



Acute post streptococcal glomerulonephritis

Acute post streptococcal glomerulonephritis

- It develops after streptococcal infection in children and young adults.
- It is most common disorder in developing countries.
- Age group: Most frequent in children between 6-10 years of age, but may develop in adults.

Etiology and Pathogenesis

- **Follows streptococcal infection** (hence post-streptococcal) rather than direct primary infection of the kidney by streptococci.
- **Primary streptococcal infection** usually involves the **pharynx** (pharyngitis) or the skin (impetigo/pyoderma).
- Infections of Skin are usually associated with scarlet fever, overcrowding and poor hygiene.
- **Certain strains of group A β -hemolytic streptococci are nephritogenic.** More than 90% are due to **types 12, 4, and 1.**
- Streptococcal antigenic component responsible for immune reaction in post-streptococcal glomerulonephritis is streptococcal pyogenic exotoxin B (SpeB) in most but not all cases.
- **latent period of 1 to 4 weeks** following primary streptococcal infection.
- Immune-complex **mediated** disease

Mechanism of Damage

- **Immune complexes** are formed in the **circulation** and gets **deposited within glomeruli**.
- Immune complexes **initiate inflammation** by **activating complement and** other humoral and **cellular mediators of inflammation**.
- The inflammatory mediators attract and **activate neutrophils and monocytes** and stimulate proliferation of mesangial and endothelial cell.
- Result is Hypercellular glomerulus.

Presentation

- Hematuria 1-3 weeks following group A streptococcal infection.
- Periorbitaloedema.

Morphology

Gross:

- The kidneys are enlarged and show pale capsular surface and cortex.

