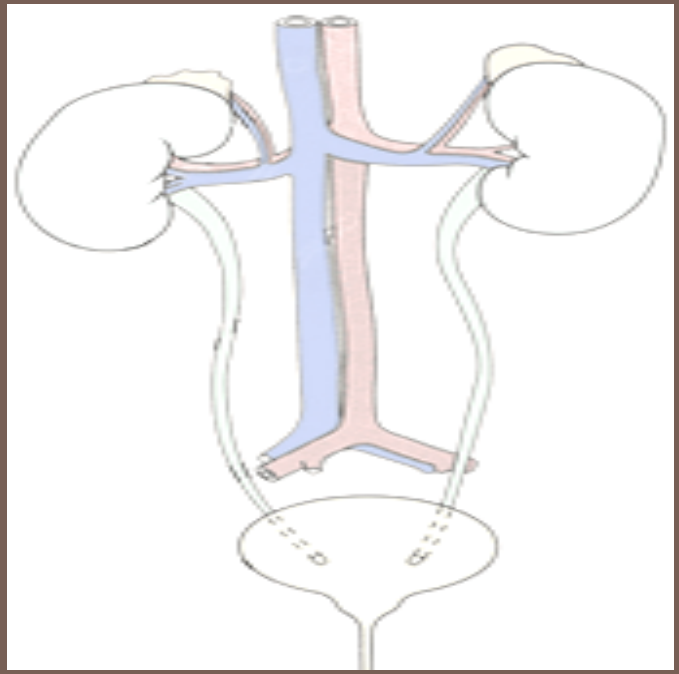
RENAL BLOCK



PATHOLOGY PRACTICAL

Prepared by:

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- *Dr. Sayed Al Esawy*

Head of Pathology Department: Dr. Hisham Al Khalidi

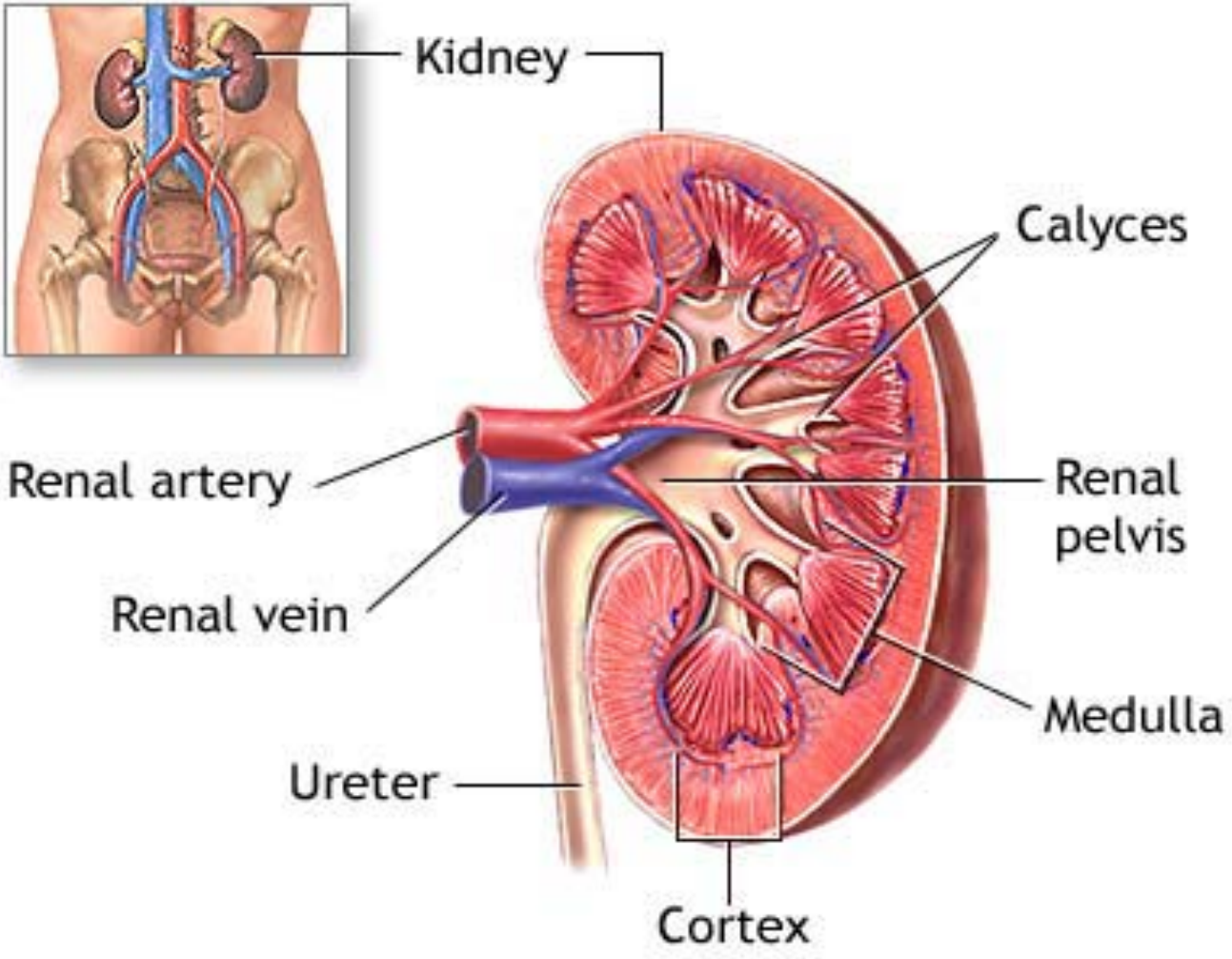
NORMAL ANATOMY AND HISTOLOGY

Objectives:

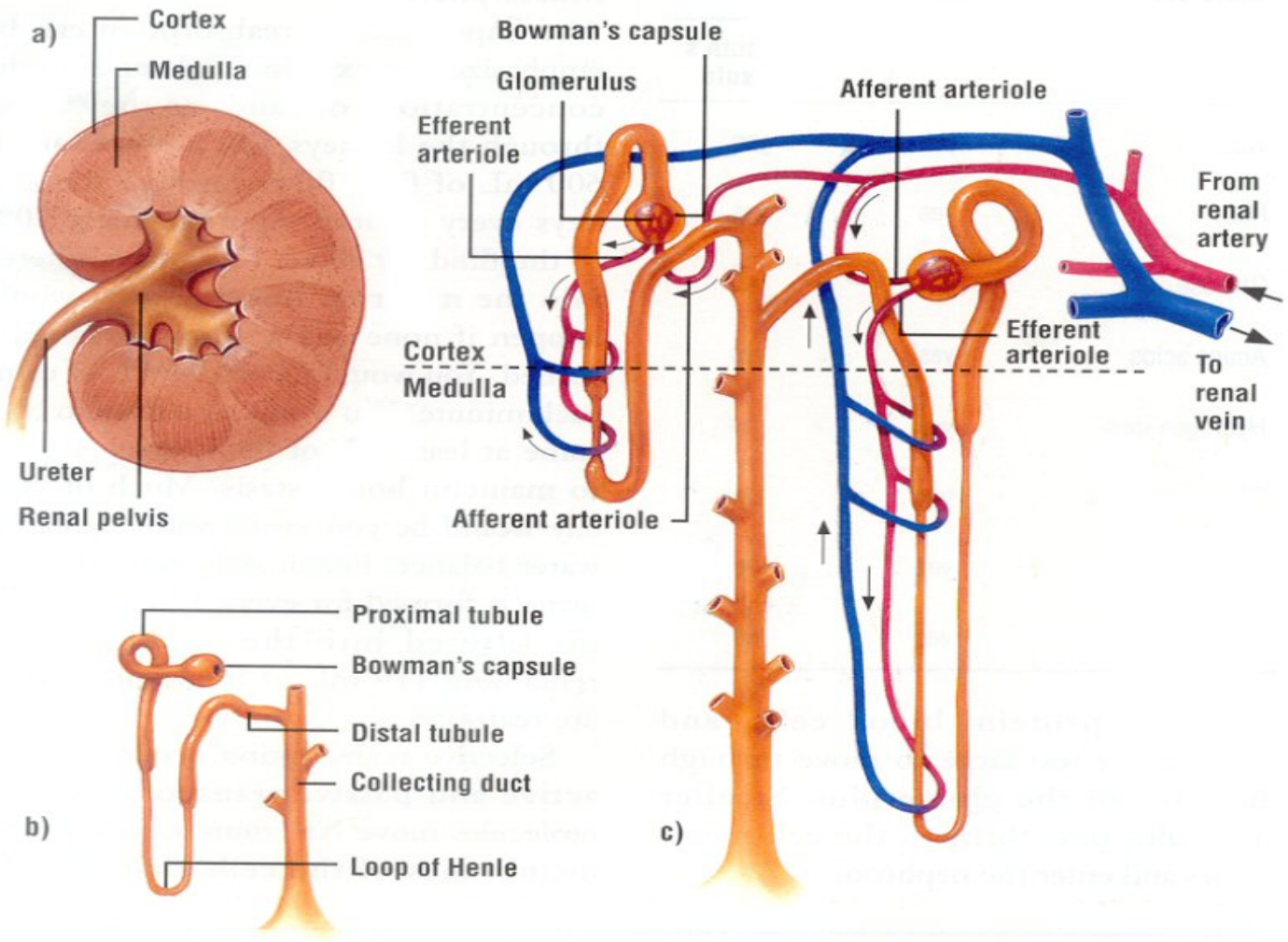
At the end of the practical sessions for the renal block, the students will be able to:

- Describe the normal constituents of the nephron, renal glomeruli and tubules.
- Identify the gross and microscopic features of:
  - Post-streptococcal glomerulonephritis.
  - Renal hydronephrosis, pyonephrosis & polycystic kidney.
  - Acute & chronic pyelonephritis.
  - Nephrotic and nephritic Syndrome.
  - Wilm's tumor.
  - Renal cell carcinoma, urothelial carcinoma of the urinary bladder.
  - Clear cell carcinoma of the kidney.
  - Pathology of renal allograft.

