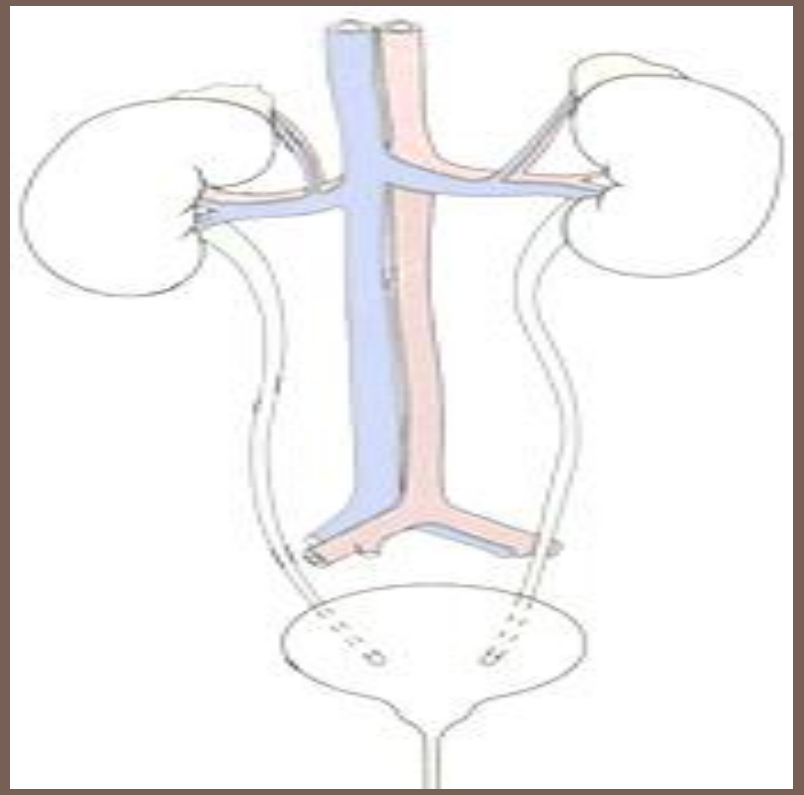
RENAL BLOCK



PATHOLOGY PRACTICAL

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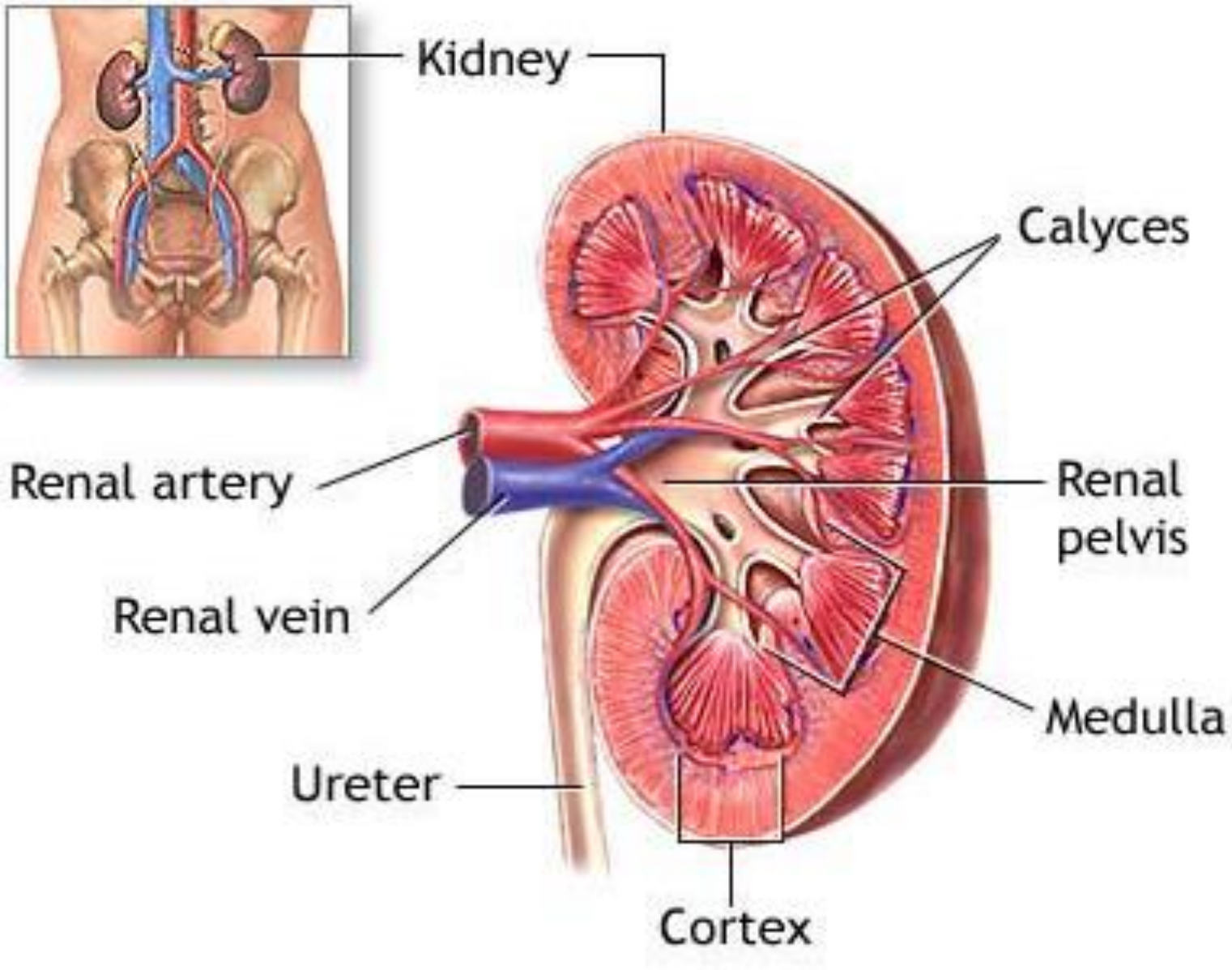
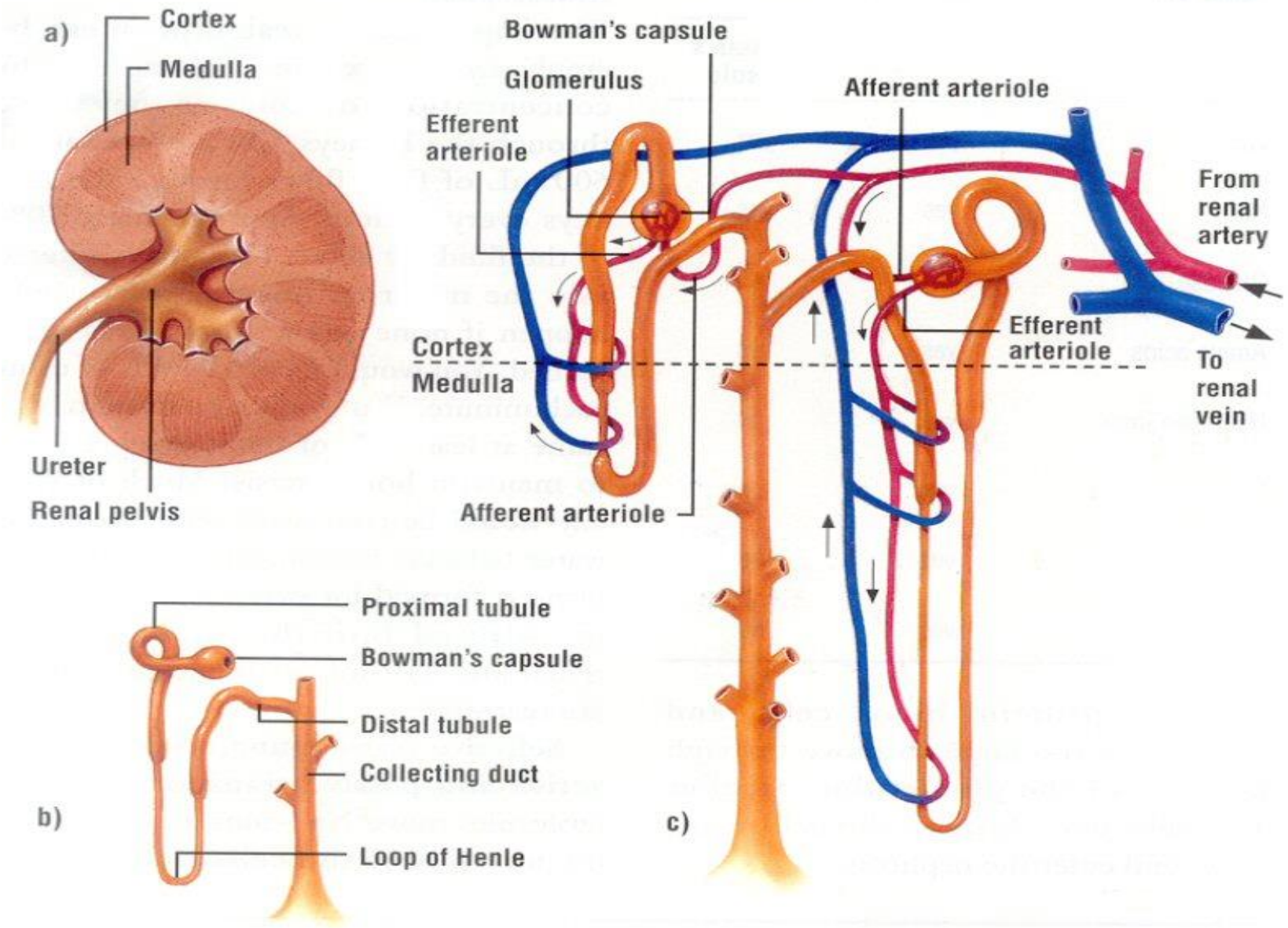
NORMAL ANATOMY AND HISTOLOGY

