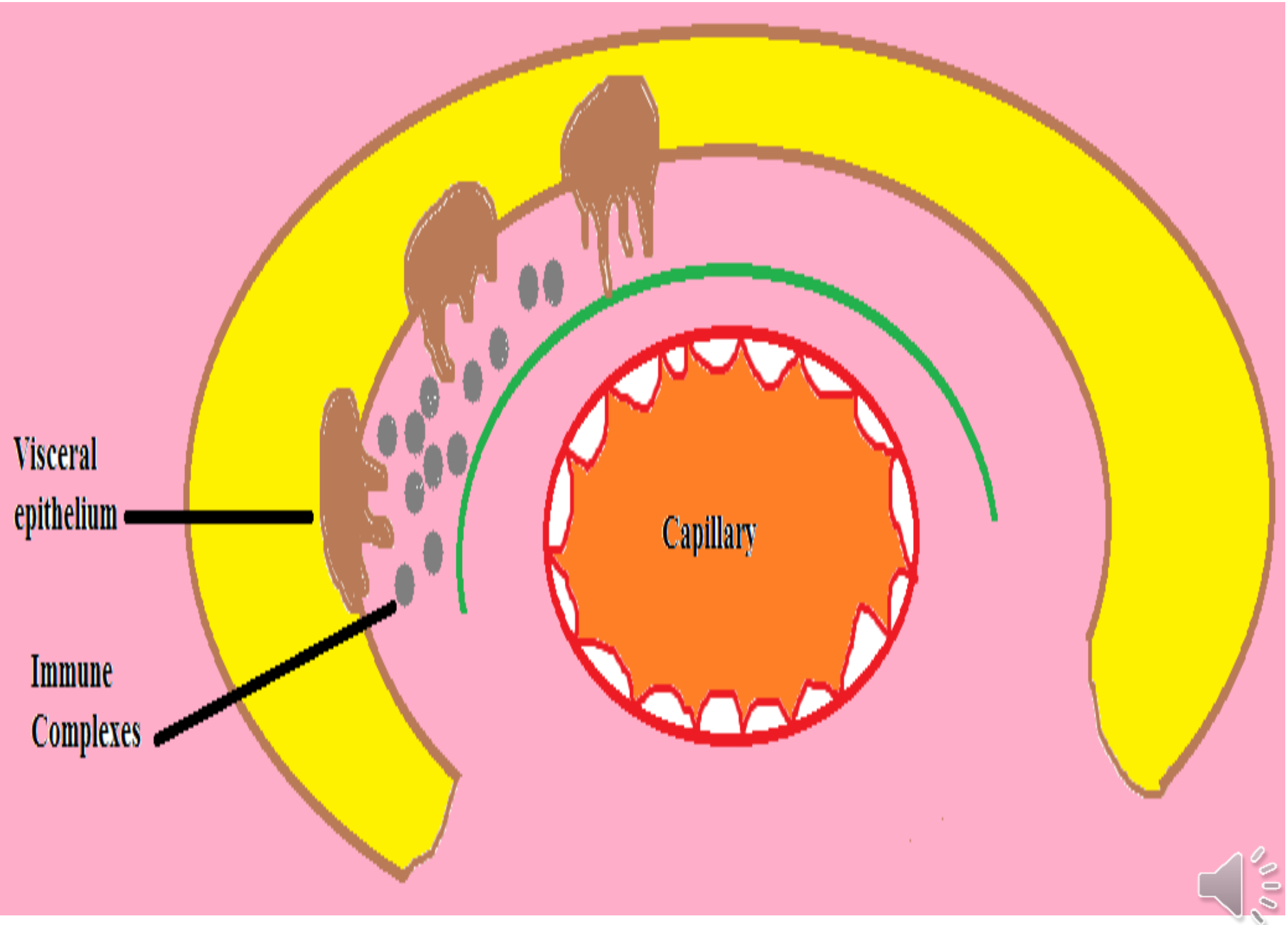
Primary Glomerulonephritis

- Acute diffuse proliferative G.N.
- Membranous G.N.
- Minimal change G.N.

Acute diffuse proliferative glomerulonephritis/ post streptococcal glomerulonephritis.

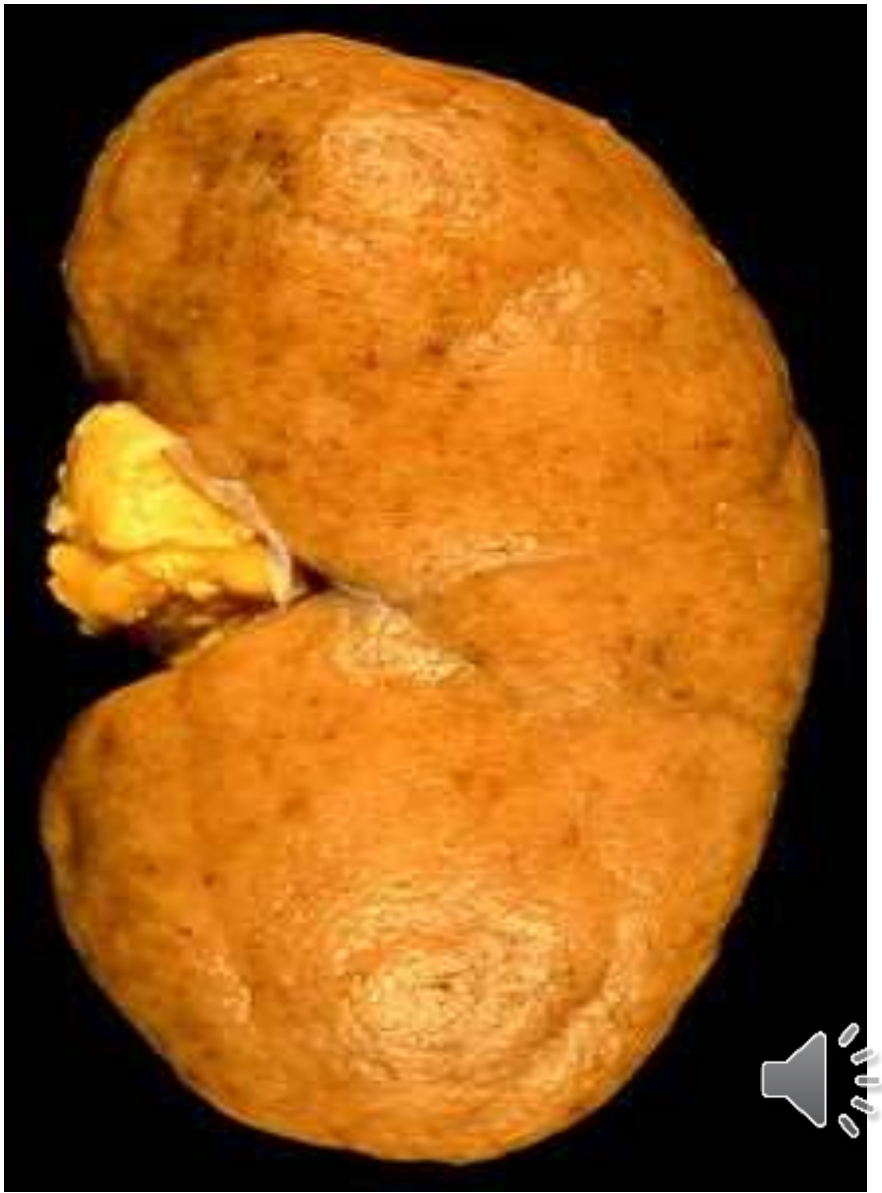
Aetiology:

