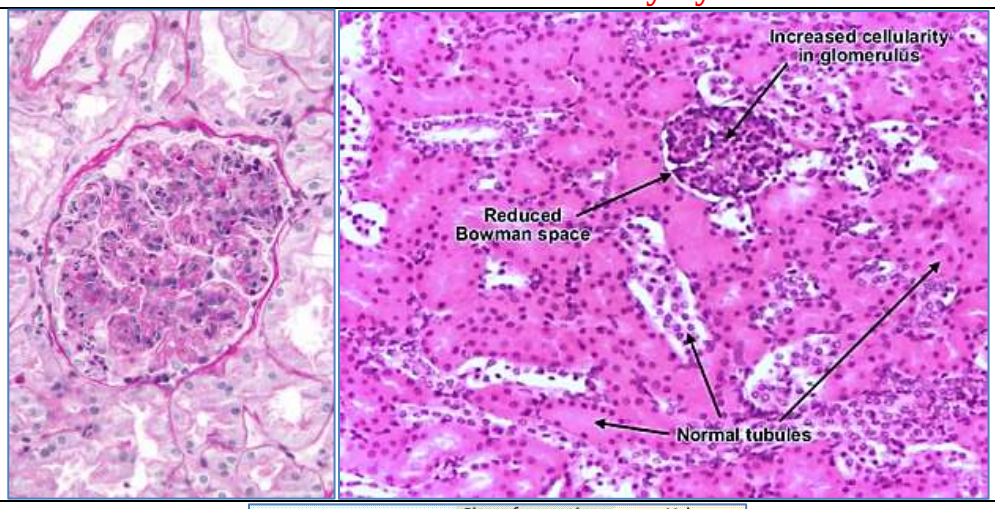
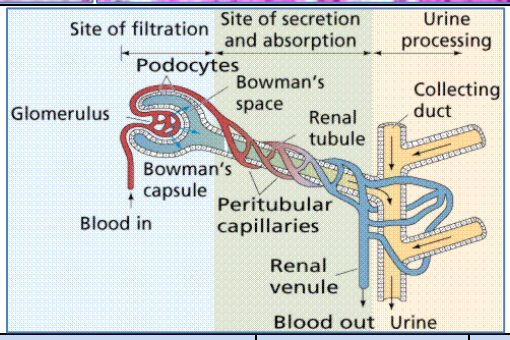
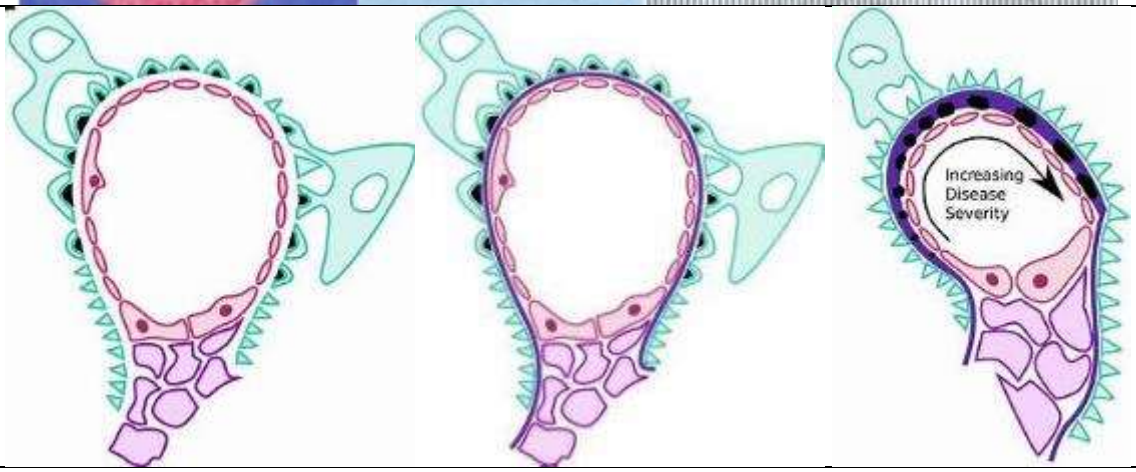
# XIX. Diseases of the Urinary System