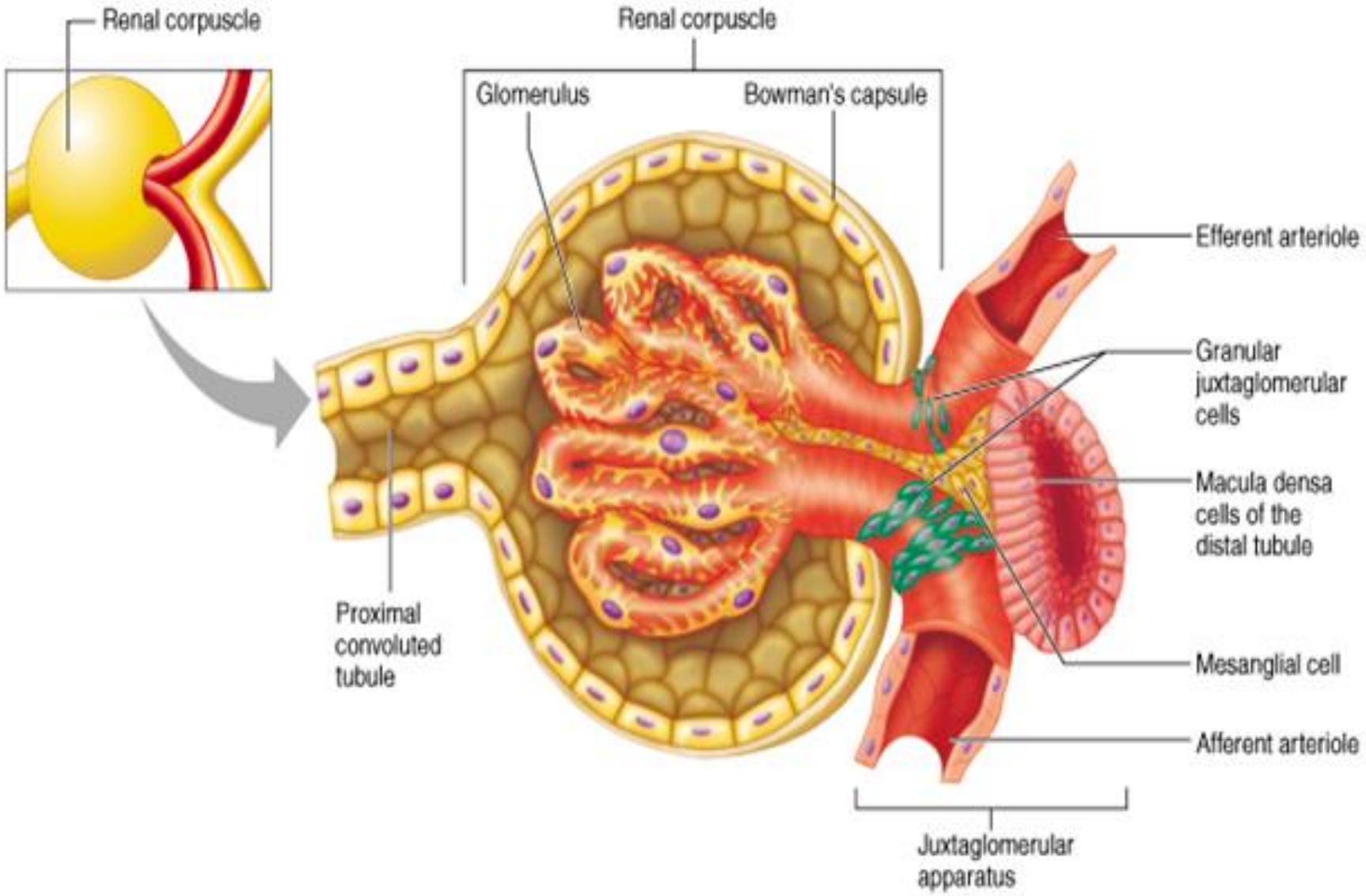
Anatomy of the Kidney



NEPHRON STRUCTURE



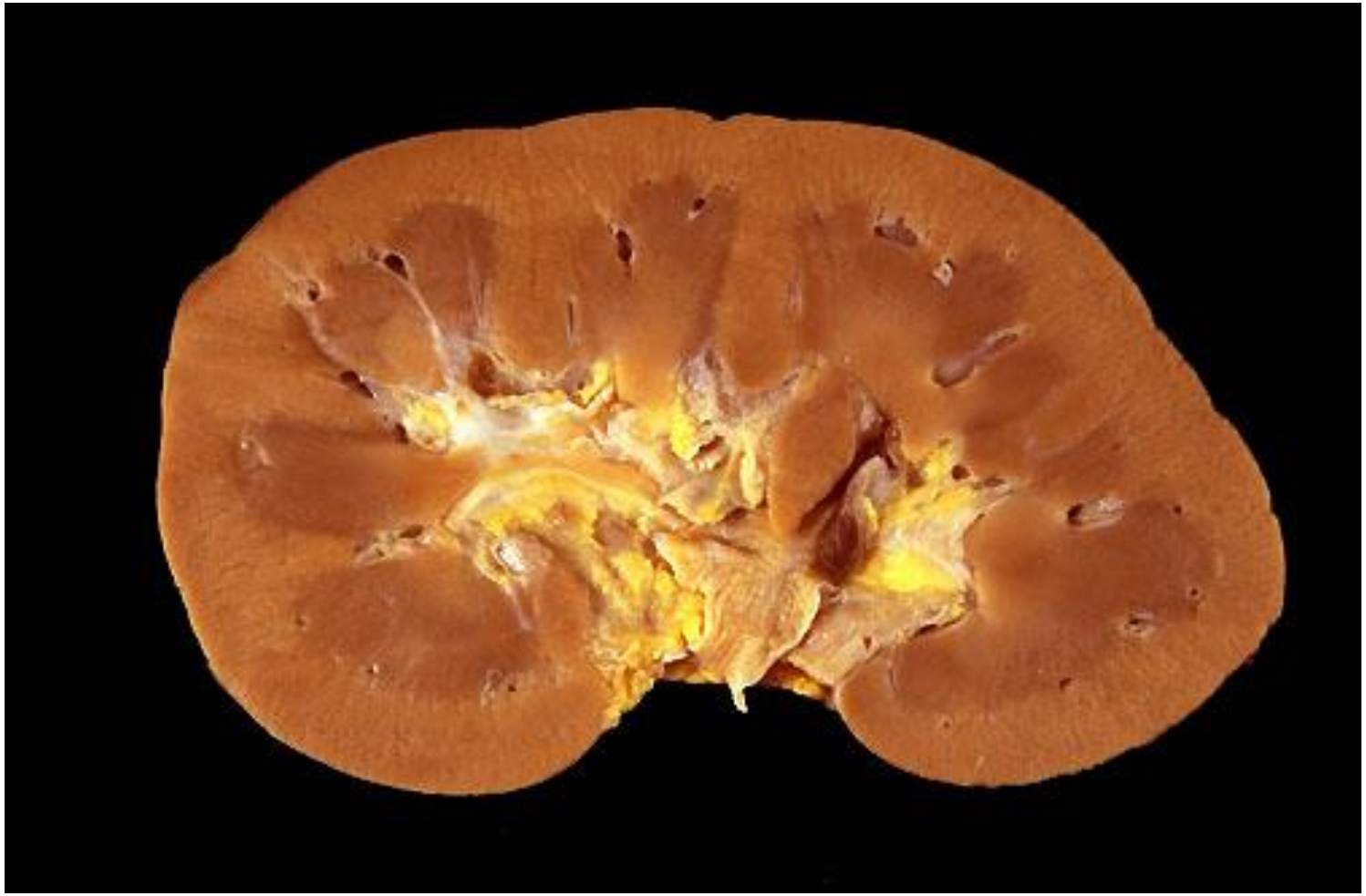
Renal Corpuscle



KIDNEY ANATOMY : NEPHRONS

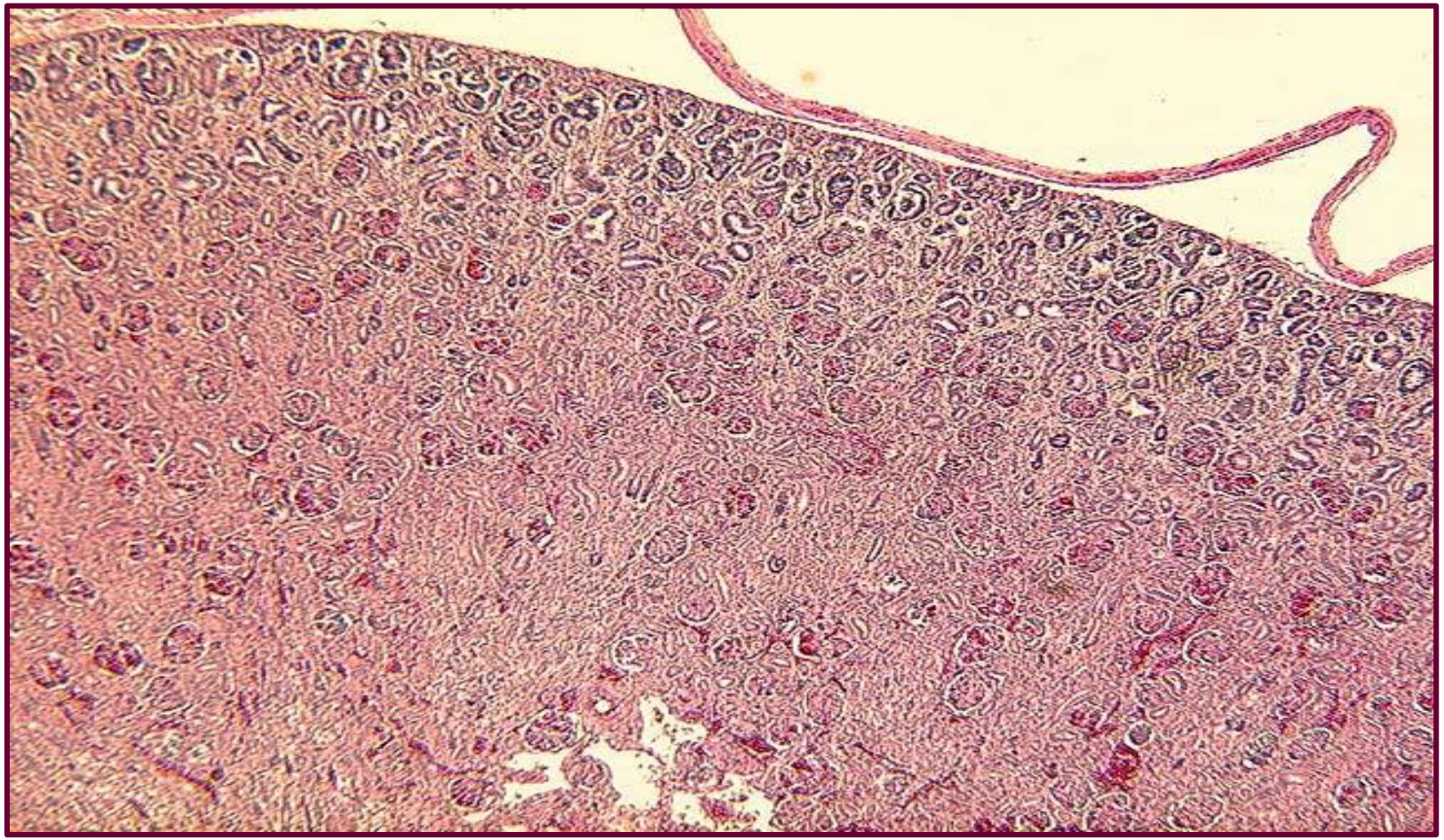
- *Nephron is the functional unit of the kidney.*
- *Each kidney contains about 1,000,000 to 1,300,000 nephrons.*
- *The nephron is composed of glomerulus and renal tubules .*
- *The nephron performs its function by ultra filtration at glomerulus and secretion and reabsorption at renal tubules.*

Normal Kidney - Gross



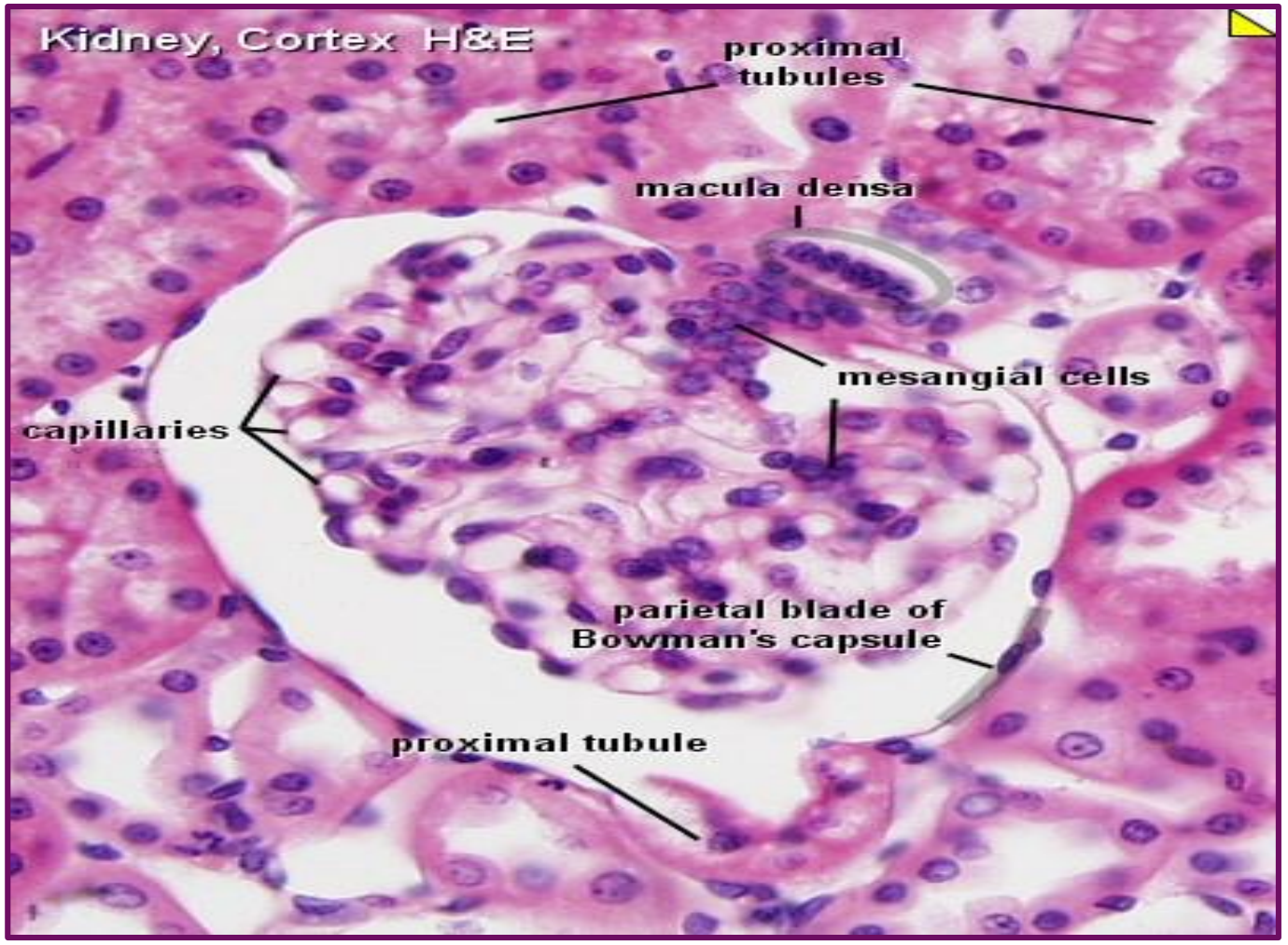
In cross section, this normal adult kidney demonstrates the lighter outer cortex and the darker medulla, with the renal pyramids into which the collecting ducts coalesce and drain into the calyces and central pelvis.

Kidney – Normal Histology

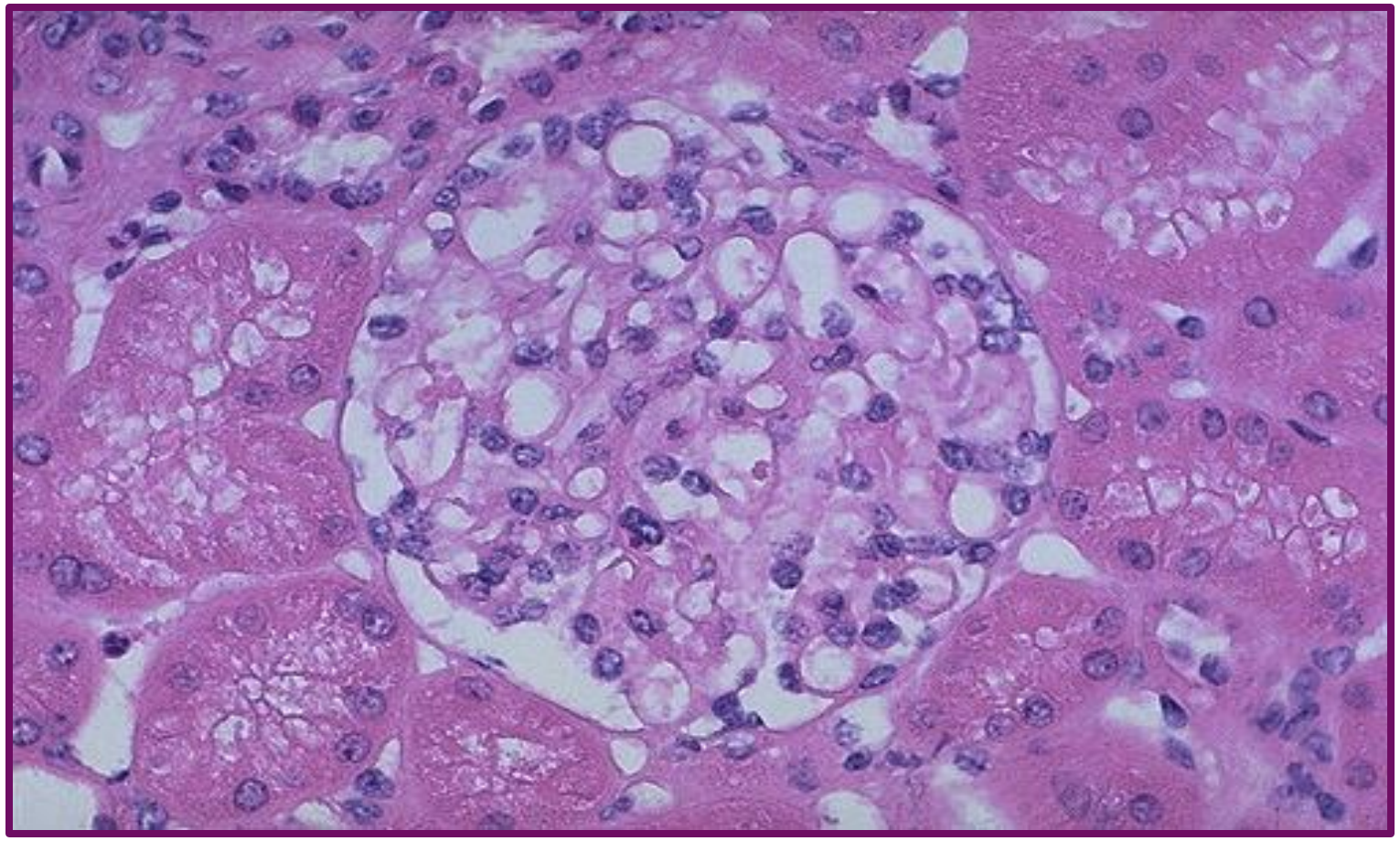


The kidneys show a well preserved lobular structure with indistinct corticomedullary demarcation

Renal Corpuscle – Normal Histology

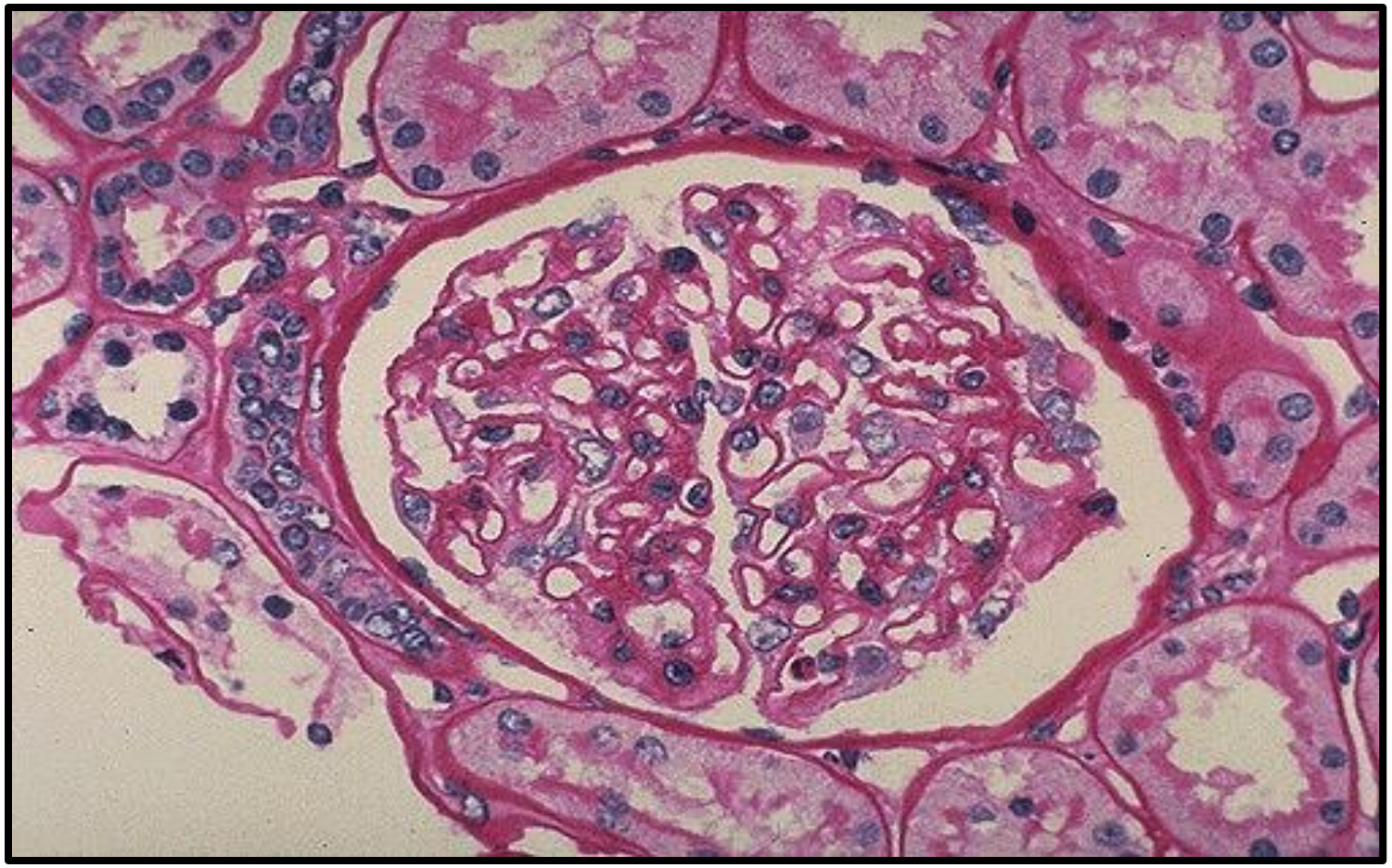


Renal Corpuscle – Normal Histology



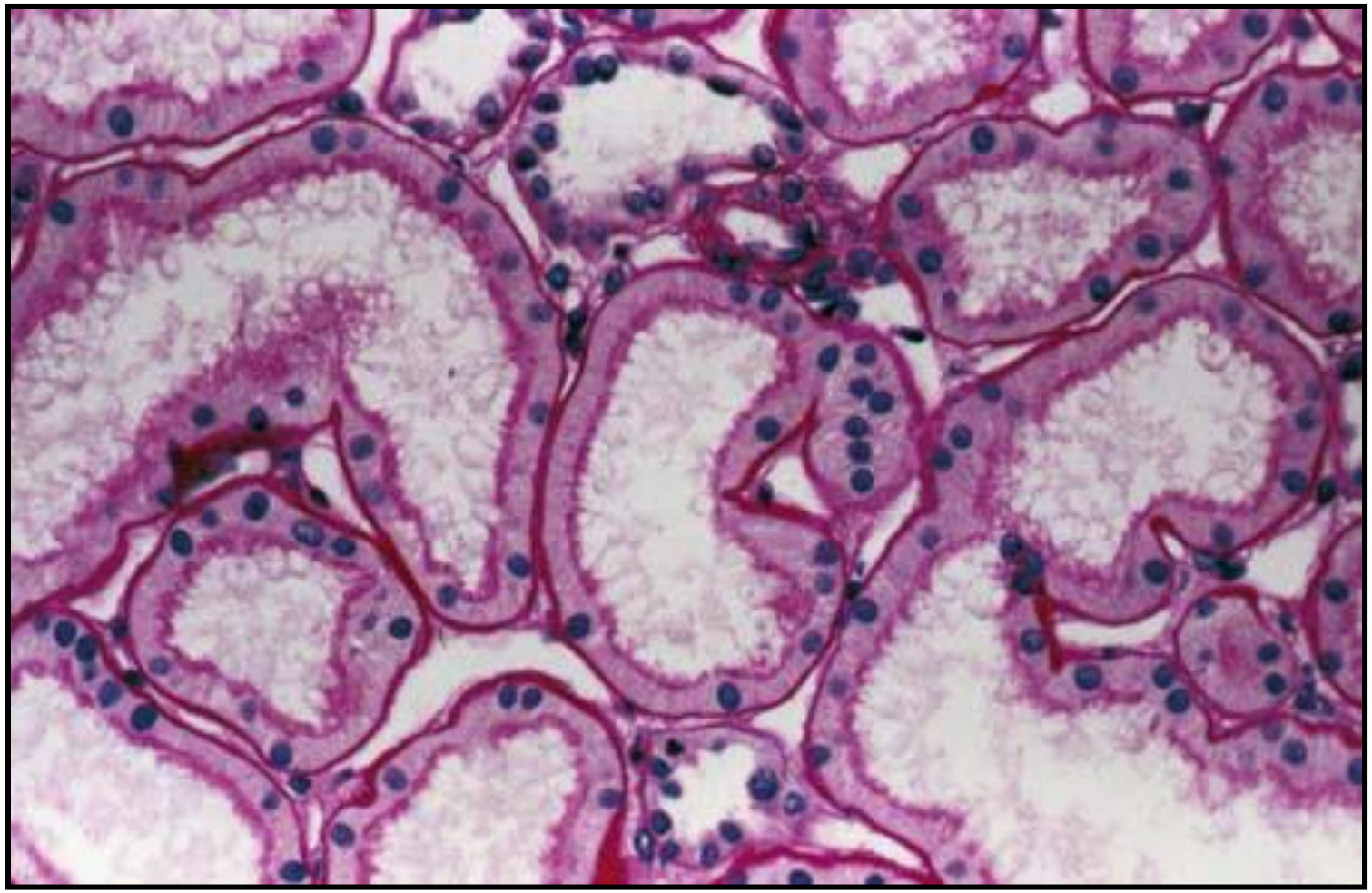
Normal glomerulus by light microscopy. The glomerular capillary loops are thin and delicate. Endothelial and mesangial cells are normal in number. The surrounding tubules are normal

Renal Corpuscle – Normal Histology



Normal glomerulus is stained with PAS to highlight basement membranes of glomerular capillary loops and tubular epithelium.