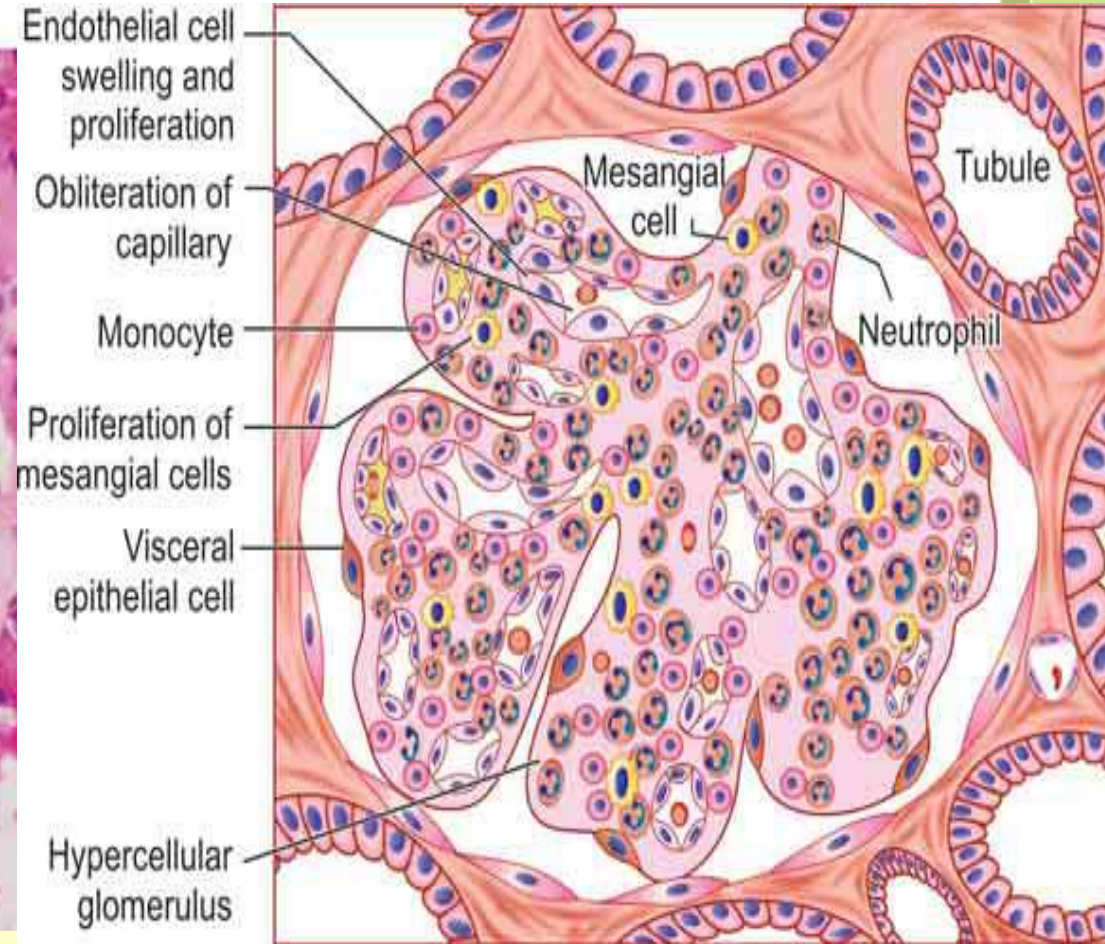
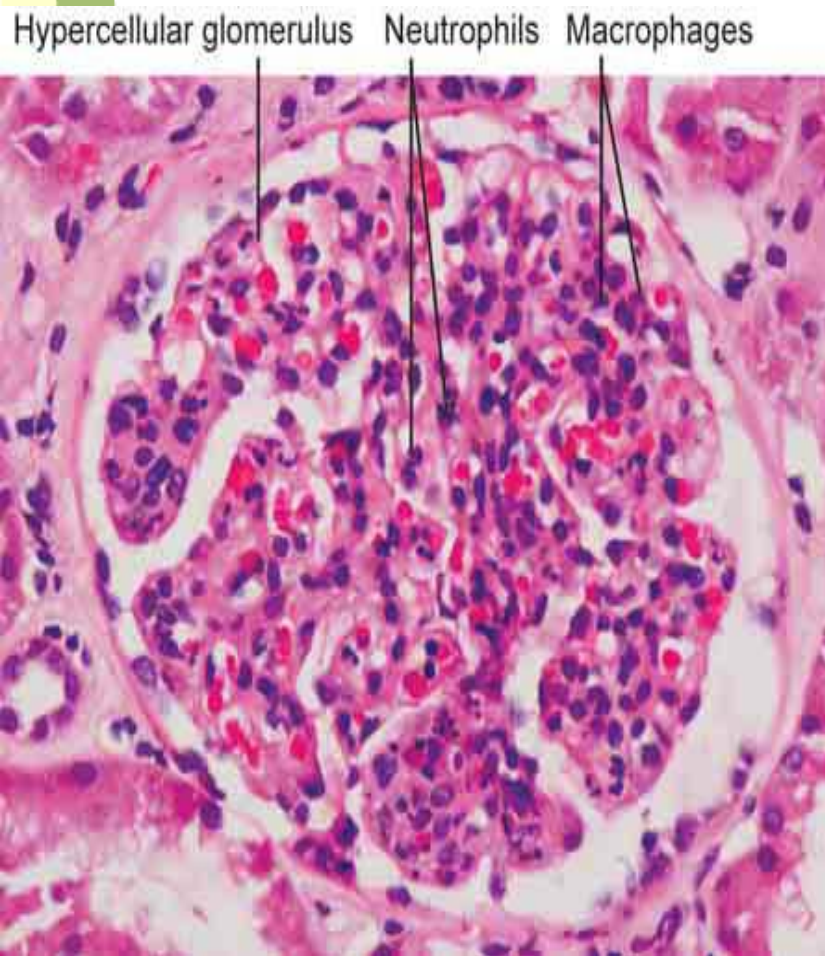
Microscopy:

Light Microscopy (LM)

1. Glomeruli:

- **Increased cellularity** (Proliferation of mesangial, endothelial and neutrophils).
 - The hypercellularity is due to:
 - Infiltration by leukocytes (neutrophils and monocytes).
 - Proliferation and swelling of endothelial and mesangial cells.
 - Rarely proliferation of parietal cells lining Bowman's capsule.
 - **Diffuse involvement.**
 - **Obliteration of glomerular capillary lumen: Due to swelling and proliferation of endothelial and mesangial cells + infiltration by leukocytes.**
2. **Tubules:** Contains **red cell casts** in the lumen and the tubular epithelial cells may show degenerative changes.
3. **Interstitium: Edema** and **inflammatory cell** infiltrate.
4. **Blood vessels:** Unremarkable.