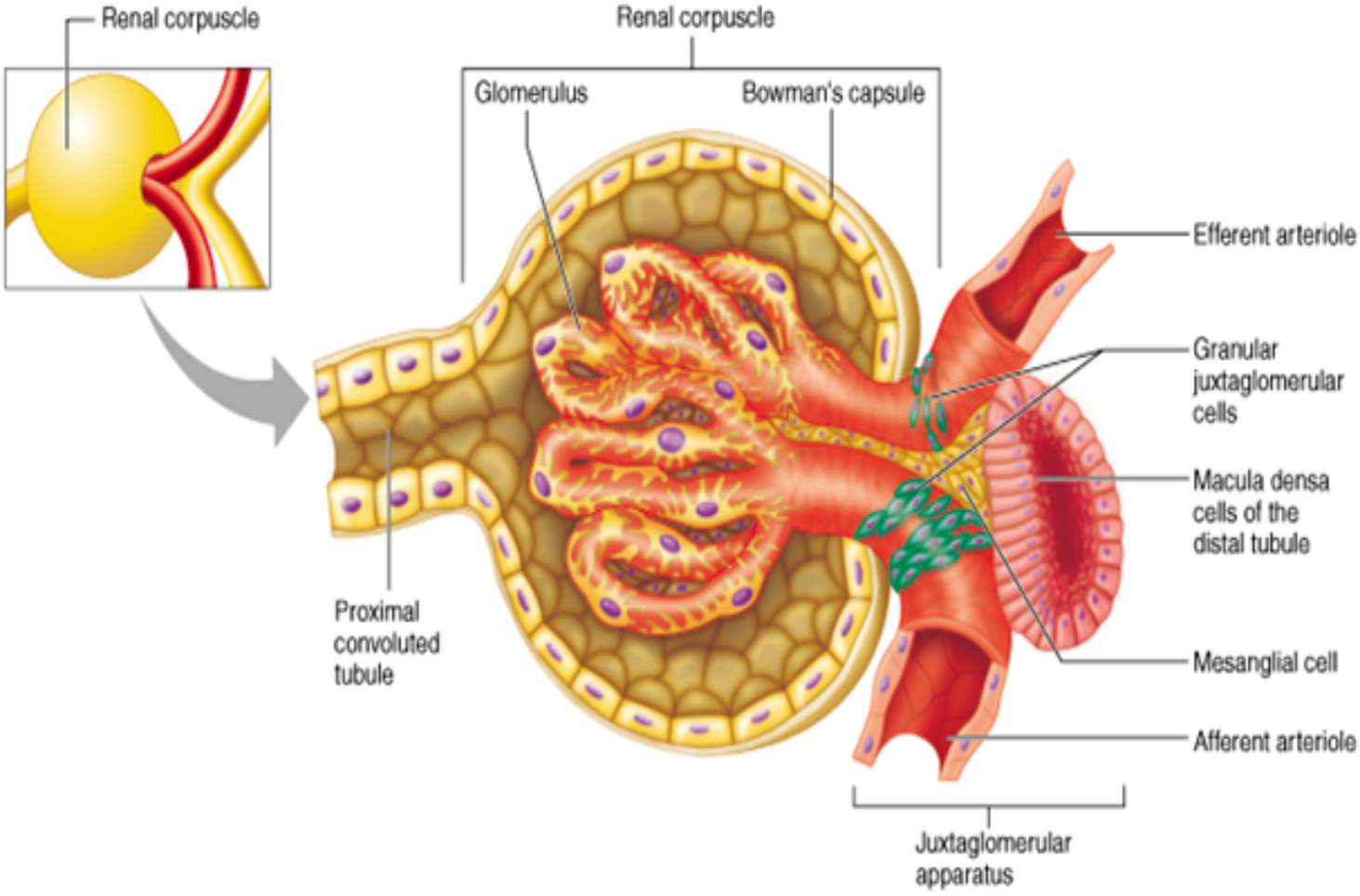
Anatomy of the Kidney



NEPHRON STRUCTURE



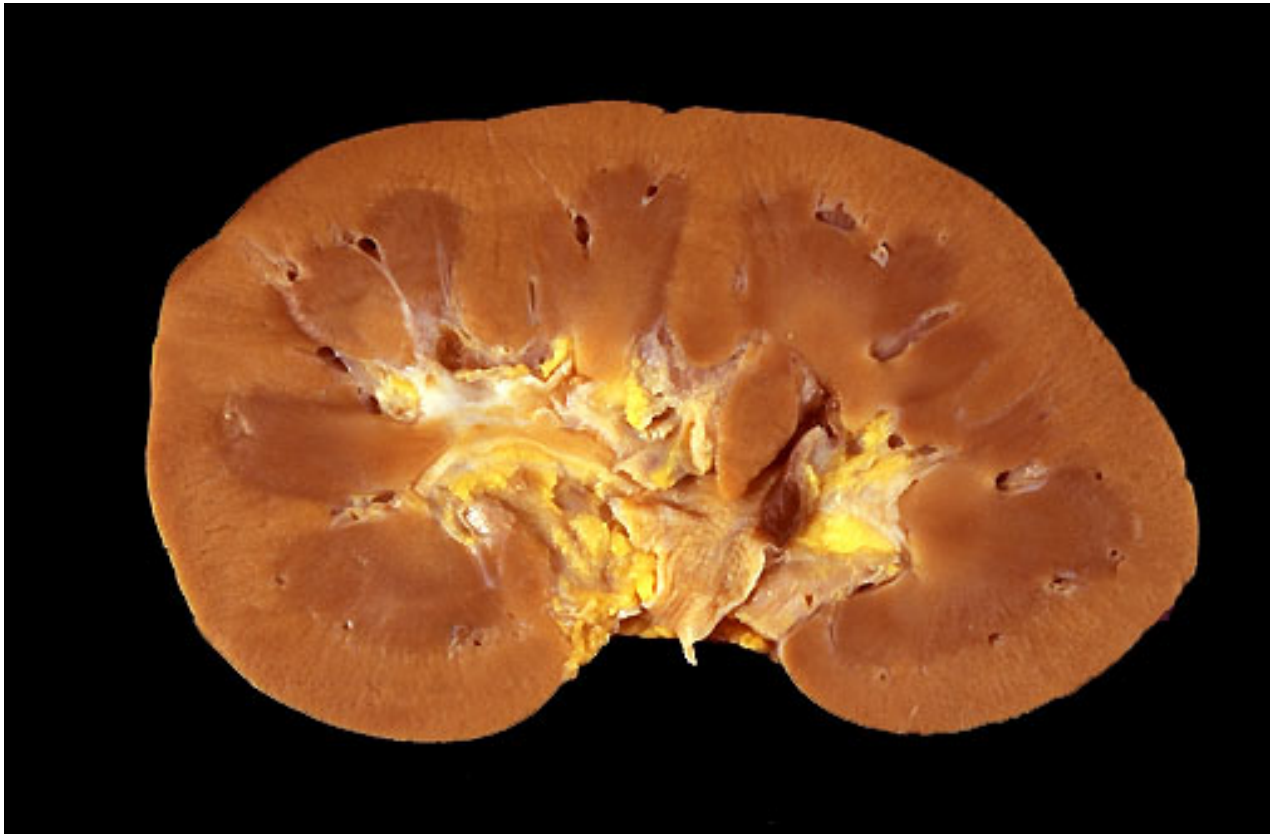
Renal Corpuscle



KIDNEY ANATOMY : NEPHRONS

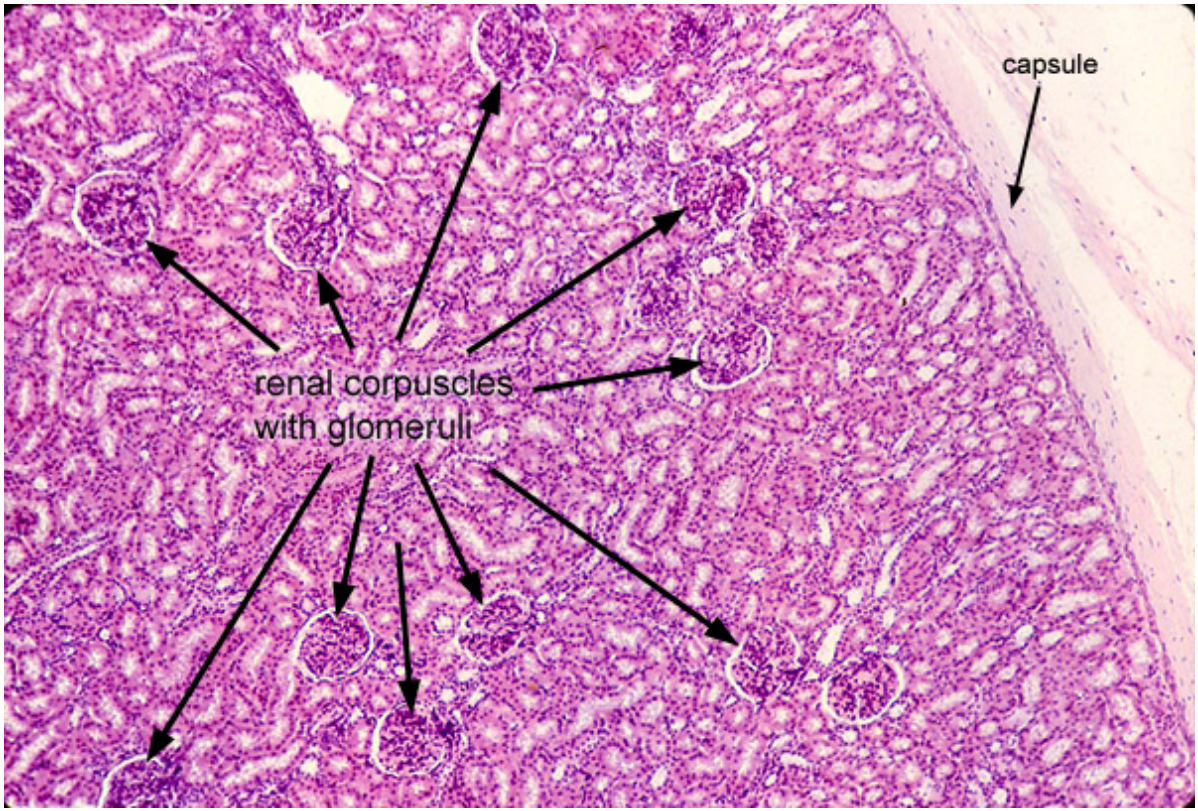
- *Nephron is the functional unit of the kidney.*
- *Each kidney contains about 1,000,000 to 1,300,000 nephrons.*
- *The nephron is composed of glomerulus and renal tubules .*
- *The nephron performs its function by ultra filtration at glomerulus and secretion and reabsorption at renal tubules.*

Normal Kidney - Gross

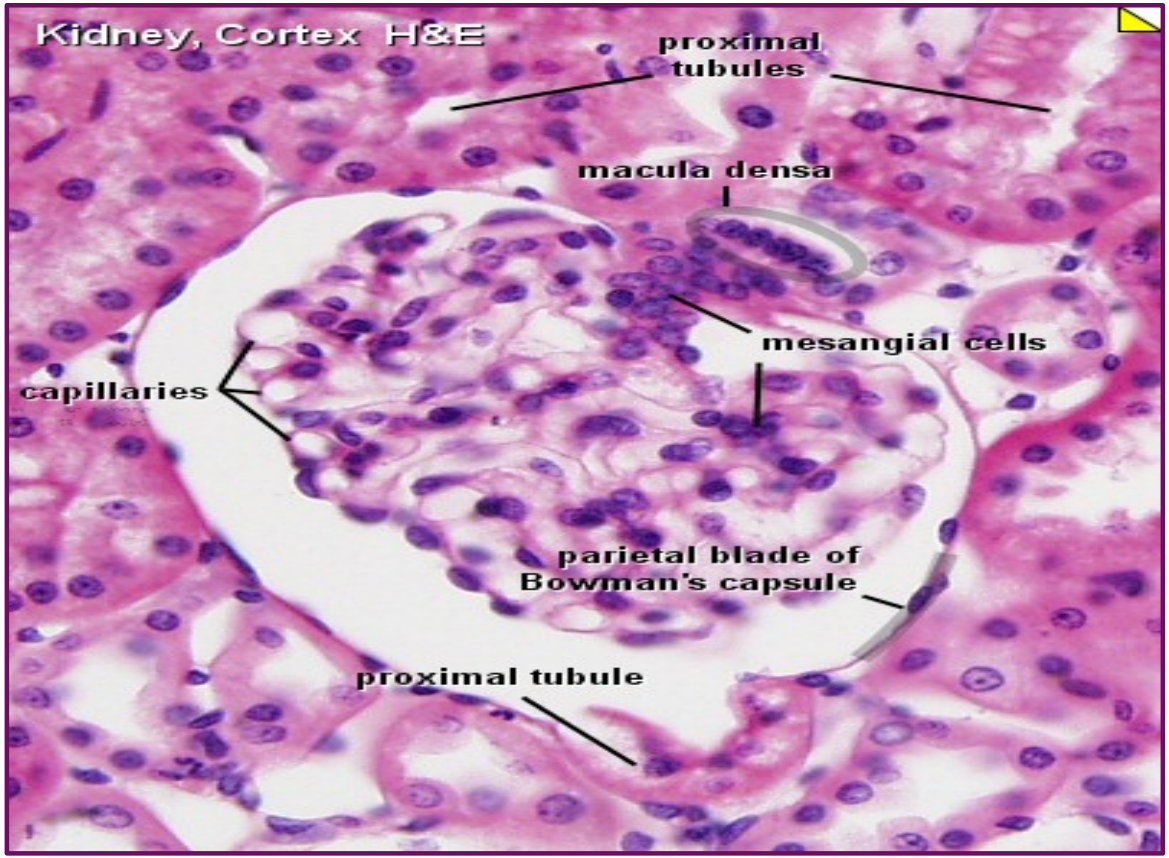


In cross section, this normal adult kidney demonstrates the lighter outer cortex and the darker medulla, with the renal pyramids into which the collecting ducts coalesce and drain into the calyces and central pelvis.

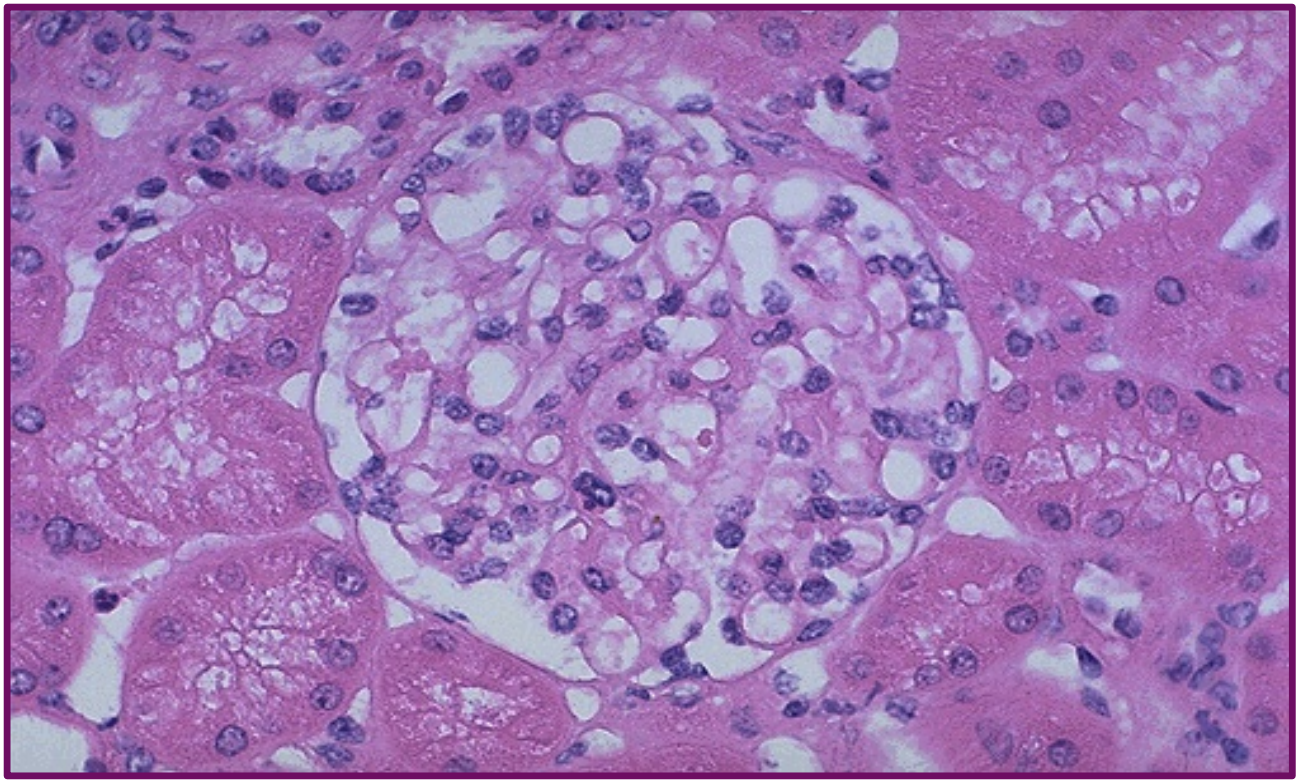
Kidney – Normal Histology



Renal Corpuscle – Normal Histology

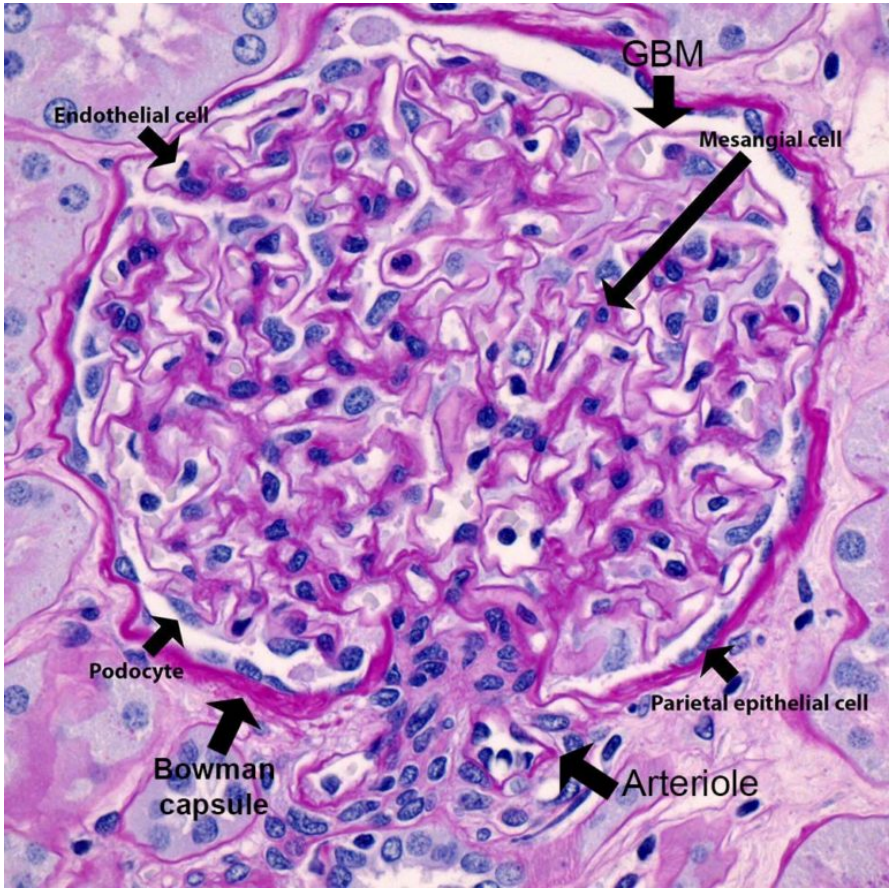


Renal Corpuscle – Normal Histology



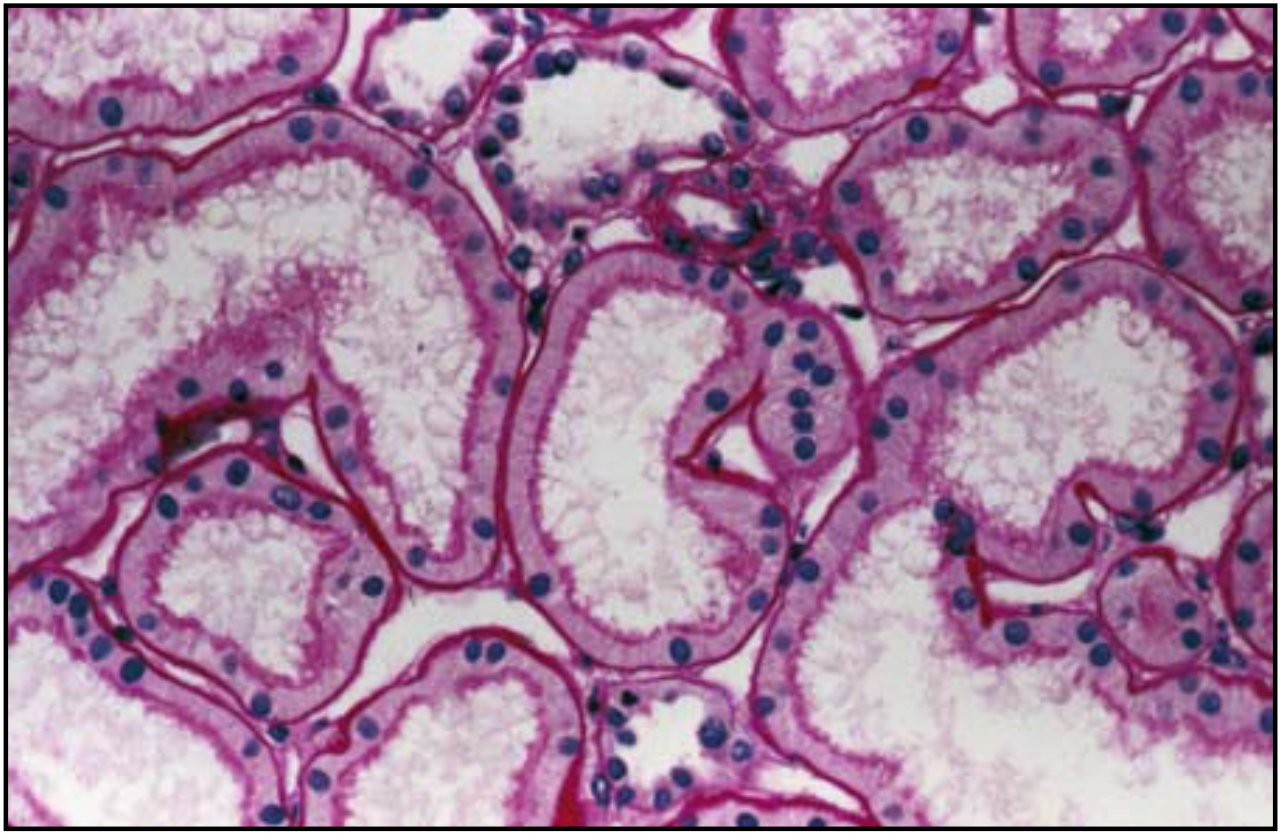
Normal glomerulus by light microscopy. The glomerular capillary loops are thin and delicate. Endothelial and mesangial cells are normal in number. The surrounding tubules are normal