Normal Cortical Tubules



Normal cortical tubules, interstitium, and peritubular capillaries; most of the tubules are proximal, with well-defined brush borders (PAS stain).

PRACTICAL SESSION : 1

ACUTE KIDNEY INJURY

Acute Kidney Injury

Causes:

Pre-renal

(All those that decrease effective blood flow to the kidney)

- *Low blood volume, low blood pressure, and heart failure.*
- *Renal artery stenosis, and renal vein thrombosis.*
- *Renal ischemia.*

Renal:

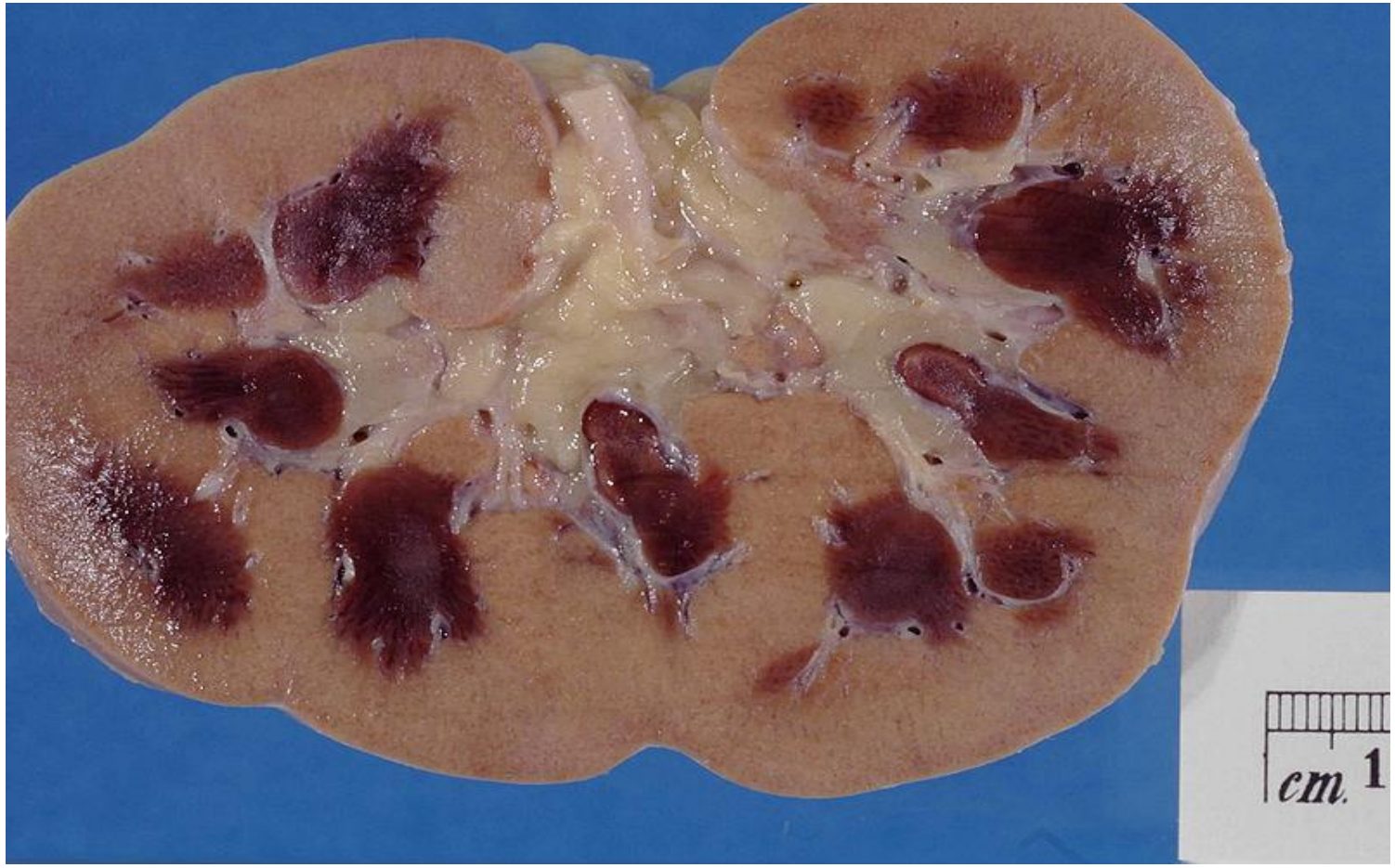
- *Glomerulonephritis (GN).*
- *Acute tubular necrosis (ATN).*
- *Acute interstitial nephritis (AIN).*

Post-renal:

(is a consequence of urinary tract obstruction)

- *Benign prostatic hyperplasia.*
- *Kidney stones.*
- *Obstructed urinary catheter.*
- *Bladder stone .*
- *Bladder, ureteral or renal malignancy.*

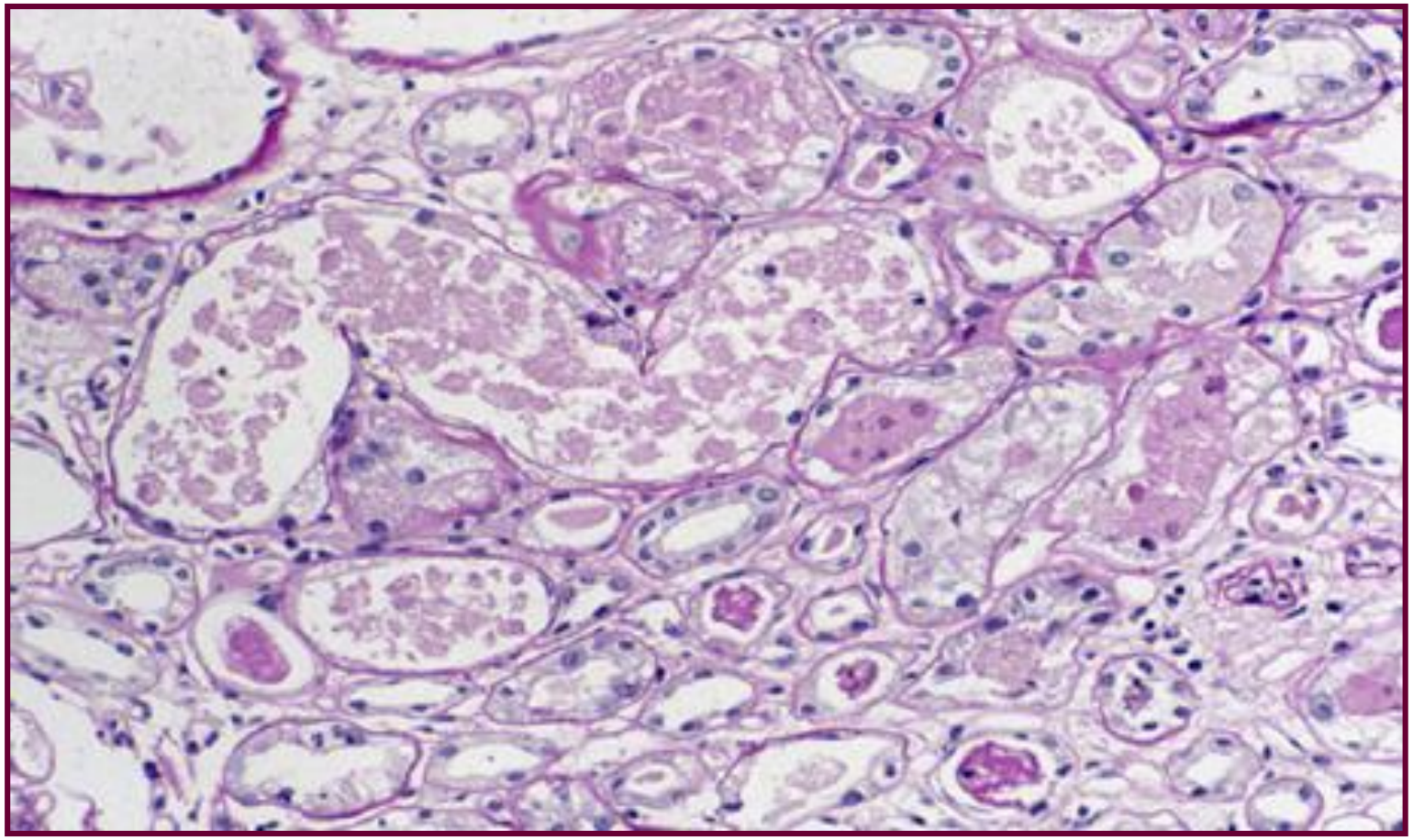
Acute Kidney Injury



Kidney showing :

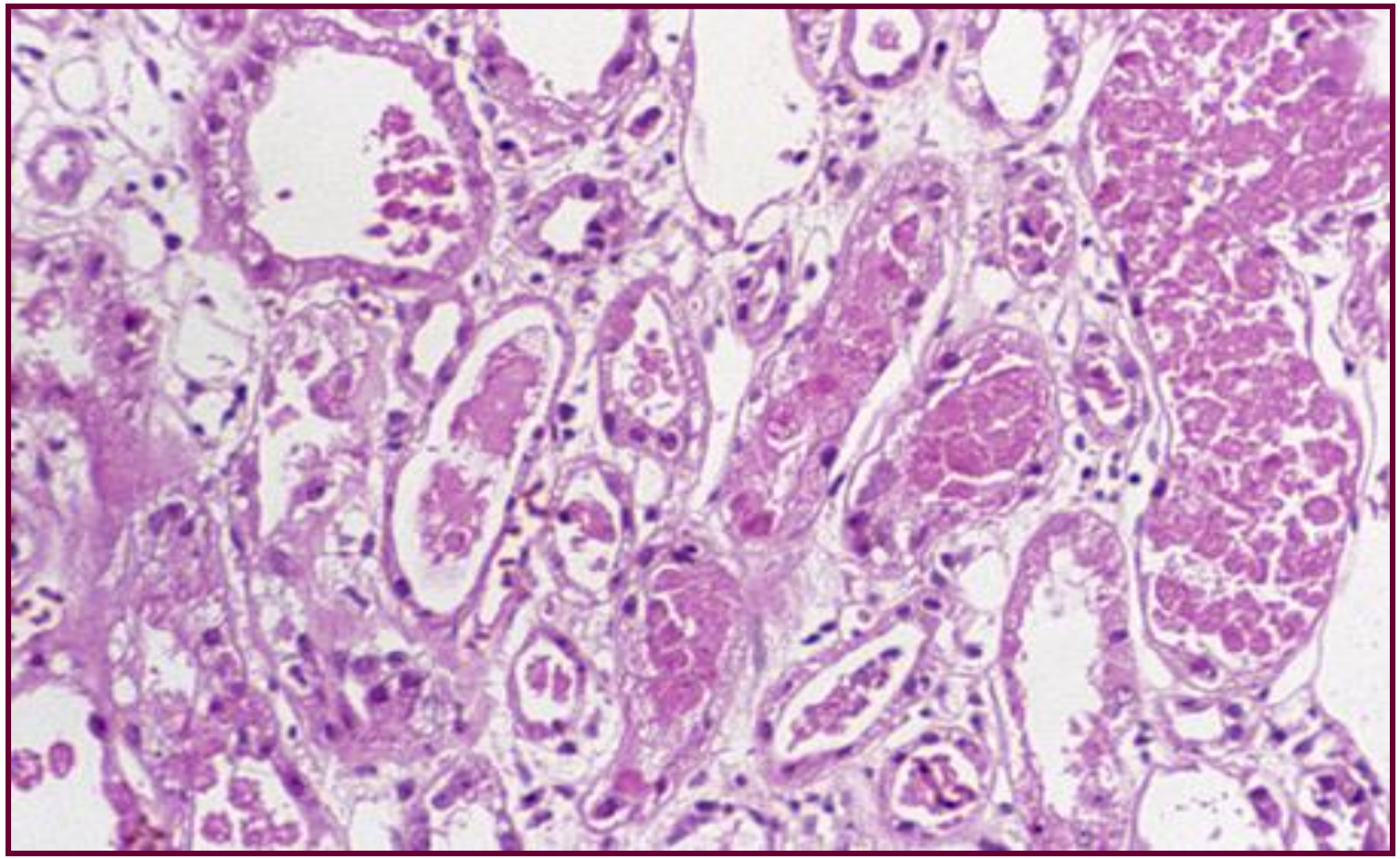
- *Marked pallor of the renal cortex.*
- *Congested renal medulla.*

Acute Tubular Necrosis



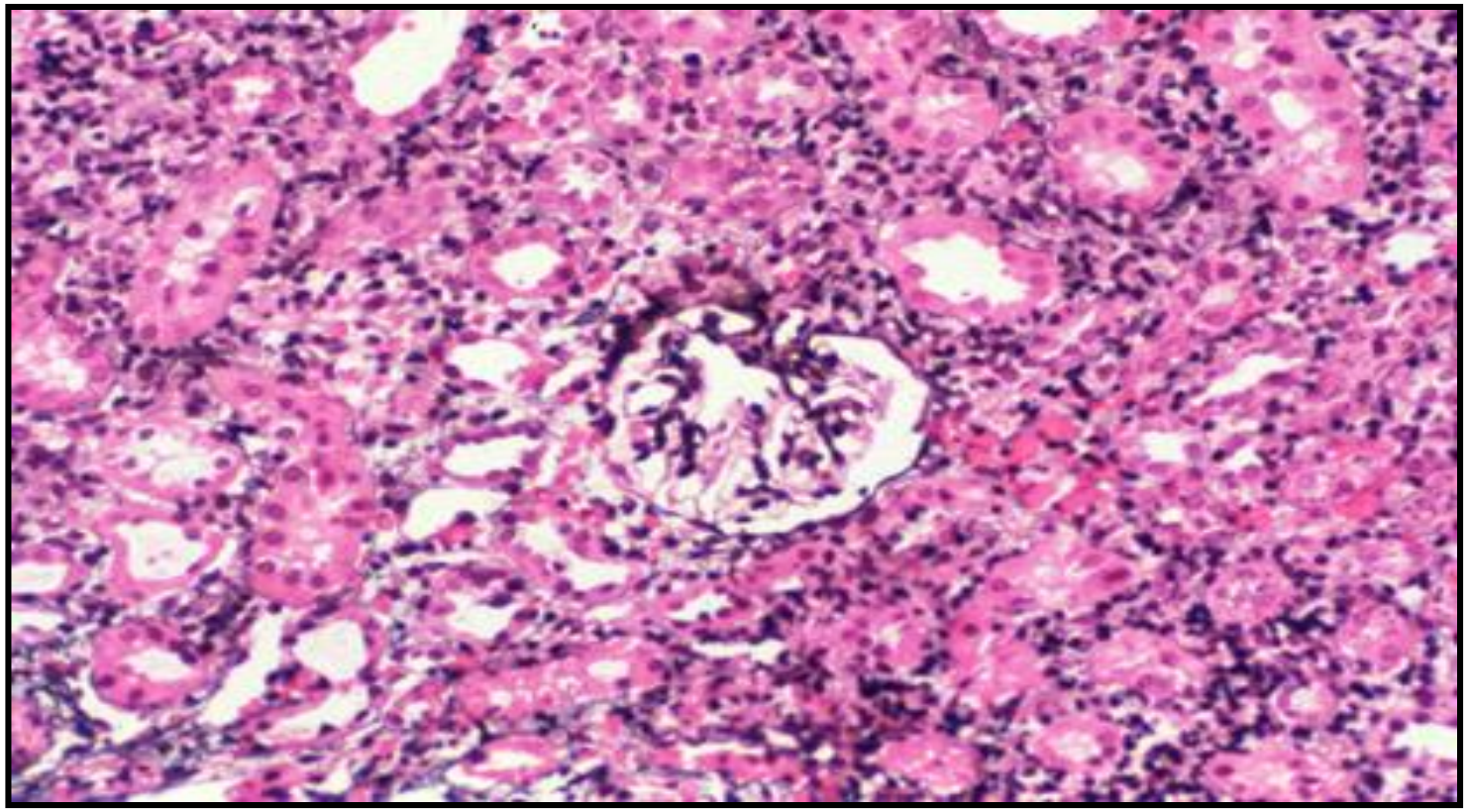
Necrotic renal tubular cells.
Some tubules lined by flat and vacuolated epithelium.

