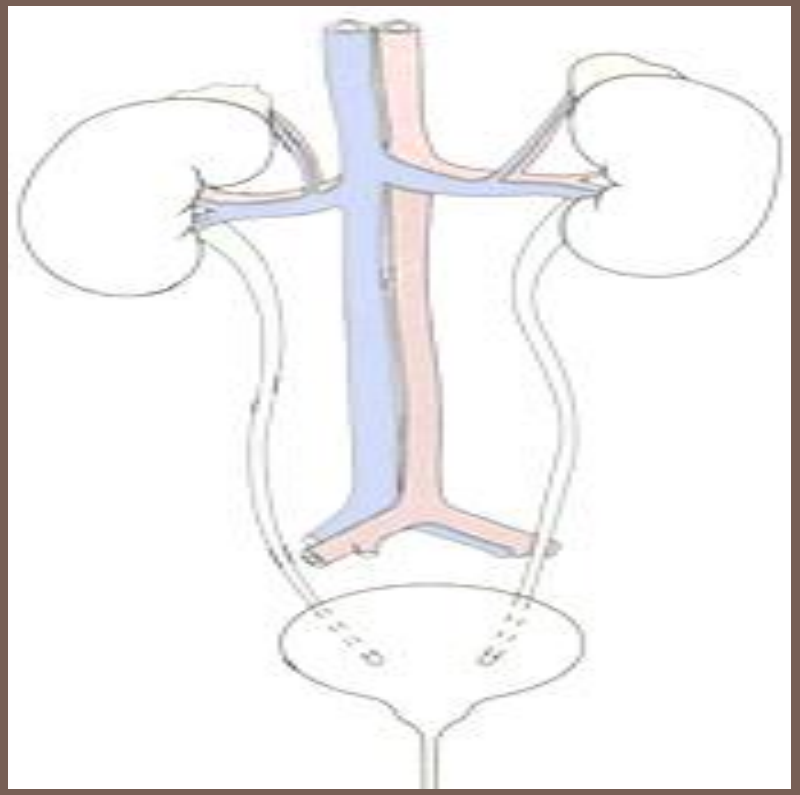
RENAL BLOCK



PATHOLOGY PRACTICAL

Prepared by:

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- *Dr. Sayed Al Esawy*

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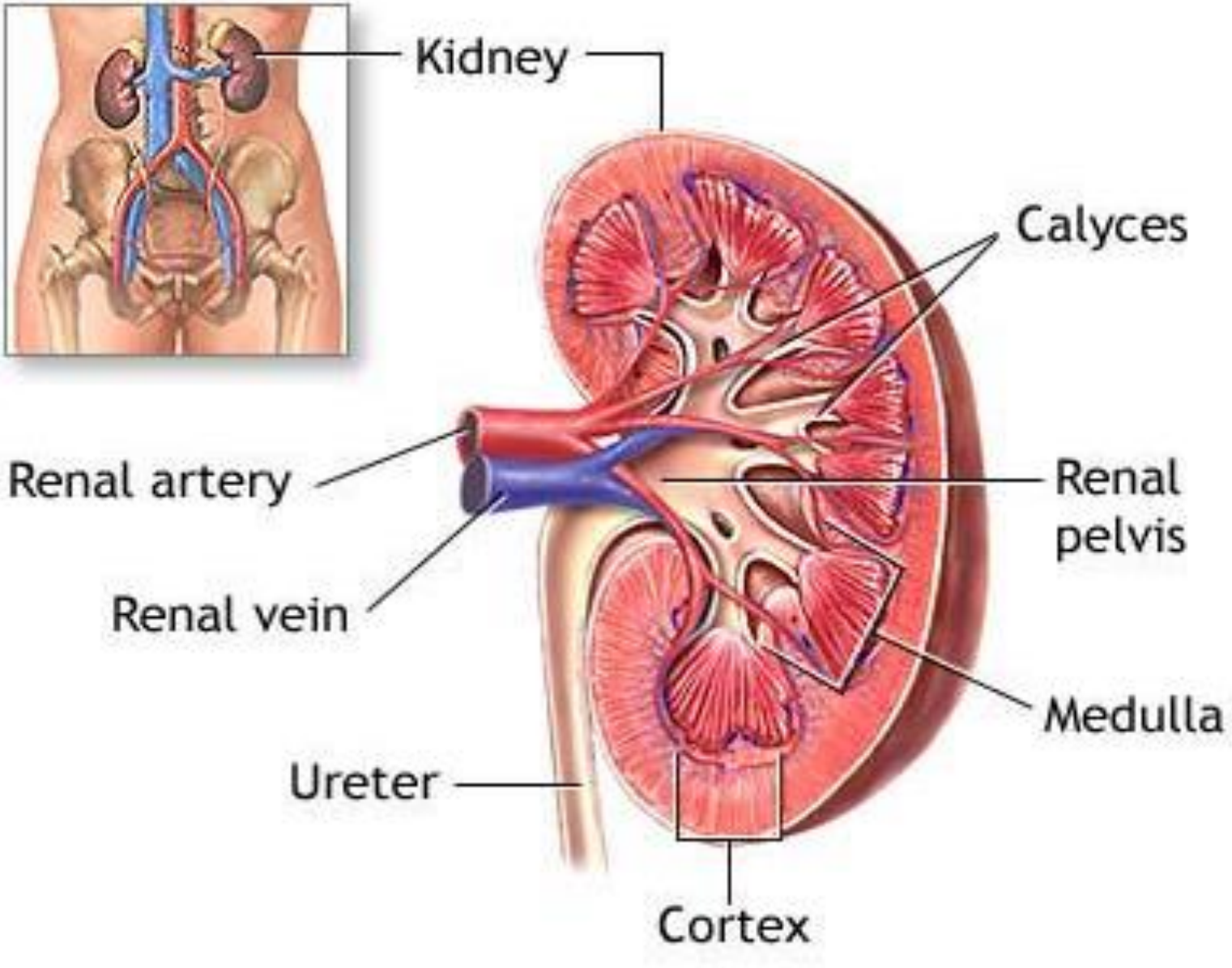
NORMAL ANATOMY AND HISTOLOGY

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.