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:

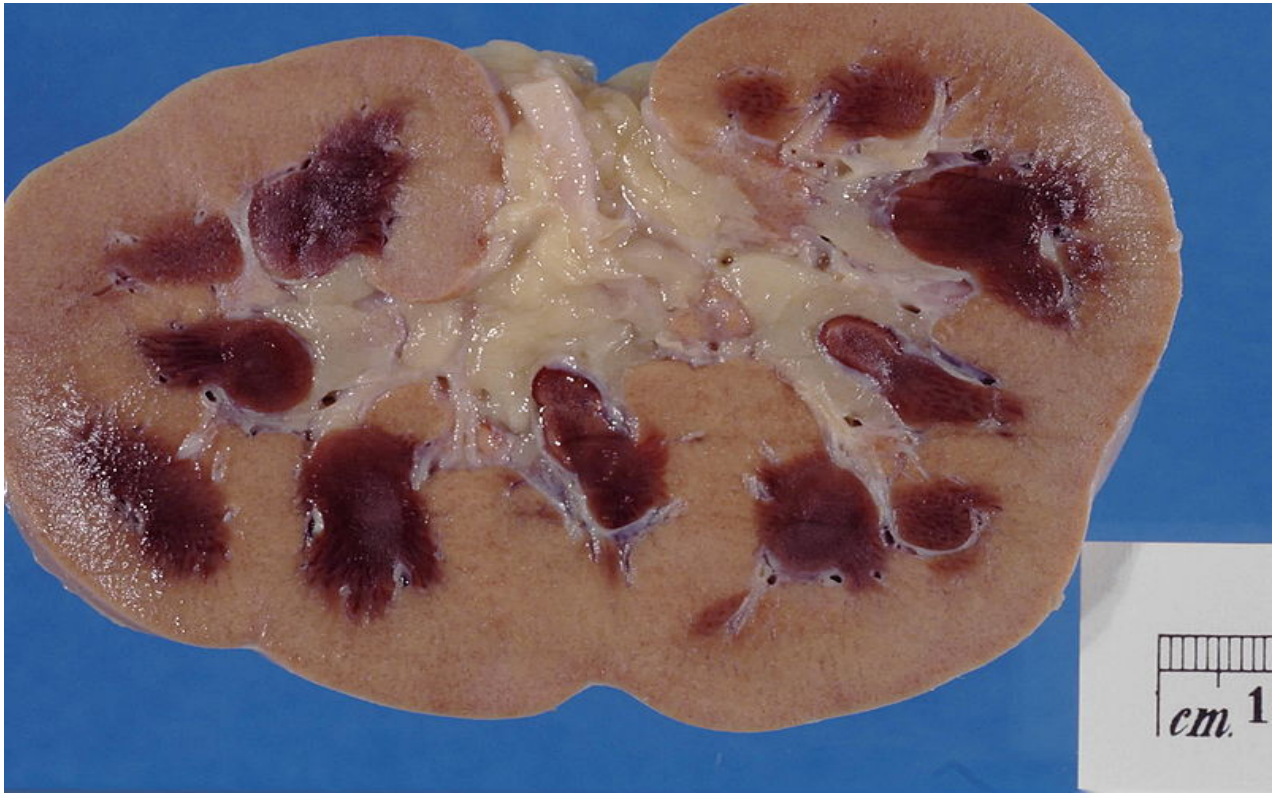
- *Glomerulonephritis (GN).*
- *Acute tubular necrosis (ATN).*
- *Acute interstitial nephritis (AIN).*

Post-renal:

(is a consequence of urinary tract obstruction)

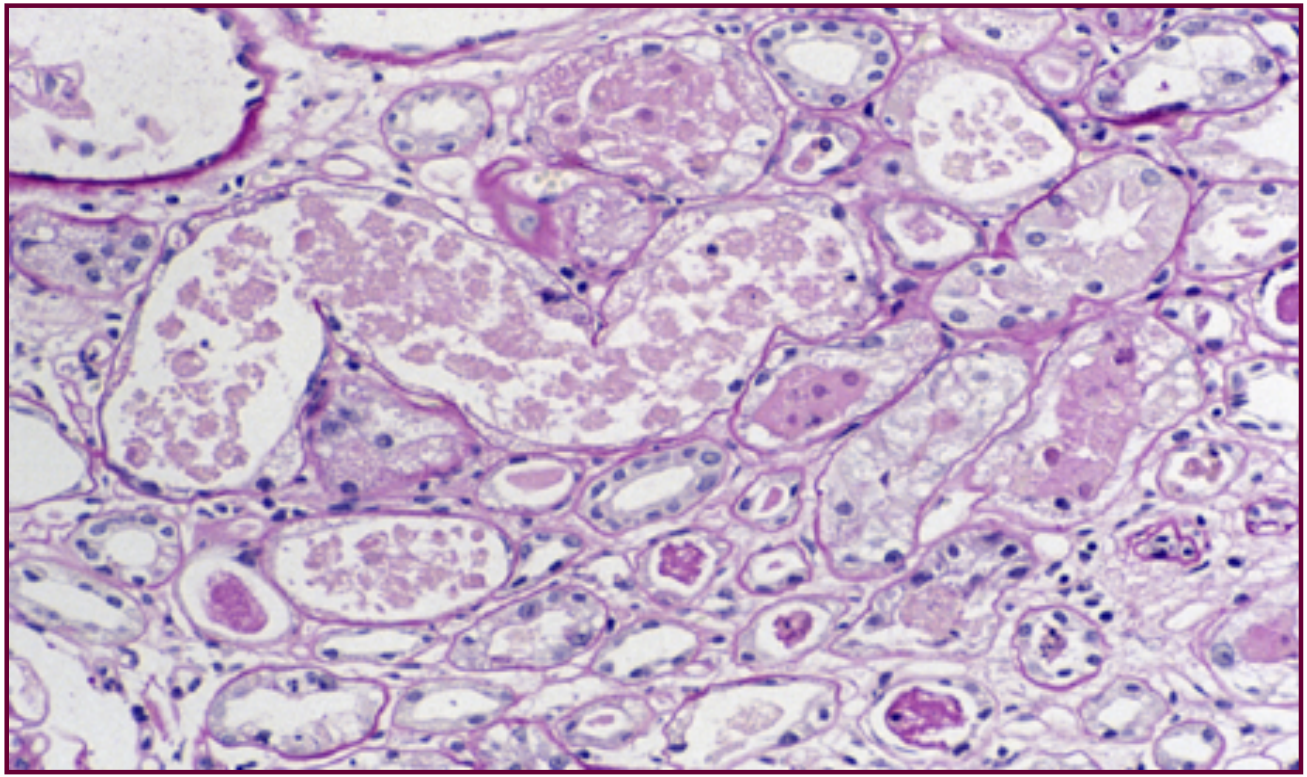
- *Benign prostatic hyperplasia.*
- *Kidney stones.*
- *Obstructed urinary catheter.*
- *Bladder stone .*
- *Bladder, ureteral or renal malignancy.*

Acute Kidney Injury



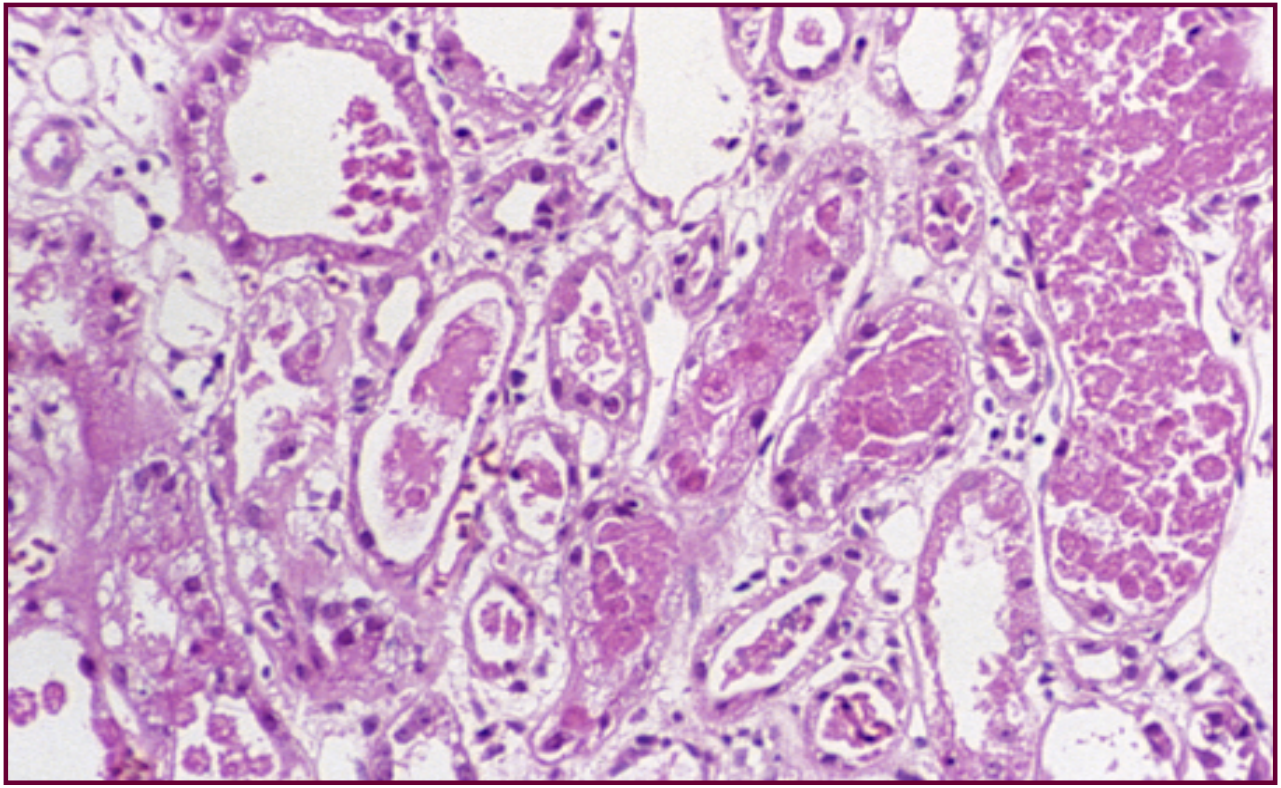
Kidney showing marked pallor of the cortex, contrasting to the darker areas of surviving medullary tissue.

Acute Tubular Necrosis



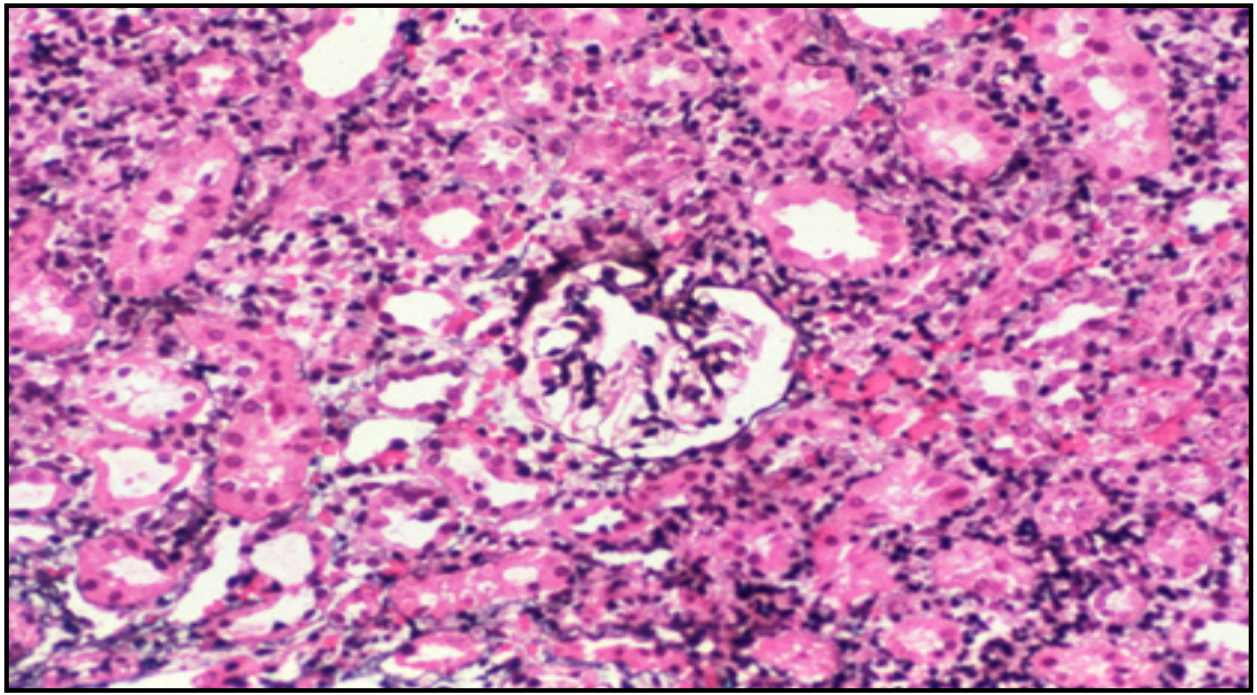
Acute tubular necrosis is manifest by vacuolated cells and sloughed, necrotic cells in tubular lumina, with some tubules lined by flattened epithelium and some showing frank necrosis (PAS stain, x 400).