Acute Tubular Necrosis



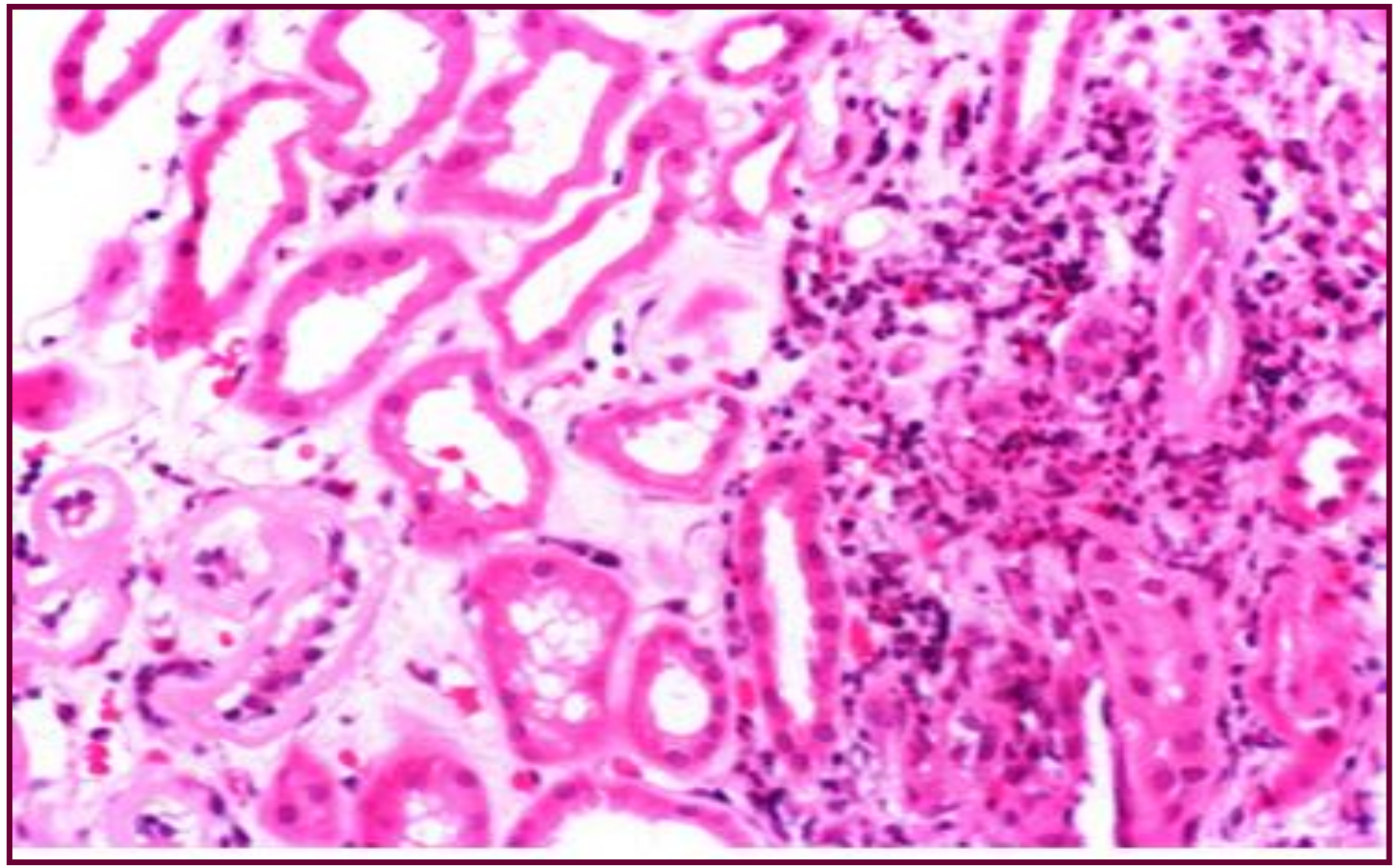
There may also be degeneration and frank necrosis of individual cells or tubular segments in acute tubular necrosis, or flattened, regenerating type epithelium with degenerated cells in the lumen (middle left) (H&E x 200).

Acute Interstitial Nephritis



There is edema associated with an interstitial lymphoplasmocytic infiltrate. There are numerous causes for acute interstitial nephritis, including toxins, viral infections and drug-induced hypersensitivity reactions. The glomeruli are uninvolved, unless there is an associated minimal change disease-type injury caused by non steroidal anti-inflammatory drugs

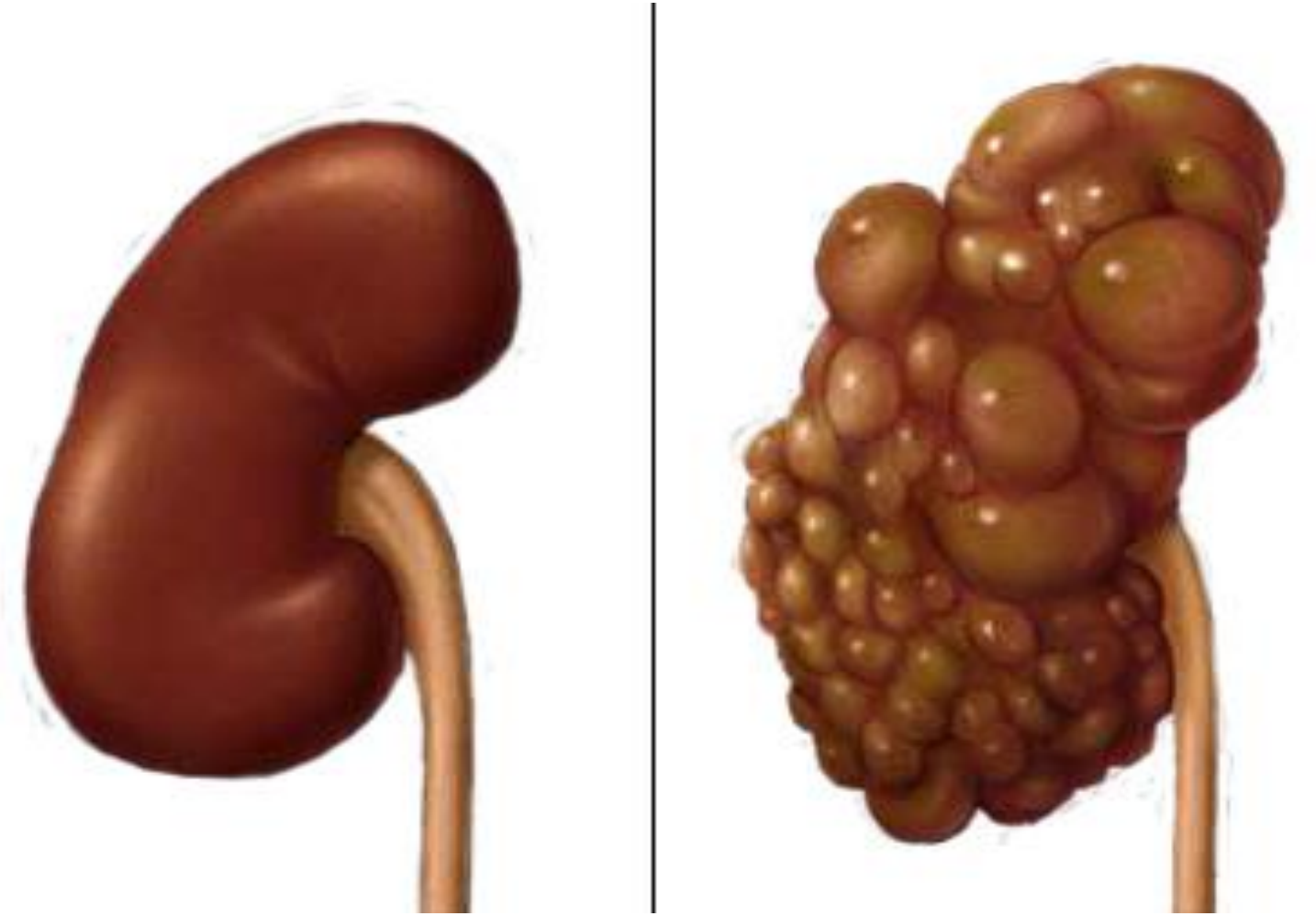
Acute Interstitial Nephritis



There is edema in addition to preexisting mild tubulointerstitial fibrosis in this case of acute interstitial nephritis caused by drug-induced hypersensitivity. There is a prominent interstitial eosinophilic component, in addition to lymphocytes and plasma cells (H&E stain x 100)

POLYCYSTIC KIDNEY

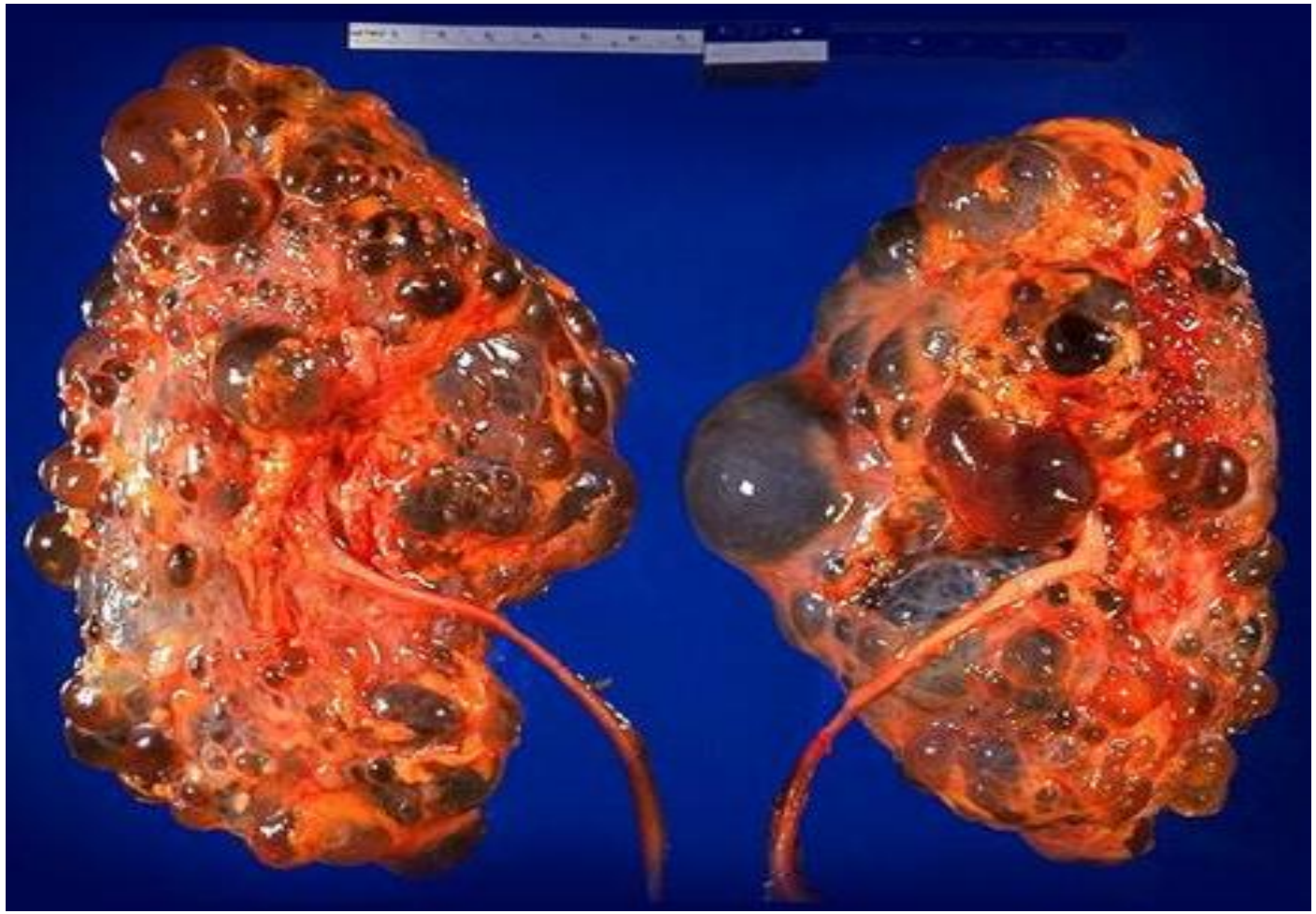
Normal vs Polycystic Kidney



Polycystic kidney – Gross Anatomy

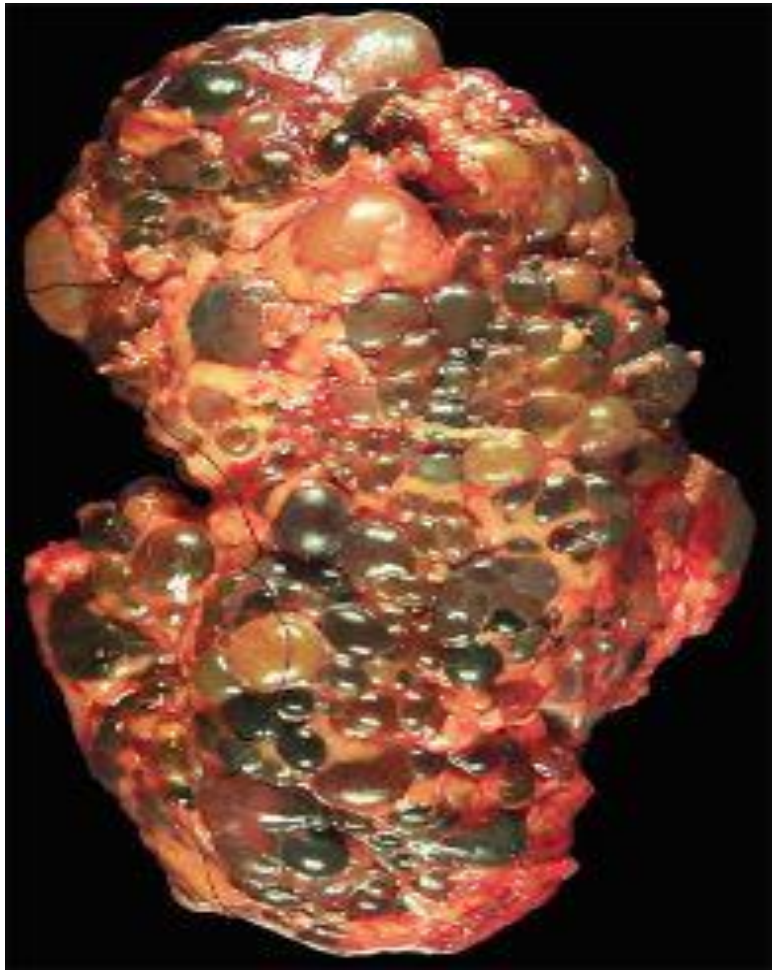
Markedly enlarged kidney and replacement of the renal parenchyma by numerous cysts of variable sizes