- A rather common auto-immune disease in children and young adults. The disease may follow acute tonsillitis, pharyngitis or rarely skin infection.
- It is caused by strains of group A beta haemolytic streptococci after a latent period of 1-4 weeks.
- During the latent period; antibodies mainly IgG are formed against streptococcal antigens.. an immune-complex reaction occurs between streptococcal antigens and the antibodies in patient's serum. The immune-complexes deposit between the visceral epithelial cells and glomerular basement membrane followed by complement activation and inflammatory injury to the glomerular capillaries.


Visceral
epithelium
Immune
Complexes
Capillary

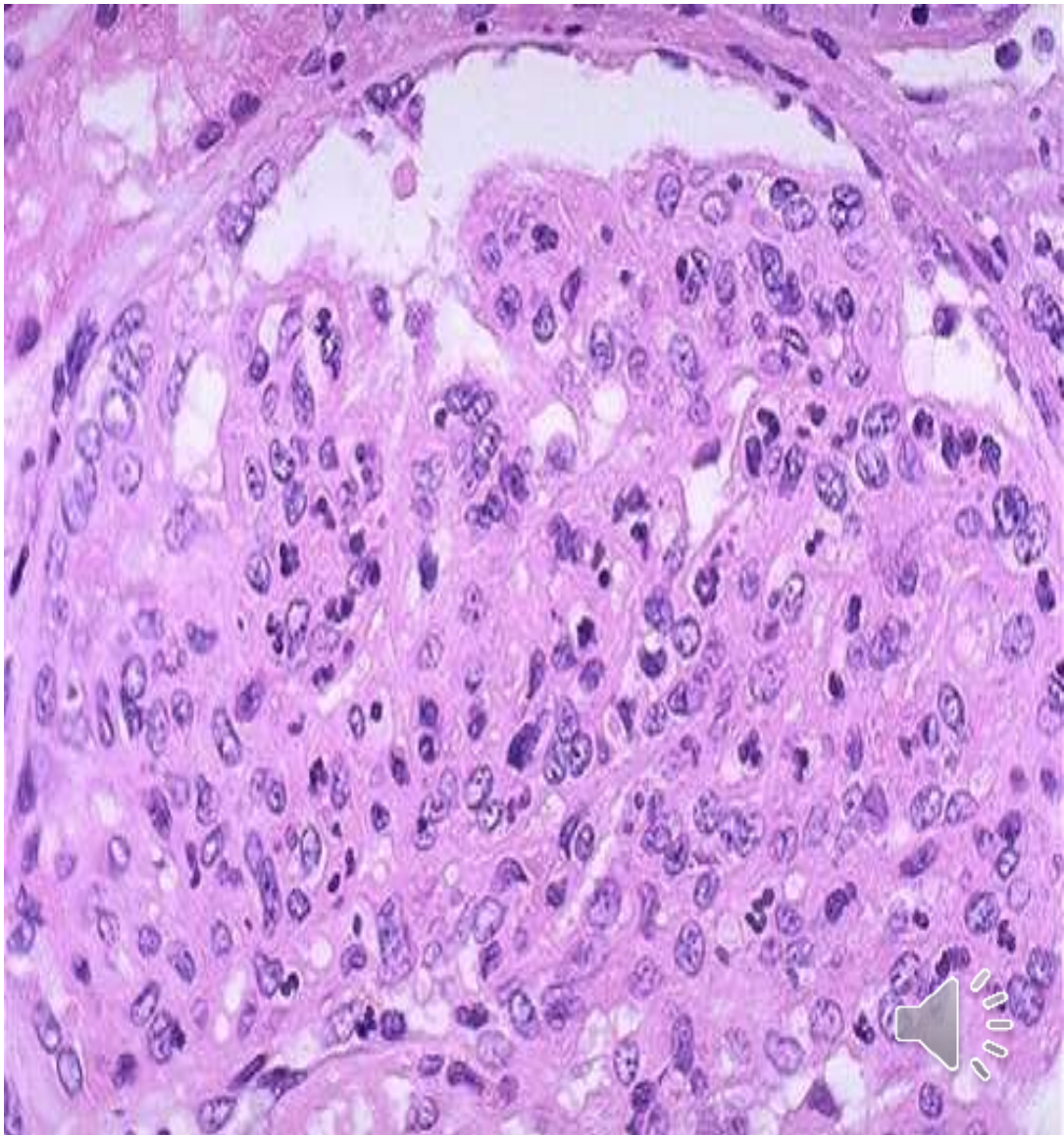
Gross picture

- Both kidneys are slightly enlarged, the capsule strips easily.
- The kidney surface is smooth. Cut section is pale due to oedema.
- In severe cases, there is red spots due to due to capsular haemorrhage.