Acute Tubular Necrosis



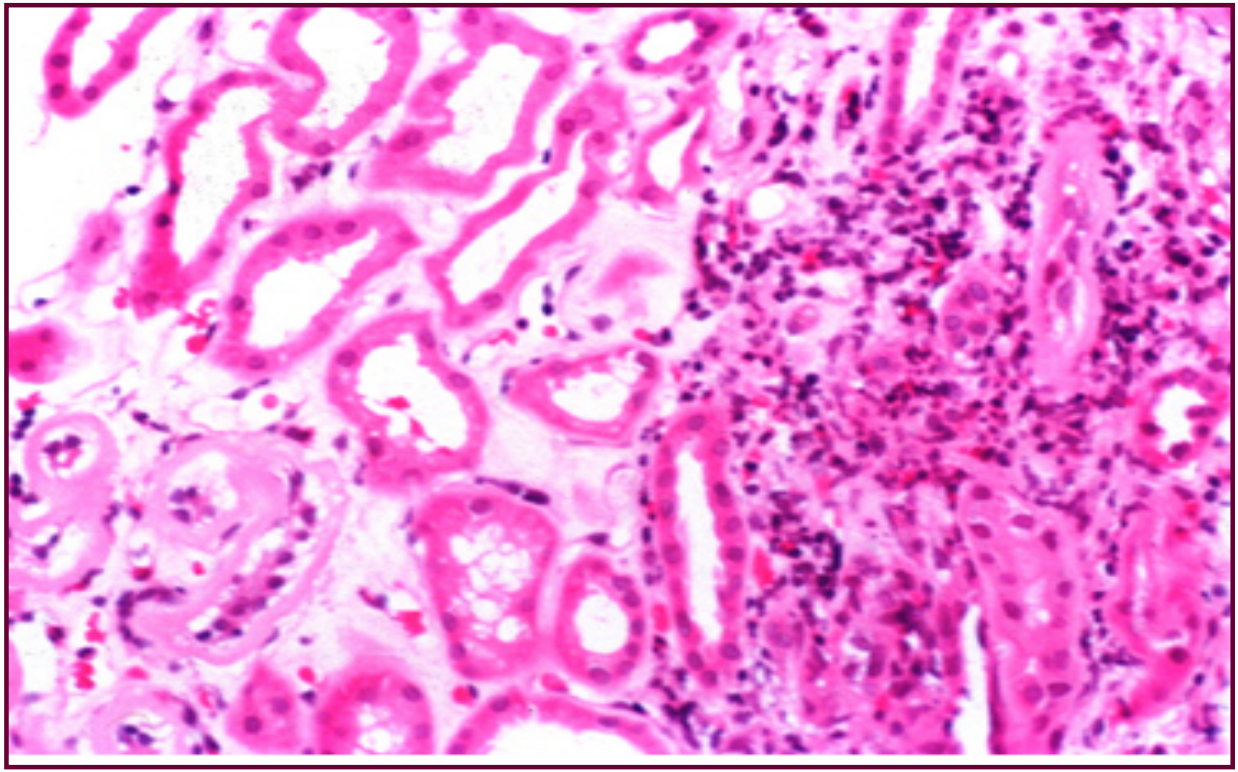
There may also be degeneration and frank necrosis of individual cells or tubular segments in acute tubular necrosis, or flattened, regenerating type epithelium with degenerated cells in the lumen (middle left) (H&E x 200).

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

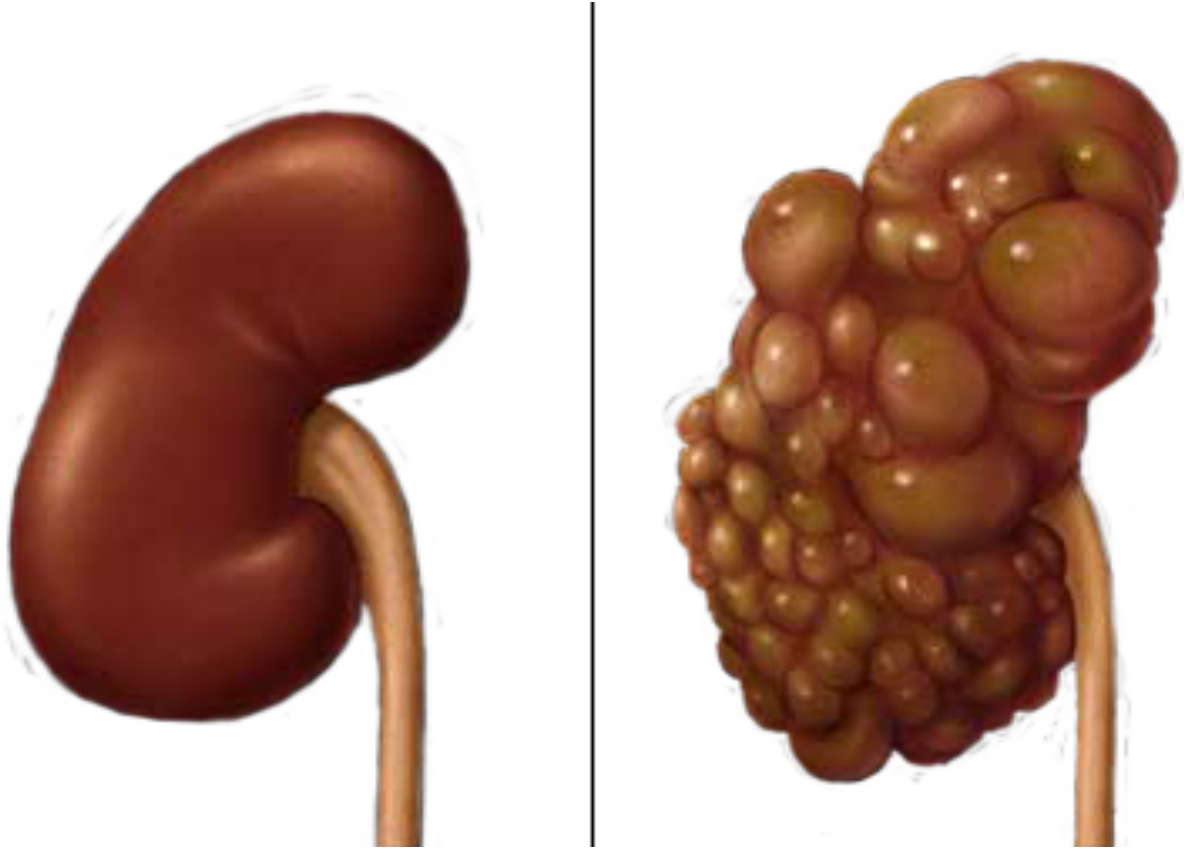
Acute Interstitial Nephritis (Drug-Induced Interstitial Nephritis)



- *Eosinophils in interstitium.*
- *Interstitial oedema.*

POLYCYSTIC KIDNEY

Normal vs Polycystic Kidney



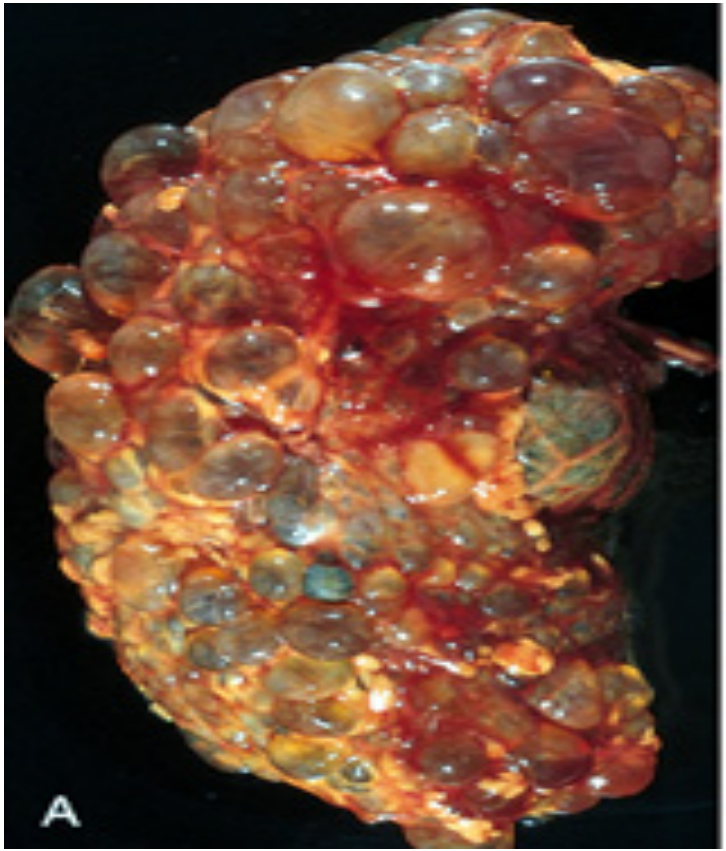
Polycystic kidney

- Autosomal Dominant (Adult) Polycystic Kidney Disease is a disease of autosomal dominant inheritance caused by mutations in the genes encoding polycystin-1 or -2. It accounts for about 10% of cases of chronic renal failure; kidneys may be very large and contain many large cysts.
- *Autosomal recessive (childhood) polycystic kidney disease* is caused by mutations in the gene encoding fibrocystin. It is less common than the adult form and strongly associated with liver abnormalities like fibrosis; the kidneys contain numerous small cysts.

Autosomal Dominant (Adult) Polycystic Kidney Disease

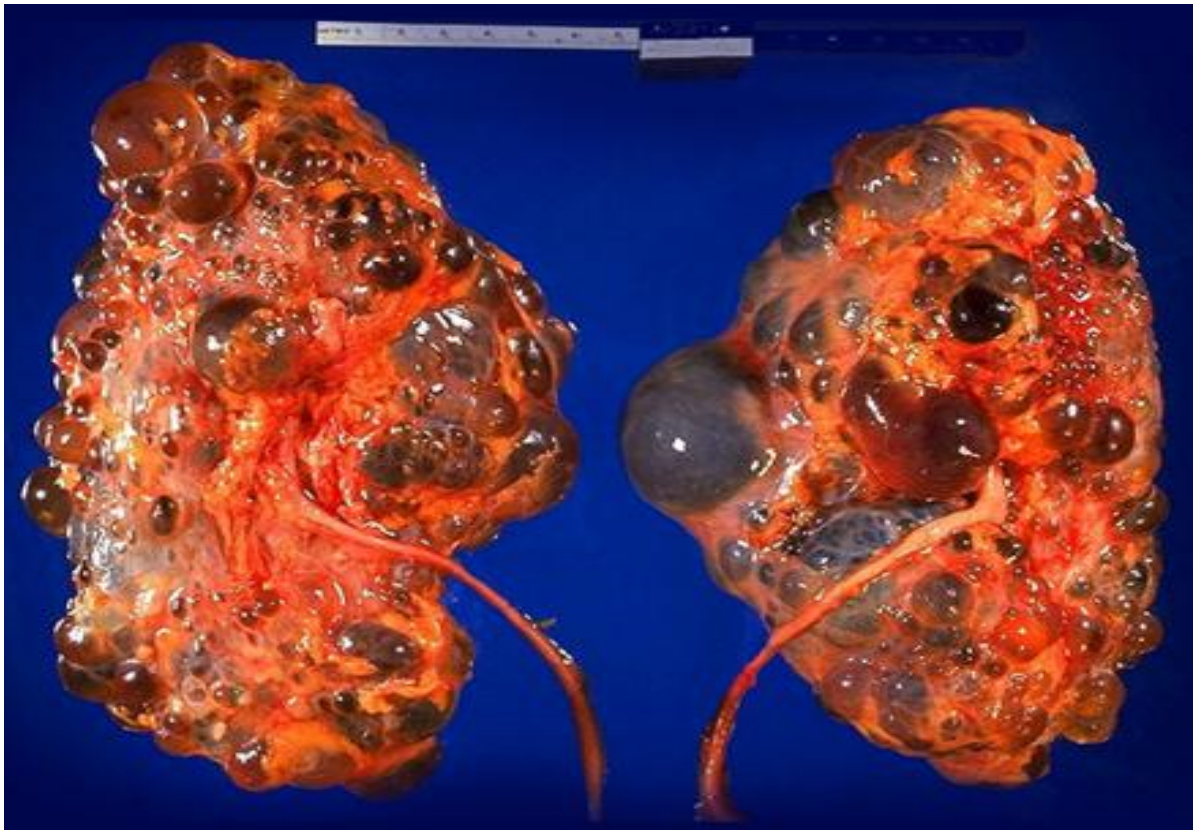
- Polycystic kidney disease in adults usually *produces symptoms by the fourth decade of life,* by which time the kidneys are quite large (the cysts start to develop in adolescence).
- Common clinical features
  - *flank pain* or a heavy, dragging sensation
  - *Intermittent gross hematuria*
  - palpable abdominal mass.
- Complications of adult polycystic kidney
  - acute distention of a cyst, either by intracystic hemorrhage or by obstruction, may cause excruciating pain.
  - *urinary infection.*
  - Hypertension of variable severity develops in about 75% of persons with this disorder.
  - Saccular aneurysms of the circle of Willis are present in 10% to 30% of patients and they can rupture → subarachnoid hemorrhage.
  - Renal failure/uremia (end-stage kidney disease) usually occurs by age 50.
- Patients in whom the disease progresses to renal failure are treated by renal transplantation.
- Death usually results from uremia or hypertensive complications.

Autosomal Dominant (Adult) Polycystic Kidney Disease – *Gross Anatomy*



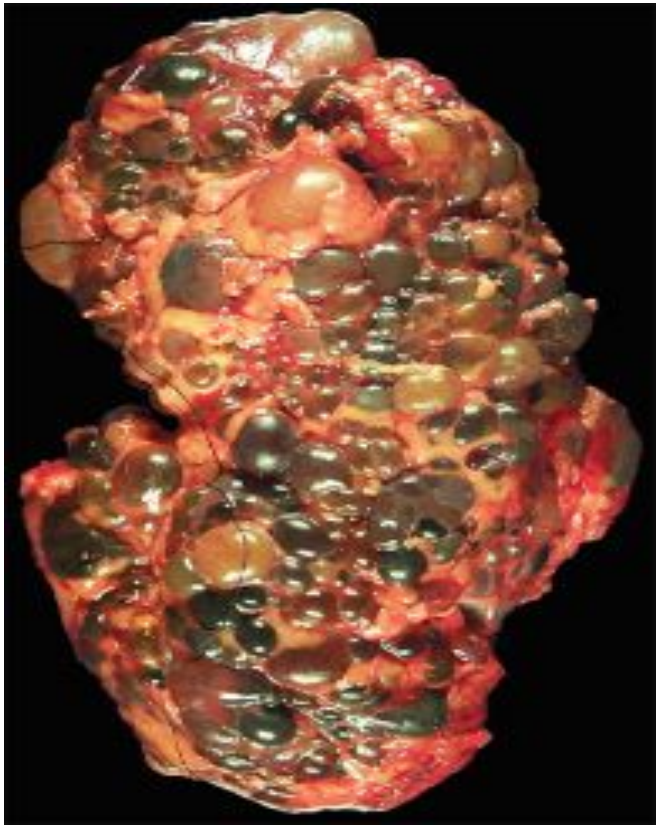
Markedly enlarged kidney and replacement of the renal parenchyma by numerous cysts of variable sizes