Polycystic kidney – Gross Anatomy

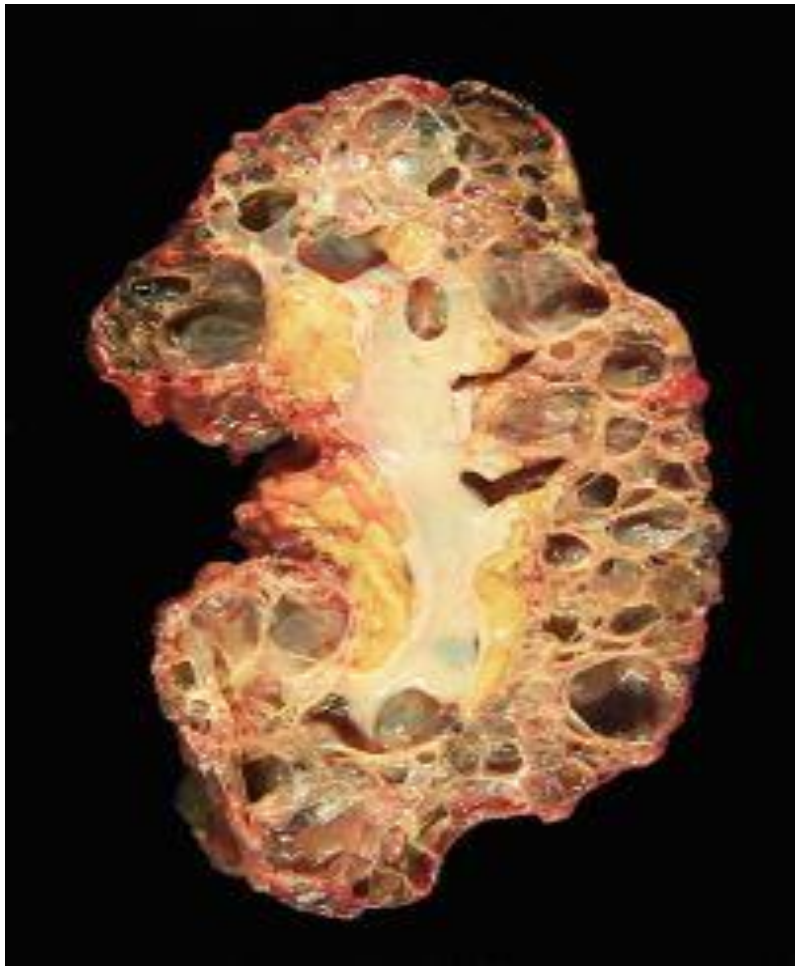


Bilateral autosomal dominant polycystic kidney disease

Gross Polycystic kidney and its Cut Section

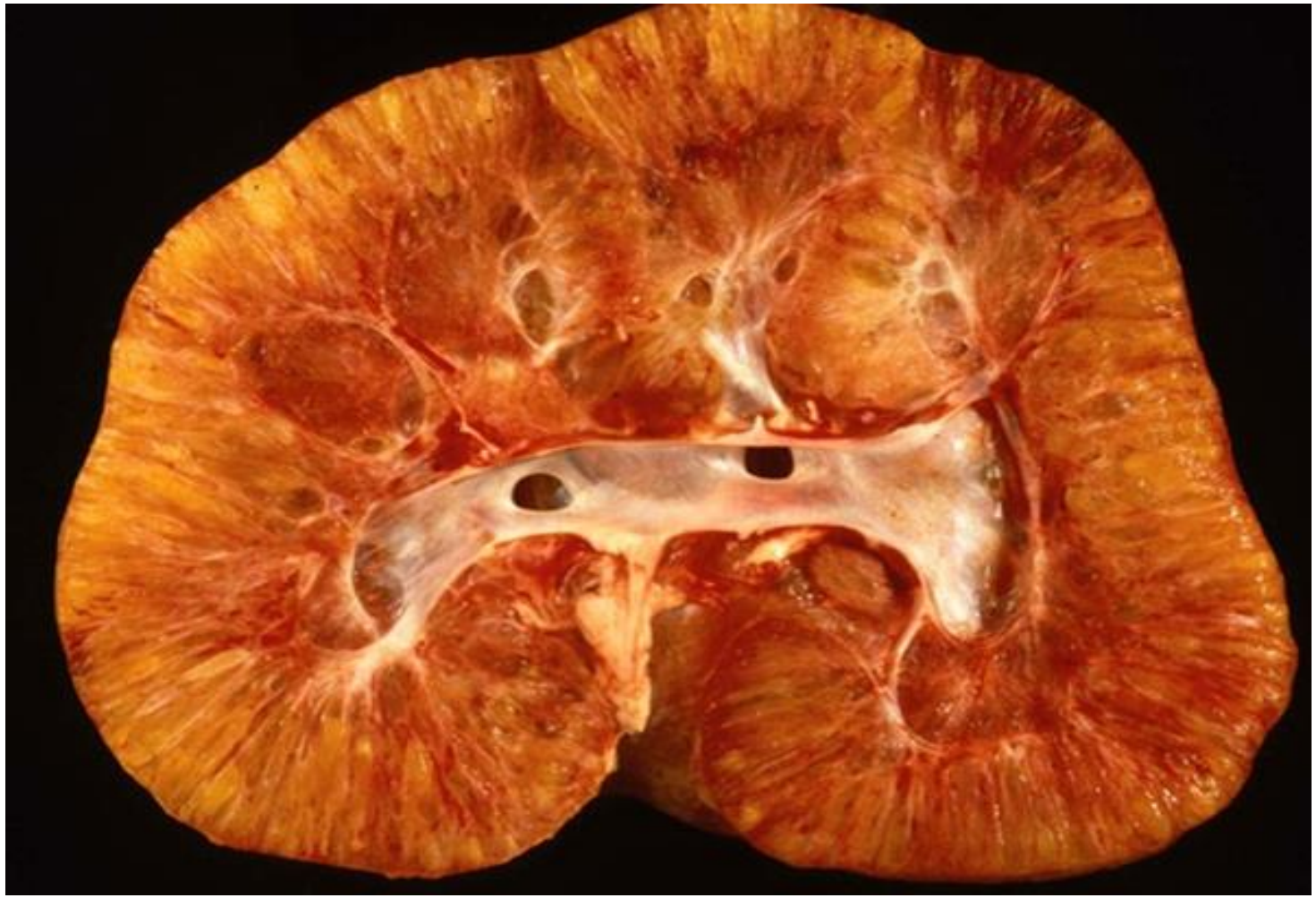


Massively enlarged kidney disrupted by numerous cysts



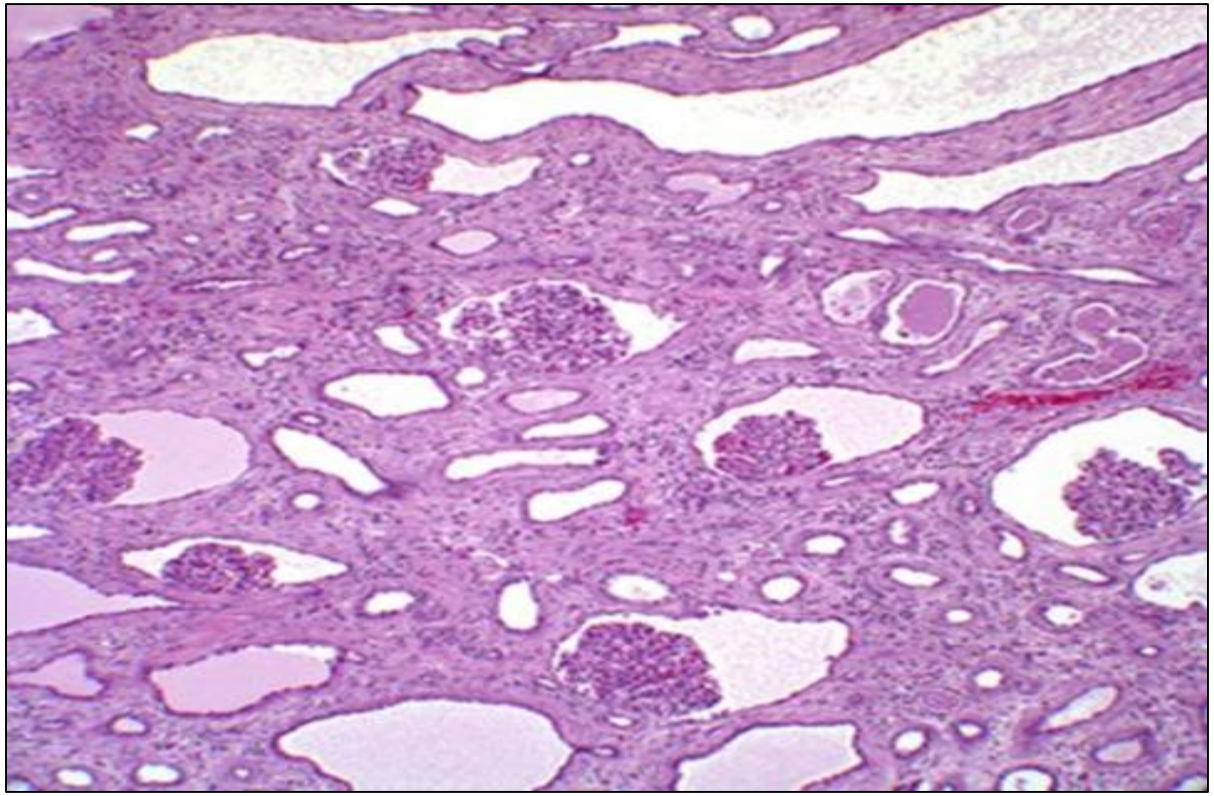
Cut surface of the kidney, showing extensive cortical destruction by cysts

Infantile Polycystic kidney – Gross



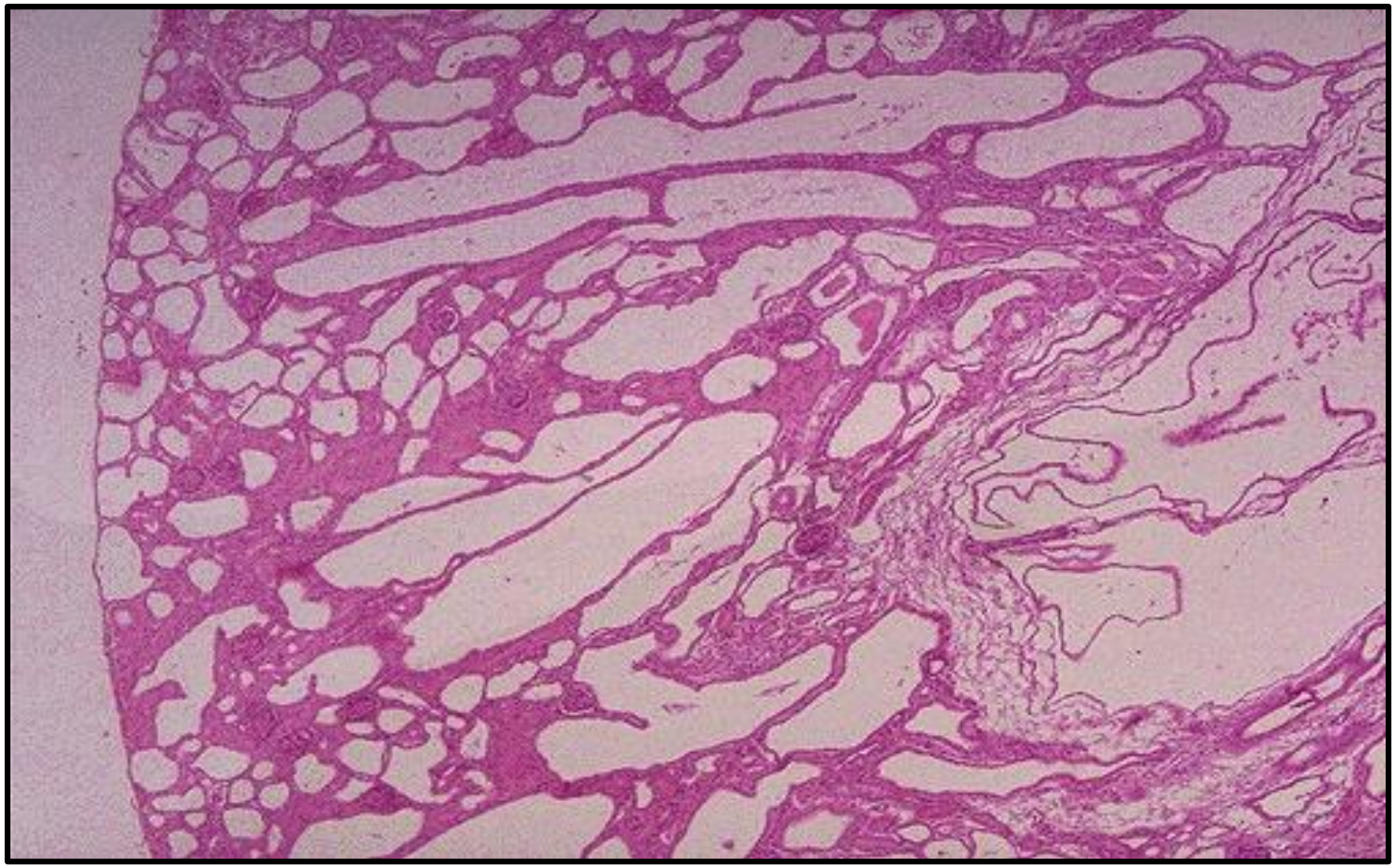
Coronal section of an infantile polycystic kidney

Polycystic kidney – Histopathology



Kidney of child with autosomal dominant PCKD.
a. Coronal section of a polycystic kidney.
b. histology demonstrating glomerular cysts .
Note the normal-sized glomeruli with the enlarged Bowman's space and tubular cystic changes

Polycystic kidney – Histopathology



Autosomal Recessive Polycystic Kidney Disease (ARPKD). Note that the cysts fill most of the parenchyma, and it is hard to find glomeruli.

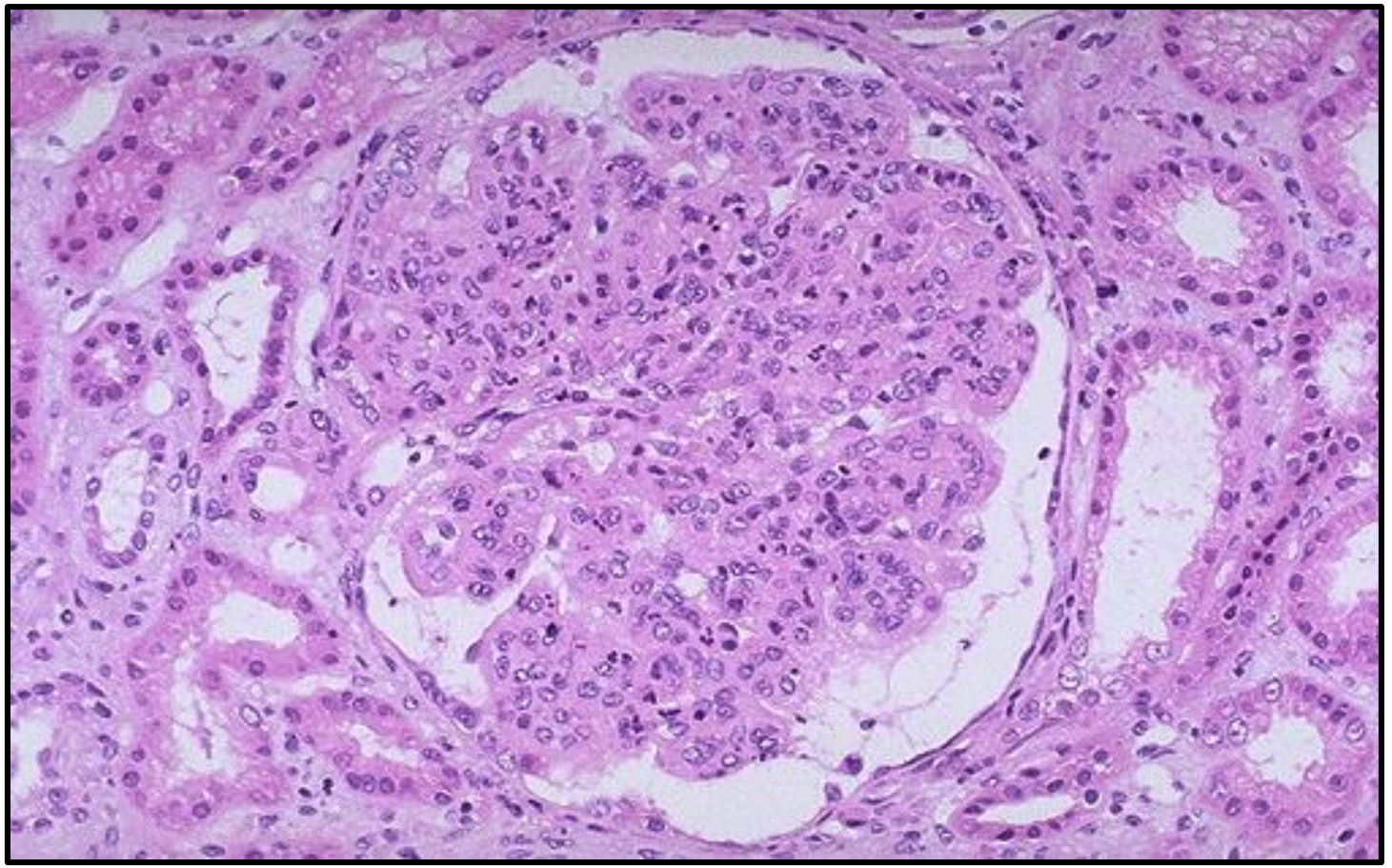
ACUTE (POST-STREPTOCOCCAL) GLOMERULONEPHRITIS

Acute (Post-streptococcal) Glomerulonephritis

Section of the kidney shows:

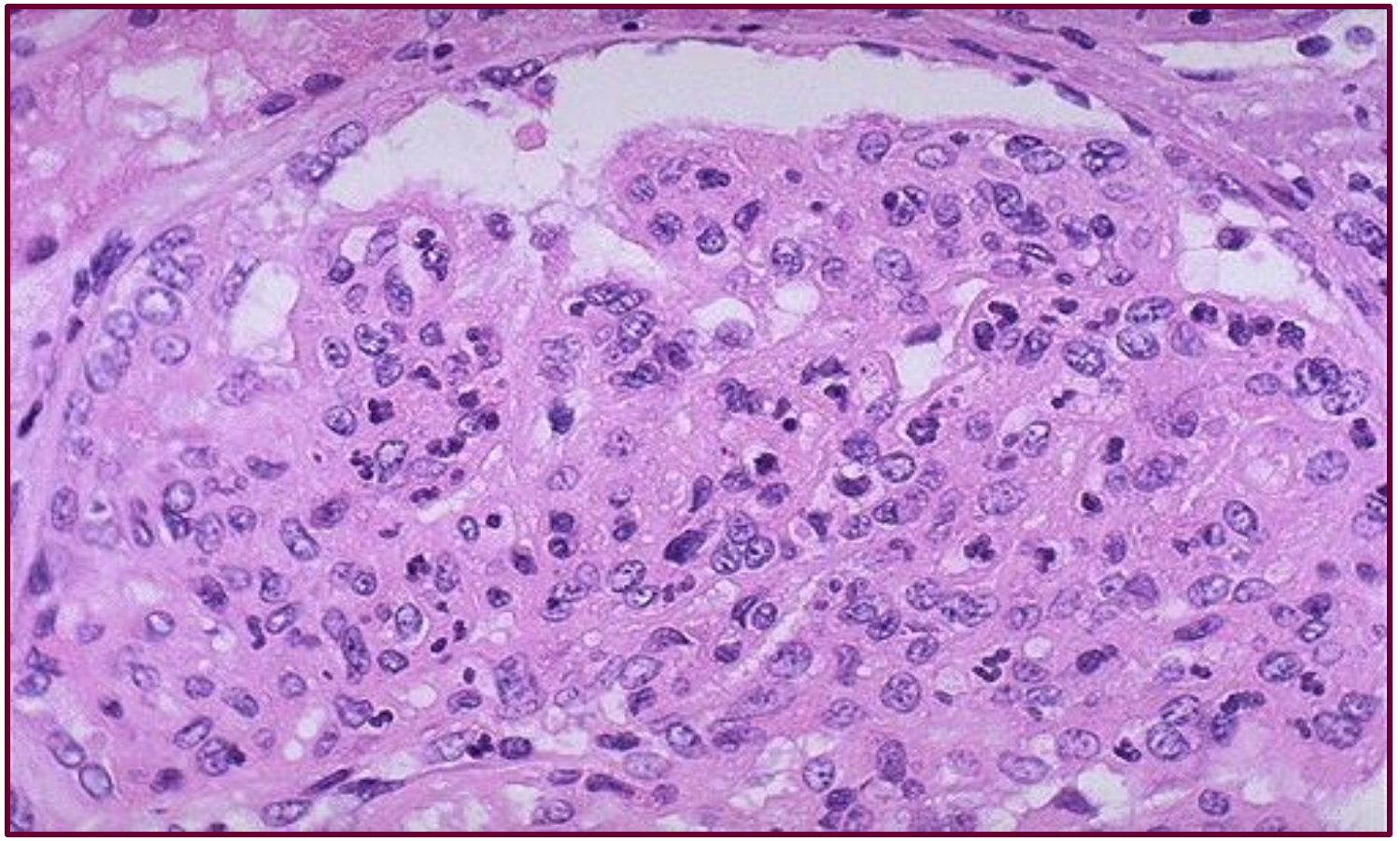
- The glomeruli are enlarged, lobulated and hypercellular with obliteration of capsular space.
- Cellularity is due to proliferation of endothelial and mesangial cells with some neutrophils.
- Many capillaries appear obliterated.
- Tubules show degenerative changes.

