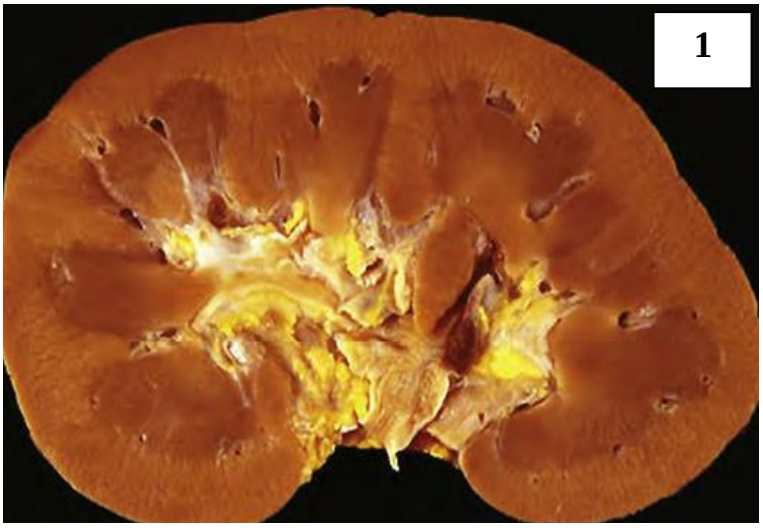
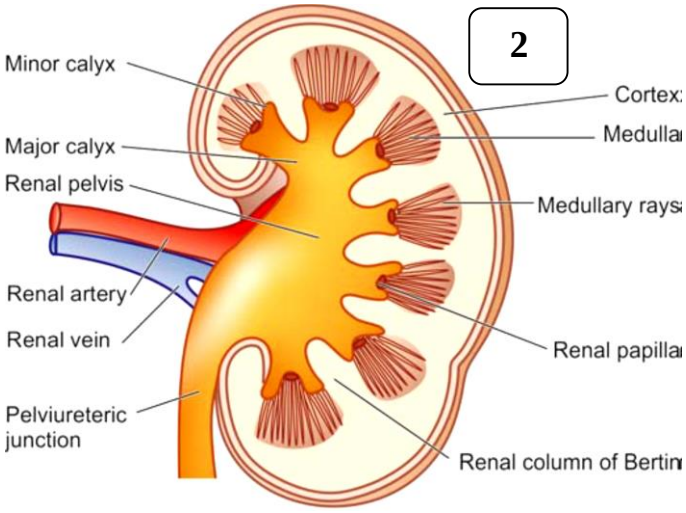
Normal structure of the Kidney

❖ **Anatomy:** The kidneys are each weighing about 150 gm in adult males and about 135 gm in adult females.



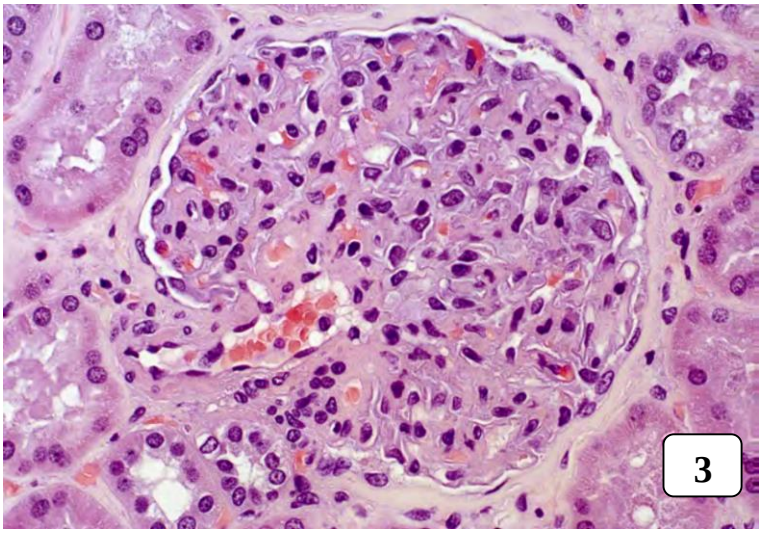
- **Gross:** In cross-section, the normal kidney shows 3 main structures: *peripheral cortex*, *medulla* and the *inner renal pelvis* (**Fig. 1 & 2**): For **fig 1**, the lighter outer renal cortex (*), normally 5 to 10 mm in thickness, and darker inner medulla (♦) with central pelvis containing adipose tissue. As **fig. 2** the hilum of the kidney is situated at the midpoint on the medial aspect where the artery, vein, lymphatics, and ureter are located. The kidney is surrounded by a thin fibrous capsule that is adherent to the hilum.