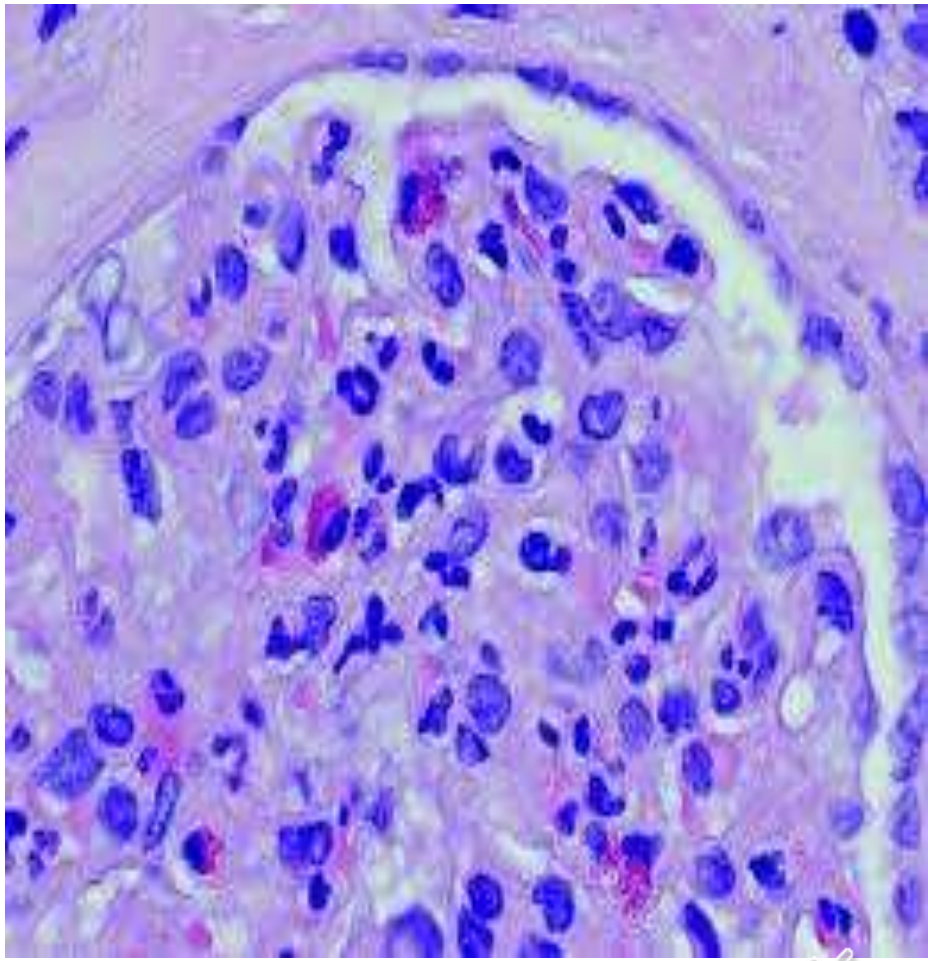


Microscopic picture

- All glomeruli show swollen, cellular, bloodless capillary tufts filling the capsular spaces. The increased cellularity is due to proliferation of capillary endothelium, glomerular epithelium, mesangial cells and infiltration by neutrophils and monocytes.
- The capsular space is narrowed and contains coagulated albumin, fibrin threads, neutrophils, RBCs and desquamated epithelial cells.



- The collecting tubules contain casts especially blood casts.
- The interstitial tissue is hyperaemic, oedematous and shows neutrophilic infiltrate.



General features

- Fever and malaise.
- **Nephritic oedema:** peri-orbital oedema which is marked in the morning. The oedema may involve the rest of the face and may become generalized.
- **Hypertension:** mild to moderate degree. Both hypertension and oedema are due to salt and water retention.
- **Blood changes:** elevation of blood urea and creatinine.

- **Urine changes:**
  - The urine is brownish and turbid (turbid or Coca-Cola like) due to presence of altered red cells.
  - Oliguria, moderate proteinuria and increased specific gravity up to 1035.
  - Microscopically; the sediment contains red cells, neutrophils, hyaline, granular, cellular and blood casts.
- ***Haematuria, proteinuria, hypertension, oedema, oliguria and uraemia constitute acute nephrtic syndrome.***

Course

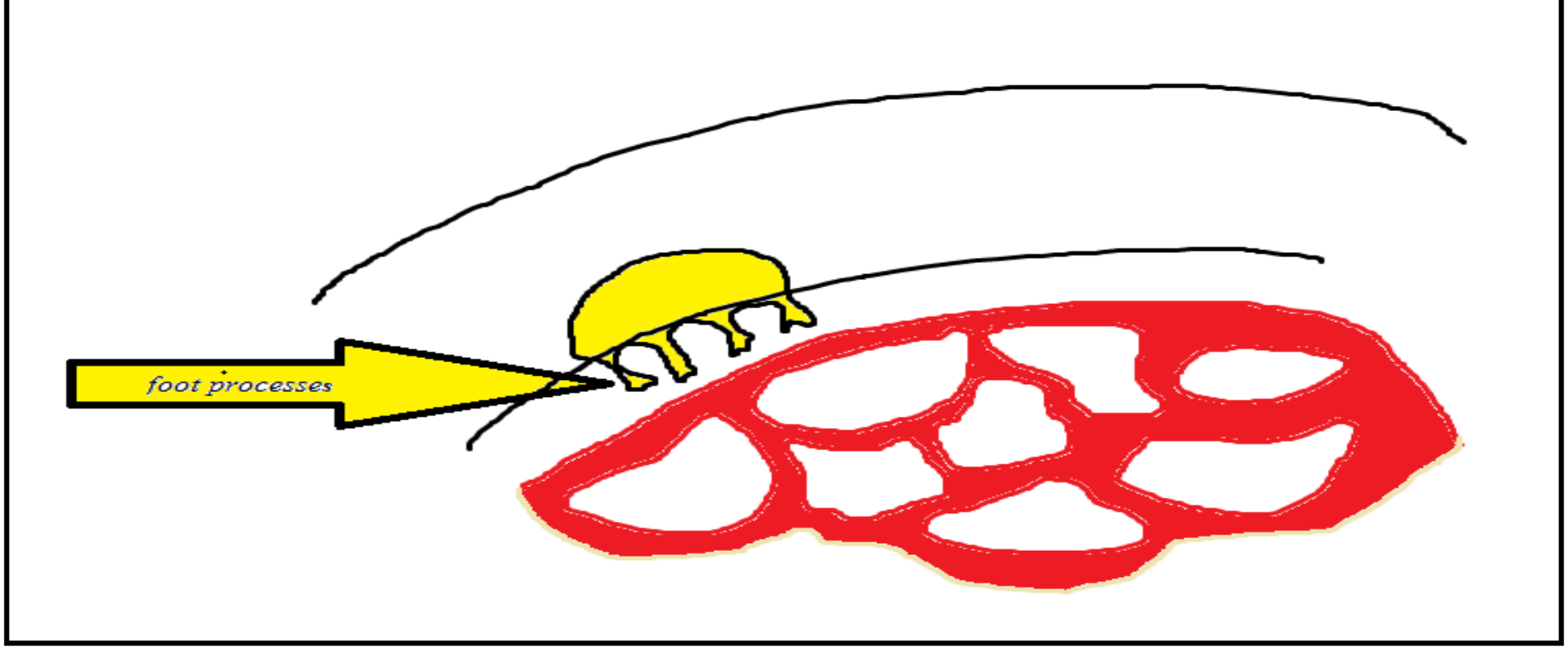
- Complete recovery in over 95% of children and 65% of adults.
- The disease may progress to rapidly progressive or chronic glomerulonephritis.
- Death from acute uraemia or acute heart failure caused by sever hypertension.