Acute (Post-streptococcal) Glomerulonephritis



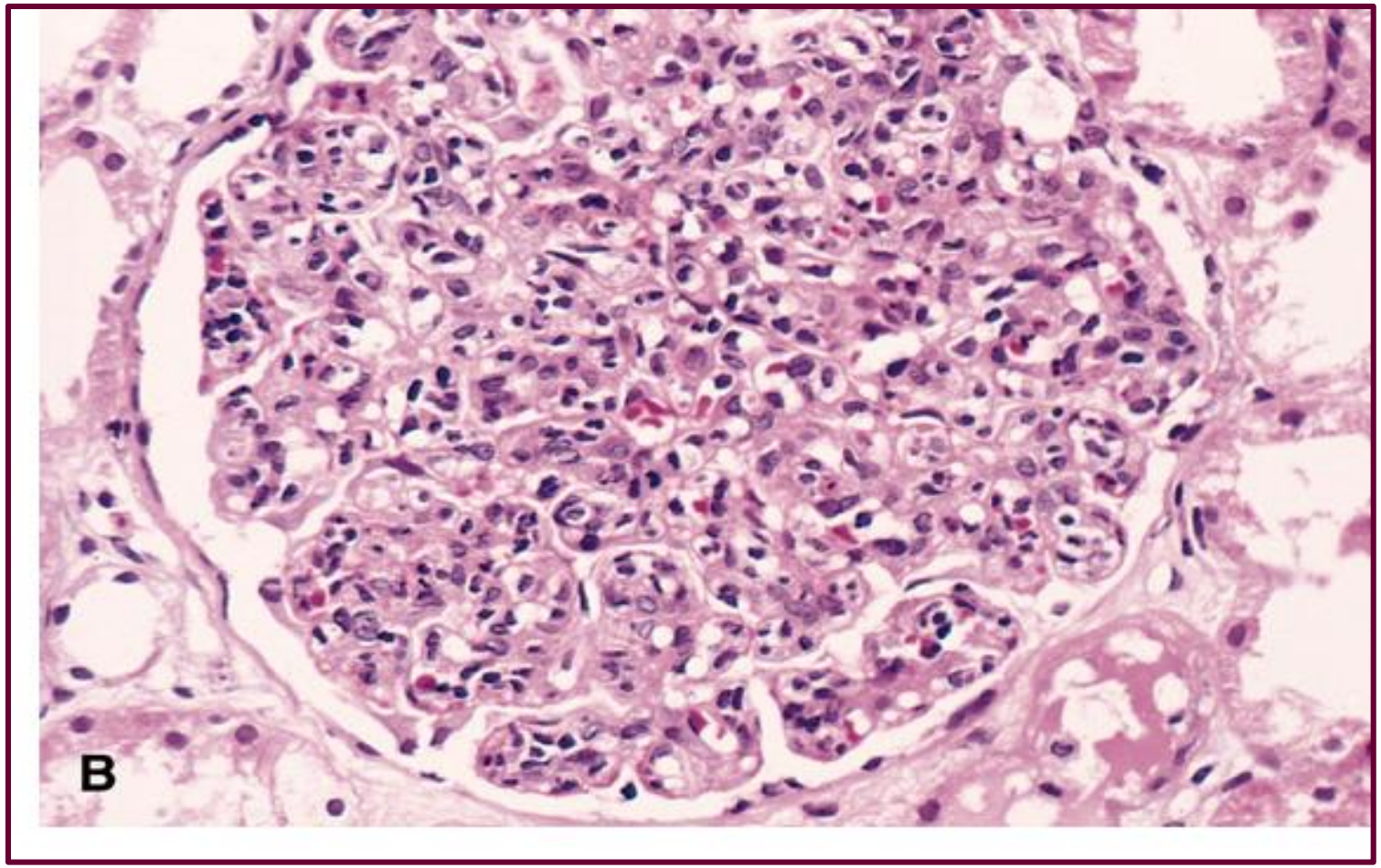
This glomerulus is hypercellular and capillary loops are poorly defined. This is a type of proliferative glomerulonephritis known as post-infectious glomerulonephritis

Acute (Post-streptococcal) Glomerulonephritis



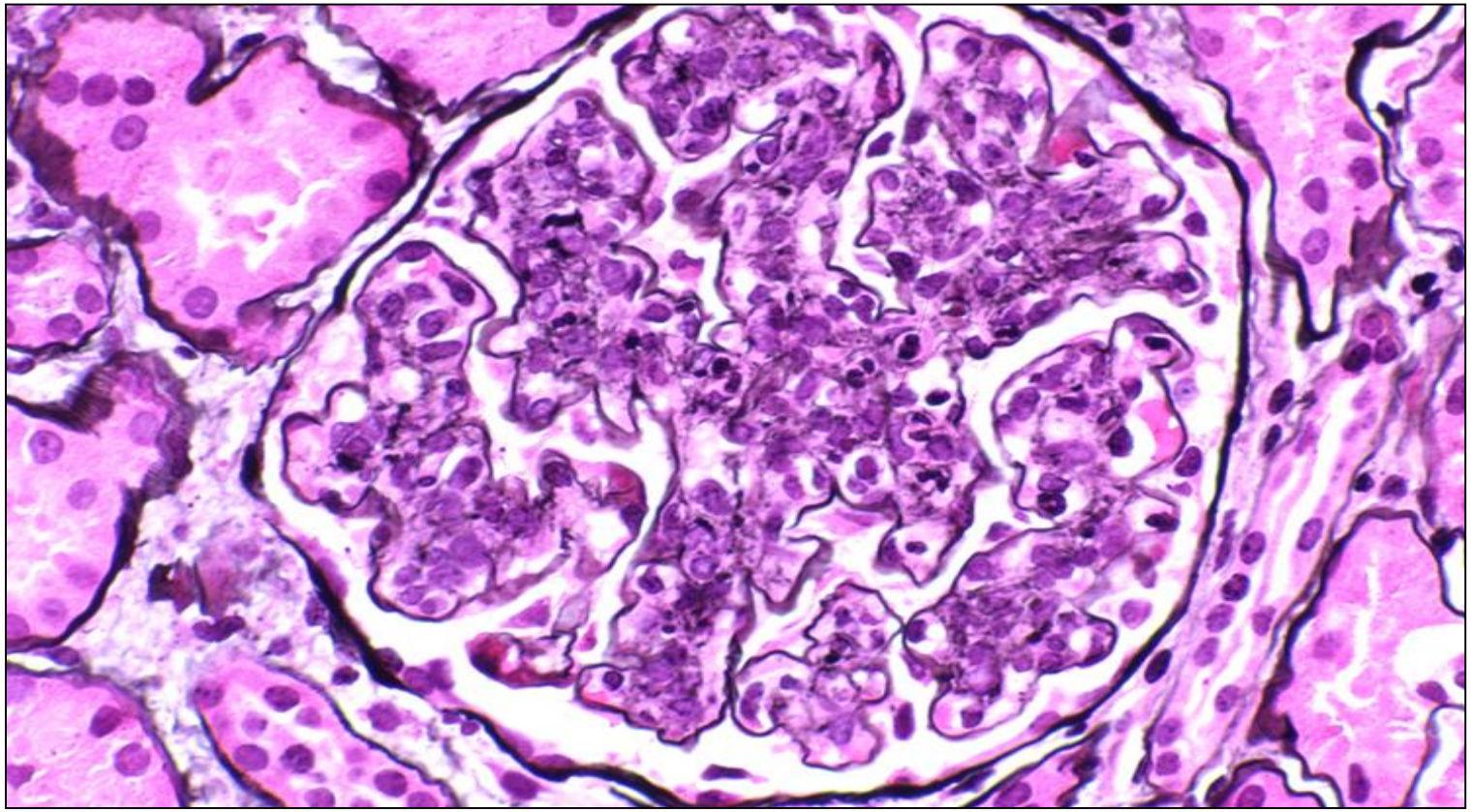
The hypercellularity of post-infectious glomerulonephritis is due to increased numbers of epithelial, endothelial, and mesangial cells as well as neutrophils in and around the glomerular capillary loops

Acute (Post-streptococcal) Glomerulonephritis



High power LM of a hypercellular glomerulus; numerous capillaries contain inflammatory cells, mostly neutrophils

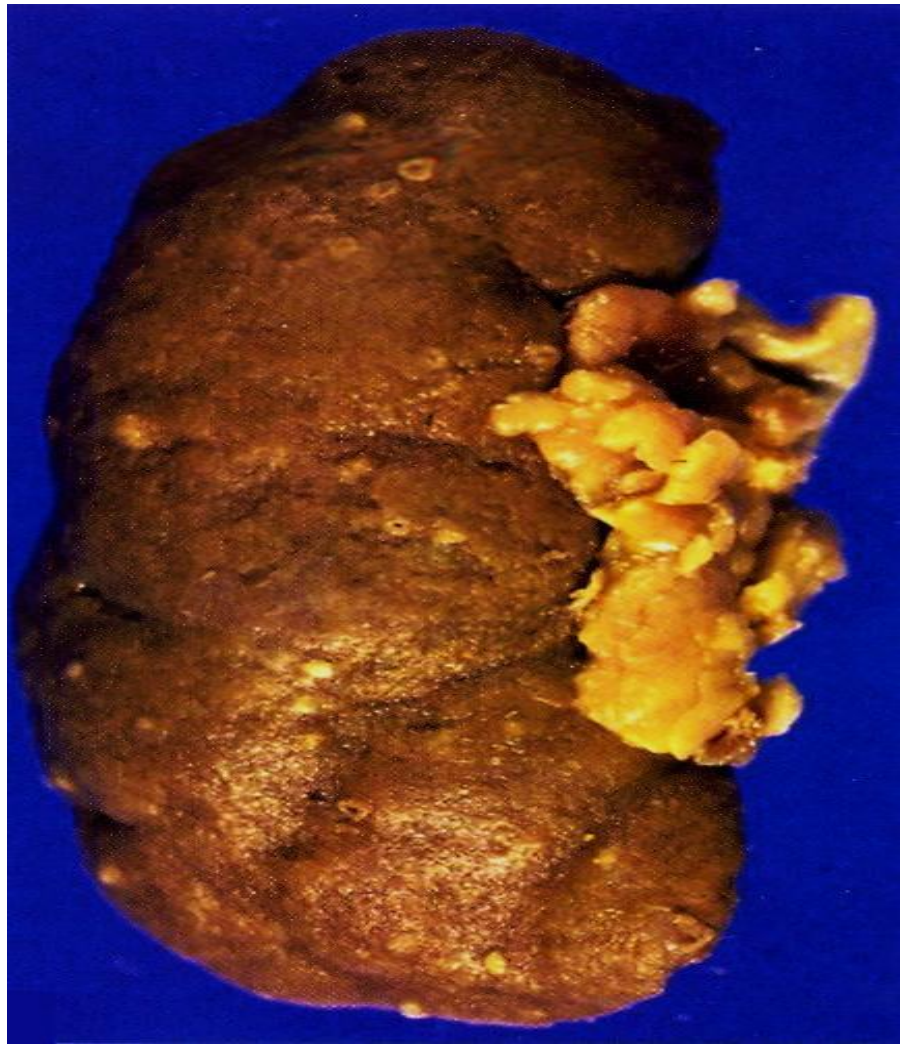
Acute Post-streptococcal Glomerulonephritis



Acute Poststreptococcal Glomerulonephritis is evident in this high-power silver stain with large number of PMNs. The glomerular basement membrane does not show splitting or spikes. There is proliferation of endothelial and mesangial cells and infiltrating cells and filling and distending capillary loops.

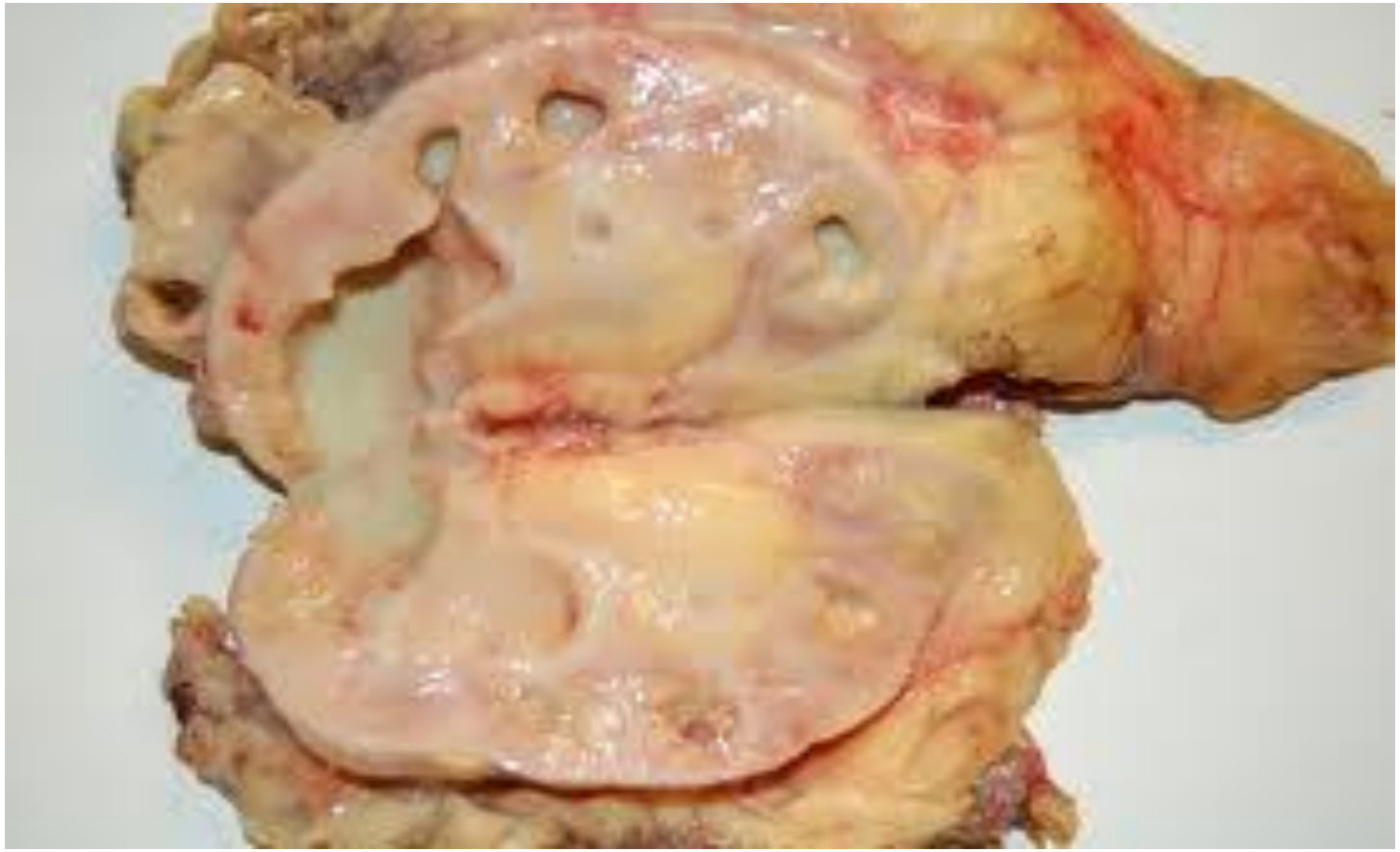
ACUTE & CHRONIC PYELONEPHRITIS

Pyelonephritis with small cortical abscesses



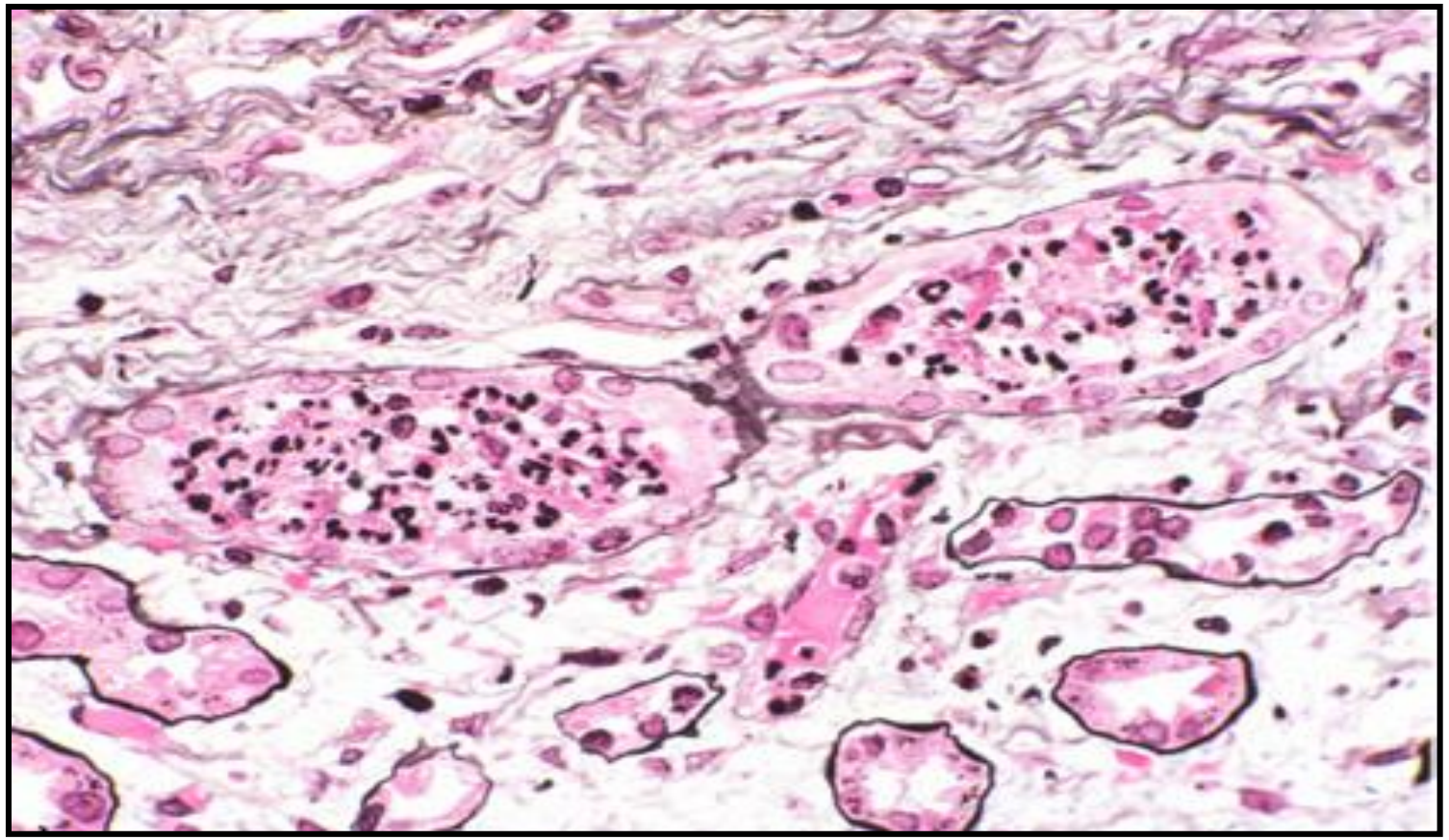
Pyelonephritis with small cortical abscesses