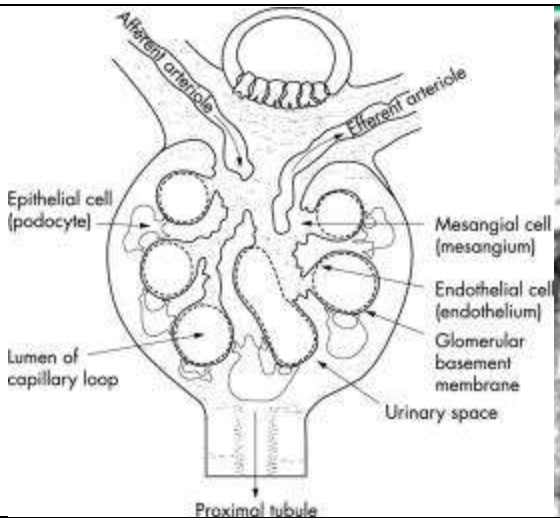

| **Acute Diffuse Glomerulonephritis** | **IV- 1. 3191** | | |
|---|---|---|---|
| ***Kidney:*** | ***Size:*** | Normal (or slightly enlarged) | |
| | ***Capsule:*** | Tense (if sectioned →parenchyma bulges)<br>Strips off easily | |
| ***Subcapsular surface:*** | | Smooth<br>Pale yellowish-grey | |
| | ***Consistence:*** | Fleshy-soft | |
| | ***Cut surface:*** | Oedematous – moist appearance | |
| | ***Cortex:*** | Normal pattern<br>Increased in width (thickness)<br>Pinkish-yellowish-grey<br>Blurring of markings<br>Petechial hemorrhages (tiny red dots) | |
| | ***Medulla:*** | Congested<br>Reddish-brown<br>Well-demarcated from cortex | |
| ***Blood vessels:*** | | No gross change | |
| ***Renal pelvis (and calyces):*** | | No gross change | |

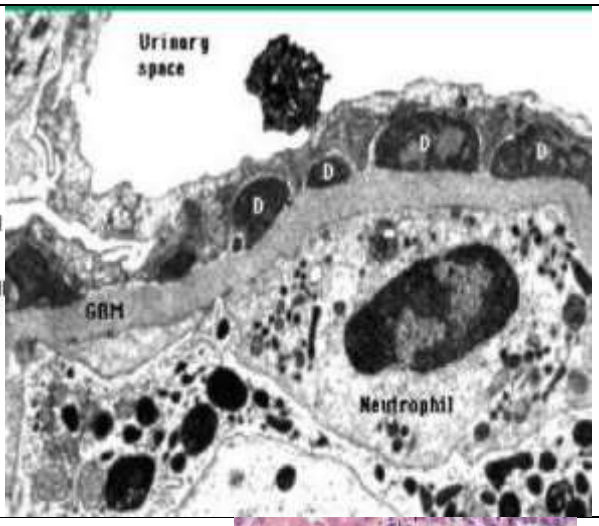


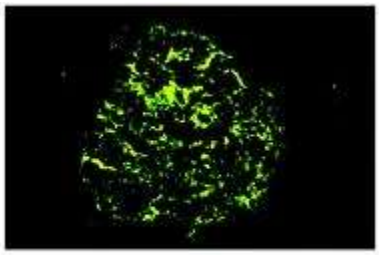



***N.B.I:***

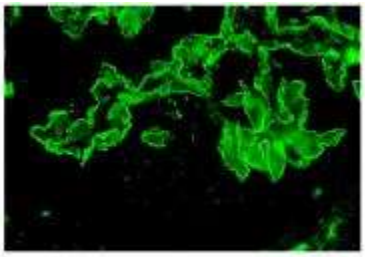
- *Usually the gross appearance of acute diffuse glomerulonephritis is not very characteristic.*
- ***Early or/and mild cases*** → *no gross changes.*
- ***In general****, both kidneys are affected and become swollen and pale.*
- The capsule is tense and the surface under the capsule is smooth, grey and with tiny red (haemorrhagic) dots; or reddish-brownish with pale areas.
- The cut surface has a moist appearance.
- Glomeruli can be seen by a hand-lens as small grey dots which appear bloodless; the reddish petechiae which may appear over the cortex are due to minute haemorrhages in the Bowman's capsules from rupture of glomerular capillaries.
- MR
- ***Typical cases*** → large bloodless (hypercellular) glomeruli (stage of proliferation).
  ***Atypical cases*** → atypical picture chiefly microscopic (exudative; proliferative; haemorrhagic; necrotizing etc.).