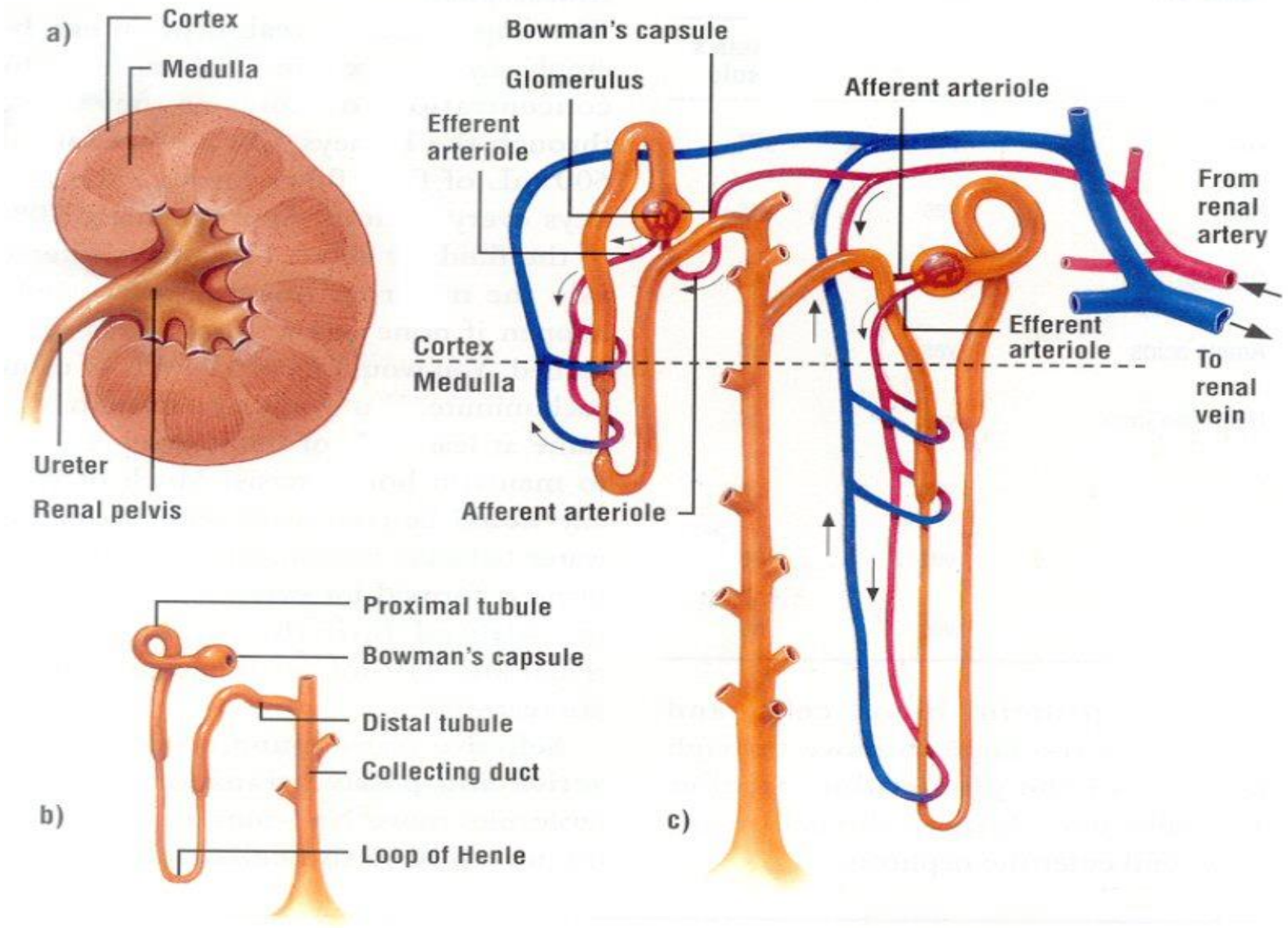
Anatomy of the Kidney



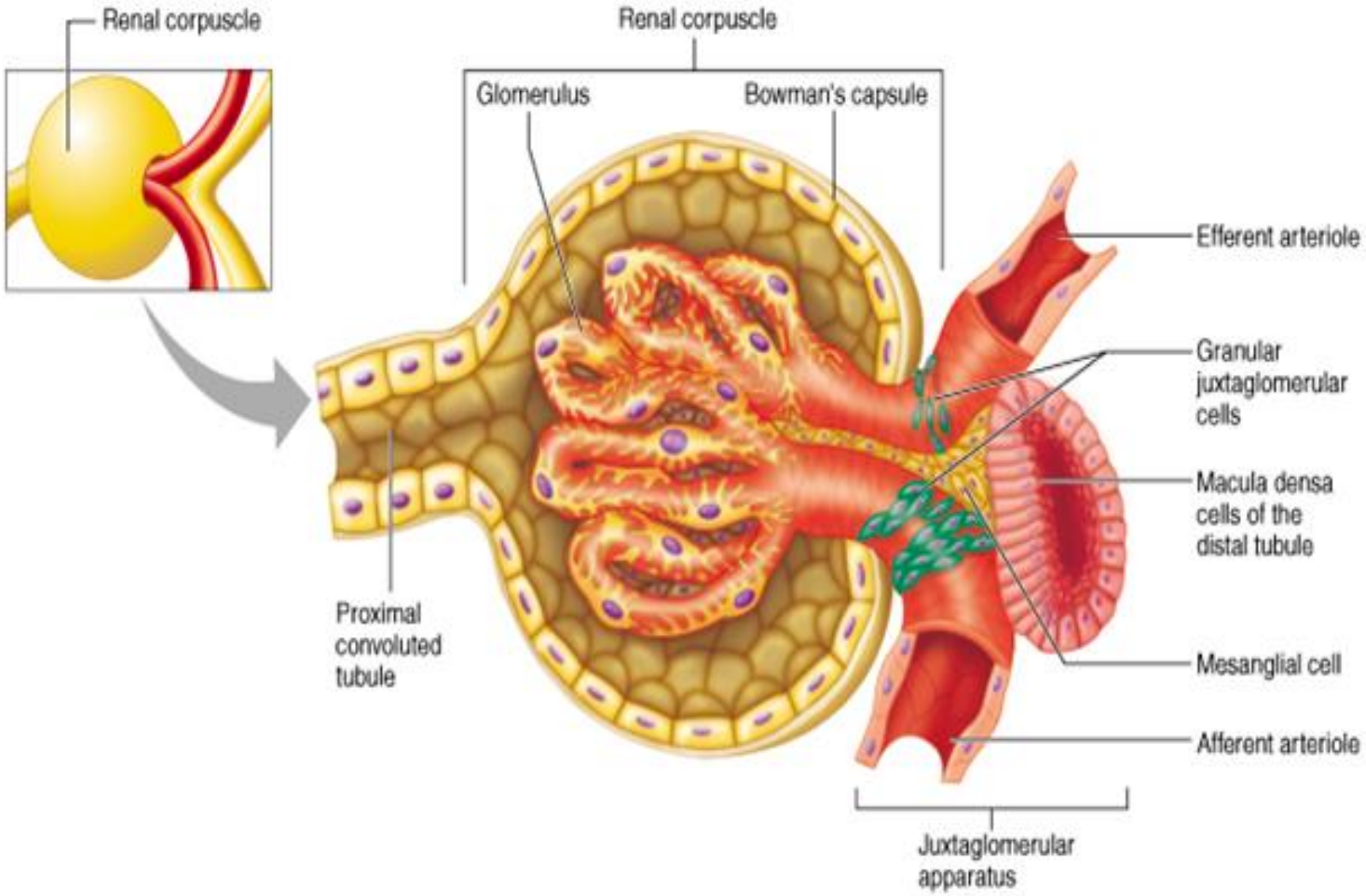
KIDNEY ANATOMY : NEPHRONS

- *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.*

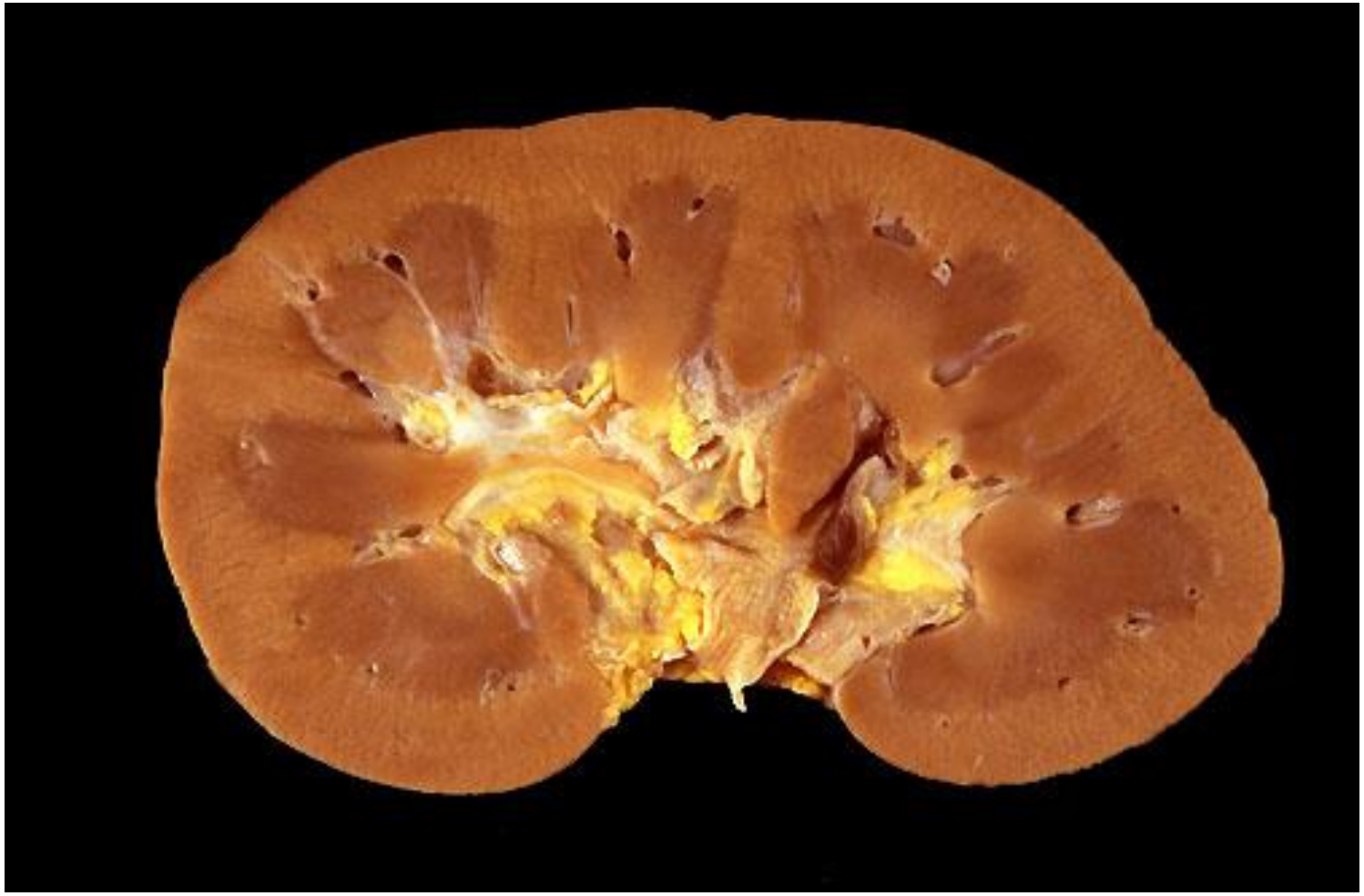
NEPHRON STRUCTURE



Renal Corpuscle

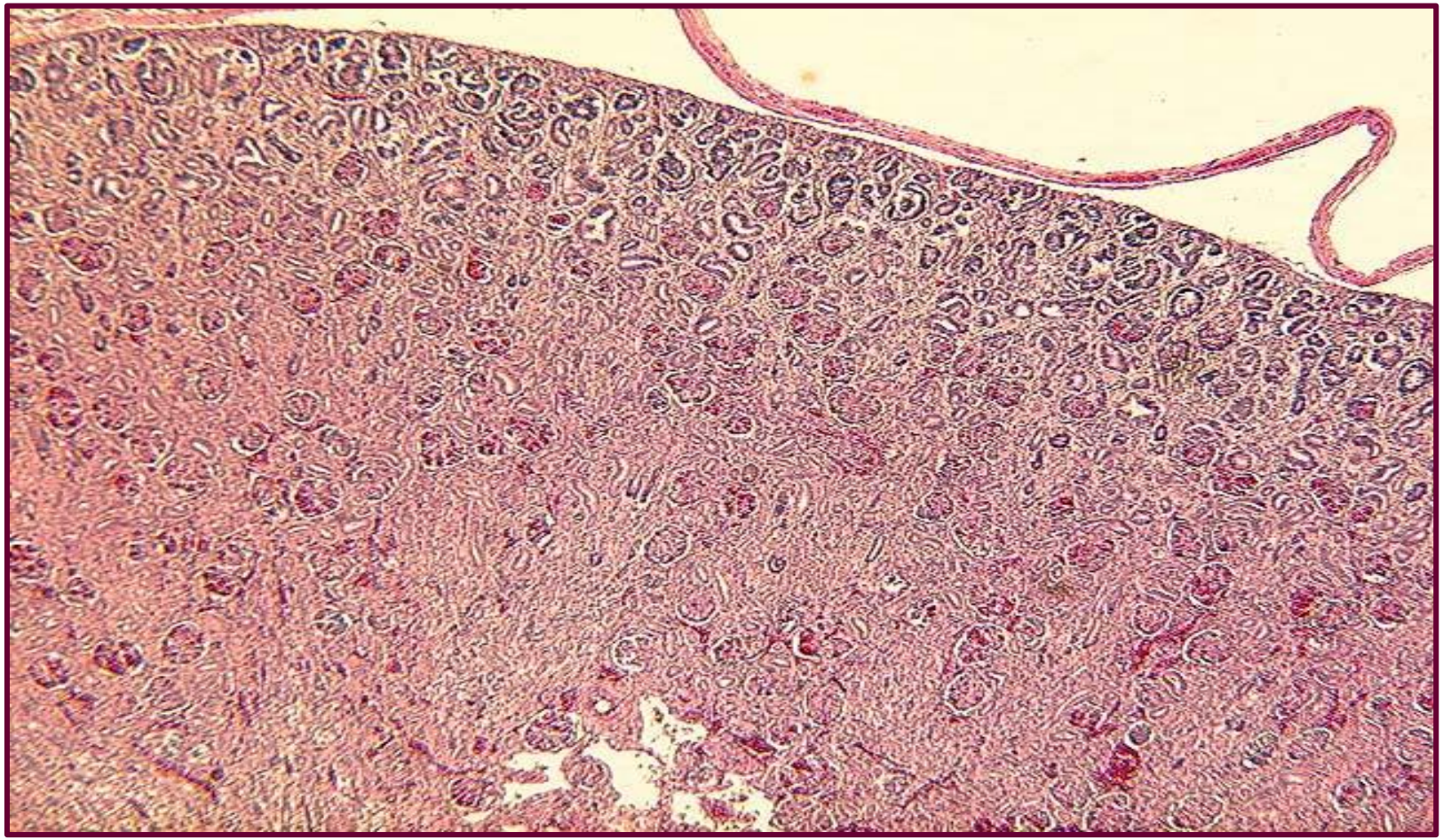


Normal Kidney - Gross



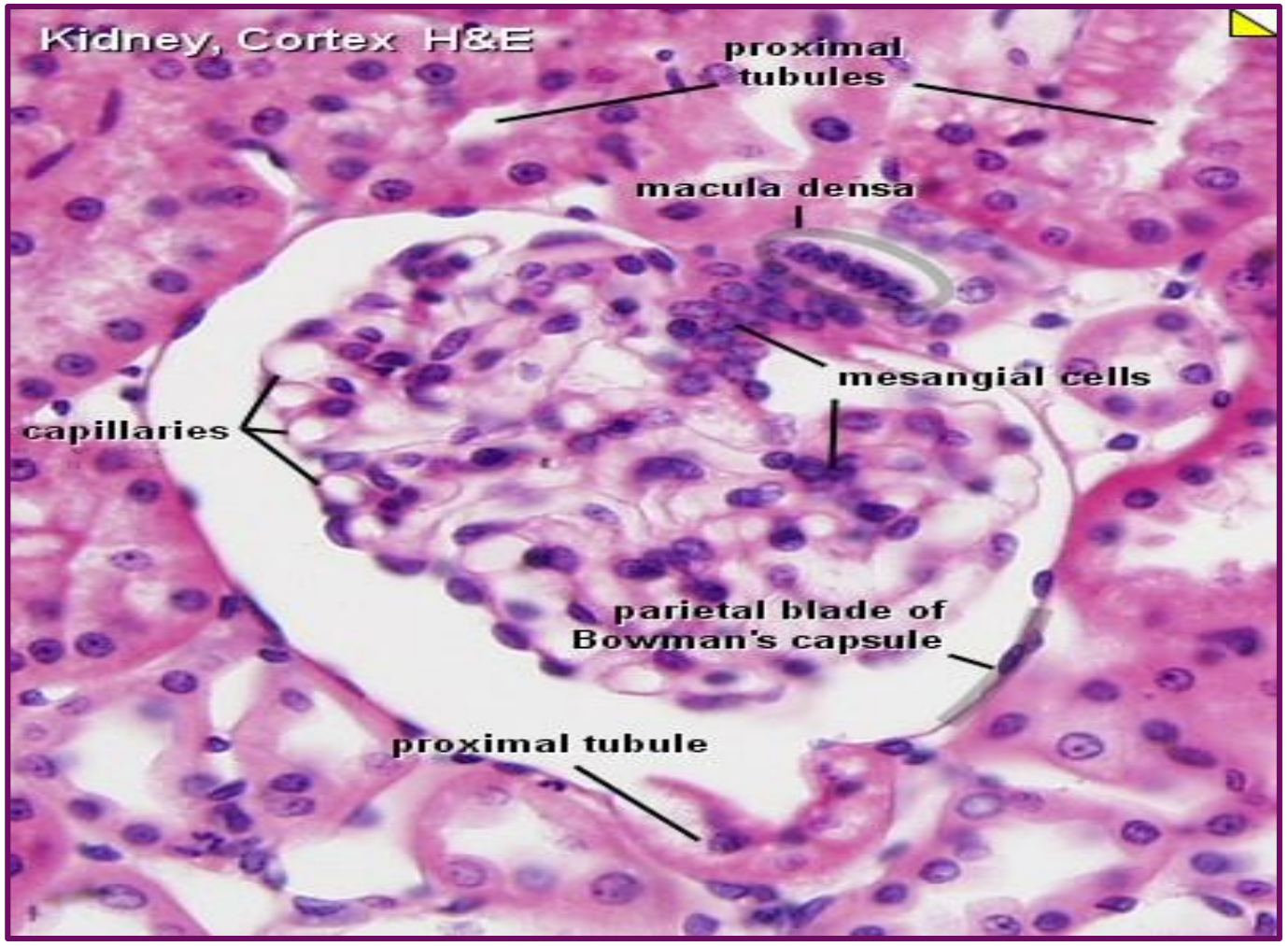