Autosomal Dominant (Adult) Polycystic Kidney Disease – *Gross Anatomy*

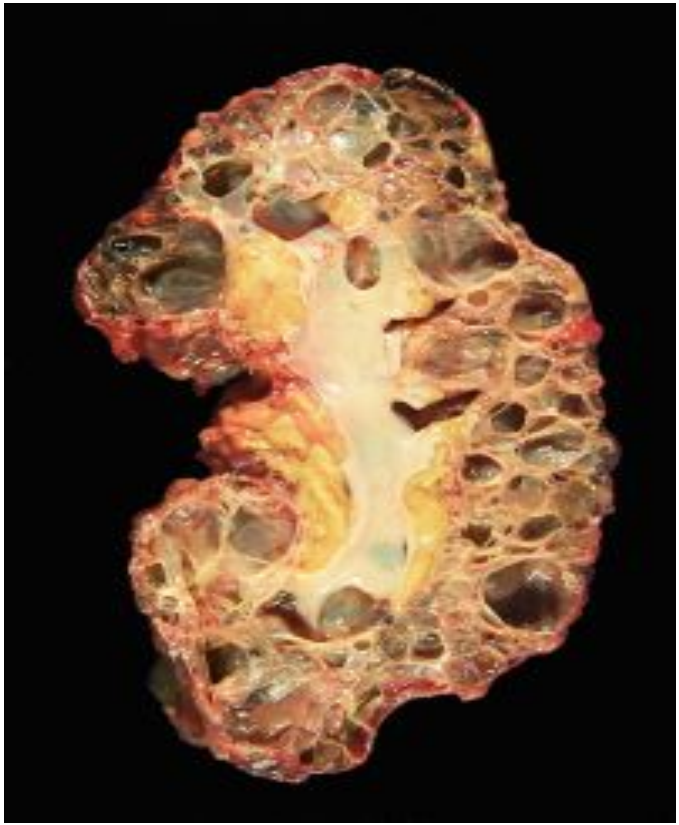


Bilateral autosomal dominant polycystic kidney disease

Gross Autosomal Dominant (Adult) Polycystic Kidney Disease *and its Cut Section*

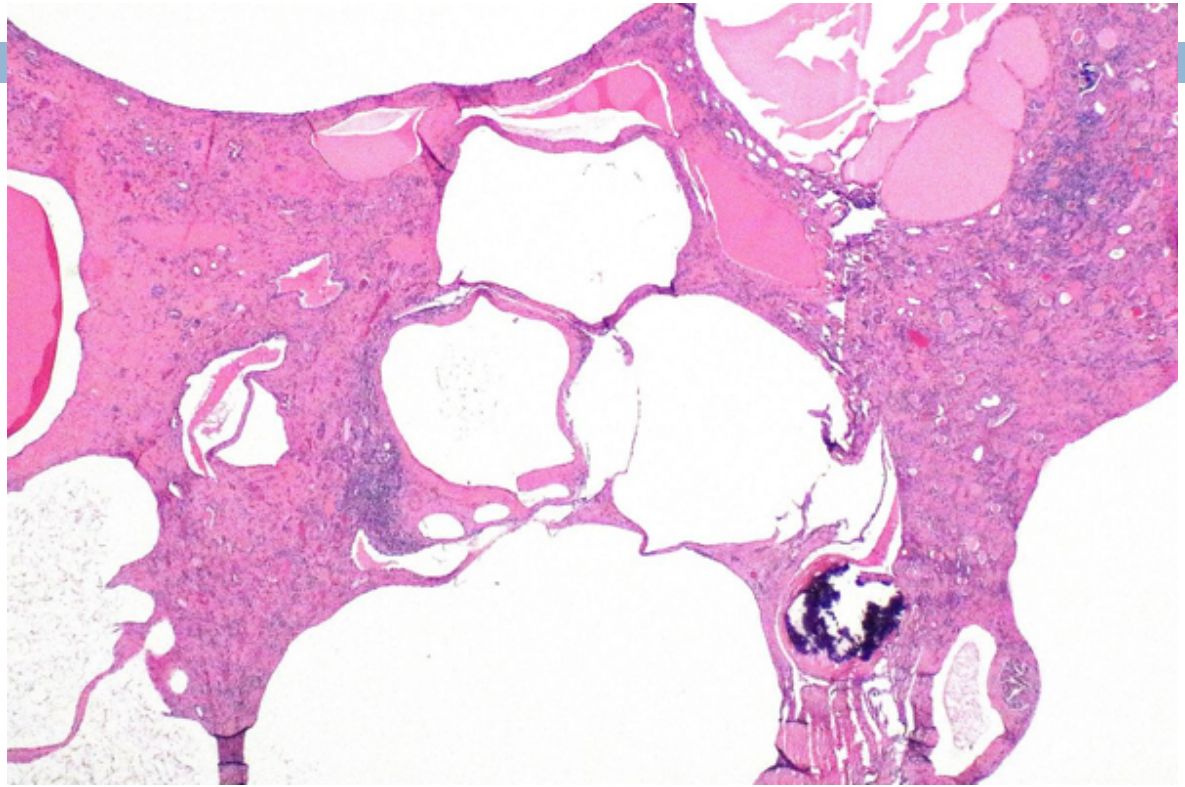


Massively enlarged kidney disrupted by numerous cysts



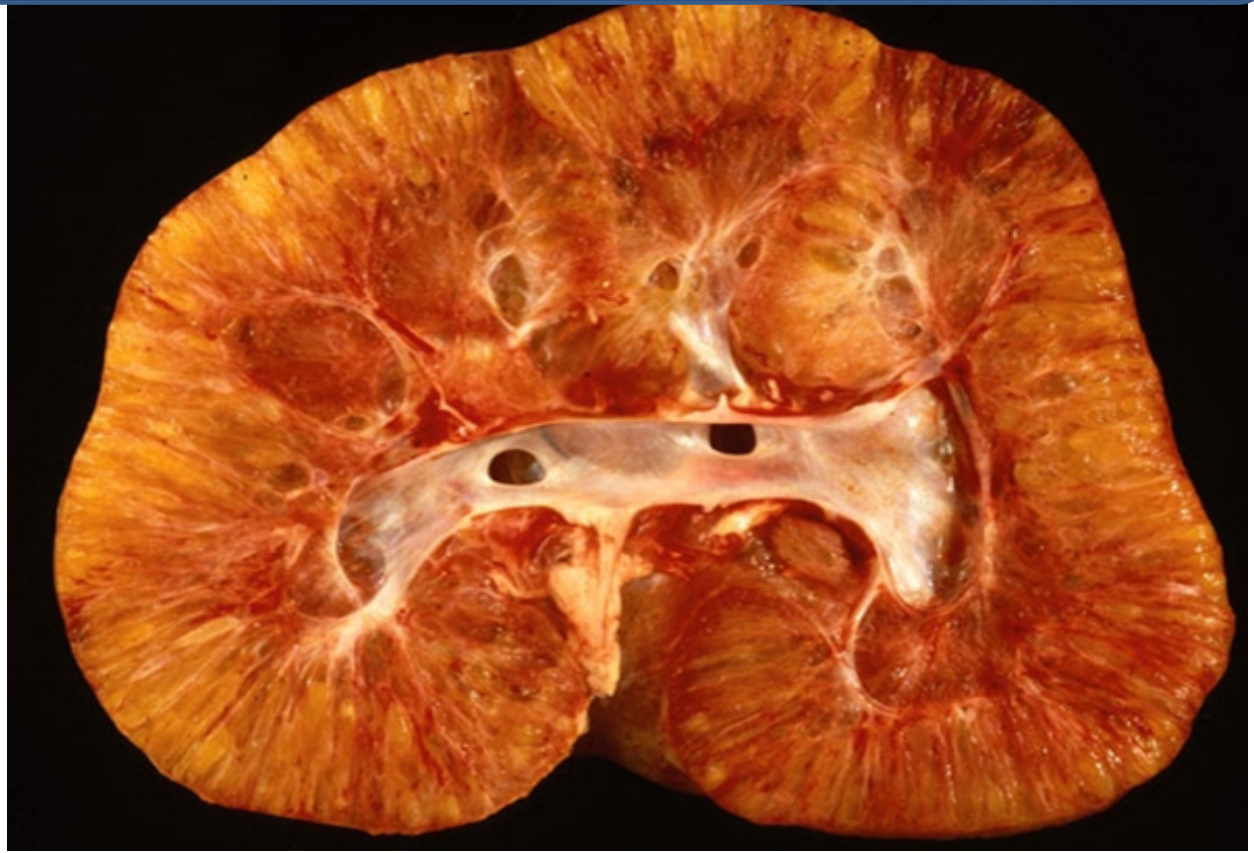
Cut surface of the kidney, showing extensive cortical destruction by cysts

Autosomal dominant polycystic kidney – histopathology



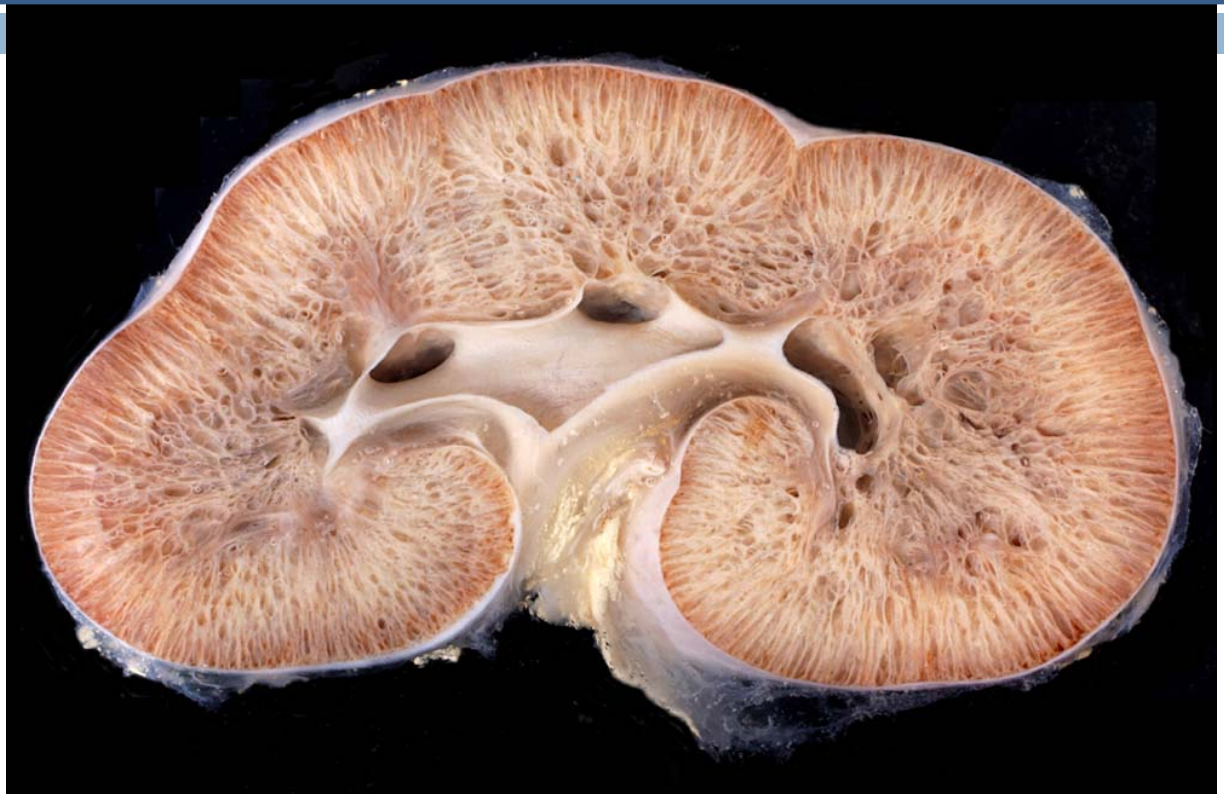
Sections show cortical cysts lined by simple flattened epithelium, with interspersed atrophic tubules, interstitial fibrosis and calcifications.

*Autosomal recessive (childhood) polycystic kidney disease – **Gross***



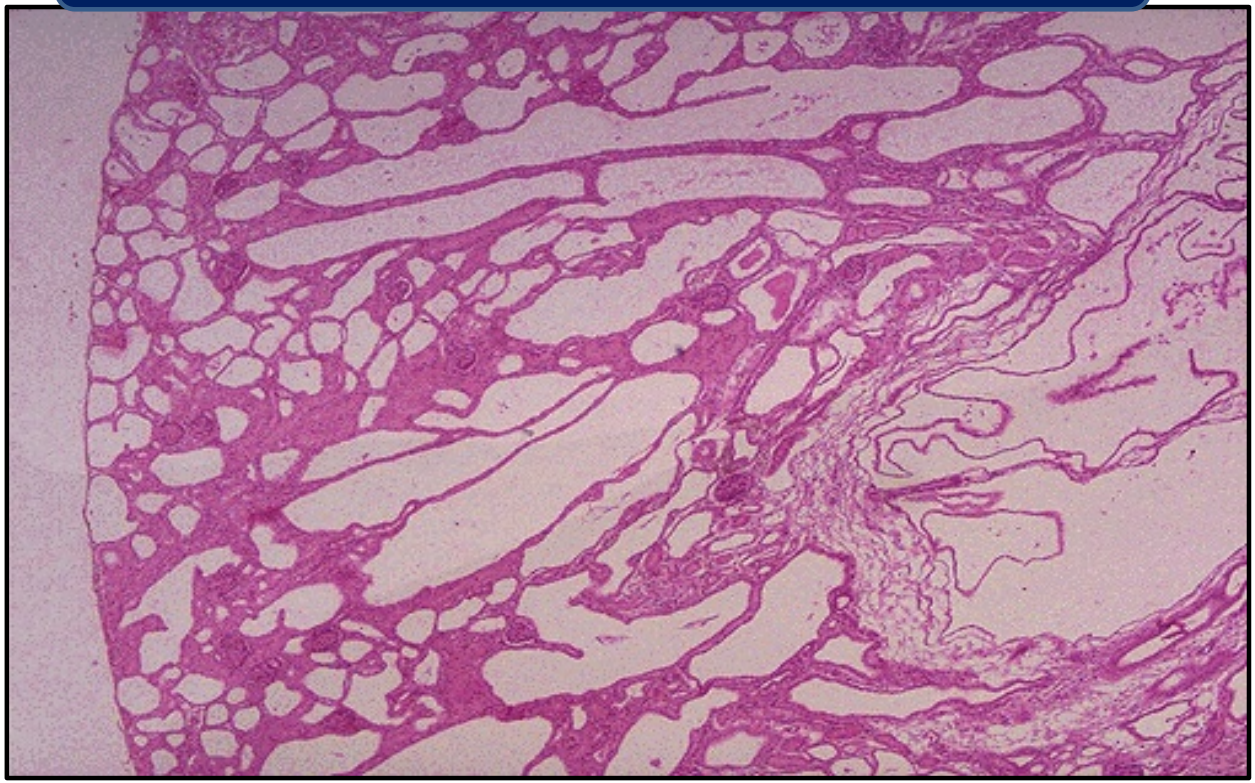
Coronal section of an infantile polycystic kidney

Autosomal recessive (childhood) polycystic kidney disease – Gross



- Markedly enlarged kidneys with smooth surface
- Small cysts in cortex and medulla
- Dilated channels are perpendicular to cortical surface
- Cysts are present in medulla (collecting ducts)

Polycystic kidney – Histopathology



The childhood form of polycystic kidney disease is a rare autosomal recessive disorder that is genetically distinct from adult polycystic kidney disease. In autosomal recessive polycystic kidney disease (ARPCKD), **numerous small cysts** in the cortex and medulla give the kidney a sponge like appearance. Dilated, elongated channels at right angles to the cortical surface completely replace the medulla and cortex. The cysts are lined by cuboidal cells. The disease is bilateral. In almost all cases, findings include multiple epithelium-lined **cysts in the liver** and proliferation of portal bile ducts. ***Note that the cysts fill most of the parenchyma, and it is hard to find glomeruli.***

PRACTICAL SESSION : 2

INFECTION OF THE URINARY TRACT

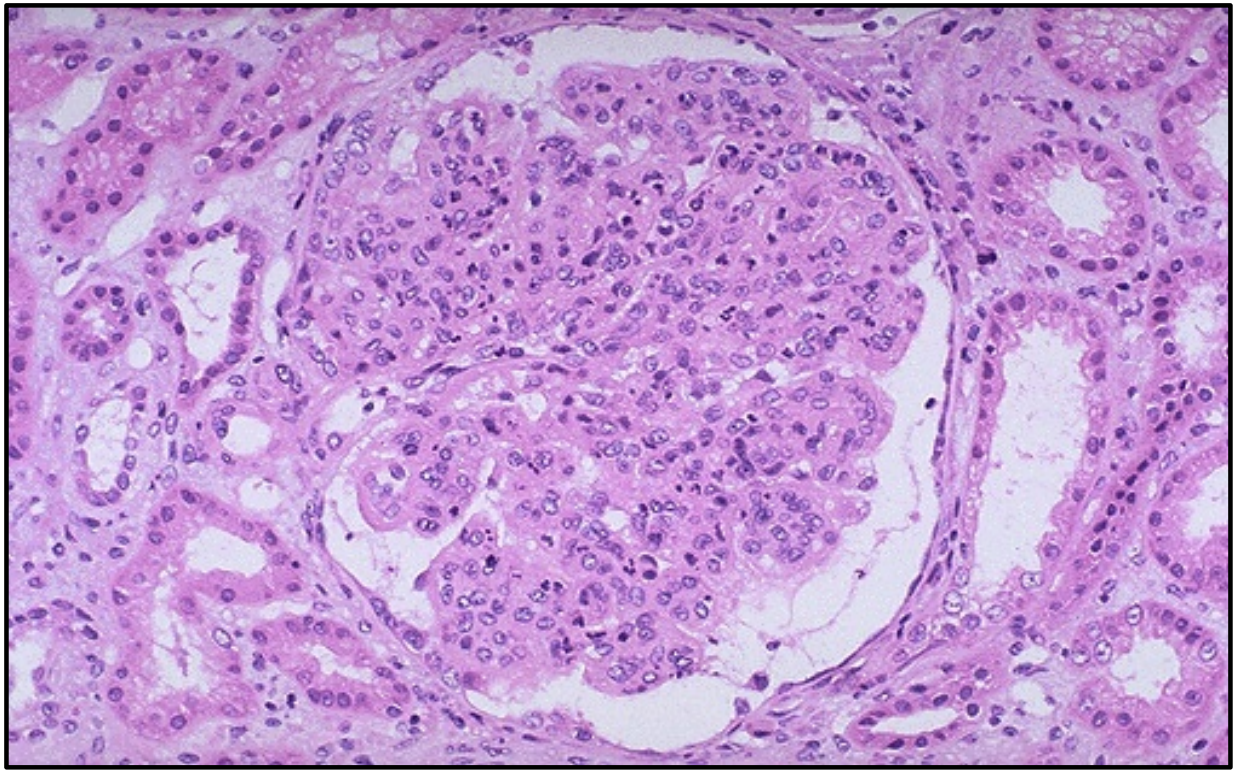
ACUTE (POST-STREPTOCOCCAL) GLOMERULONEPHRITIS