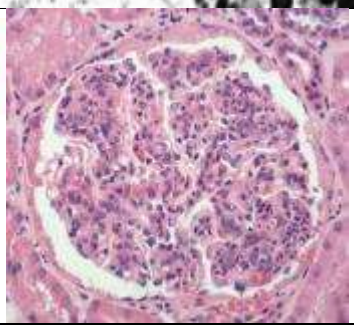



2a: Granular (lumpy, bumpy) deposition of C3 such in Poststreptococcal Glomerulnephritis



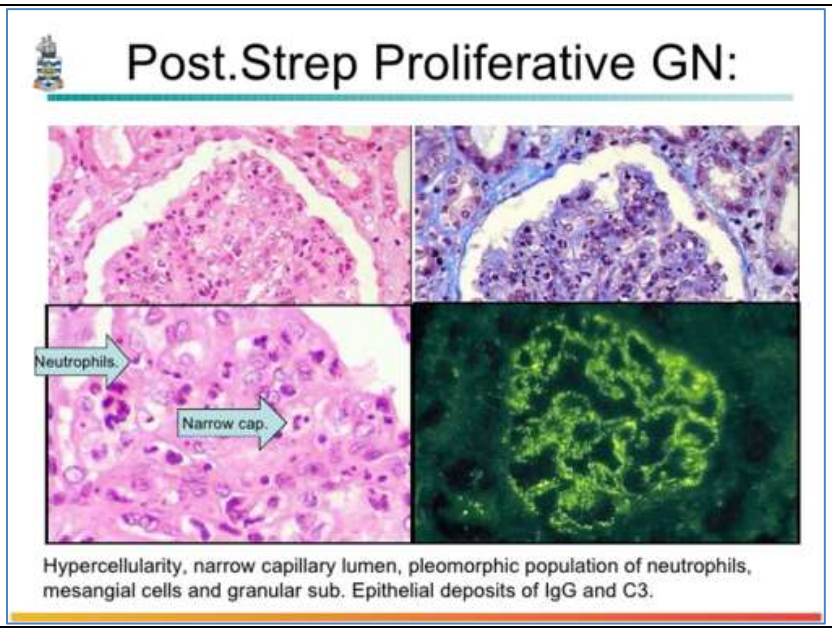
2b: Linear, ribbon-like deposition of IgG such in Goodpasture's Syndrome



**N.B.2:**

- Glomerulonephritis (originally-described by "Bright" as a diffuse inflammatory condition affecting the glomeruli of both kidneys).
- Can occur at all ages (particularly at childhood and senility) and
- In both sexes (more in males).