In cross section, this normal adult kidney demonstrates the lighter outer cortex and the darker medulla, with the renal pyramids into which the collecting ducts coalesce and drain into the calyces and central pelvis.

Kidney – Normal Histology

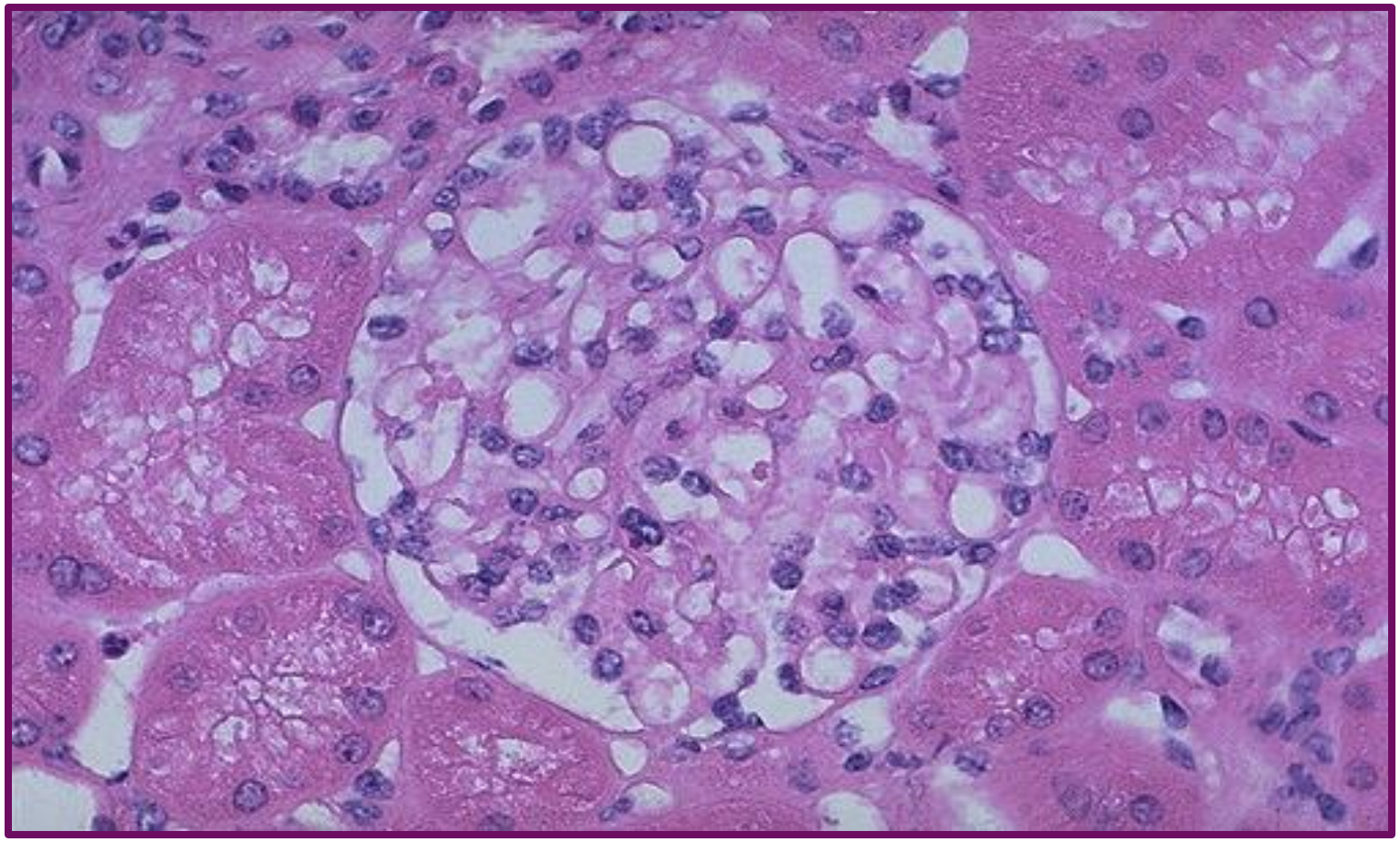


The kidneys show a well preserved lobular structure with indistinct corticomedullary demarcation