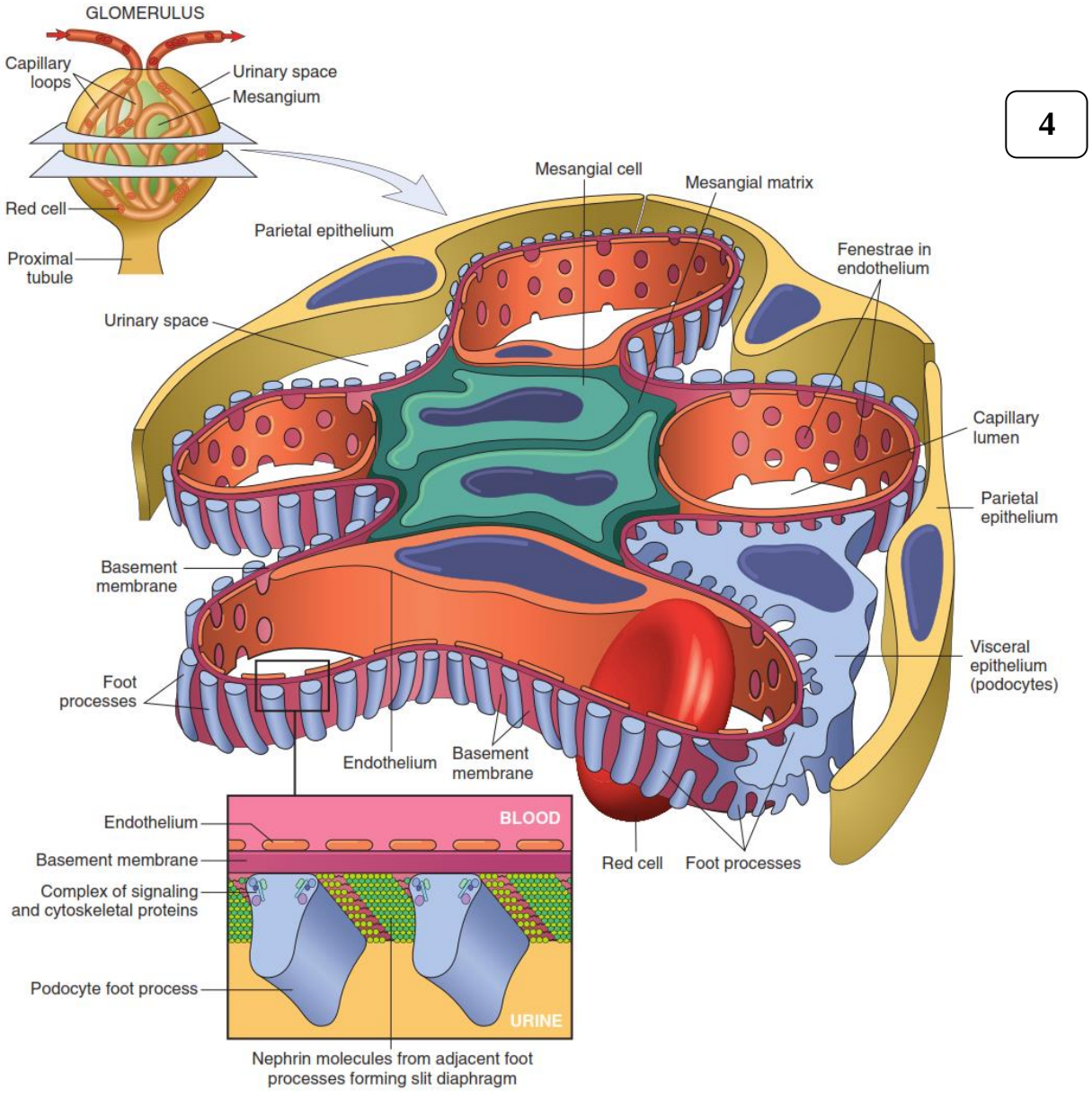


When back to **fig. 1**, note the renal papillae (▲) projecting into the calyces, through which collecting ducts the excreted urine into the renal pelvis.

❖ **Histology:** Each kidney is composed of approximately one million microstructures called nephrons. A nephron consists of 5 major parts, each having a functional role in the formation of urine: the glomerular capsule (glomerulus and Bowman's capsule), the proximal convoluted tubule (PCT), the loop of Henle, the distal convoluted tubule (DCT), and the collecting ducts.

The **normal glomerulus** of the kidney has thin, delicate capillary loops around the mesangial regions (*) which are not prominent. Most glomerular filtration occurs through the capillary loops inside the Bowman space (♦). The visceral epithelial cells (podocytes) that surround the capillary loops (▼) are not easily recognized by light microscopy. (**See slide 3 & picture 4**)





Abnormal structure of the kidney

❖ Pathophysiology of Renal Disease:

1. ***Glomerular diseases****:* These are most often immunologically mediated and may be acute or chronic.

2. ***Tubular, Interstitial diseases****:* These are more likely to be caused by toxic or infectious agents and are often acute (tubulointerstitial diseases).

3. ***Vascular diseases****:* These include changes in the nephron as a consequence of increased intra-glomerular pressure such as hypertension or impaired blood flow.

Regardless of the cause, renal disease is usually divided into two major pathological syndromes: *acute renal failure* and *chronic renal failure (or chronic kidney disease, CKD).*

I. Glomerular diseases

Including a large, clinically significant group of renal diseases. Glomerulonephritis (GN) or Bright's disease is the term used for diseases that primarily involve the renal glomeruli. It is classified into 2 groups:

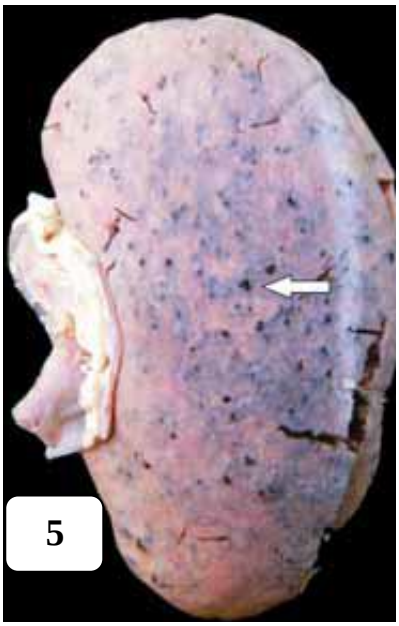
I. *Primary glomerulonephritis* in which the glomeruli are the target site.

II. *Secondary glomerular diseases* include certain systemic and hereditary diseases that secondarily affect the glomeruli.