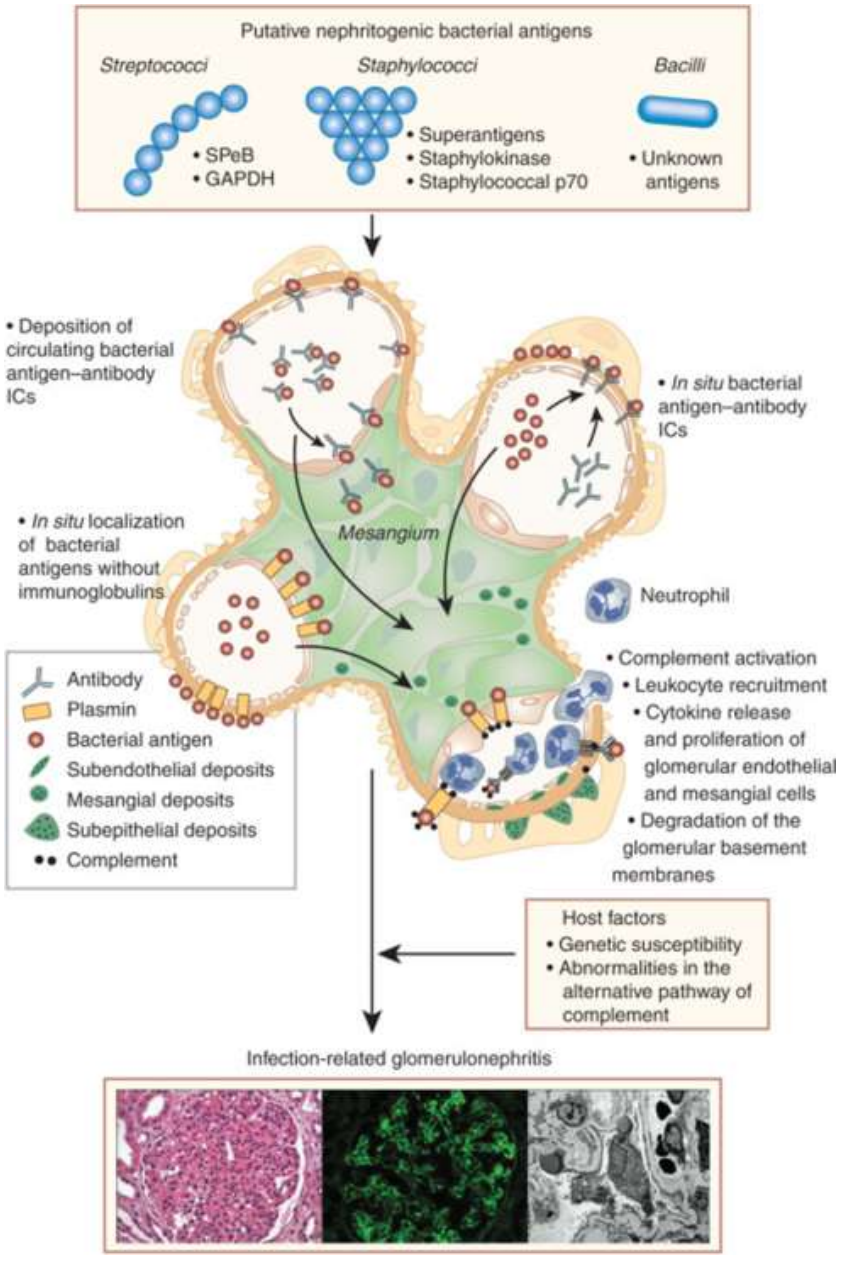
- It usually (though not always) follows (within 5 weeks) a previous bacterial infection (often tonsillitis; scarlet fever; upper respiratory infections; rheumatic fever etc...) with **streptococcus haemolyticus** (specific varieties of **group A,** beta hemolytic, which are characterized or/and followed by high levels of **anti-streptolysins** and, an **antigen-antibody immunological reaction** → hypersensitivity state → Immune complex deposition (antigen-antibody-complement complex)in the basement membrane of vessels of glomeruli →***post streptococcal acute diffuse proliferative glomerular inflammation***).




- Therefore, the forms of glomerulonephritis (affecting primarily the glomeruli), depend on hypersensitivity, infection and embolization.
- *The diffuse glomerulonephritis, from the pathological point of view, and to a much less extent clinically, may be subdivided (at least for descriptive purposes) into :*
  ***(1) Acute stage (or phase),***
  ***(2) Subacute stage (or phase) and***
  ***(3) Chronic stage (or phase).***

**N.B.3:**

- Clinically, chills, fever, anorexia, weakness, flank-pains, haematuria (smoky urine), oliguria and possibly pyuria, albuminuria and casts.
- Later on, moderate elevation of blood pressure followed by hypertensive encephalopathy and retinopathy.
- The fatality is about 5% of cases only.


Putative nephritogenic bacterial antigens
Streptococci
Staphylococci
Bacilli
• SPeB
• GAPDH
• Superantigens
• Staphylokinase
• Staphylococcal p70
• Unknown antigens
• Deposition of circulating bacterial antigen–antibody ICs
• In situ bacterial antigen–antibody ICs
• In situ localization of bacterial antigens without immunoglobulins
Mesangium
Neutrophil
Antibody
Plasmin
Bacterial antigen
Subendothelial deposits
Mesangial deposits
Subepithelial deposits
Complement
• Complement activation
• Leukocyte recruitment
• Cytokine release and proliferation of glomerular endothelial and mesangial cells
• Degradation of the glomerular basement membranes
Host factors
• Genetic susceptibility
• Abnormalities in the alternative pathway of complement
Infection-related glomerulonephritis