Renal Corpuscle – Normal Histology

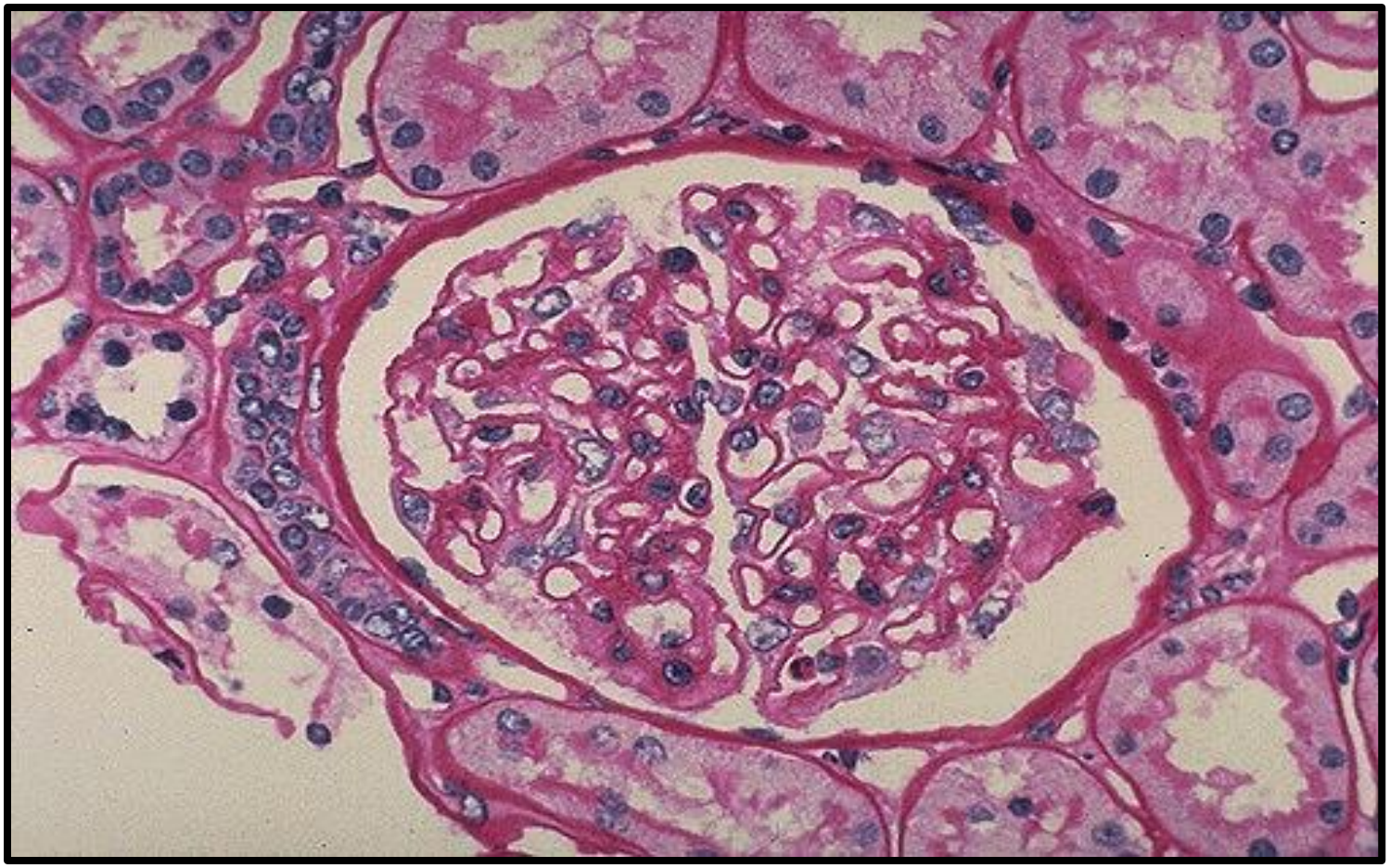


Renal Corpuscle – Normal Histology



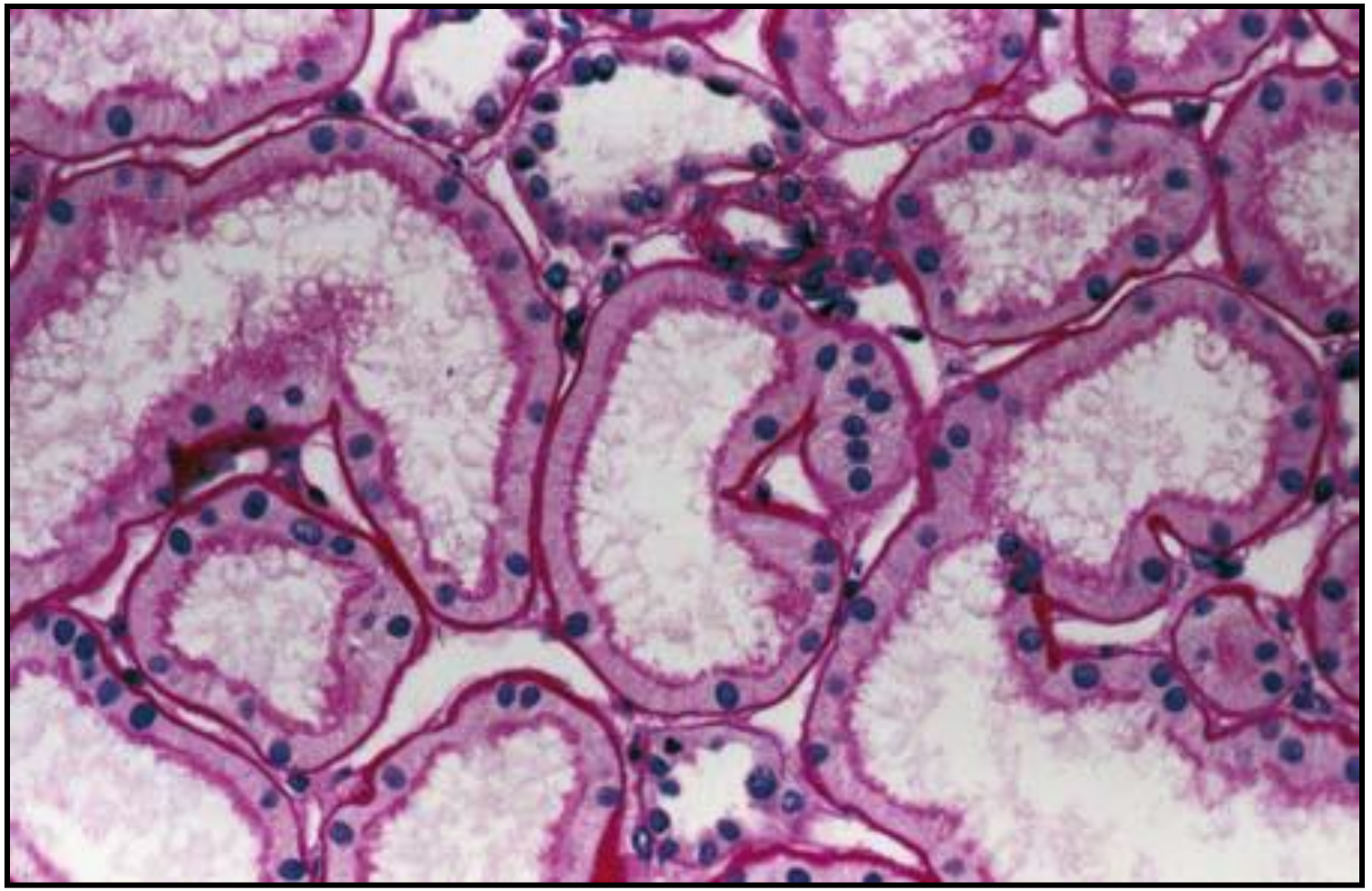
Normal glomerulus by light microscopy. The glomerular capillary loops are thin and delicate. Endothelial and mesangial cells are normal in number. The surrounding tubules are normal

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

- Defined as an abrupt or rapid decline in renal filtration function. This condition is usually marked by a rise in serum creatinine concentration or a rise in blood urea nitrogen
- Acute tubular necrosis (ATN) is the most common cause of acute kidney injury (AKI)
- is a medical condition involving the death of tubular epithelial cells that form the renal tubules of the kidneys.

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:

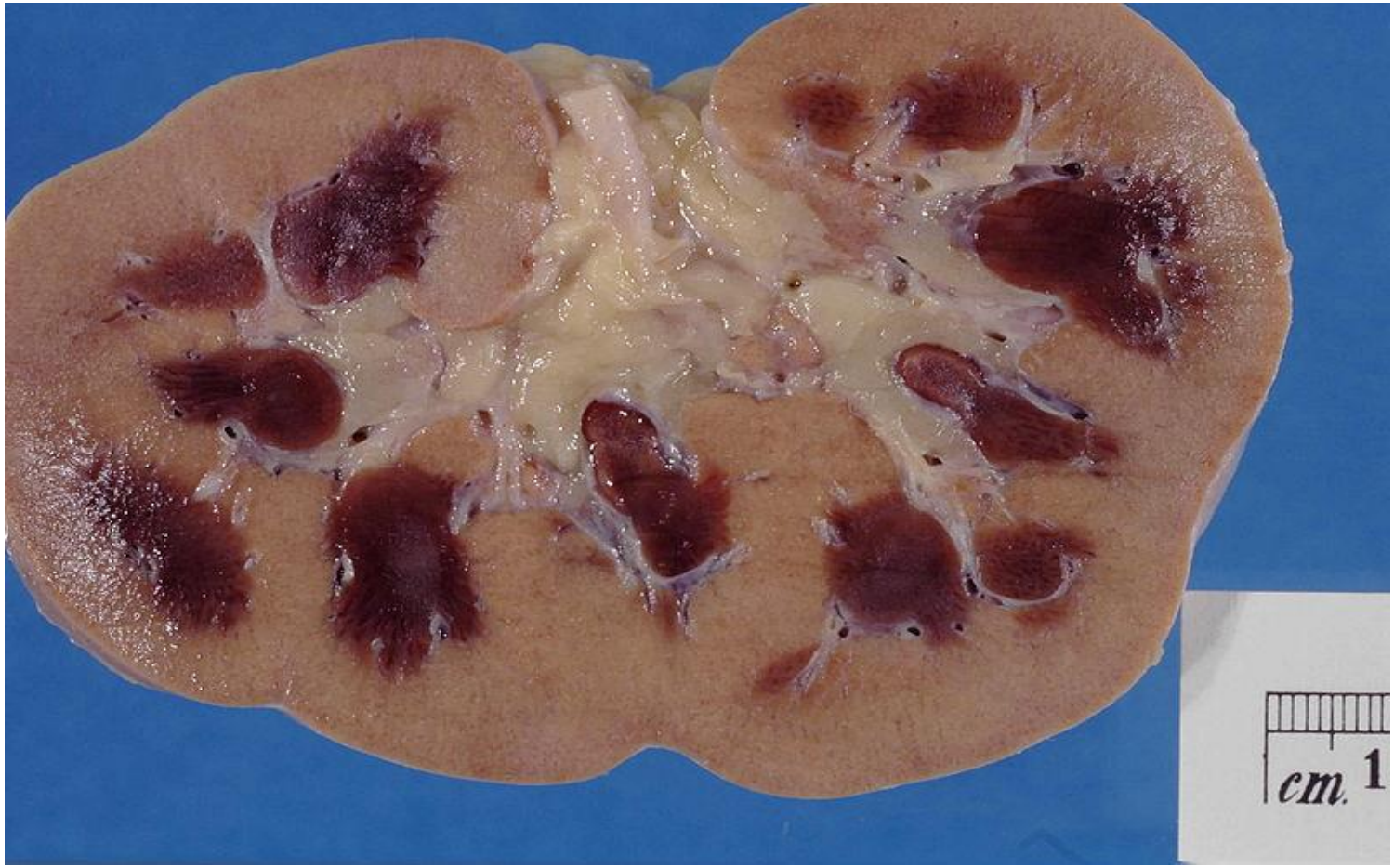
- *Glomerulonephritis (GN).*
- *Acute tubular necrosis (ATN).*
- *Acute interstitial nephritis (AIN).*

Post-renal:

(is a consequence of urinary tract obstruction)

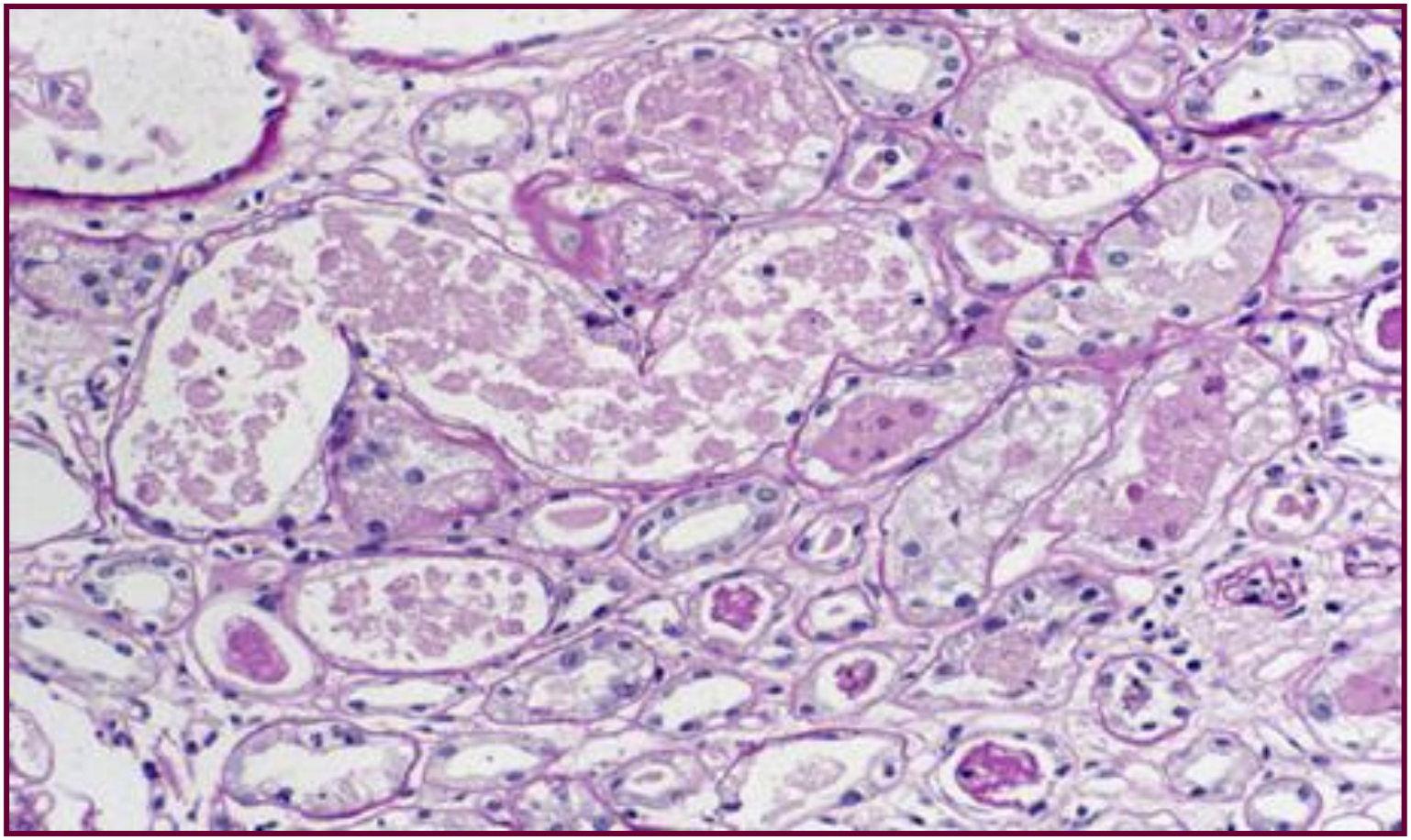
- *Benign prostatic hyperplasia.*
- *Kidney stones.*
- *Obstructed urinary catheter.*
- *Bladder stone .*
- *Bladder, ureteral or renal malignancy.*