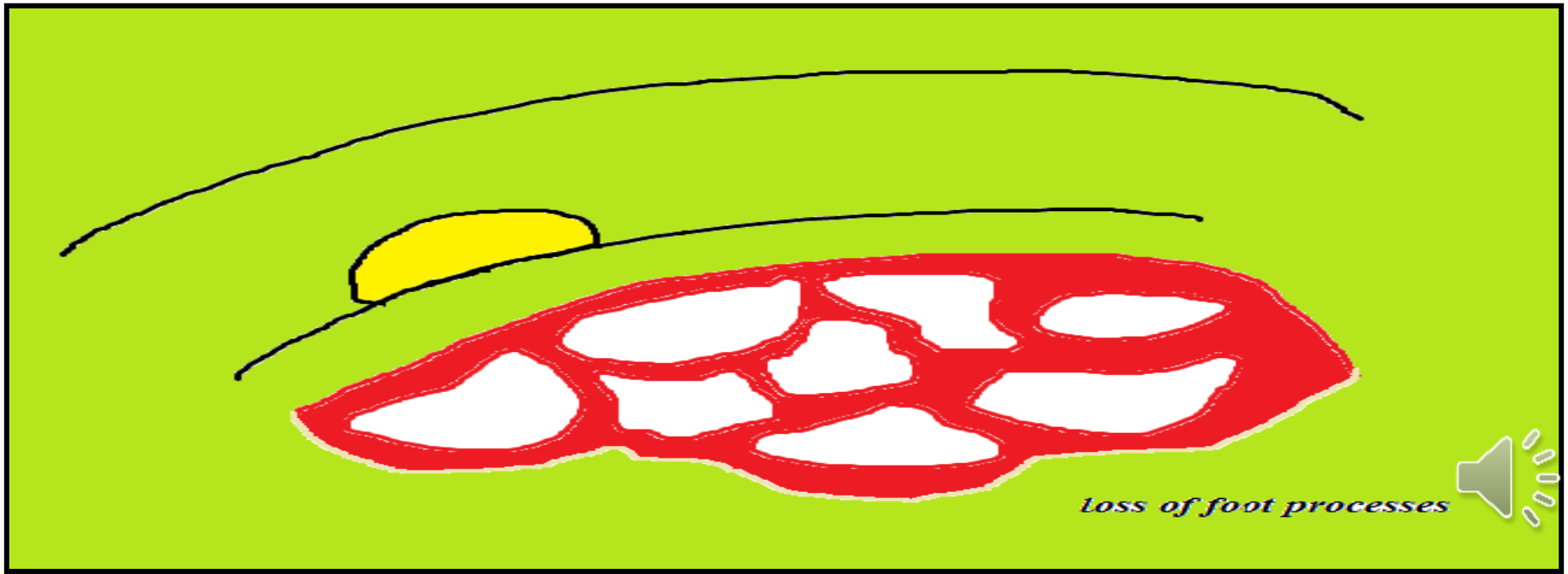
Minimal change glomerulonephritis/ lipoid disease/ light negative GN/ Foot process disease.

It is a disease of small children (2-6 years), but may occur in older children and adults. The exact aetiology is unknown but the disease may follow respiratory infection. This disease is the commonest cause of nephrotic syndrome in children.

Pathological features

- The kidneys don't show any gross abnormalities.
- **By light microscope;** the glomeruli show no pathological changes. However, the proximal convoluted tubules show lipid deposition in the lining cells. this lipid reflects glomerular leakage and reabsorption of lipoproteins.
- **By electron microscopy;** diffuse loss of foot processes of the visceral epithelial cells.
- Most cases cure with corticosteroid therapy.