Acute (Post-streptococcal) Glomerulonephritis

Section of the kidney shows:

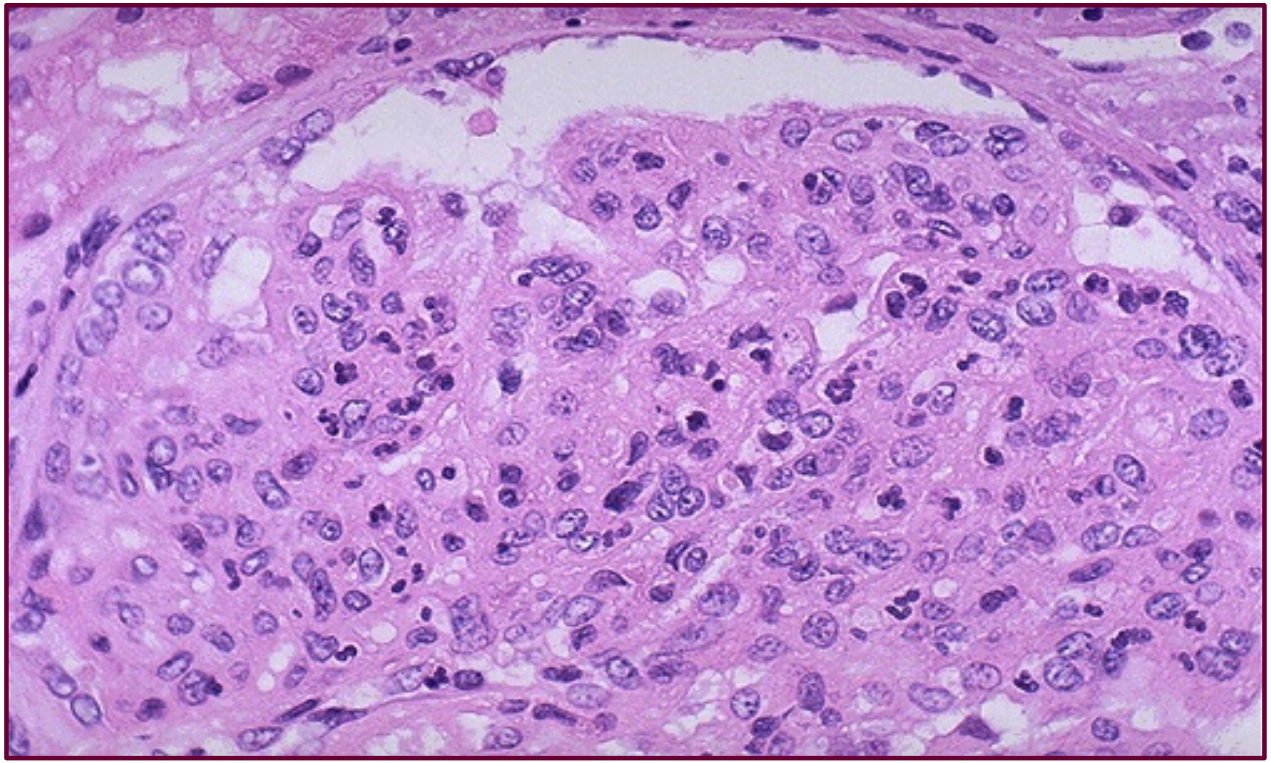
- The glomeruli are enlarged, lobulated and hypercellular with obliteration of capsular space.
- Cellularity is due to proliferation of endothelial and mesangial cells with some neutrophils.
- Many capillaries appear obliterated.
- Tubules show degenerative changes.

Acute (Post-streptococcal) Glomerulonephritis



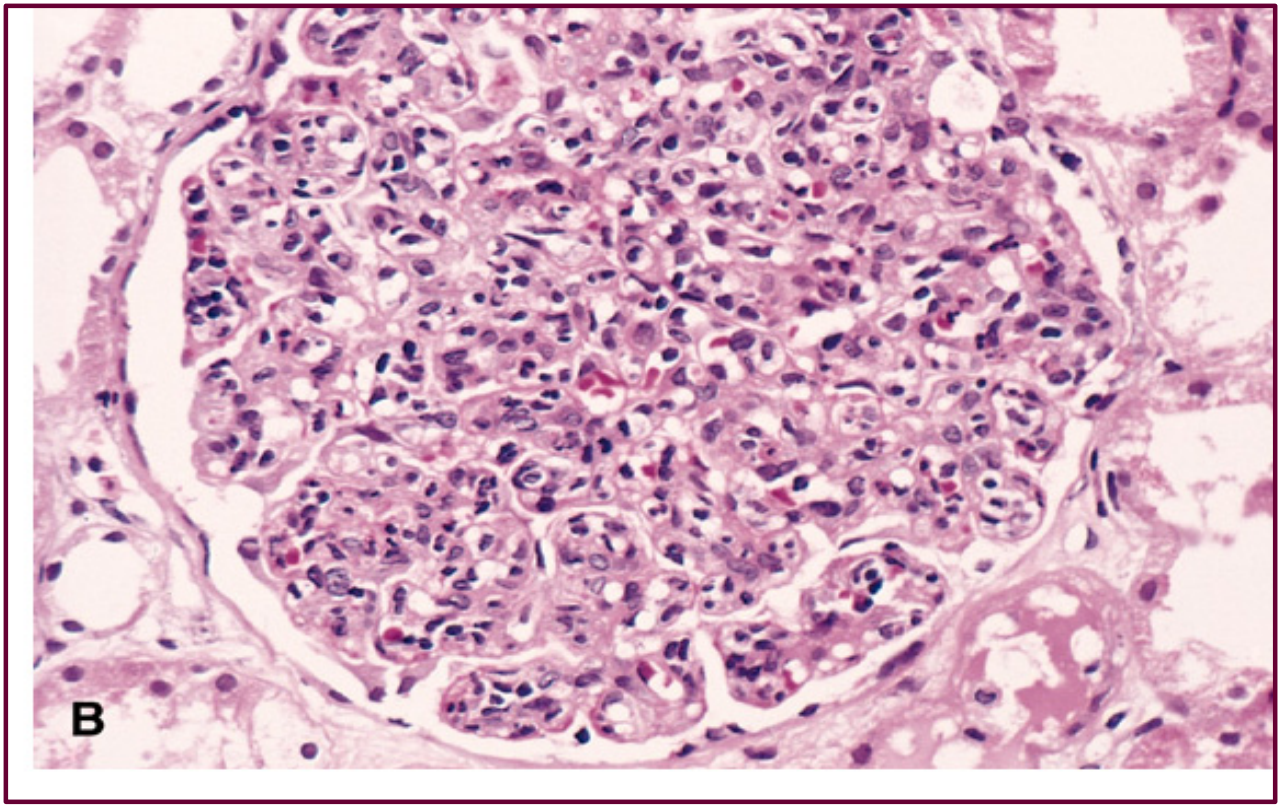
This glomerulus is hypercellular and capillary loops are poorly defined. This is a type of proliferative glomerulonephritis known as post-infectious glomerulonephritis

Acute (Post-streptococcal) Glomerulonephritis



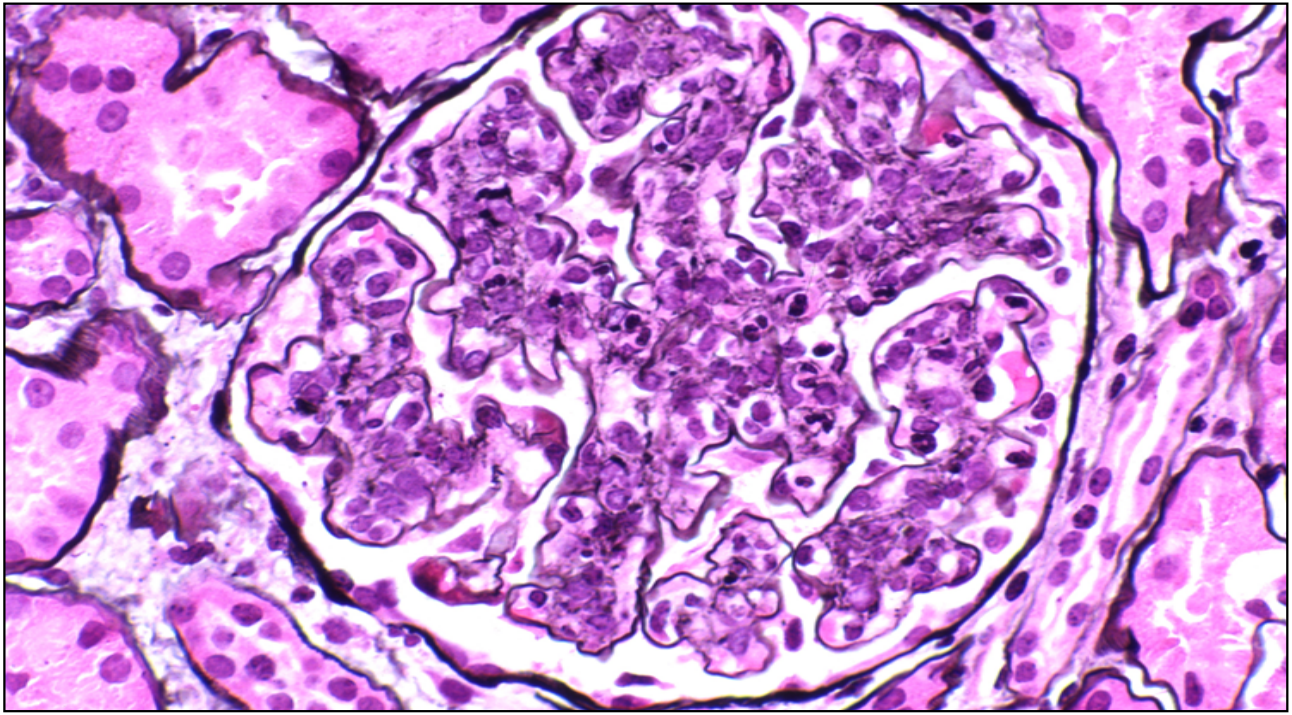
The hypercellularity of post-infectious glomerulonephritis is due to increased numbers of epithelial, endothelial, and mesangial cells as well as neutrophils in and around the glomerular capillary loops

Acute (Post-streptococcal) Glomerulonephritis



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

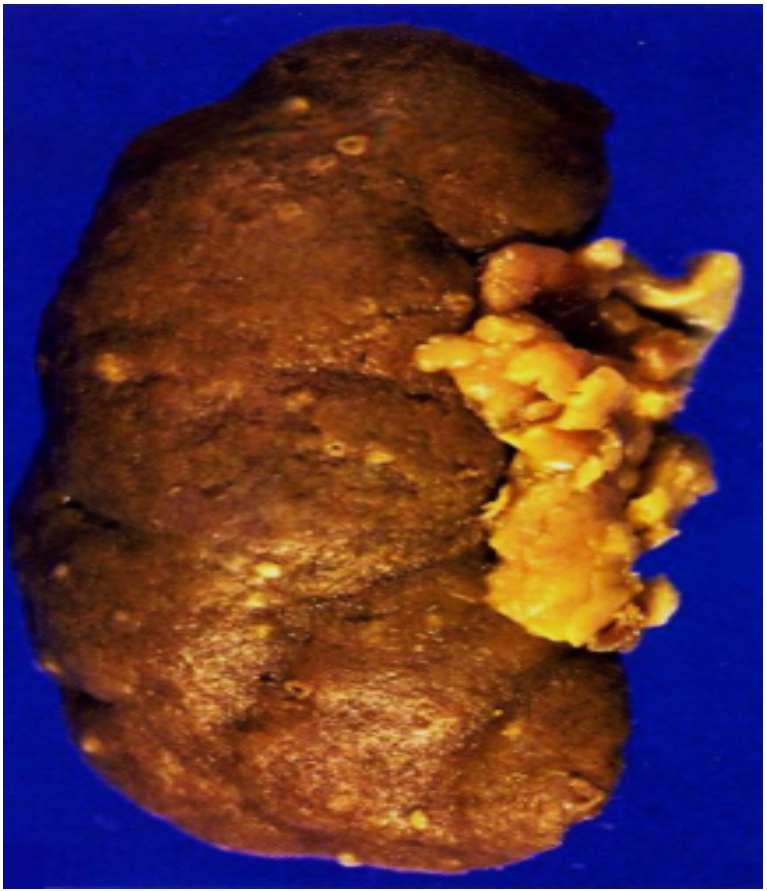
Acute Post-streptococcal Glomerulonephritis



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 filling and distending capillary loops.