Acute Kidney Injury



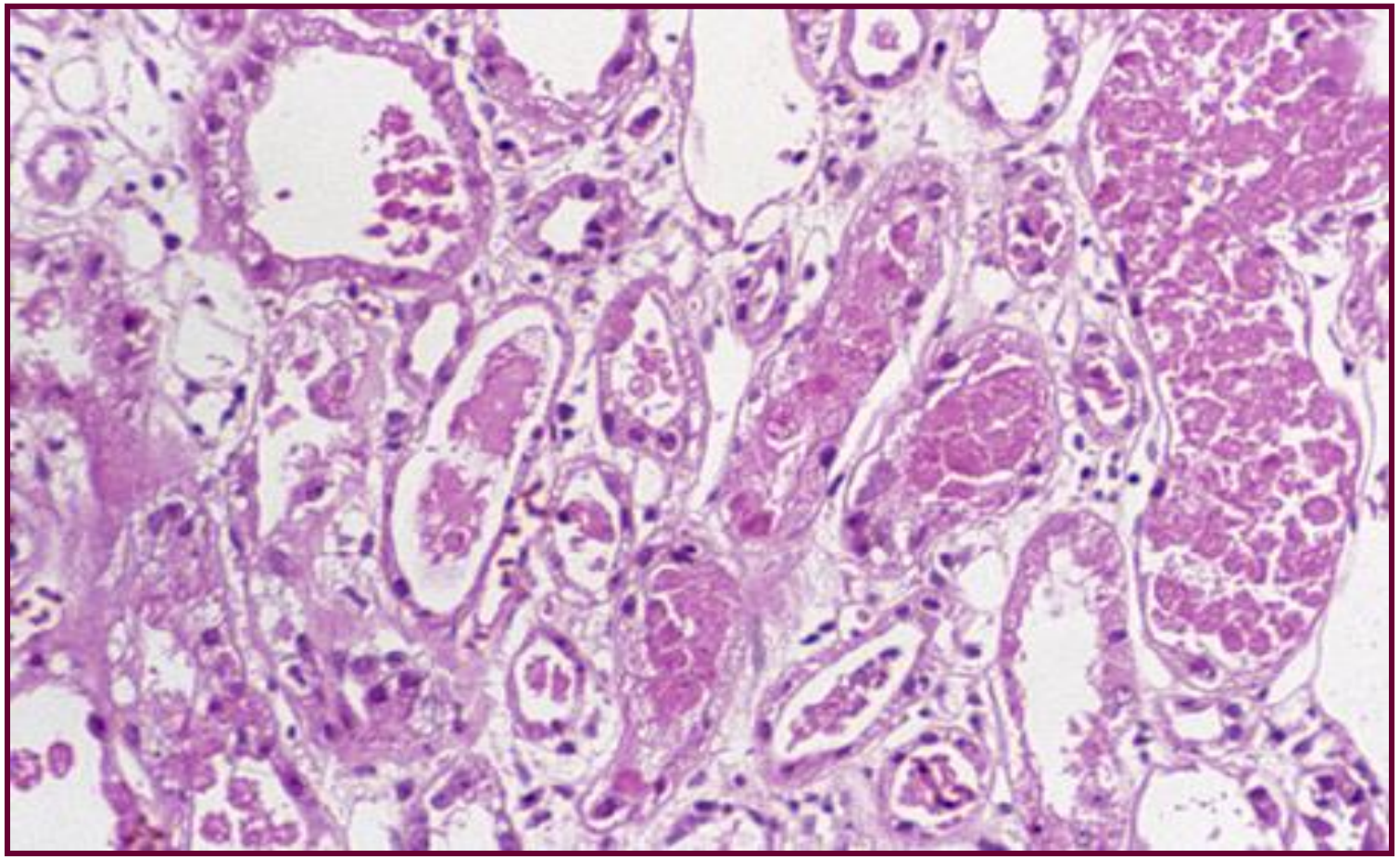
Kidney showing marked pallor of the cortex, contrasting to the darker areas of surviving medullary tissue.

Acute Tubular Necrosis



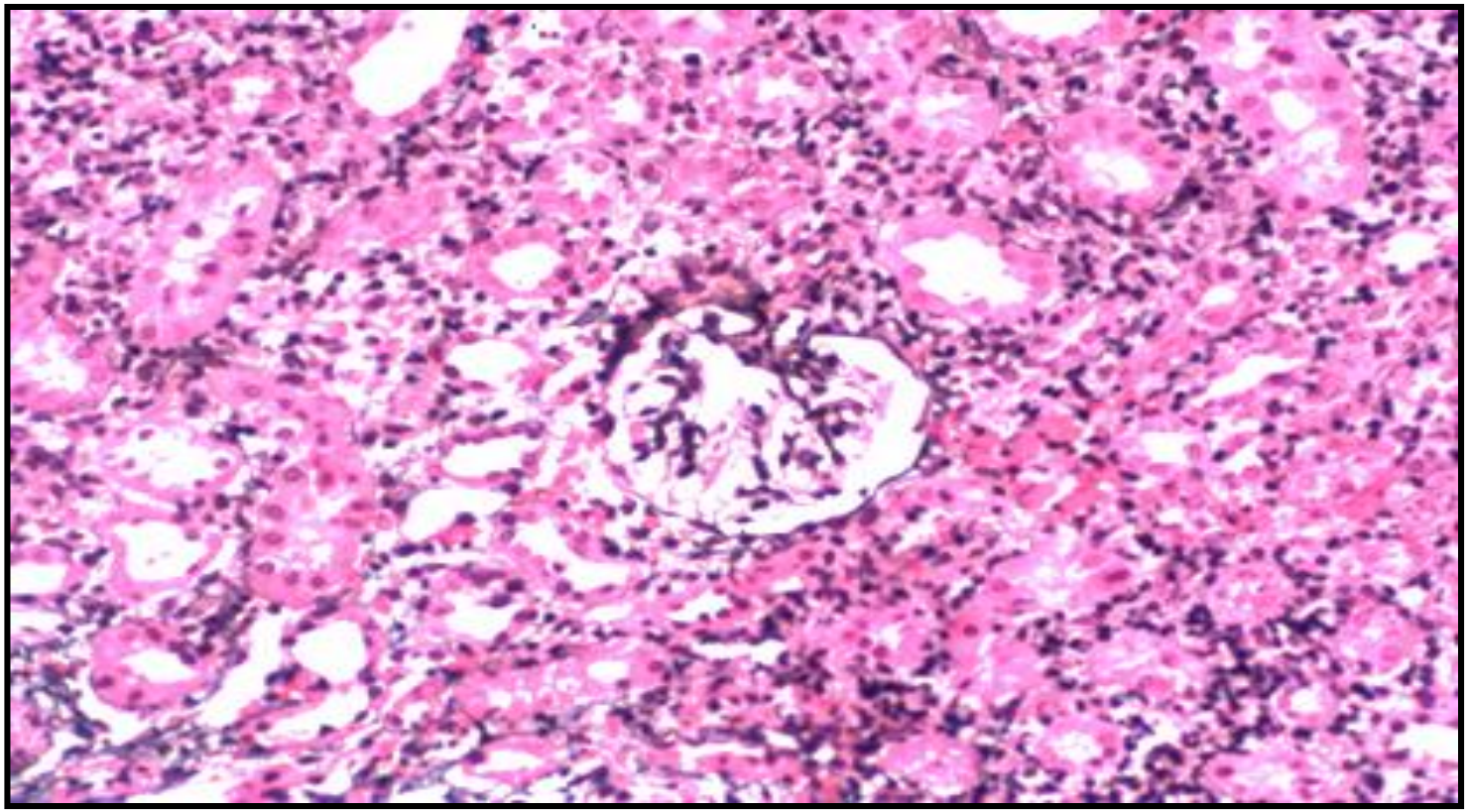
Acute tubular necrosis is manifest by vacuolated cells and sloughed, necrotic cells in tubular lumina, with some tubules lined by flattened epithelium and some showing frank necrosis (PAS stain, x 400).

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).