foot processes


loss of foot processes

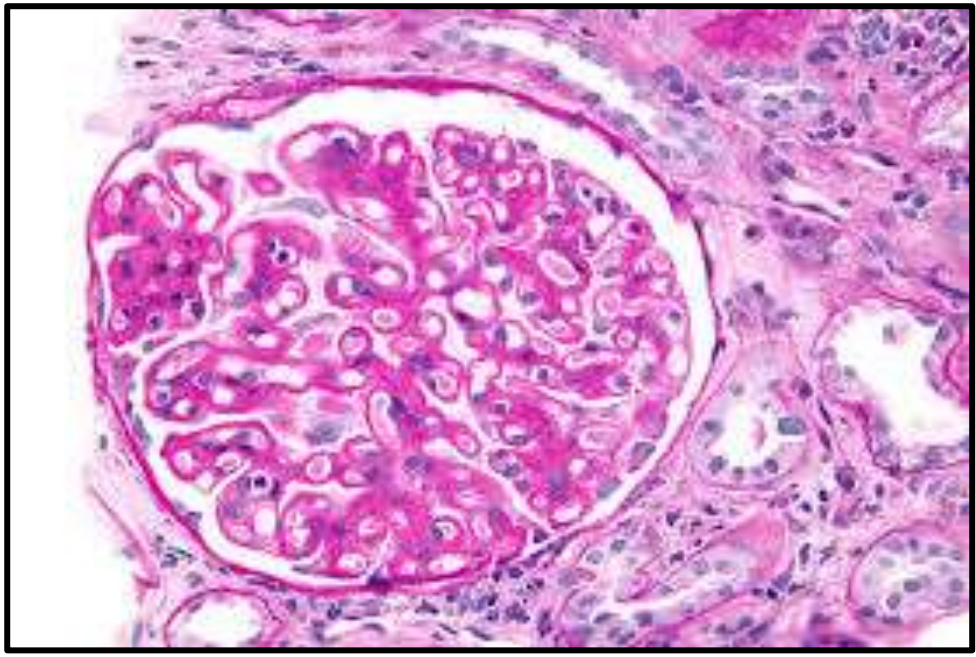
Membranous glomerulonephritis

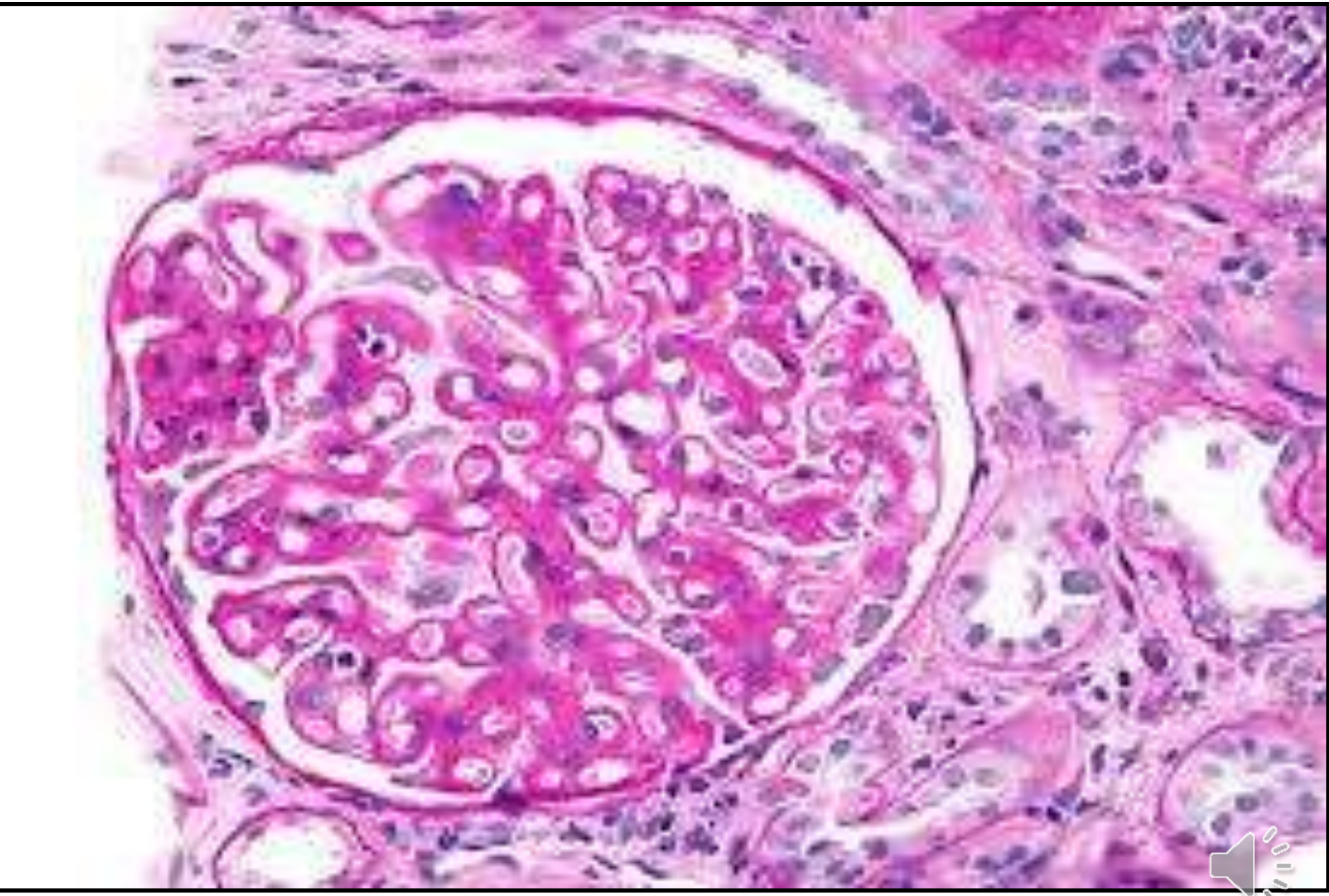
Aetiology:

- Idiopathic in 85% of cases.
- May occur in association with:
  - Systemic lupus erythematosis.
  - Infections as hepatitis B, bilharziasi, malaria, syphilis.
  - Metabolic diseases as diabetes mellitus.
  - Malignant epithelial tumors as carcinoma of lung, carcinoma of colon.

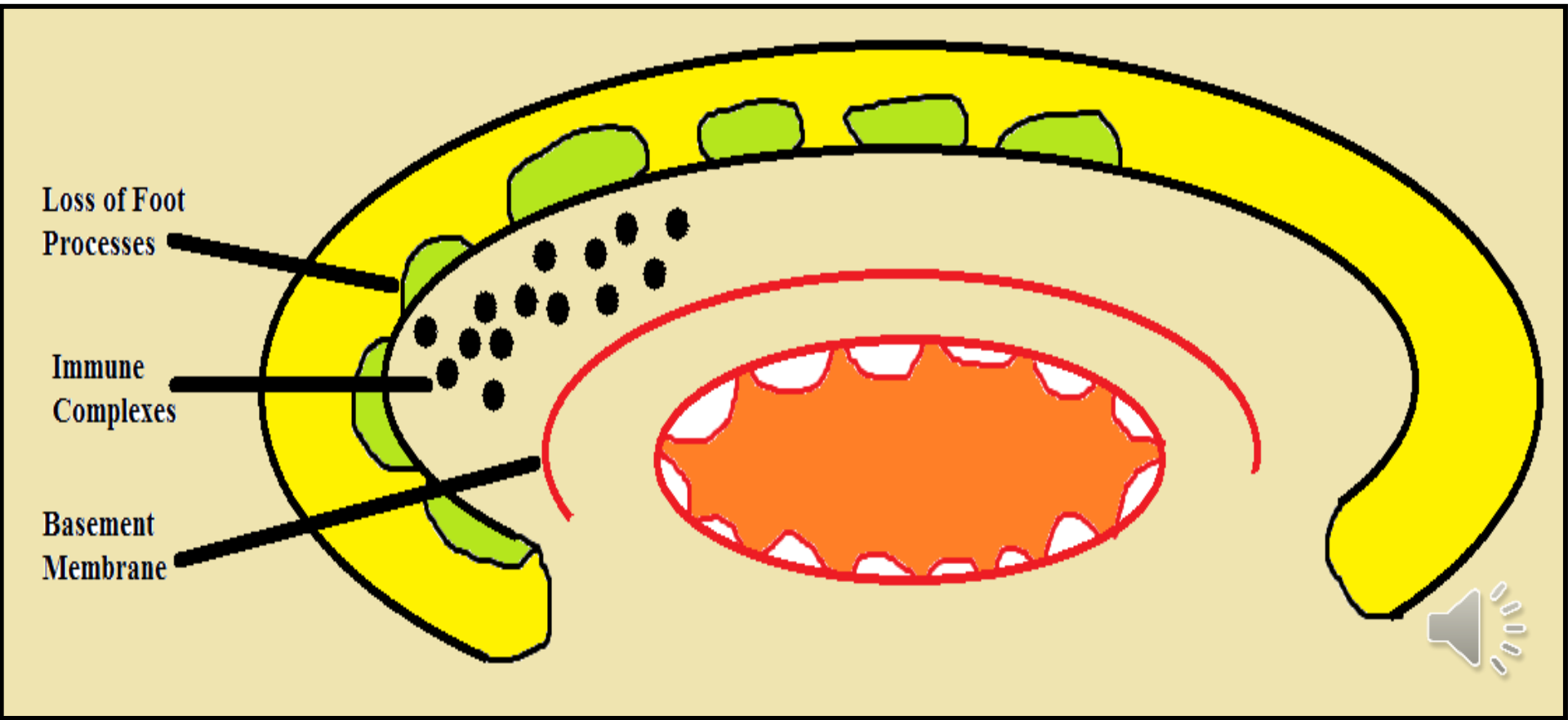
Pathological features

- The kidneys are enlarged and pale.
- **Light microscopy:** diffuse thickening of the glomerular basement membrane easily demonstrated by PAS stain. No cellular proliferation of the glomeruli.