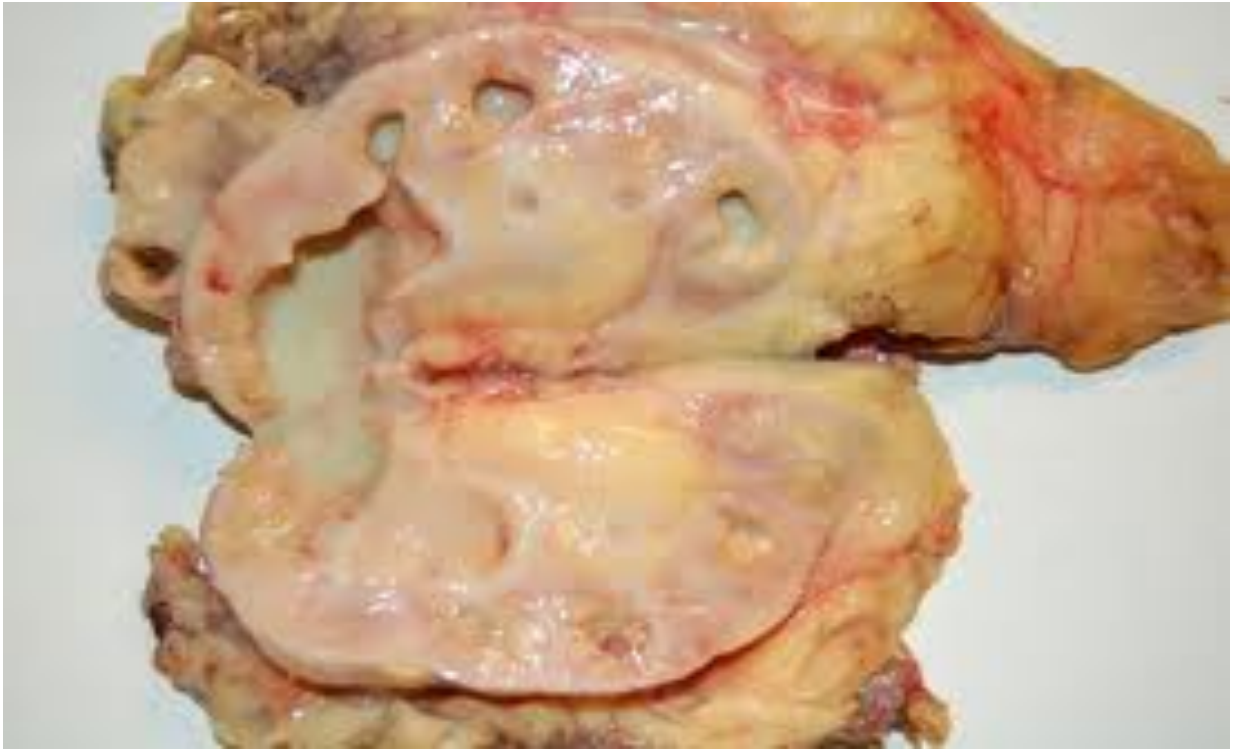
ACUTE & CHRONIC PYELONEPHRITIS

Acute Pyelonephritis with small cortical abscesses



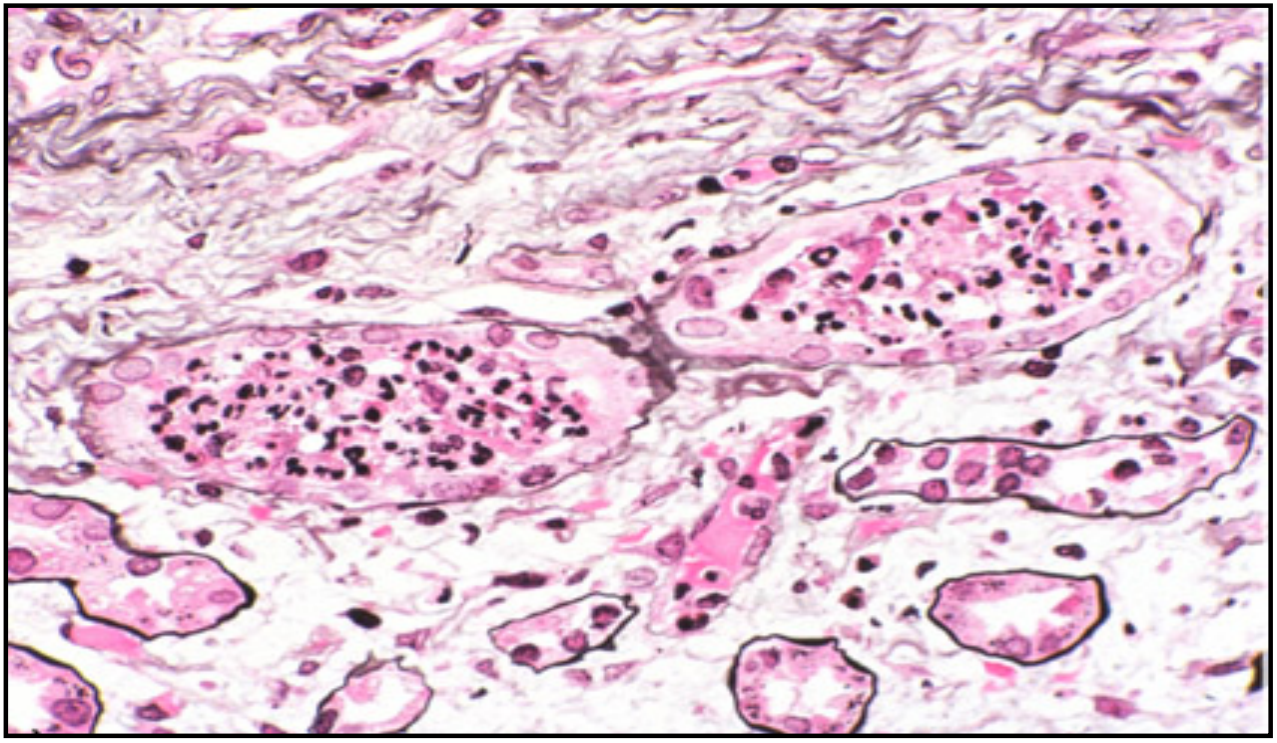
Acute Pyelonephritis with small cortical abscesses

Classic picture of Acute Pyelonephritis



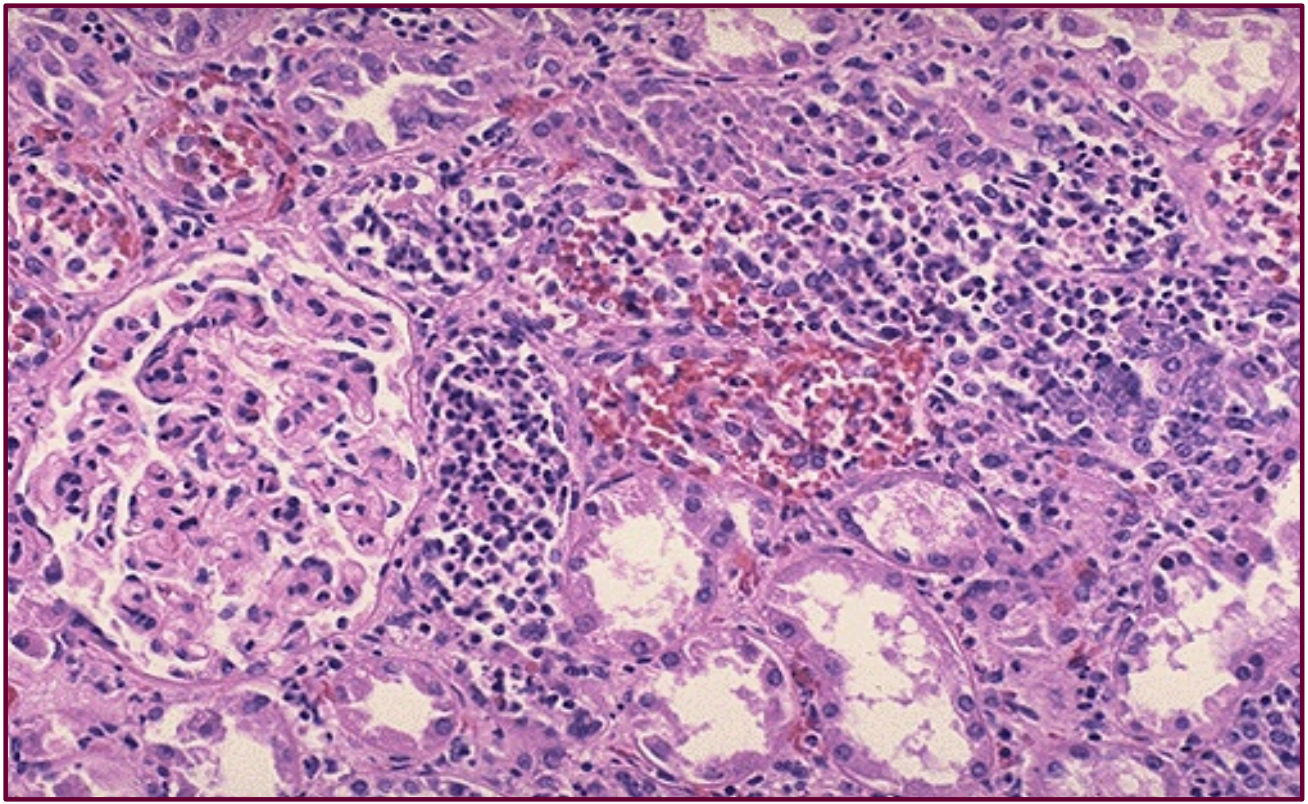
This kidney is bisected to reveal a dilated pelvis and calyxes filled with a yellow-green purulent pus which is consistent with a pyelonephritis. The cortex and medulla are pale and the corticomedullary junction is ill-defined.
No tumors are seen.

Acute Pyelonephritis - Histopathology



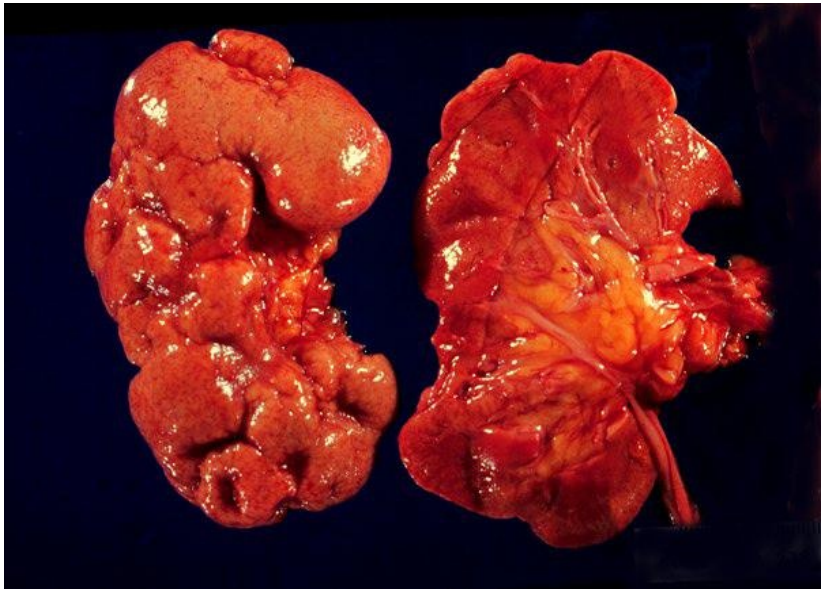
Acute pyelonephritis is diagnosed by intratubular aggregations of polymorphonuclear neutrophils (PMNs). There may be surrounding interstitial inflammation with a mixture of PMNs, lymphocytes, and plasma cells, but the predominant inflammation is within the tubule

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

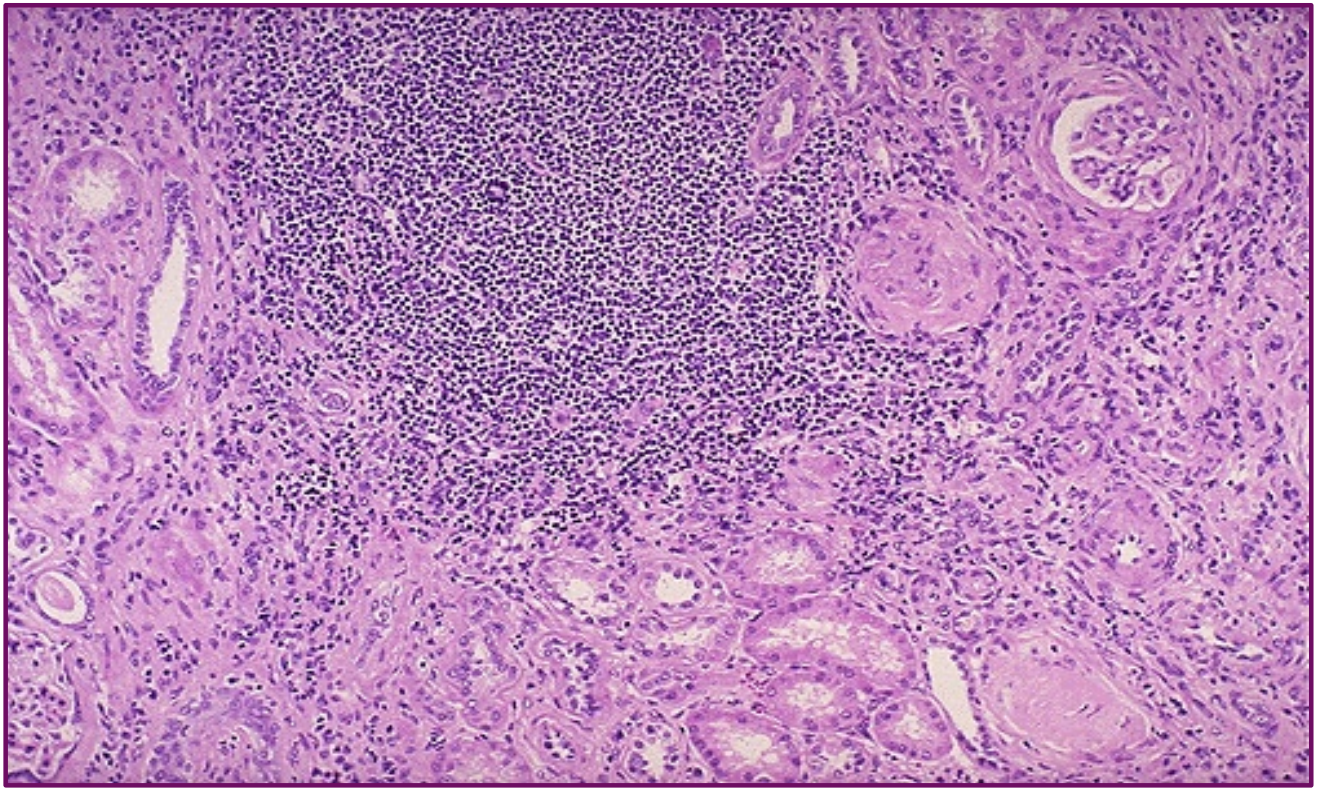


The picture shows slightly atrophic and deformed kidneys with cortical coarse scars .

The most common causes are:

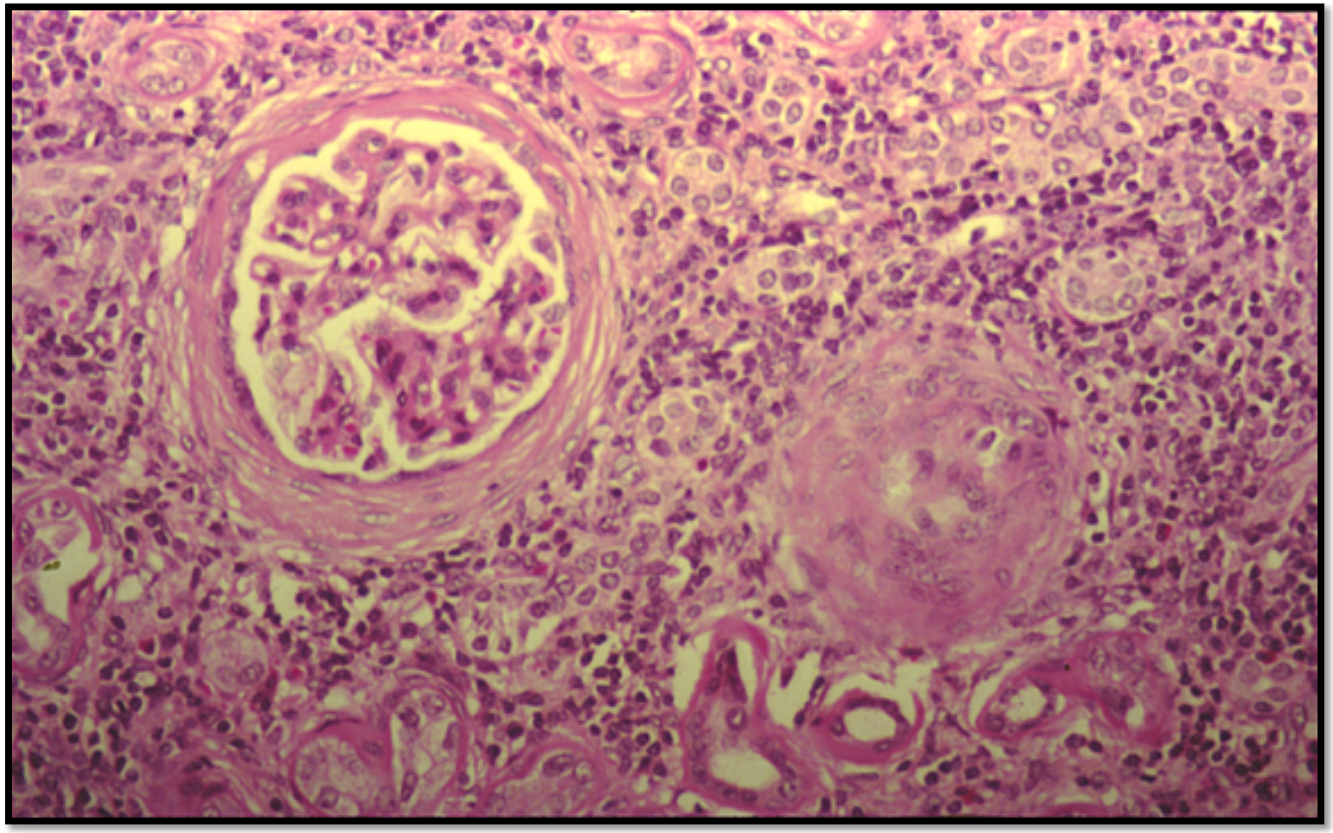
- ***Obstruction by renal stones and others.***
- ***Reflux uropathy – urinary reflux.***
- ***Drugs like NSAID's, methicillin etc.***
- ***Recurrent urinary tract infections.***