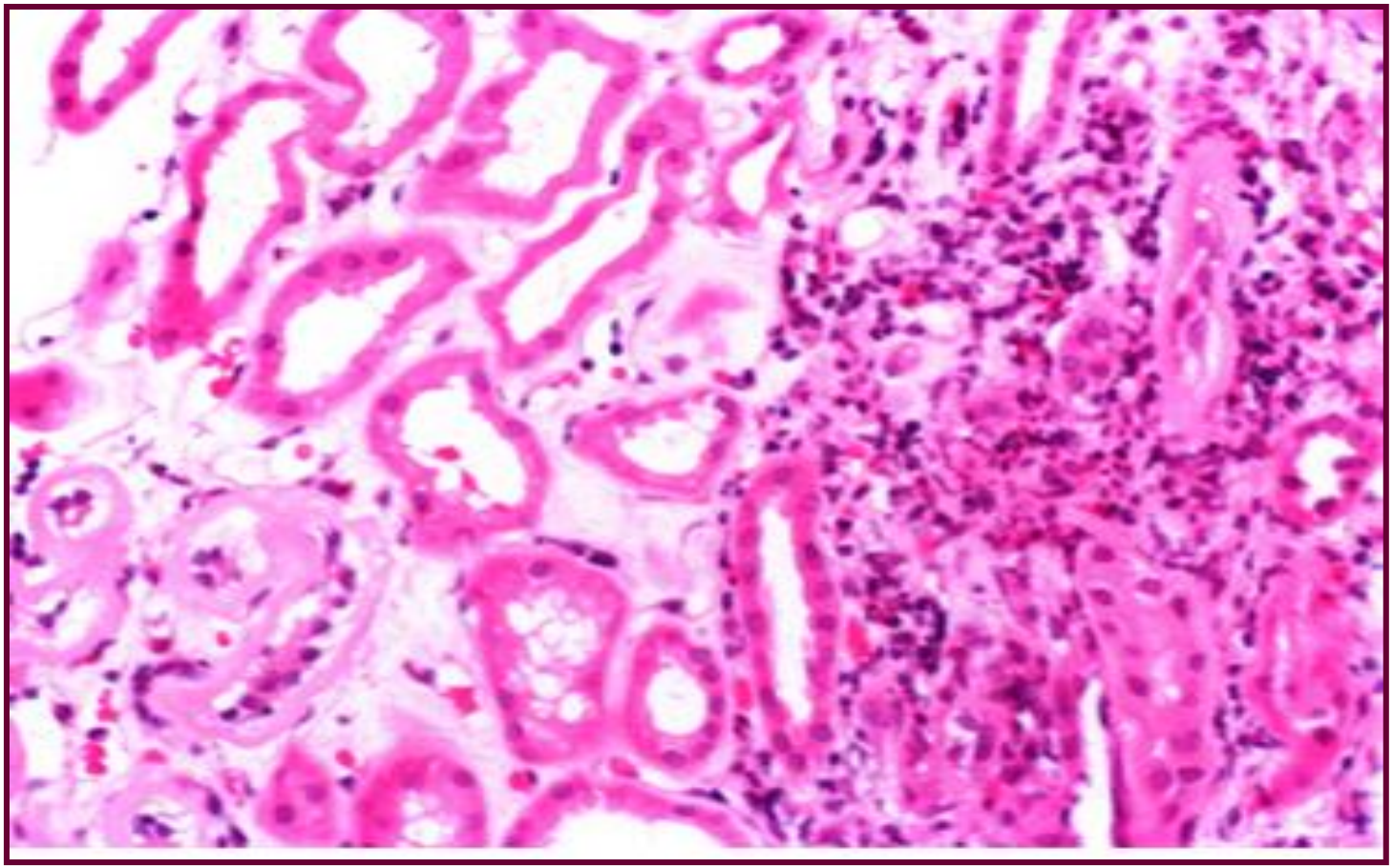
Acute Interstitial Nephritis



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



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

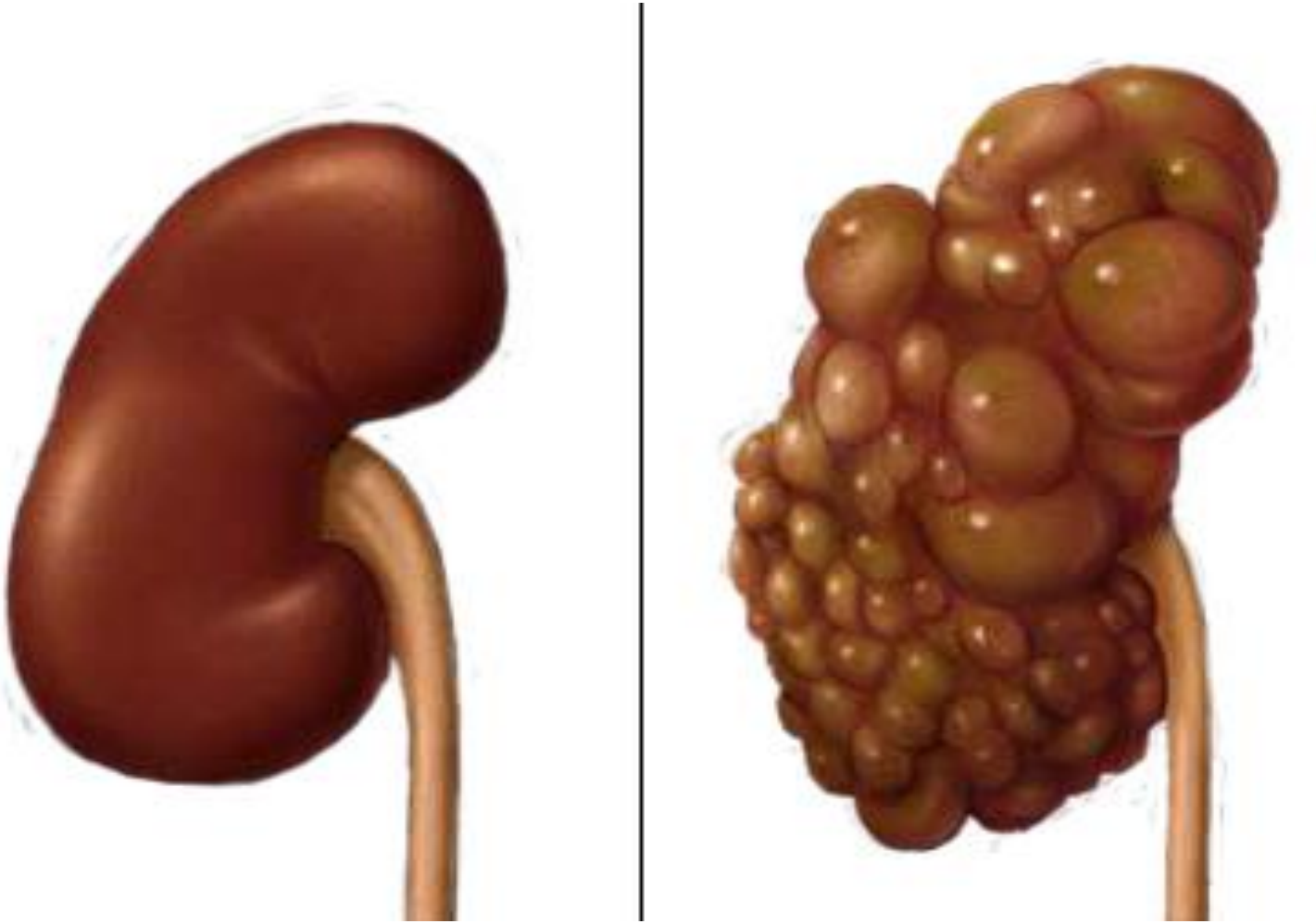
POLYCYSTIC KIDNEY

- is an inherited disorder in which clusters of cysts develop primarily within your kidneys, causing your kidneys to enlarge and lose function over time.
- Cysts are noncancerous round sacs containing fluid. The cysts vary in size, and they can grow very large.

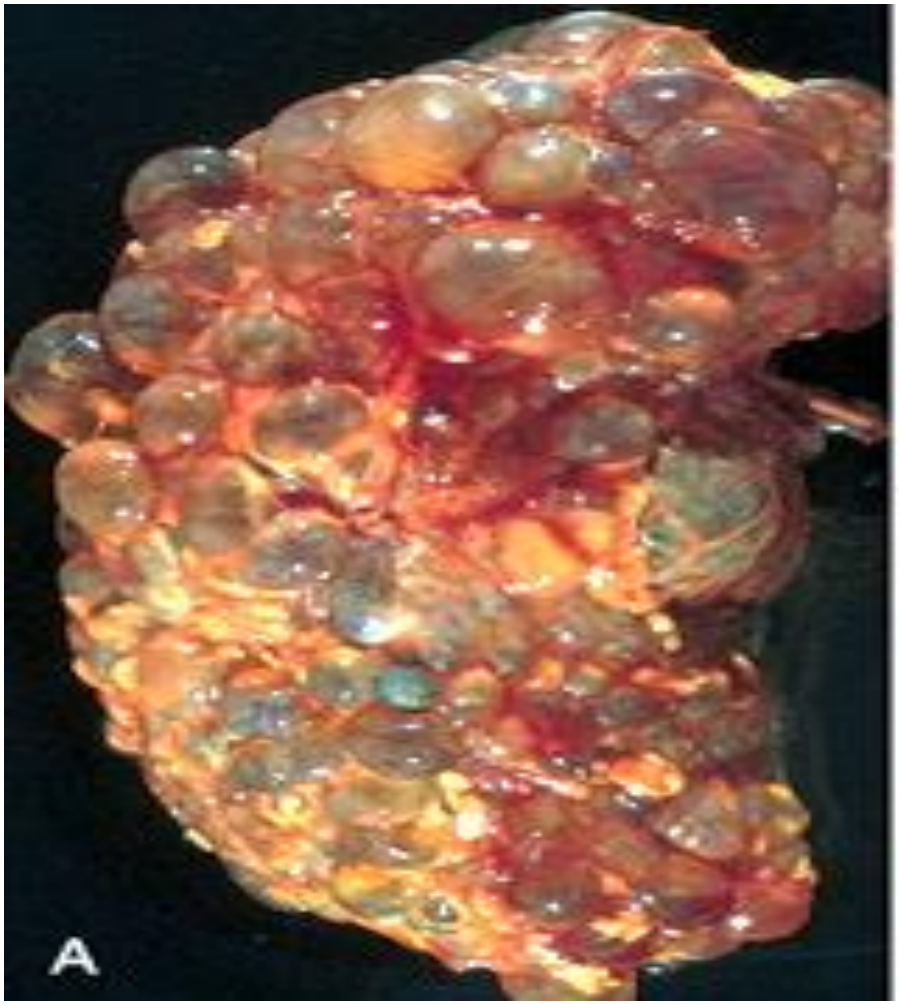
- Autosomal dominant polycystic kidney disease, Signs and symptoms of often develop between the ages of 30 and 40.

- Autosomal recessive polycystic kidney disease, less common than is ADPKD. The signs and symptoms often appear shortly after birth