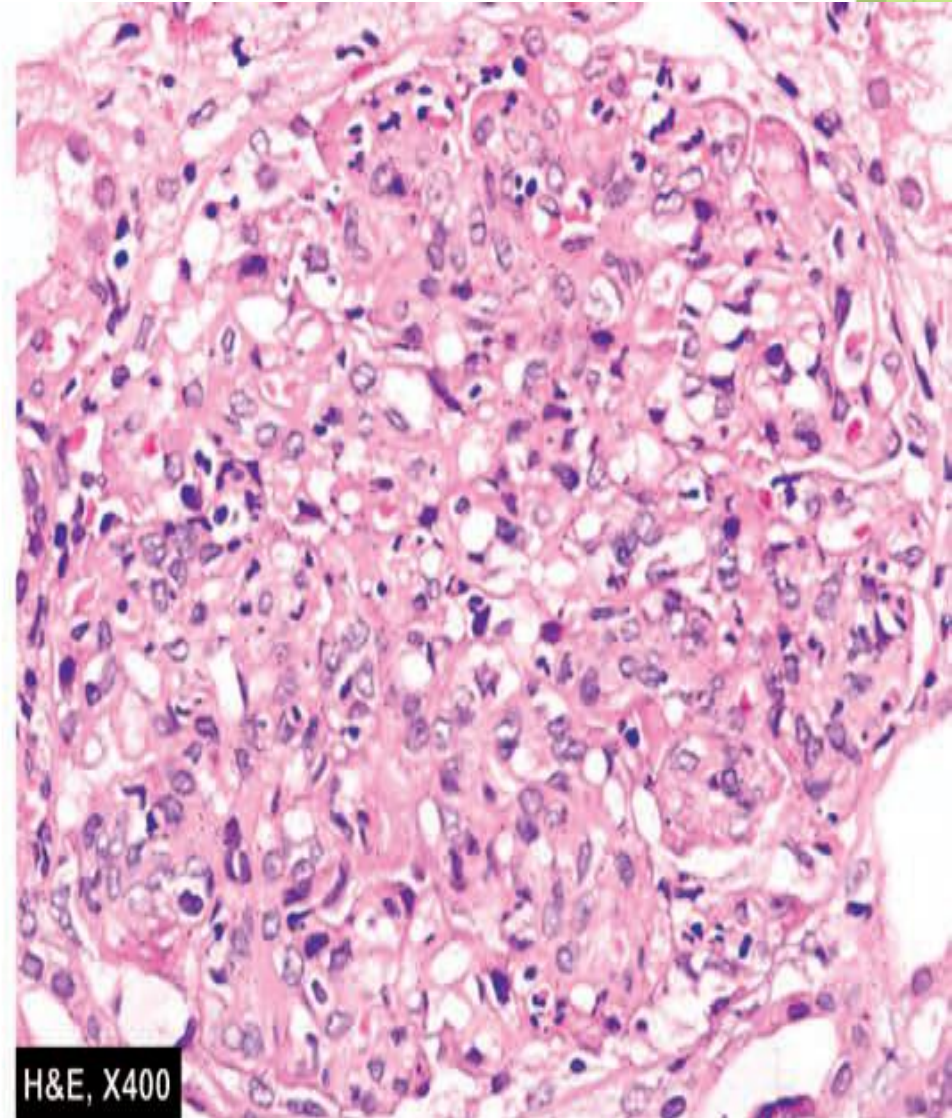