I. PRIMARY GLOMERULONEPHRITIS (Acute)

A: Acute Post-Streptococcal GN

Mostly affecting children between 2 to 14 years of age but 10% of cases are seen in adults above 40 years.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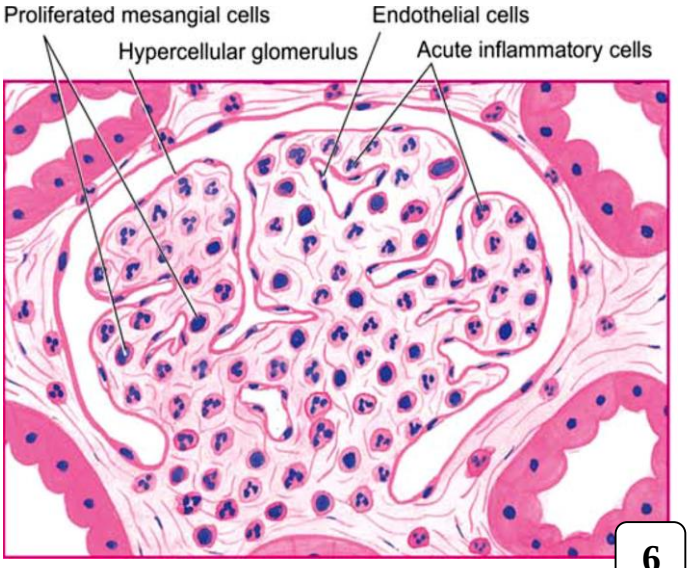
Diagnosis

- ***Grossly:*** As **fig. 5**, kidneys are enlarged, weighing one and a half to twice the normal weight. The cortical surface shows petechial haemorrhages (⟸) giving the characteristic appearance of a flea-bitten kidney.

• ***Microscopic:*** is as under (**slide 6**)



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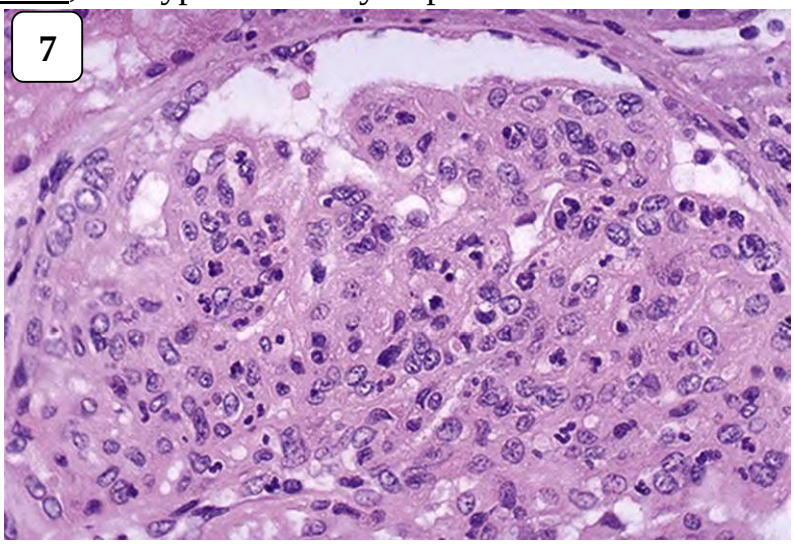
i)Glomeruli: They are enlarged and hypercellular. The diffuse hypercellularity of the tuft is due to the proliferation of mesangial, endothelial and occasionally epithelial cells as well as by infiltration of leucocytes, sometimes monocytes. There may be small deposits of fibrin within the capillary lumina and in the mesangium.

The hypercellular with increased inflammatory cells and loops are poorly defined. The infectious agent induces an immune response with antibodies that react with glomerular antigens or lead to antigen-antibody complex formation with glomerular deposition.

At **slide 7**, higher magnification, the hypercellularity of post-infectious GN is caused by increased numbers of epithelial, endothelial, and mesangial cells and neutrophils (▲) infiltrating in and around the capillary loops.



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ii) Tubules: Tubular changes are not very observed. There may be swelling and hyaline droplets in tubular cells and tubular lumina may contain red cells.

iii) Vessels Changes in arteries and arterioles are seldom present in acute GN.

B: Acute Non-Streptococcal GN.

These include other bacteria, viruses, parasitic infections. The appearance of renal biopsy by light microscopy is similar to that seen in acute post-streptococcal GN.