Normal vs Polycystic Kidney

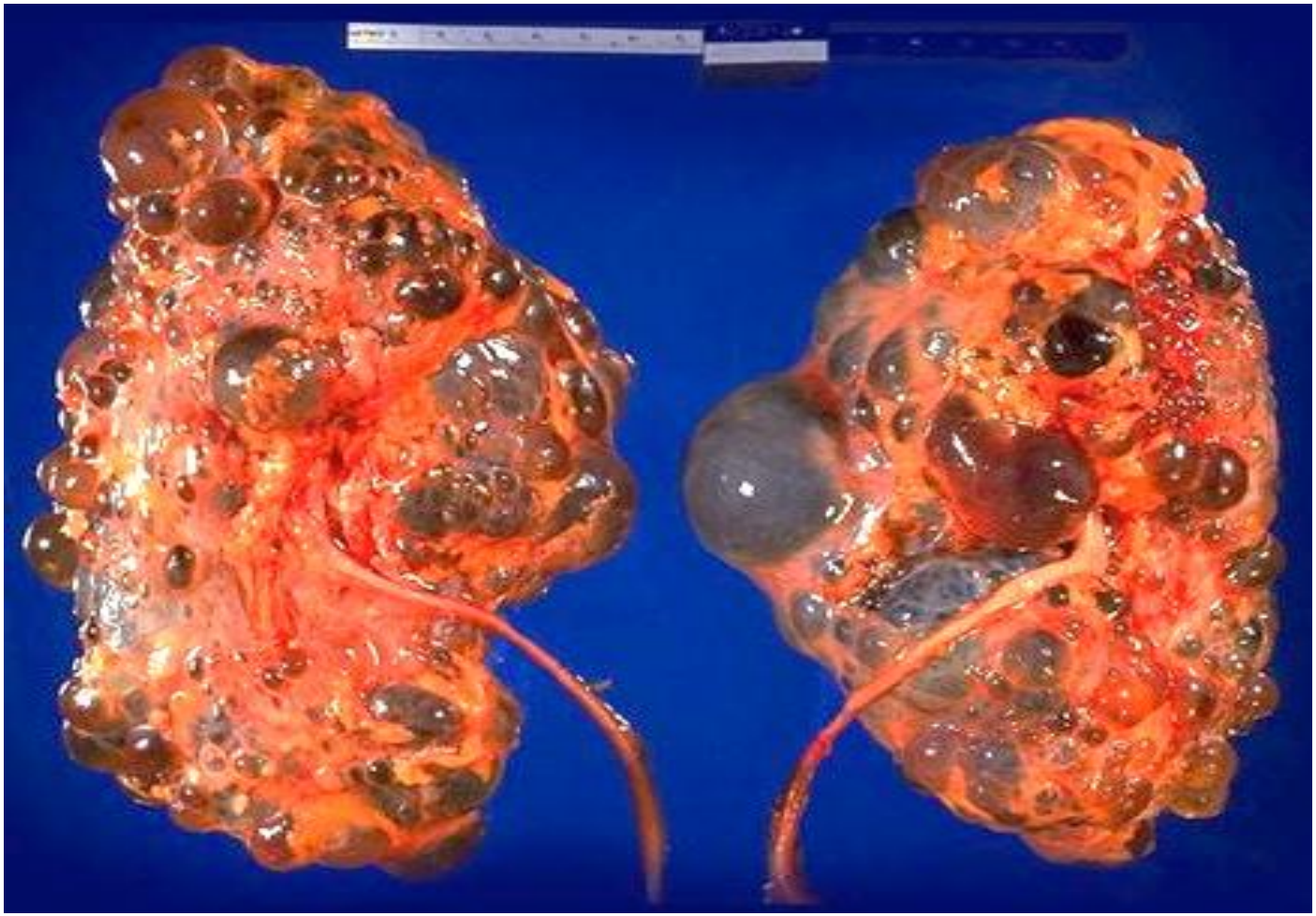


Polycystic kidney – Gross Anatomy



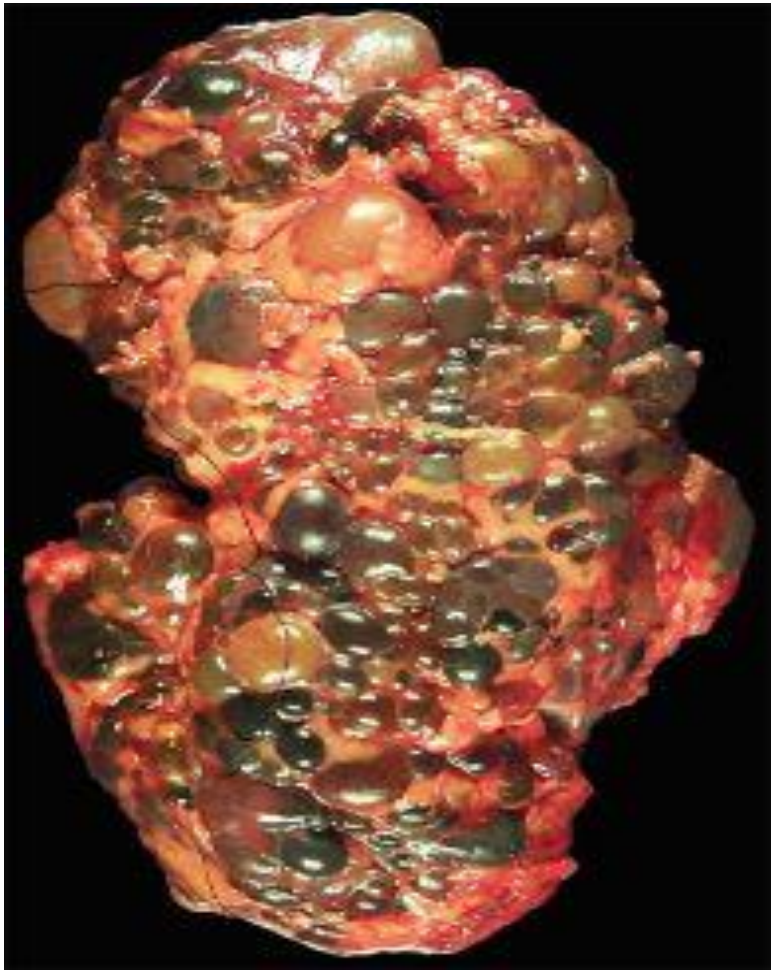
Markedly enlarged kidney and replacement of the renal parenchyma by numerous cysts of variable sizes