Chronic Pyelonephritis - Histopathology



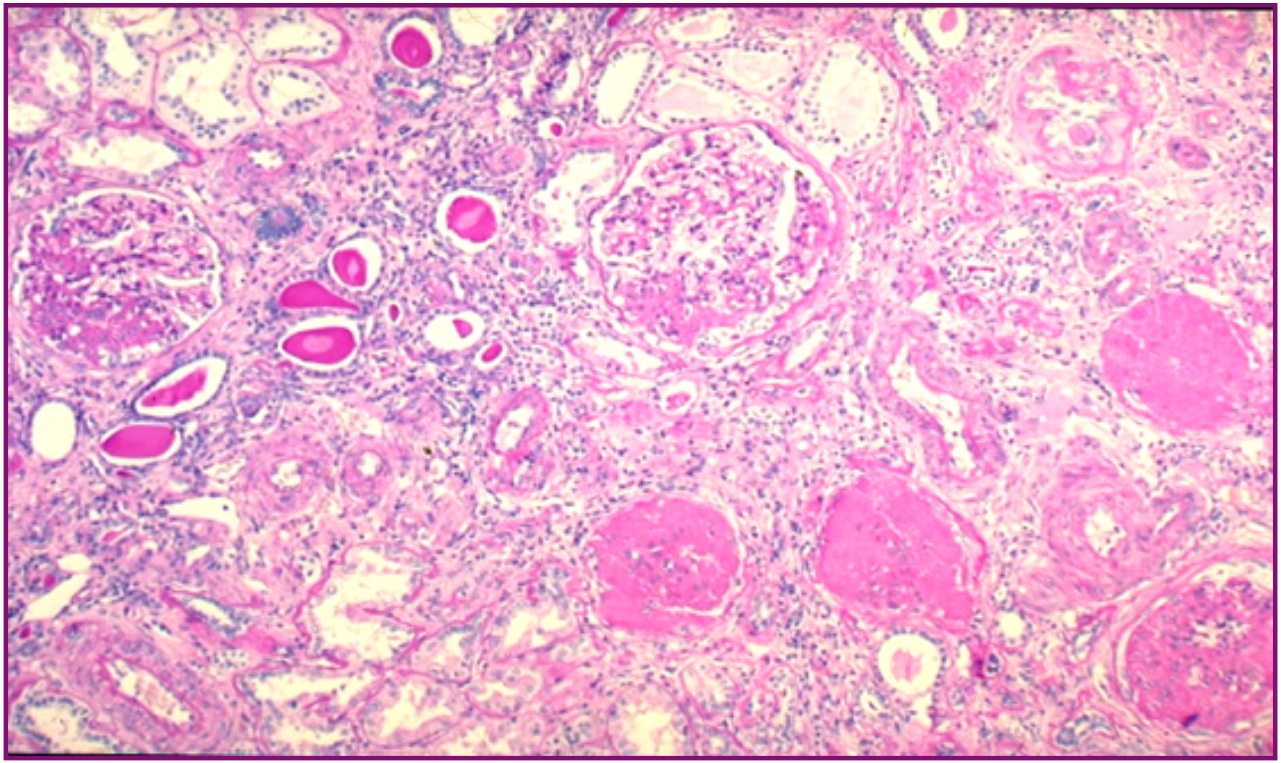
This is chronic pyelonephritis where a large collection of chronic inflammatory cells . The severity of disease depends upon the amount of remaining functional renal parenchyma **(Renal failure – insufficiency or end – stage renal disease)**.

Chronic Pyelonephritis - Histopathology



- Periglomerular fibrosis.
- Glomerulosclerosis.
- Marked chronic interstitial inflammation.
- Hyalinization and atrophy of renal tubules.

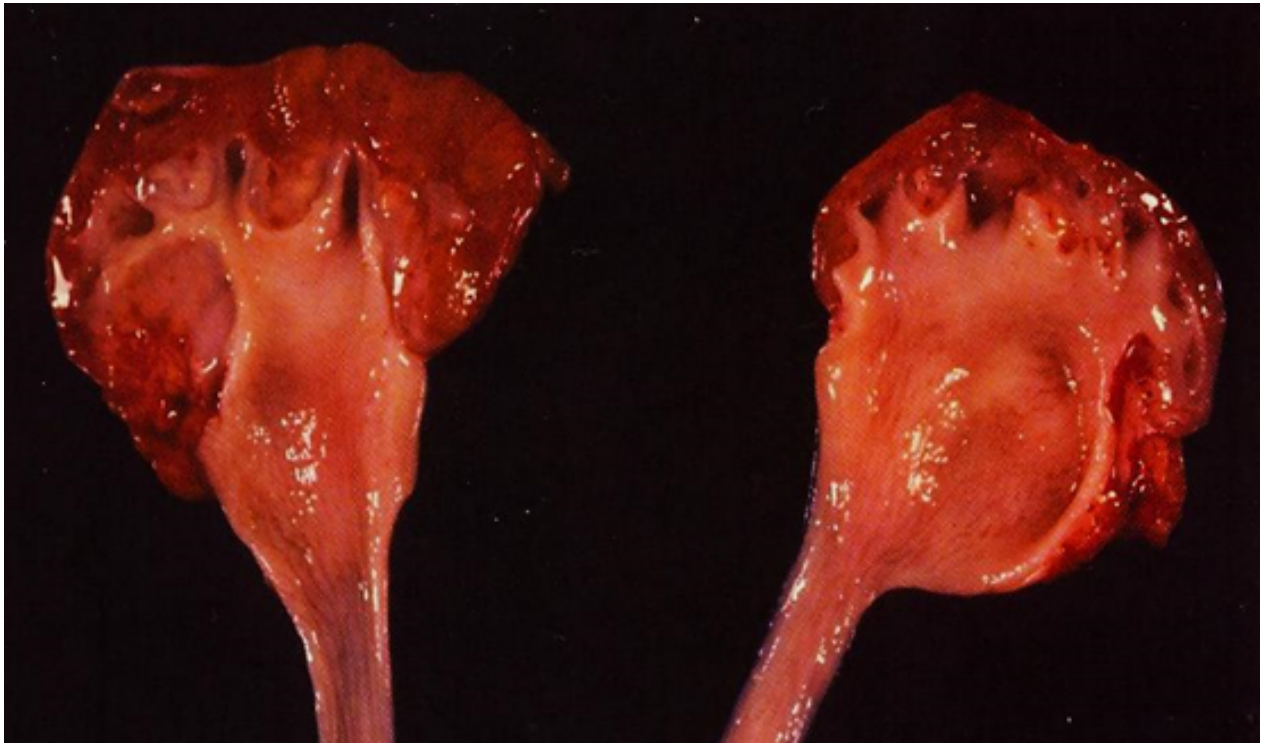
Chronic Pyelonephritis - Histopathology



- ***Glomeruli*** *show varying degrees of sclerosis & periglomerular fibrosis.*
- ***Tubules*** *show varying degrees of atrophy, Some tubules are dilated and filled with Eosinophilic hyaline casts resembling colloid (thyroidization).*
- ***Interstitial tissue*** *shows chronic inflammatory cells infiltrate and fibrosis.*

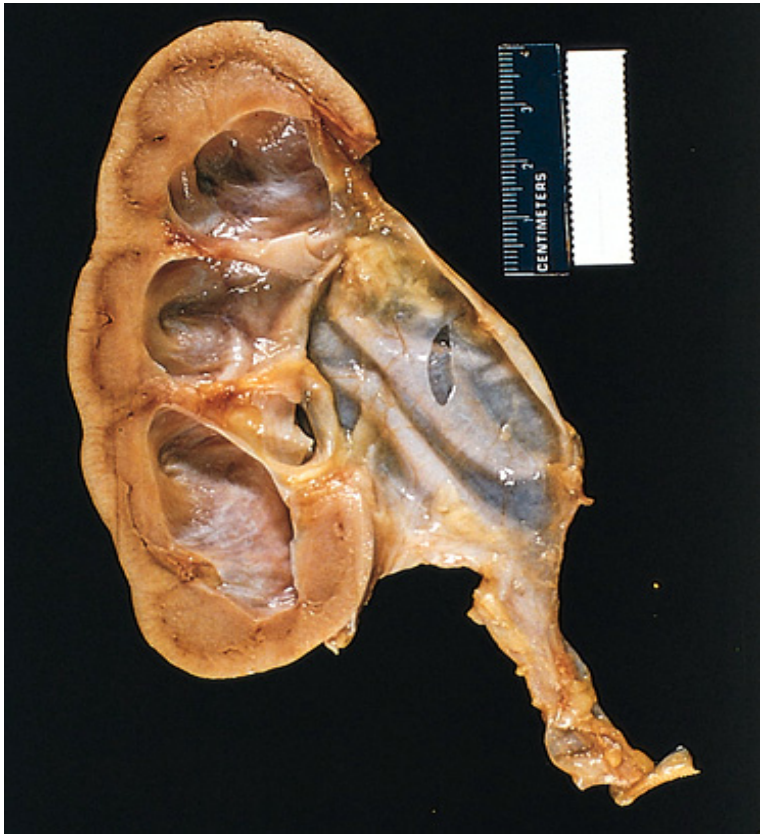
HYDRONEPHROSIS

Hydronephrosis



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

Hydronephrosis

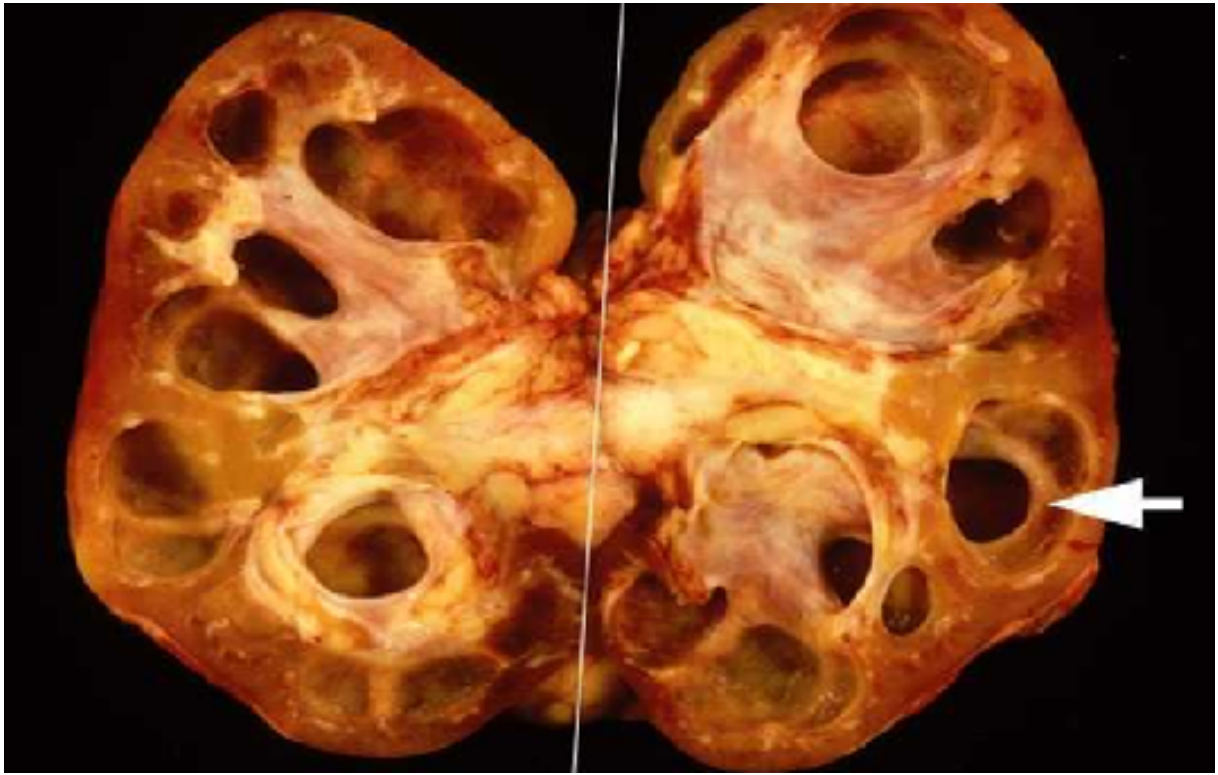


The picture shows markedly dilated renal pelvis and calyces with atrophic and thin renal cortex /parenchyma

The most common causes are:

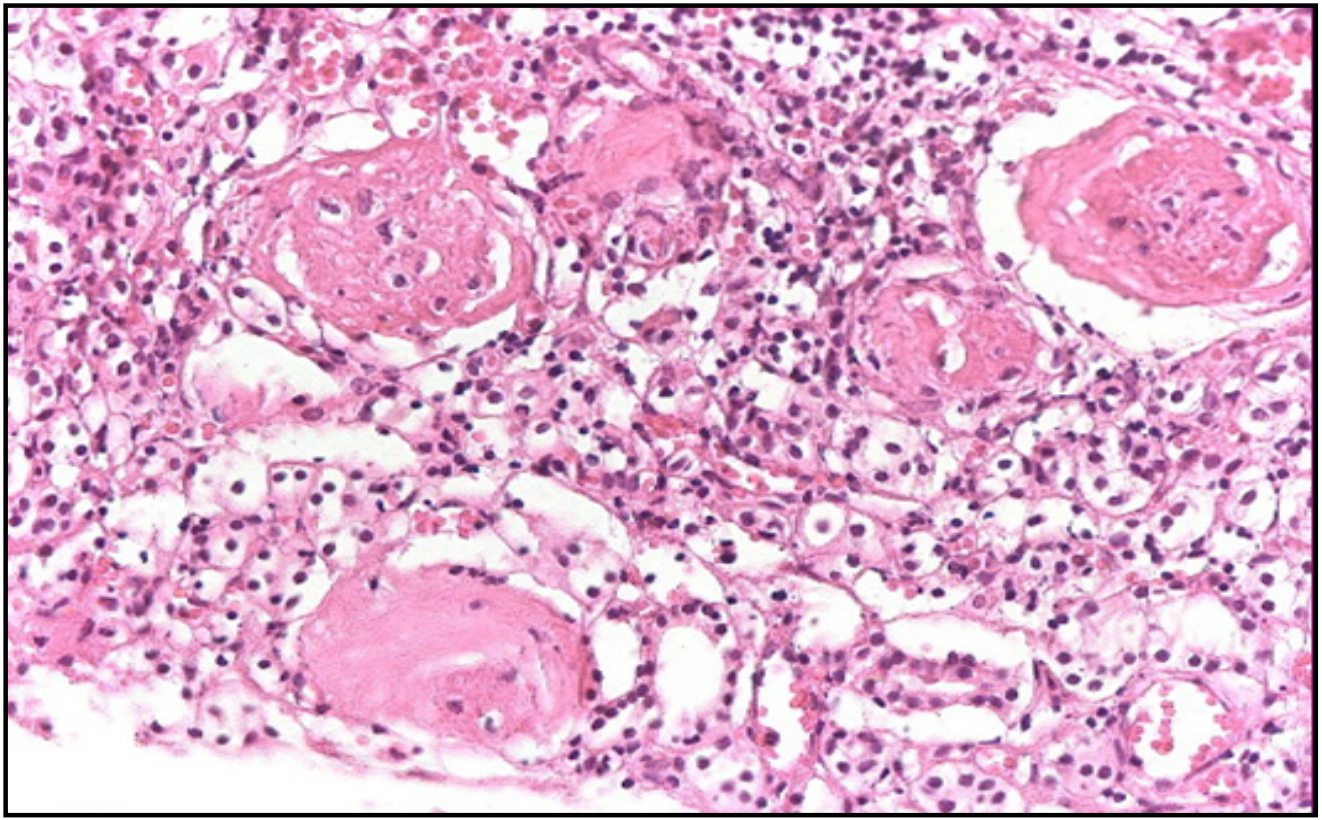
- *Foreign bodies like calculi with obstruction,*
- *Atresia of the urethra,*
- *Benign prostatic hyperplasia ,*
- *Neoplasia of the prostate and bladder*
- *Spinal cord damage with paralysis of the bladder .*

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

THE END