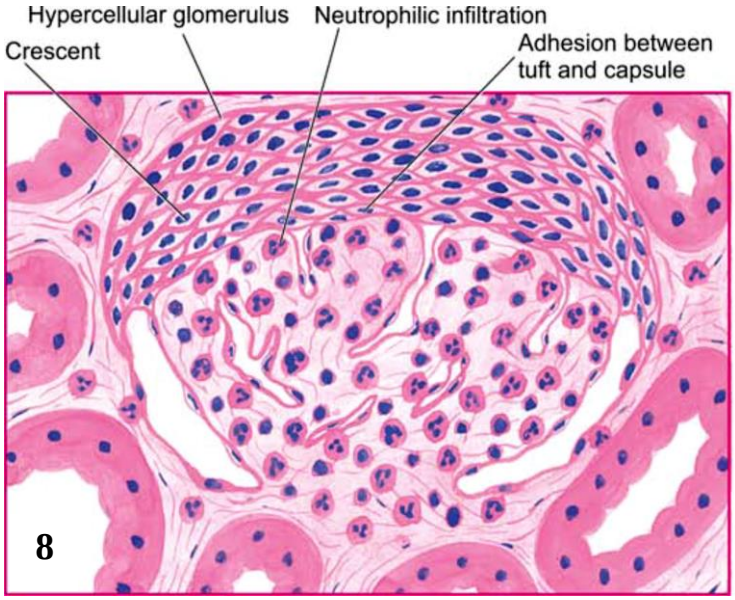
C: Rapidly Progressive Glomerulonephritis

RPGN presents with an acute reduction in renal function resulting in acute renal failure in a few weeks or months. It is characterized by the formation of 'crescents' (crescentic GN) outside the glomerular capillaries. 'Crescents' are formed from the proliferation of parietal epithelial cells lining Bowman's capsule with contribution from visceral epithelial cells and the invading mononuclear cells. The stimulus for crescent formation appears to be the presence of fibrin in the capsular space. RPGN occurs most frequently in adults, with a slight male increase.

MORPHOLOGIC FEATURES

Gross: the kidneys are usually enlarged and pale with a smooth outer surface. Cut surface shows the pale cortex and congested medulla.

Microscopic: vary according to the cause but, in general, the following features are present **(Fig. 8)**.



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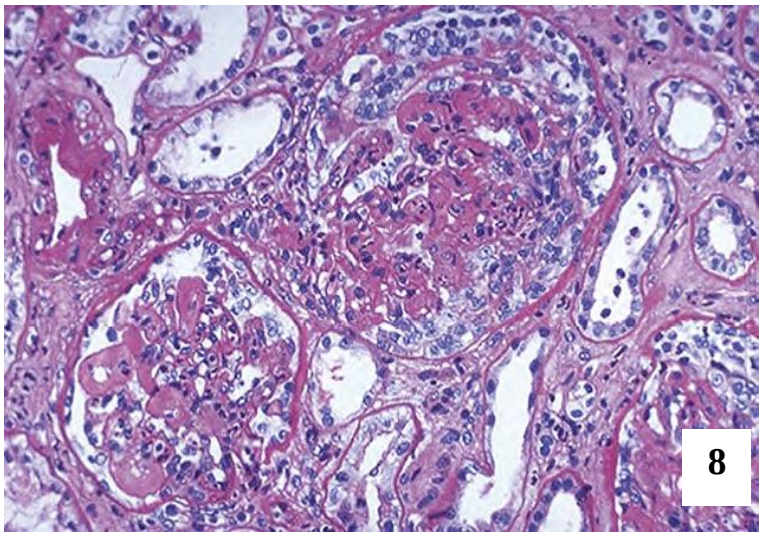
i) Glomeruli. All forms of RPGN show *'crescents'* inside of Bowman's capsules.

Crescents obliterate the Bowman's space and compress the glomerular tuft. Fibrin deposition is present alongside crescents. Besides the crescents, glomerular tufts may show increased cellularity as a result of the proliferation of endothelial and mesangial cells and leucocytic infiltration. Fibrin thrombi are frequently present in the glomerular tufts.

ii) Tubules: Tubular epithelial cells may show hyaline droplets. Tubular lumina may contain casts, red blood cells, and fibrin.

iii) Vessels Arteries and arterioles may show no change, but cases associated with hypertension usually show severe vascular changes.



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