Classic picture of Pyelonephritis



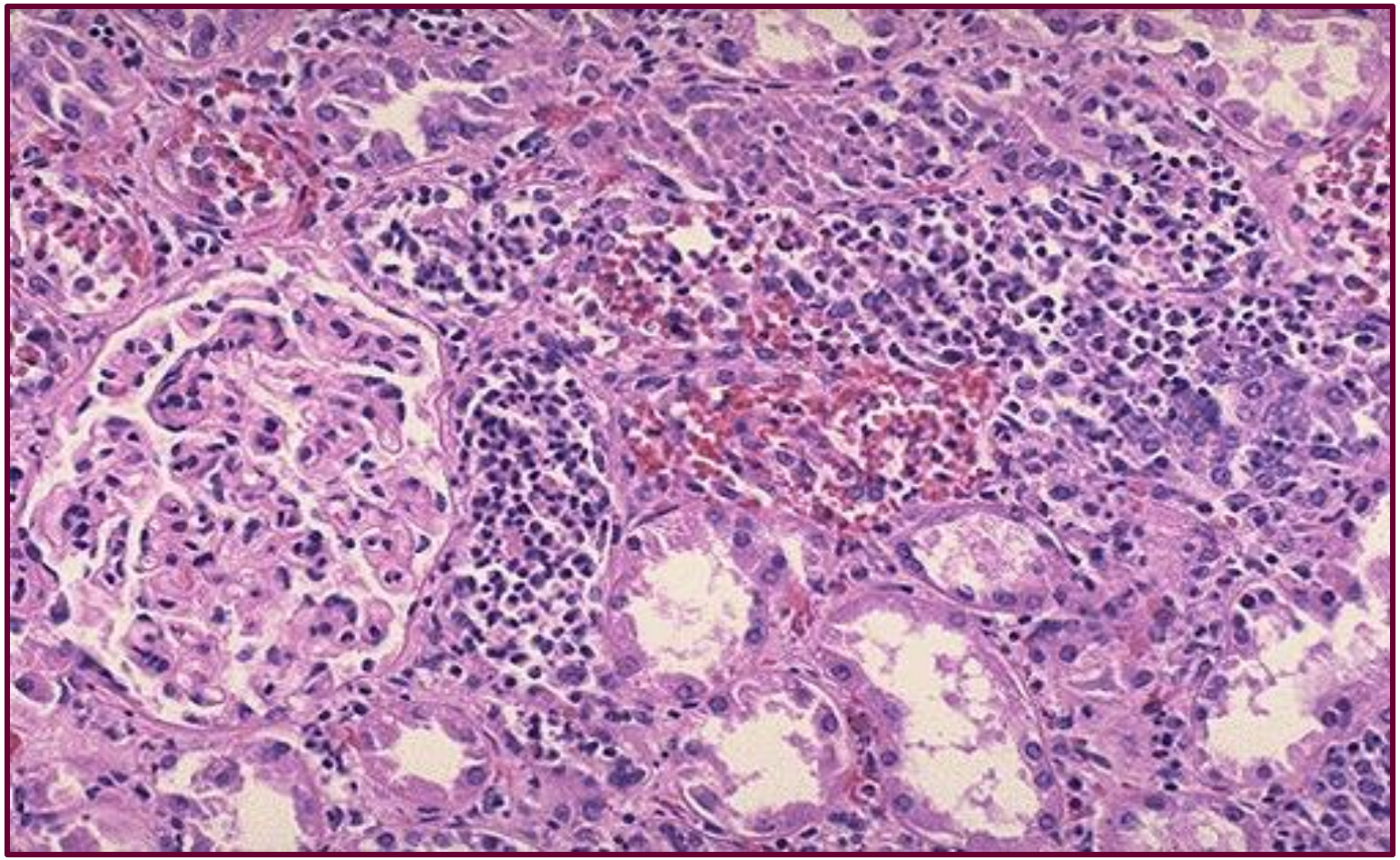
This kidney is bisected to reveal a dilated pelvis and calyxes filled with a yellow-green purulent pus which is consistent with a pyelonephritis. The cortex and medulla are pale and the corticomedullary junction is ill-defined.
No tumors are seen.

Acute Pyelonephritis - Histopathology



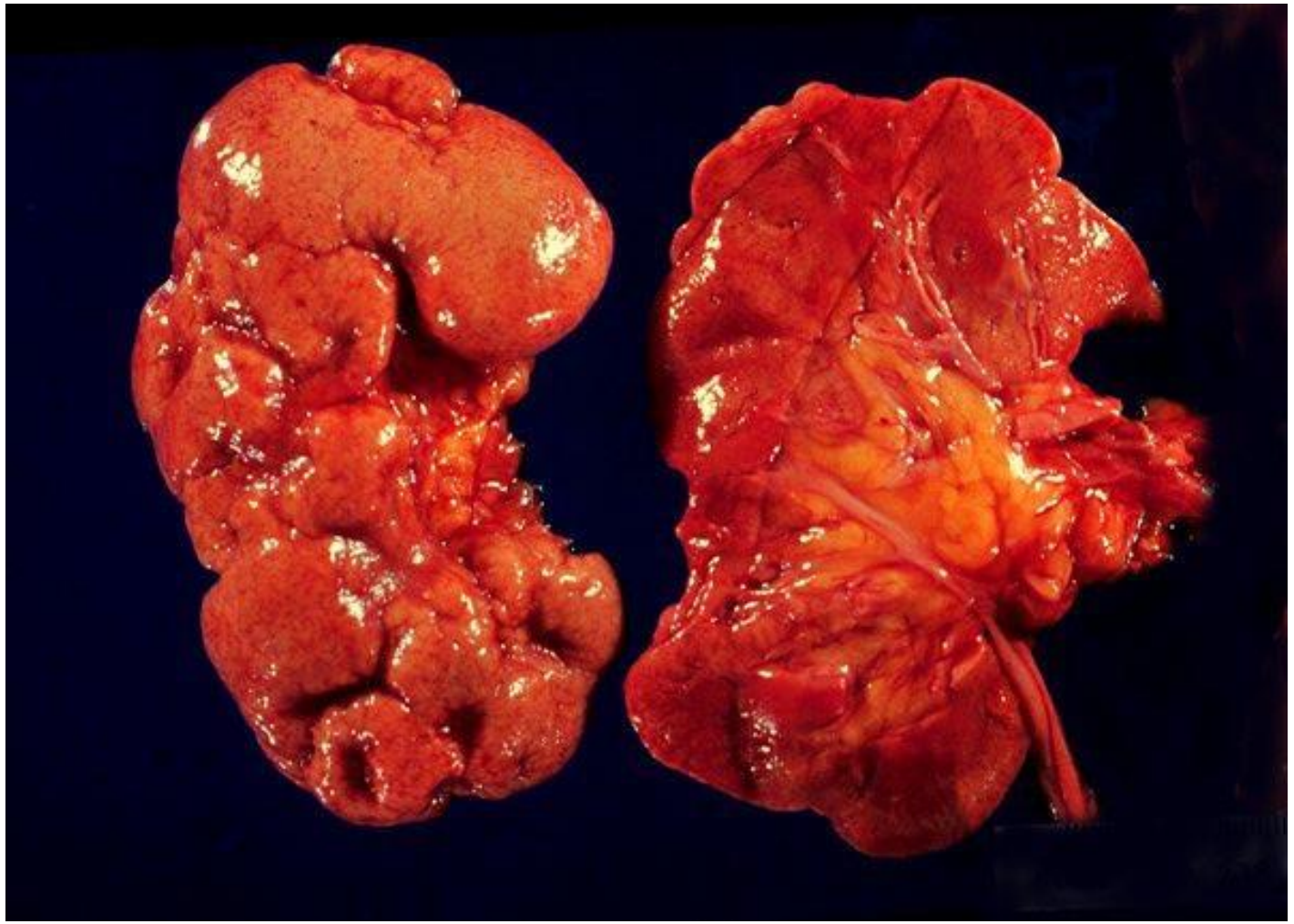
Acute pyelonephritis is diagnosed by intratubular aggregations of polymorphonuclear neutrophils (PMNs). There may be surrounding interstitial inflammation with a mixture of PMNs, lymphocytes, and plasma cells, but the predominant inflammation is within the tubule

Acute Pyelonephritis - Histopathology



Numerous PMN's are seen filling renal tubules across the center and right of this picture. These leukocytes may form into a cast within the tubule. Casts appearing in the urine originate in the distal renal tubules and collecting ducts

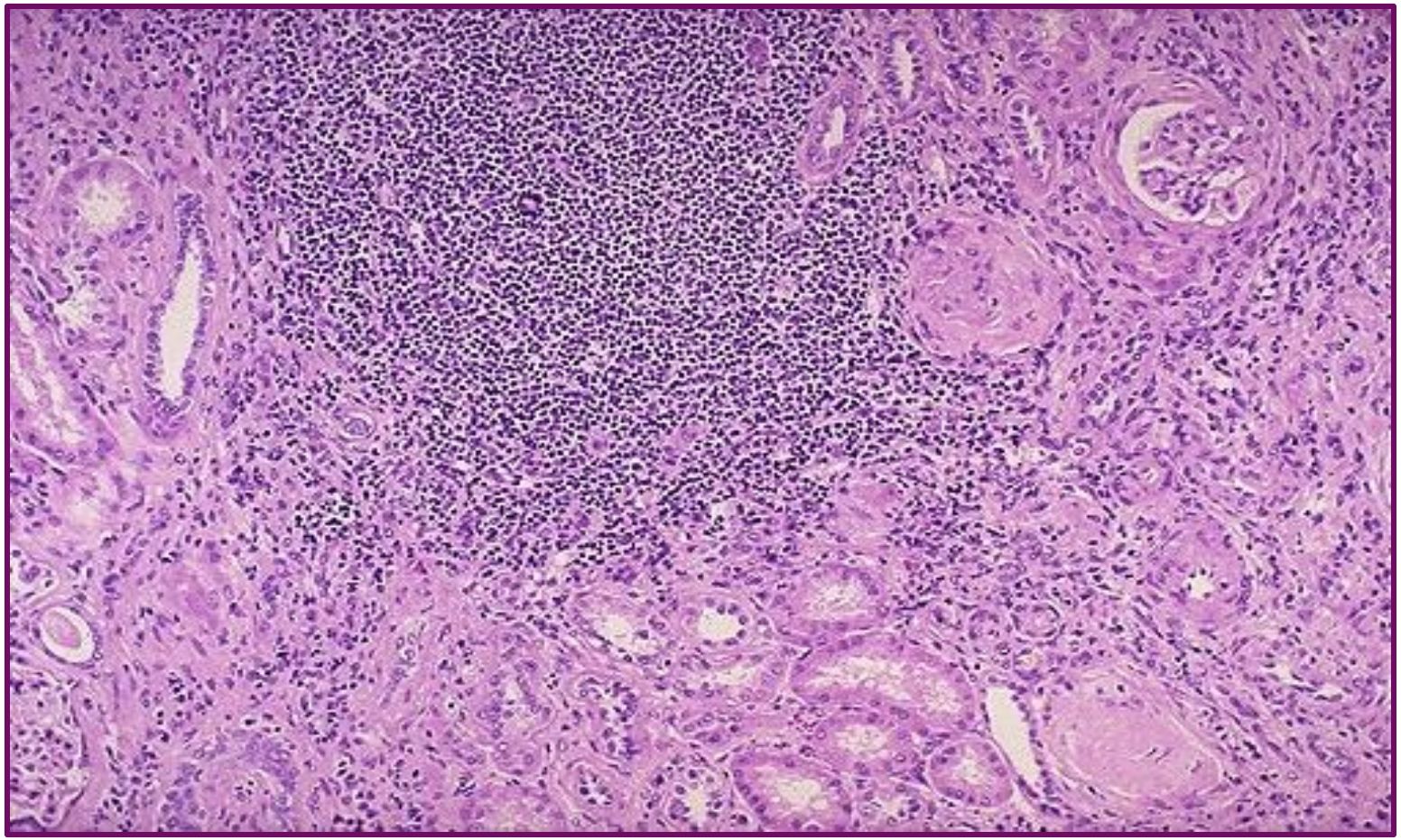
Chronic Pyelonephritis - Gross Pathology



a- Deformed and atrophic kidneys.

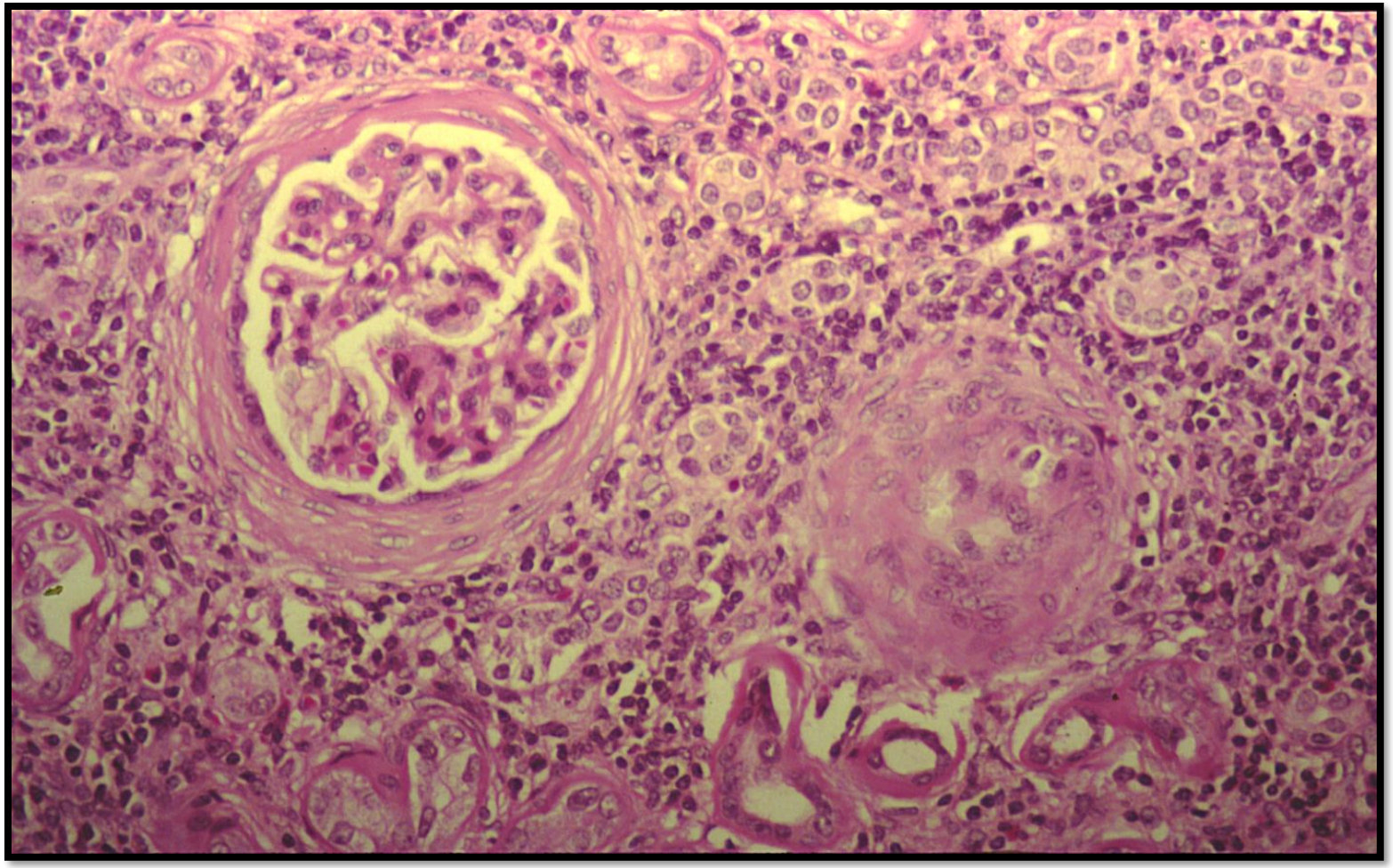
b- Deep cortical scars.