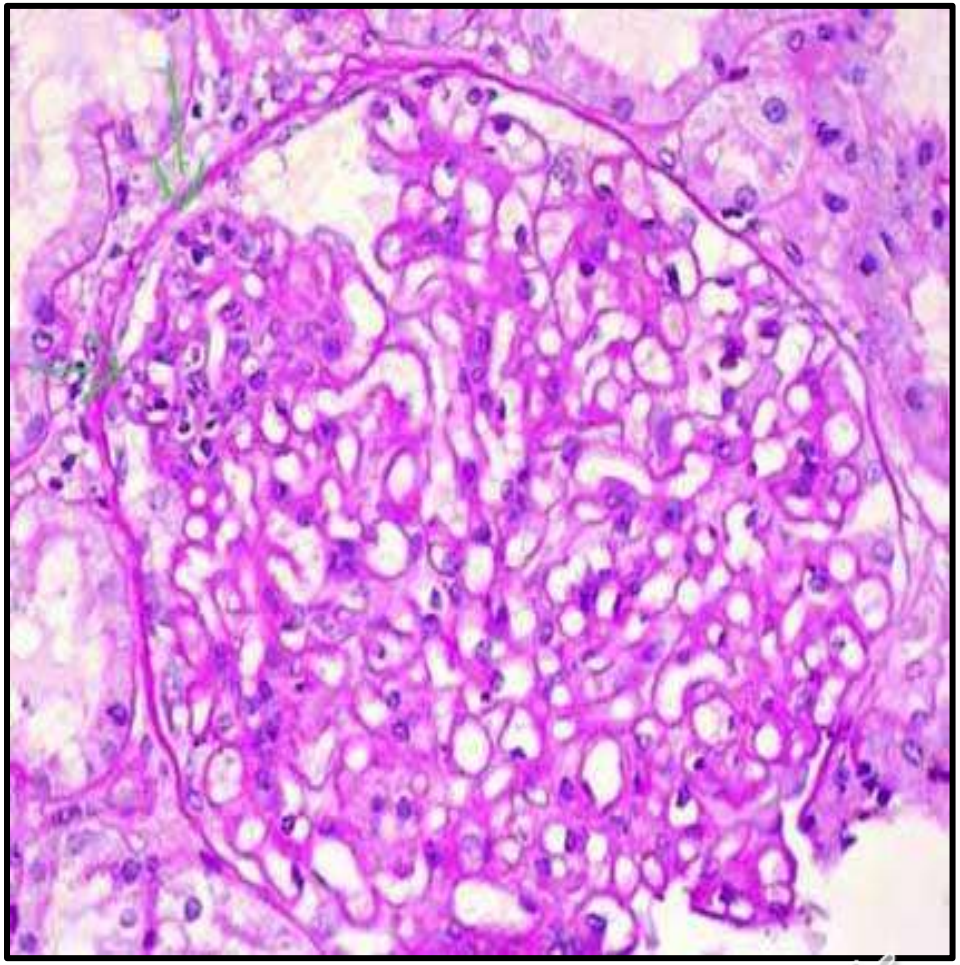




➢**By Electron microscopy:** the epithelial cells are swollen and loss their foot processes, dense IgG deposited between basement membrane and overlying epithelial cells.



- In advanced cases, glomerular sclerosis and hyalinosis occur, atrophy of tubules and interstitial fibrosis.
- Patients suffer from nephrotic syndrome. In advanced cases, renal insufficiency and hypertension develop.



Secondary Glomerulonephritis

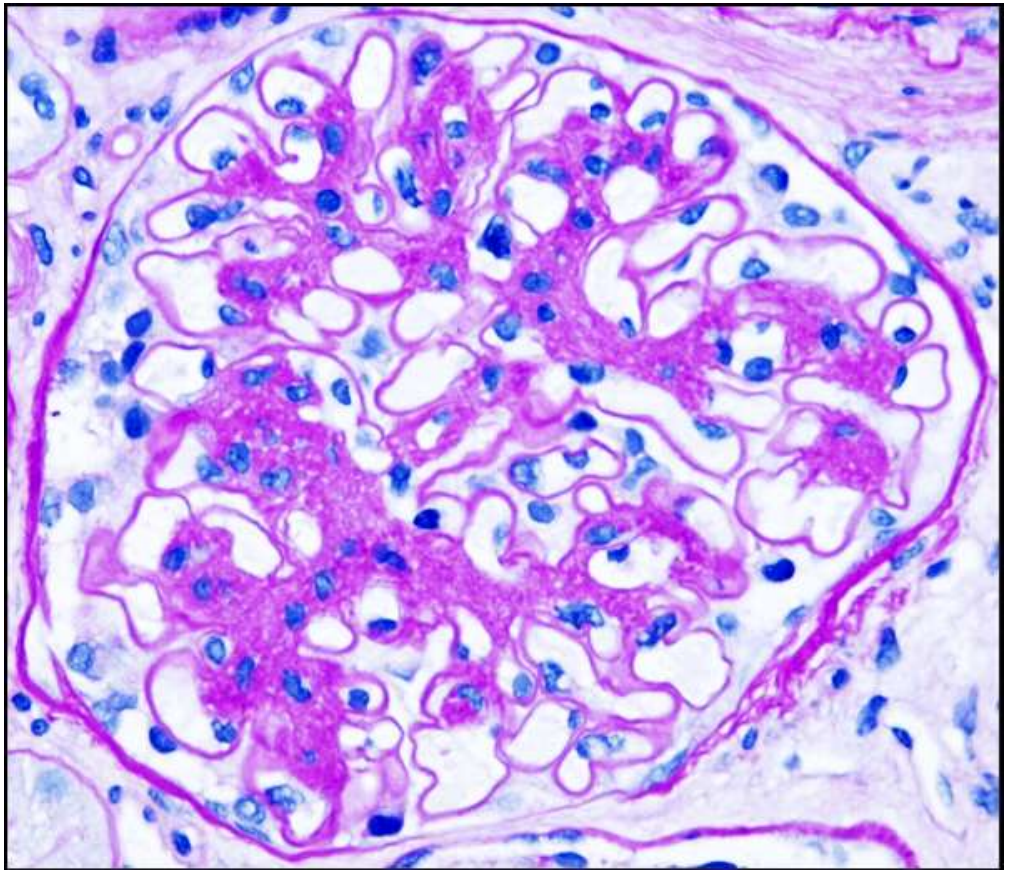
- **Diabetic Glomerulosclerosis**.