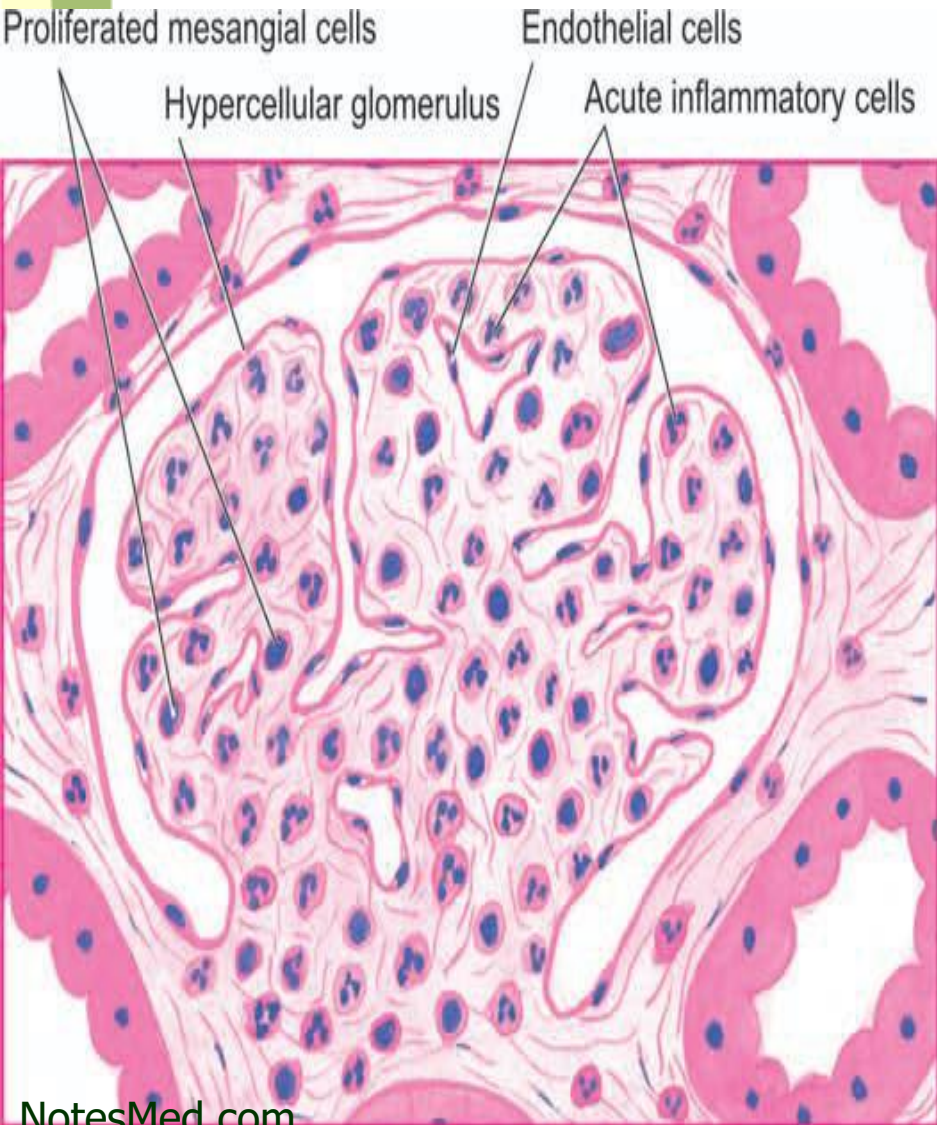
Microscopy