Chronic Pyelonephritis - Histopathology



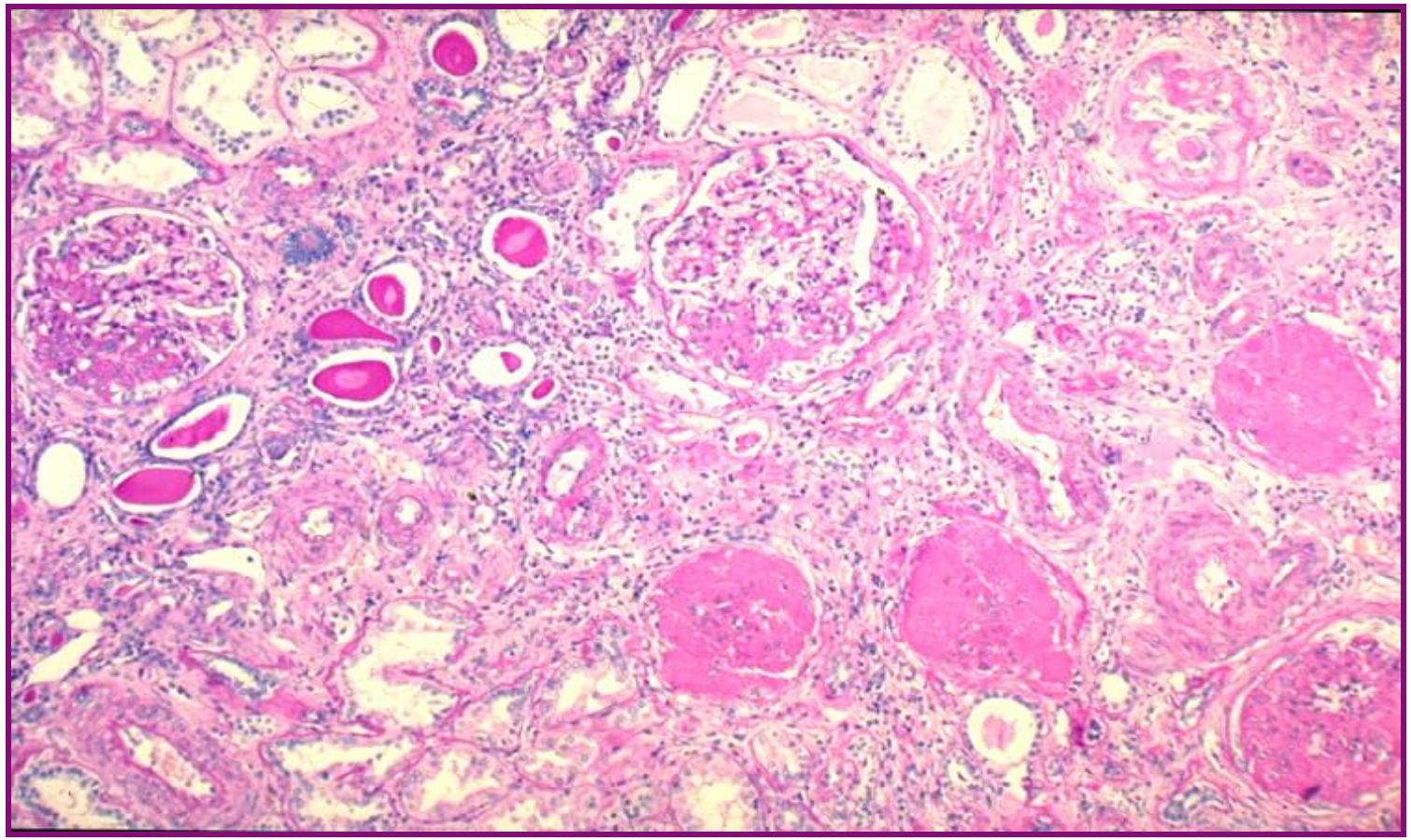
This is chronic pyelonephritis where a large collection of chronic inflammatory cells . The severity of disease depends upon the amount of remaining functional renal parenchyma

Chronic Pyelonephritis - Histopathology



High power shows periglomerular fibrosis , glomerular sclerosis and hyalinization with marked chronic interstitial inflammation .

Chronic Pyelonephritis - Histopathology



a- Chronic interstitial inflammation.

b- Atrophy and thyroidization of renal tubules.

c- Hyalinization of glomeruli and interstitial fibrosis.

Predisposing factors:

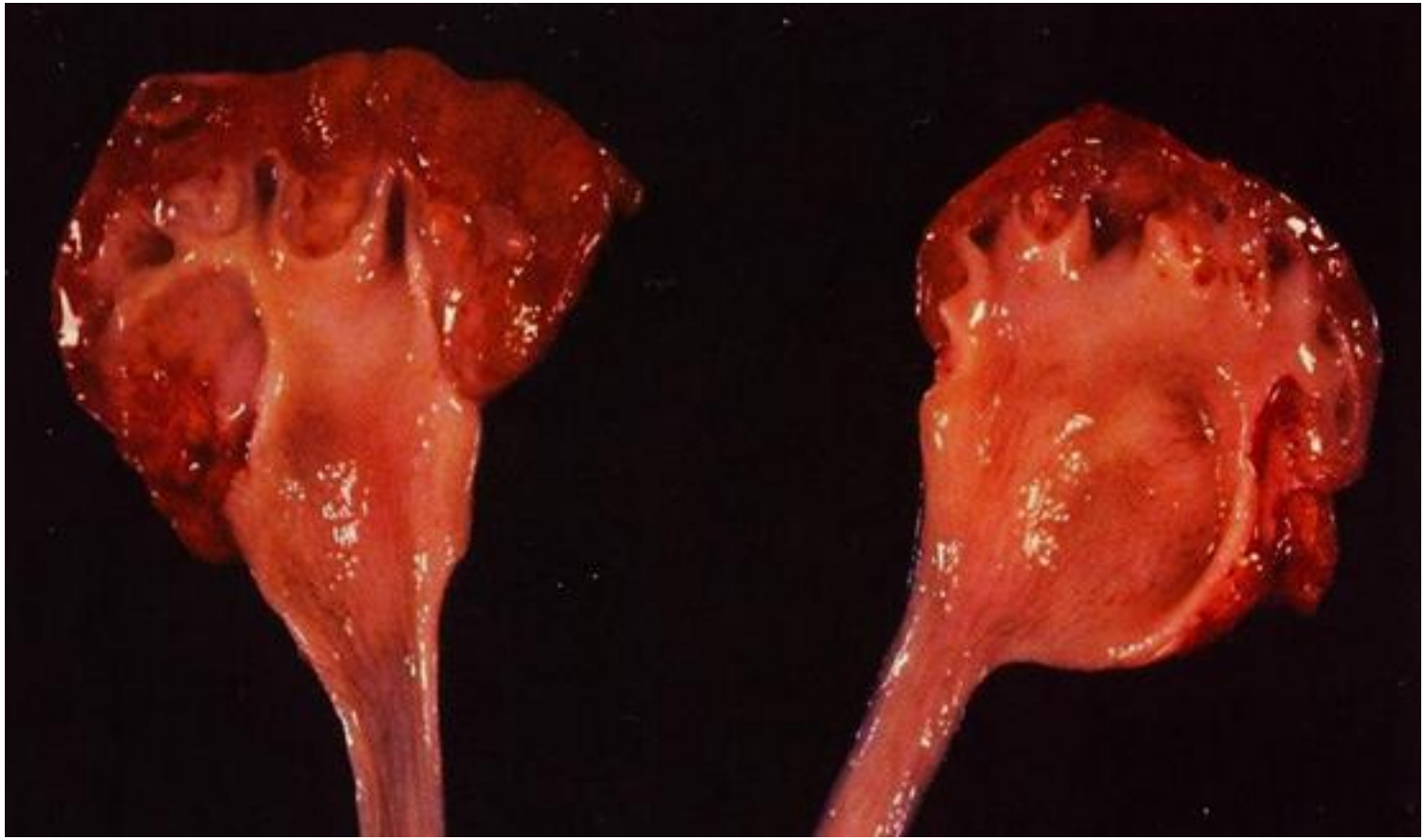
-Recurrent attacks of acute pyelonephritis.

-Drug-induced interstitial nephritis.

- Urinary tract obstruction or reflux.

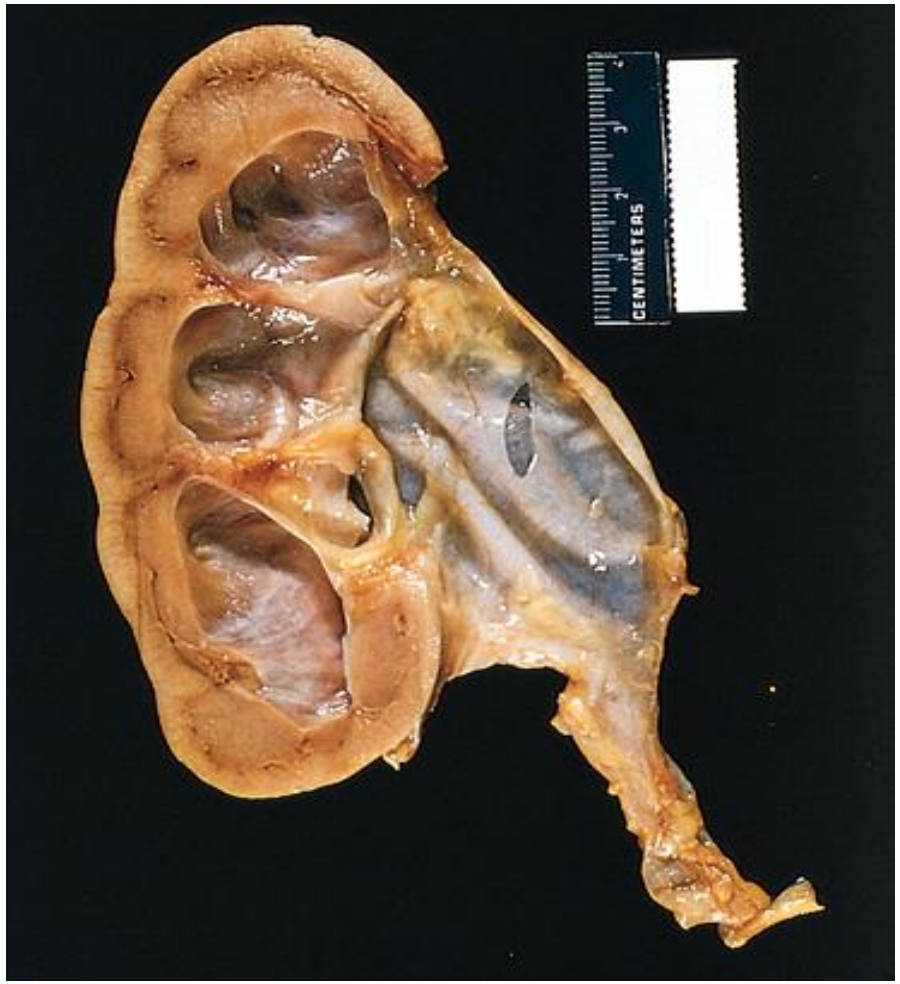
HYDRONEPHROSIS

Hydronephrosis



Bisected kidney shows markedly dilated renal pelvis and calyces with atrophic and thin renal cortex /parenchyma

Hydronephrosis

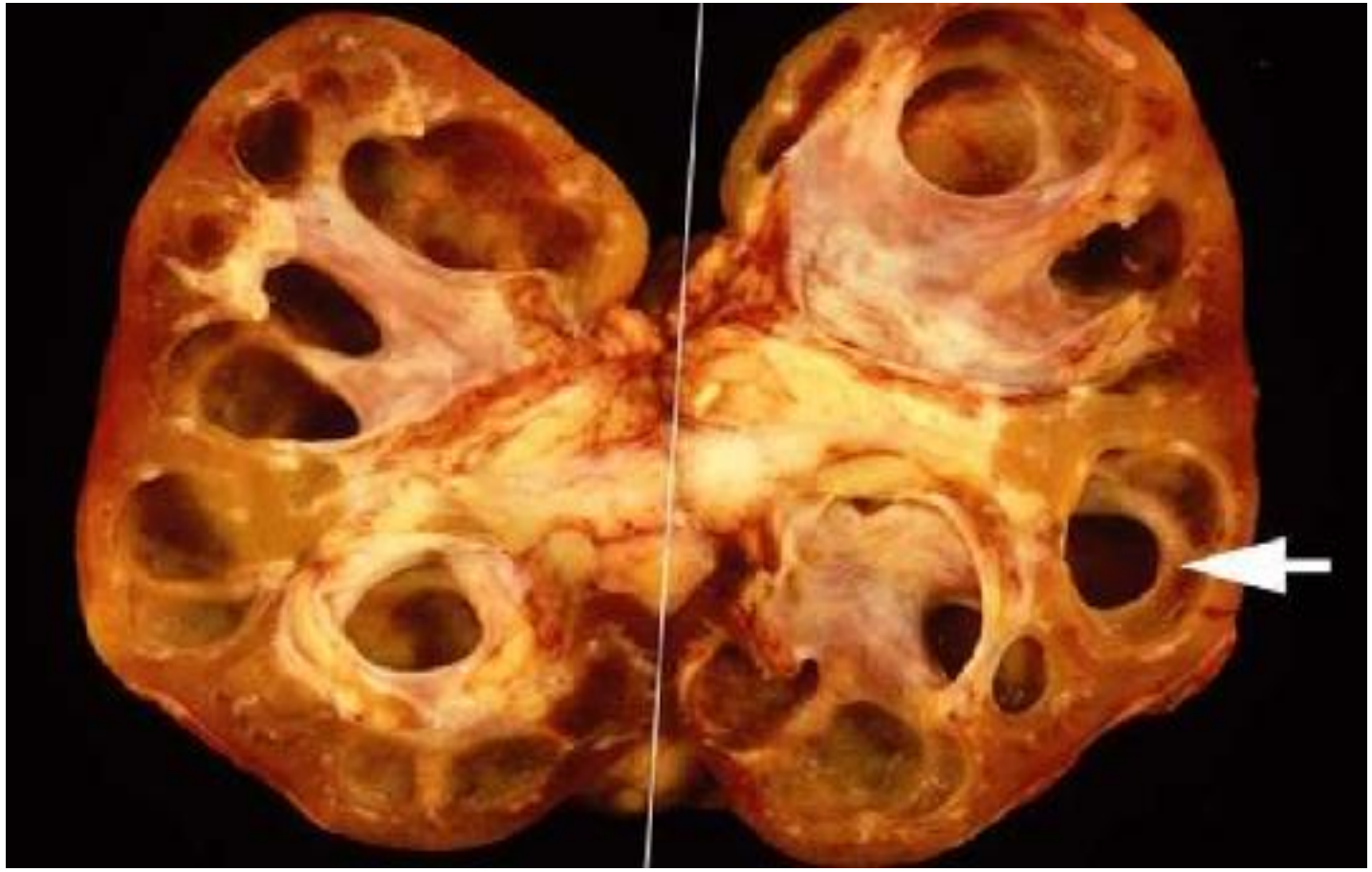


The picture shows markedly dilated renal pelvis and calyces with atrophic and thin renal cortex /parenchyma

The most common causes are:

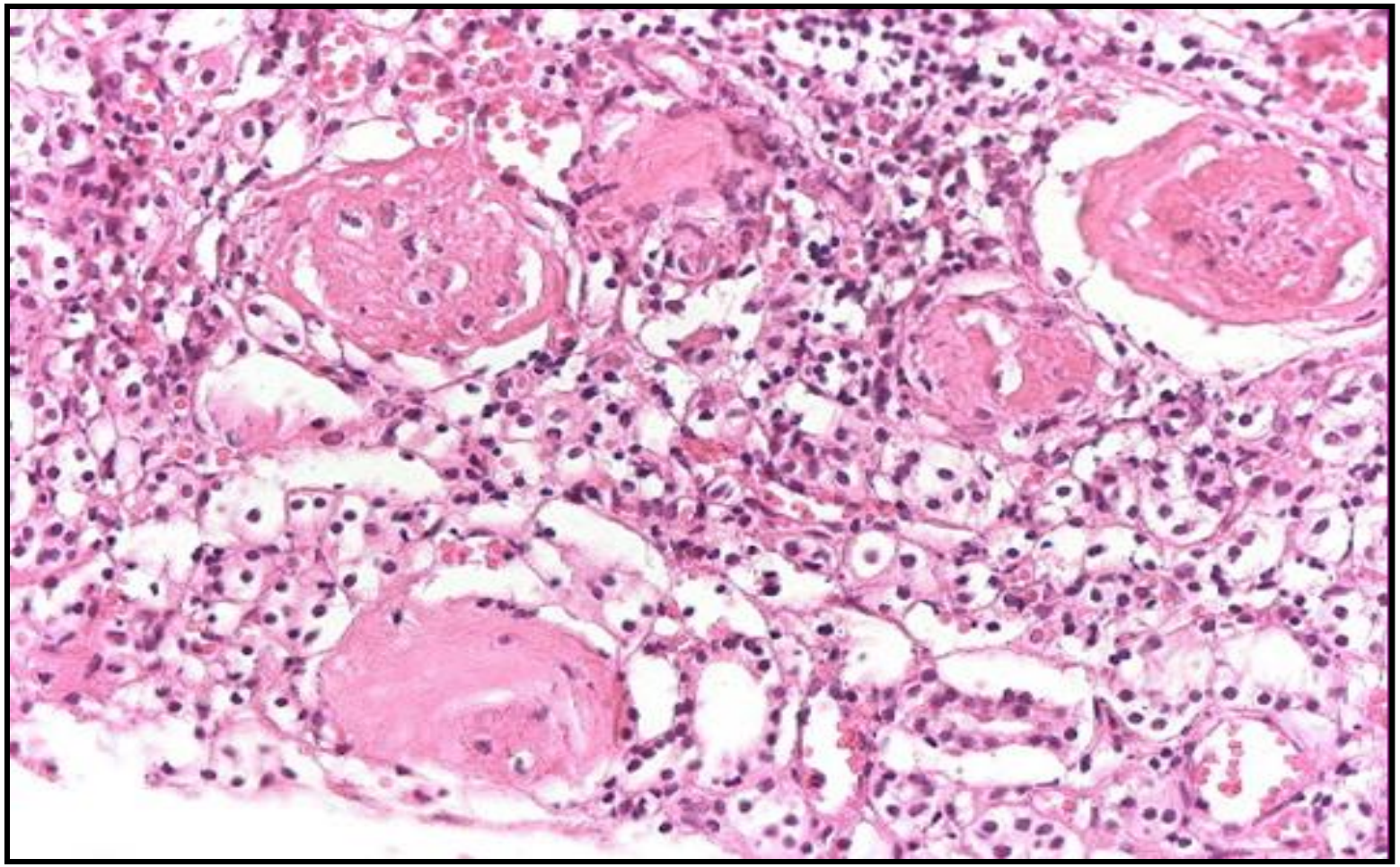
- *Foreign bodies like calculi with obstruction,*
- *Atresia of the urethra,*
- *Benign prostatic hyperplasia ,*
- *Neoplasia of the prostate and bladder*
- *Spinal cord damage with paralysis of the bladder .*

Hydronephrosis



Markedly dilated renal pelvis and calyces with atrophic and thin renal cortex

Chronic Pyelonephritis presenting as complication to Hydronephrosis



Thinning renal parenchyma with residual large renal vessels in the hilum. Sclerosis of glomeruli with atrophic tubules