The characteristic renal changes in DM are best seen in the glomeruli and arterioles.

By light microscopy

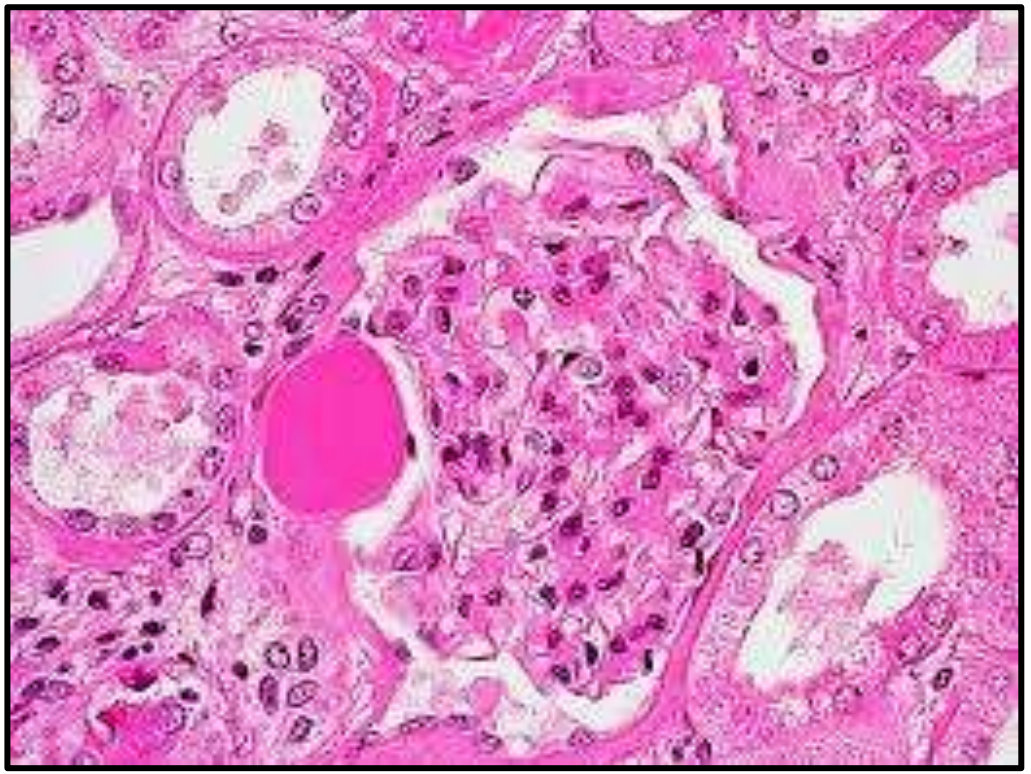
1) Diffuse diabetic glomerulosclerosis:

This is due to increase amount of mesangeal matrix in all msangeal regions in all glomeruli.



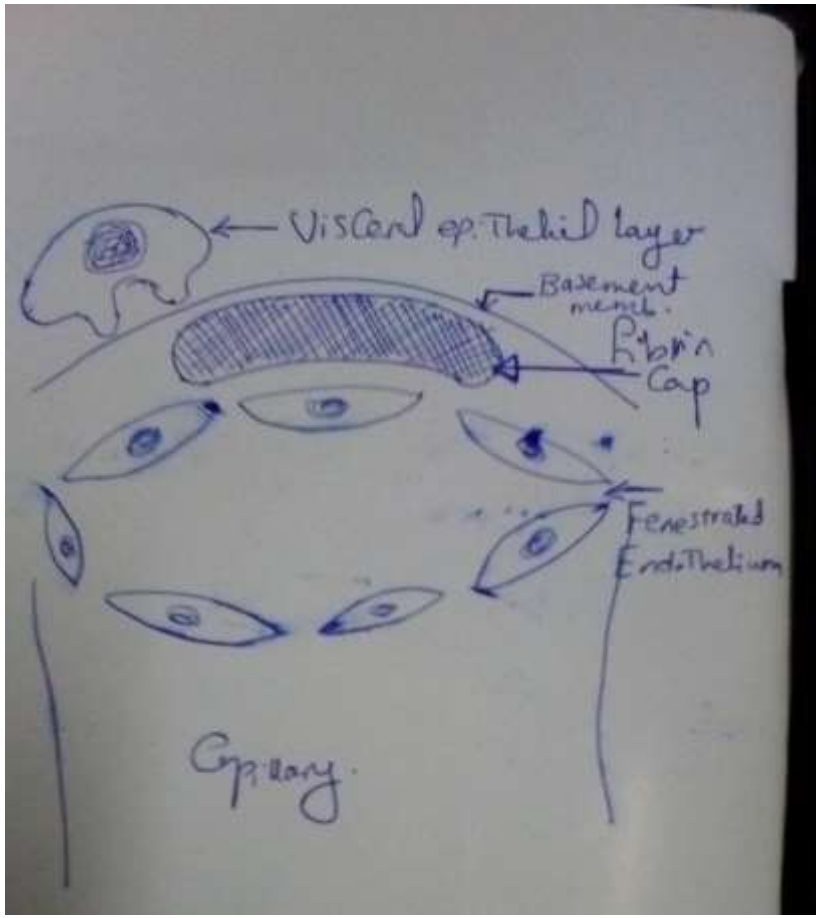
2) Nodular diabetic glomerulosclerosis (Kimmelstiel-Wilson nodule):

- This is a localized sclerotic acellular nodule located at one side of the glomerular capillary tuft.
- It is almost always superimposed on a pre-existing diffuse diabetic glomerulosclerosis.
- It appears as amorphous, eosinophilic material. It has the same staining properities as mesangeal matrix.