(A) Hematoxylin and Eosin stain
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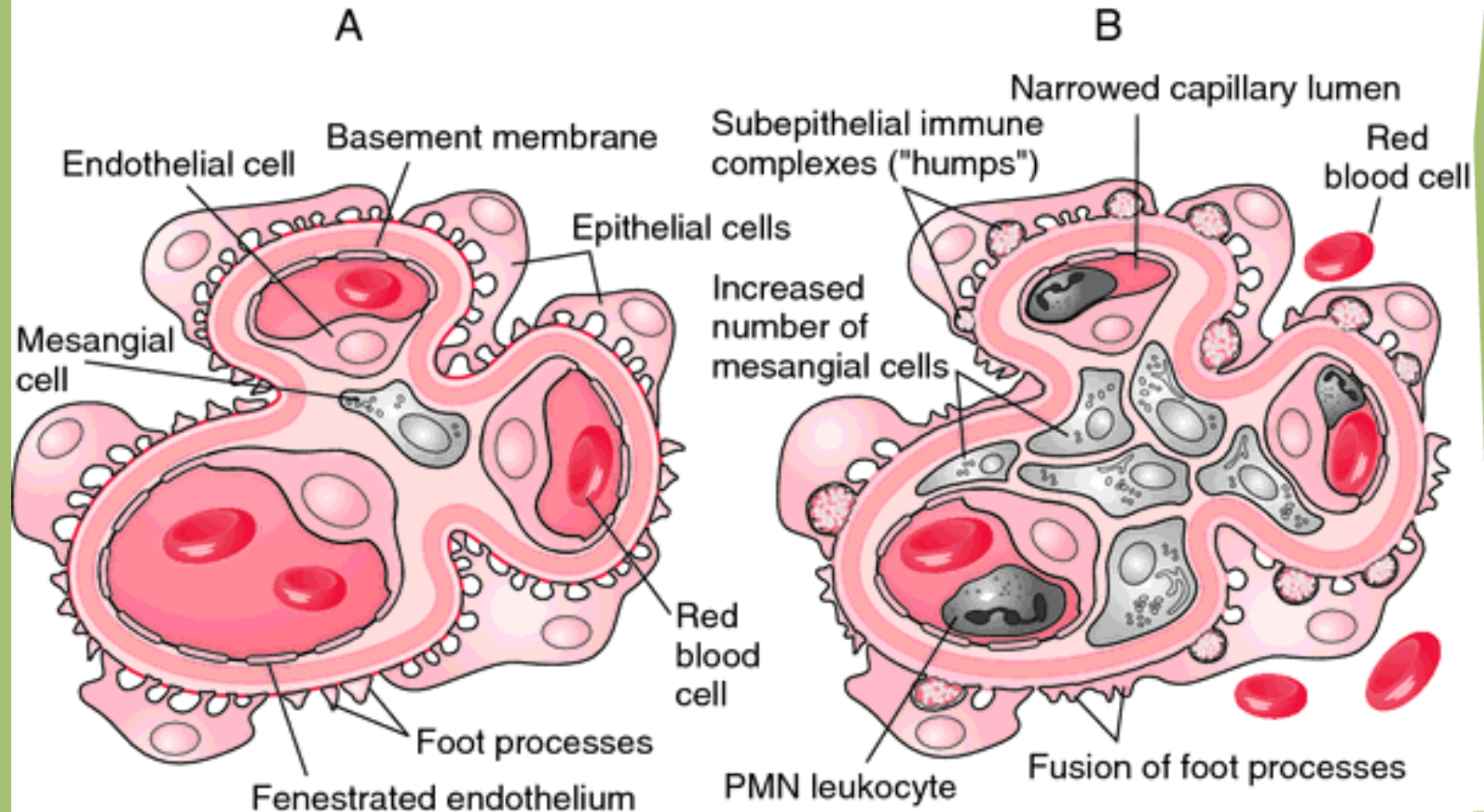
(B) diagrammatic

Microscopy



H&E, X400

Acute Post-infectious (Post-streptococcal) Glomerulonephritis



Immunofluorescence Microscopy:

- **Granular deposits of IgG, IgM, and C3** in the mesangium and along the GBM
→ **granular fluorescence.**

Electron microscopy:

- Sub-epithelial "**humps**" (**Immune complexes** deposit).
- **Sub-epithelial deposits of discrete, amorphous, electron-dense deposits** is a characteristic feature.

Clinical Course

Acute proliferative glomerulonephritis:

- Periorbital edema
- Mild to moderate hypertension
- Affected **child develops malaise, fever, nausea, oliguria, and hematuria (smoky or cola-colored urine) 1 to 2 weeks** after recovery from a **sore throat**.