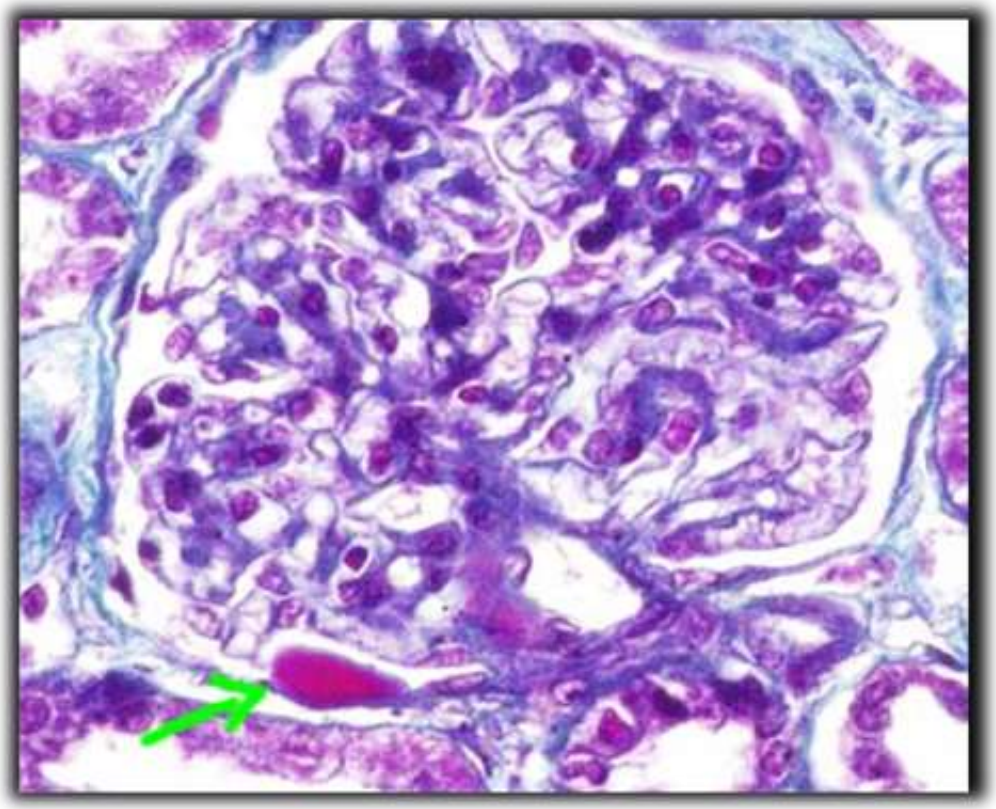


3) Insudative glomerular lesions (fibrin caps and capsular drops):

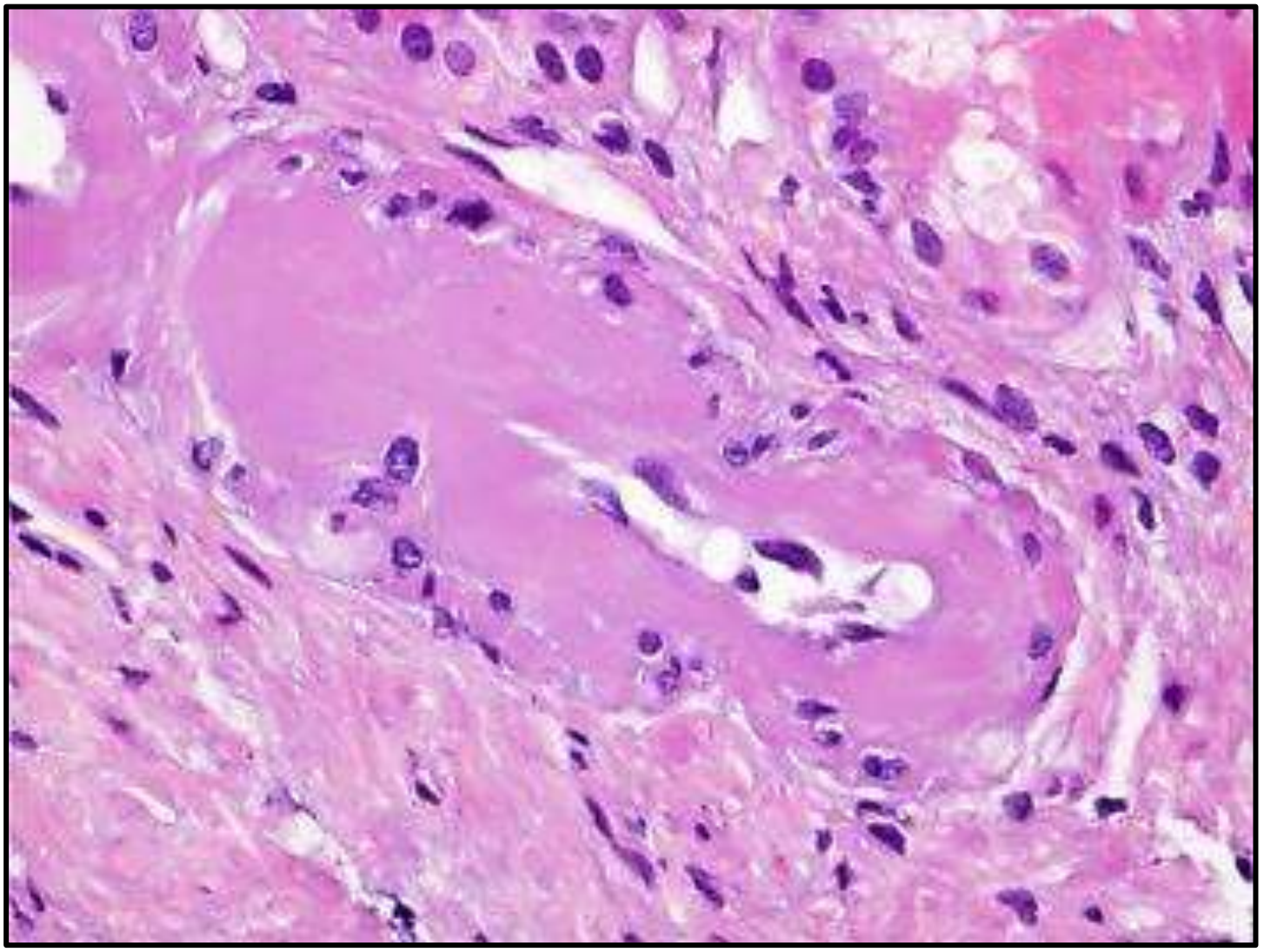
- These insudative lesions resulting from accumulation of plasma proteins in different regions in the glomeruli.
- If plasma proteins are accumulated between glomerular capillary endothelium and glomerular basement membrane; the resulting lesion is called **fibrin cap.**



- If plasma proteins are accumulated between parietal and visceral epithelial cells (in Bowman's space); the resulting lesion is called **capsular drops.**



4) Prominent arteriolar hyalinosis.




THANK YOU