Polycystic kidney – Gross Anatomy

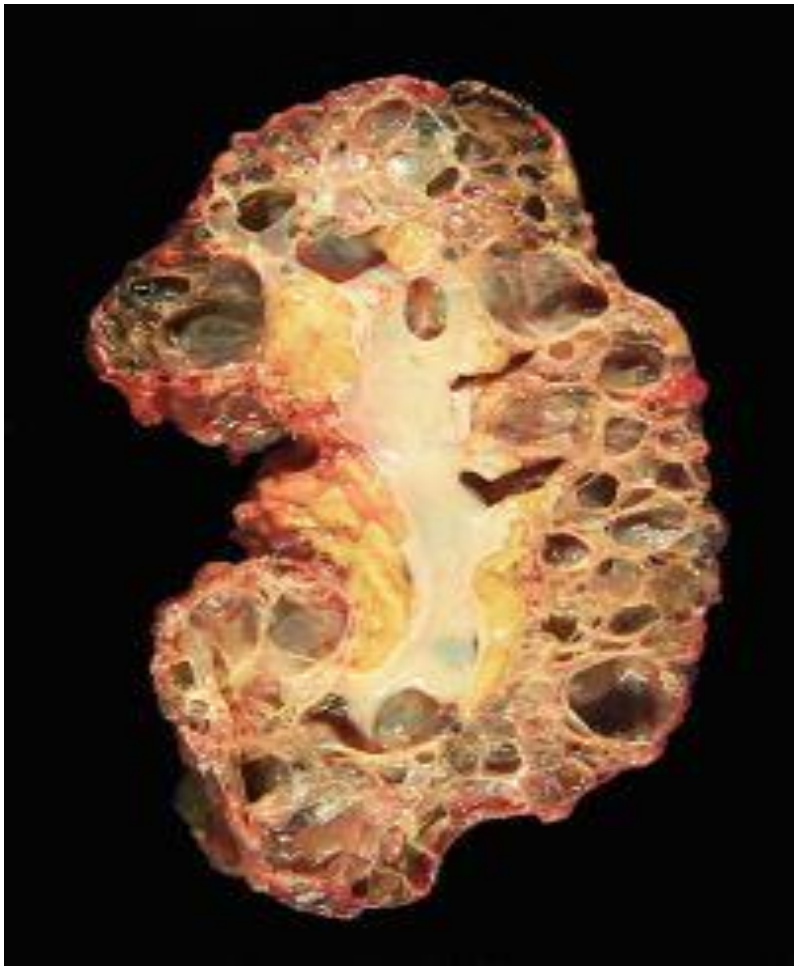


Bilateral autosomal dominant polycystic kidney disease

Gross Polycystic kidney and its Cut Section

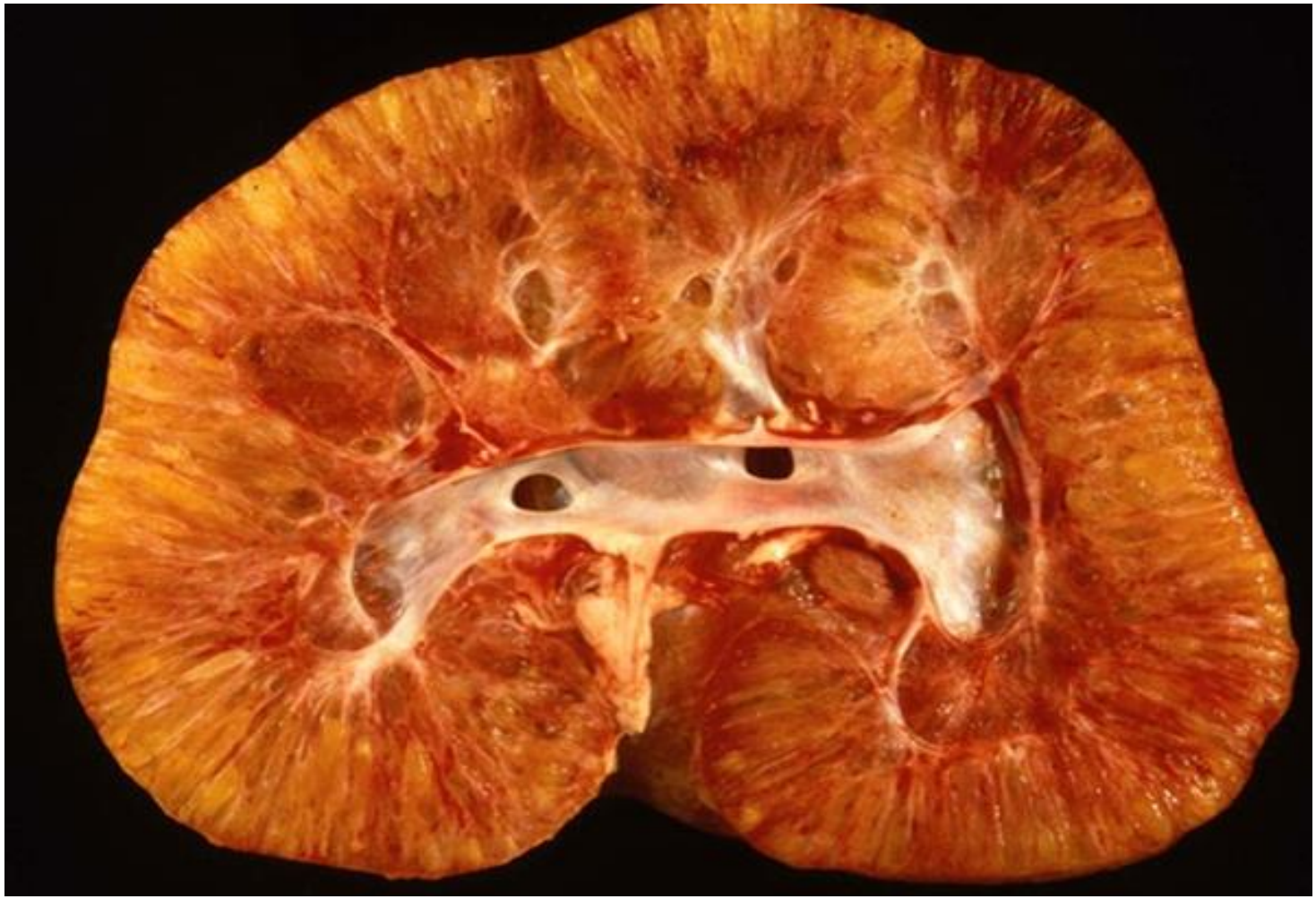


Massively enlarged kidney disrupted by numerous cysts



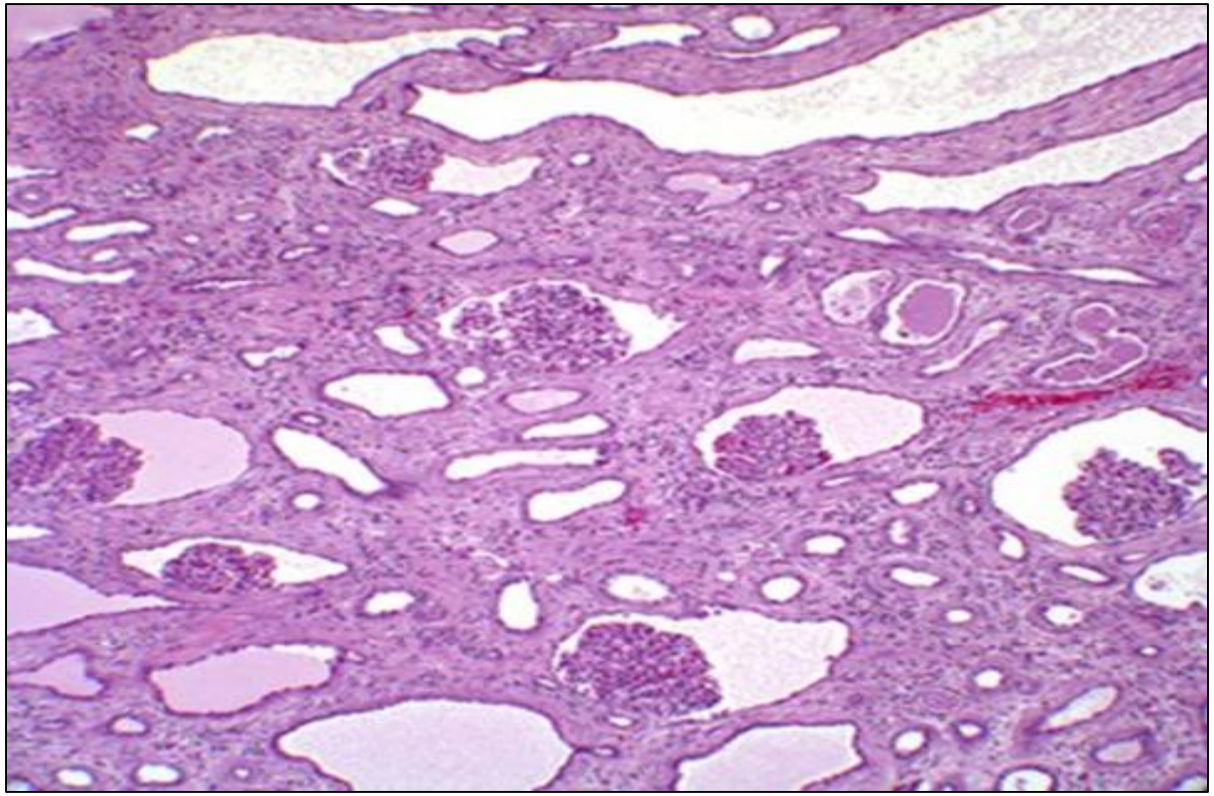
Cut surface of the kidney, showing extensive cortical destruction by cysts

Infantile Polycystic kidney – Gross



Coronal section of an infantile polycystic kidney

Polycystic kidney – Histopathology



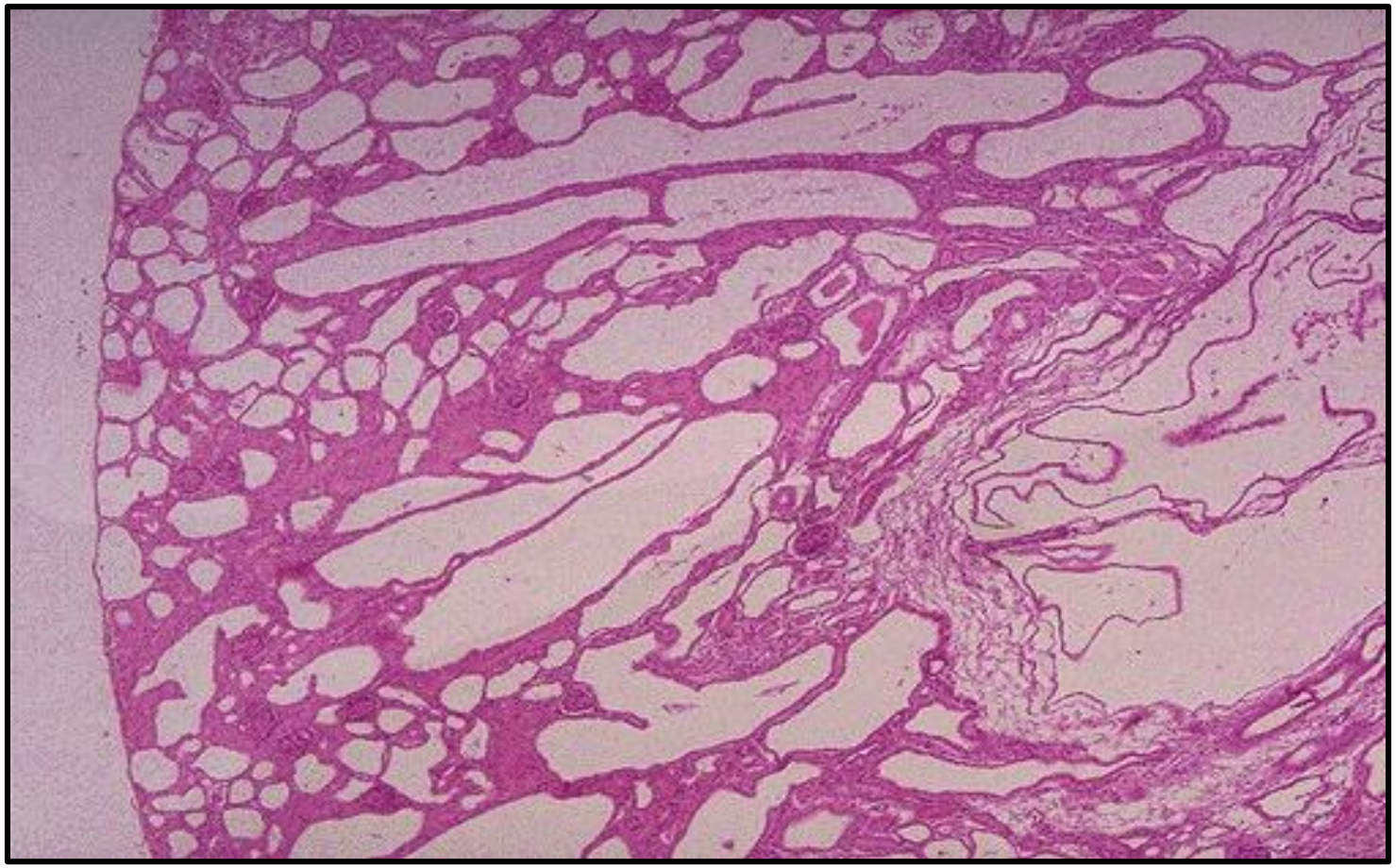
Kidney of child with autosomal dominant PCKD.

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.

PRACTICAL SESSION : 2

INFECTION OF THE URINARY TRACT

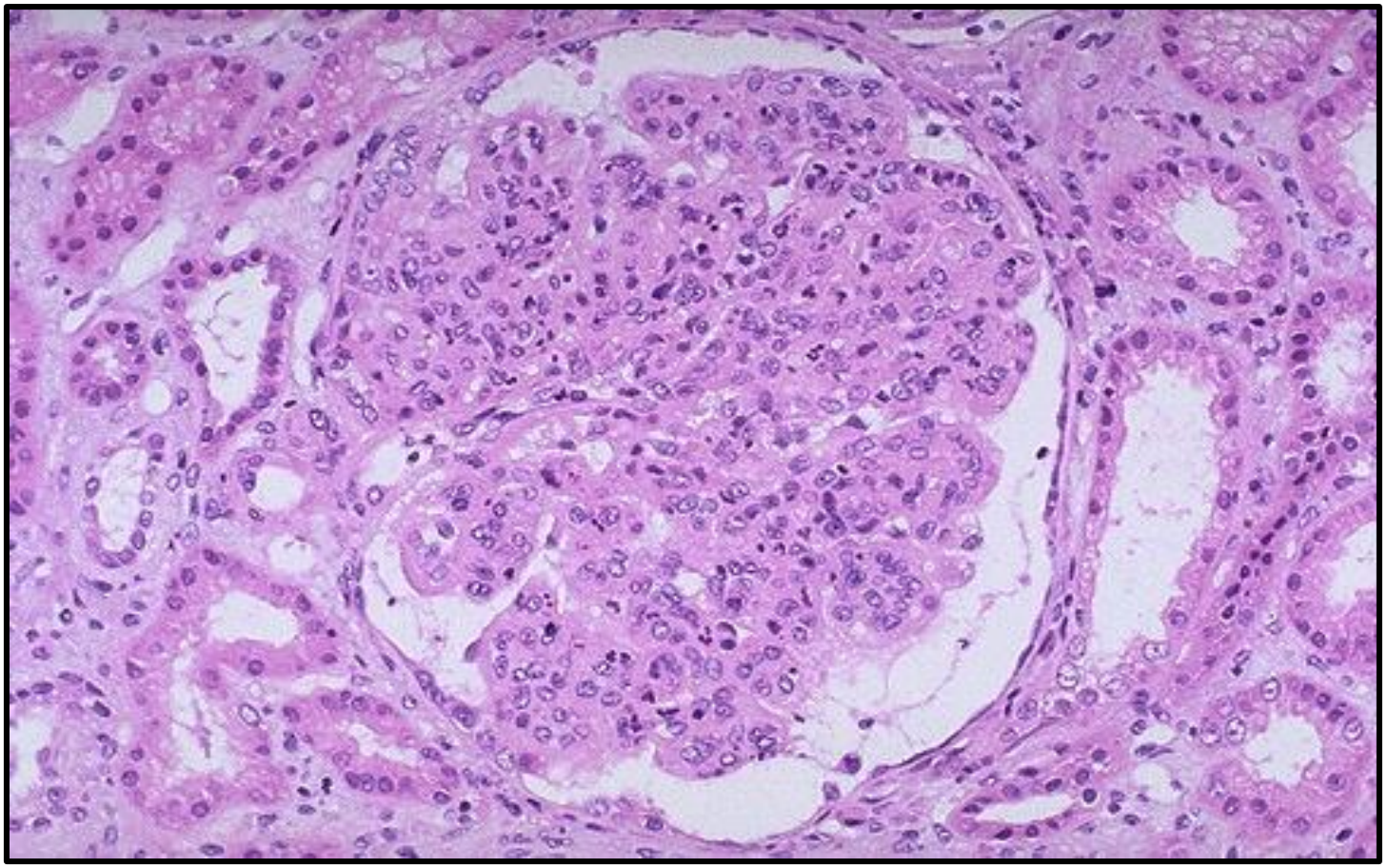
ACUTE (POST-STREPTOCOCCAL) GLOMERULONEPHRITIS

Acute (Post-streptococcal) Glomerulonephritis

Section of the kidney shows:

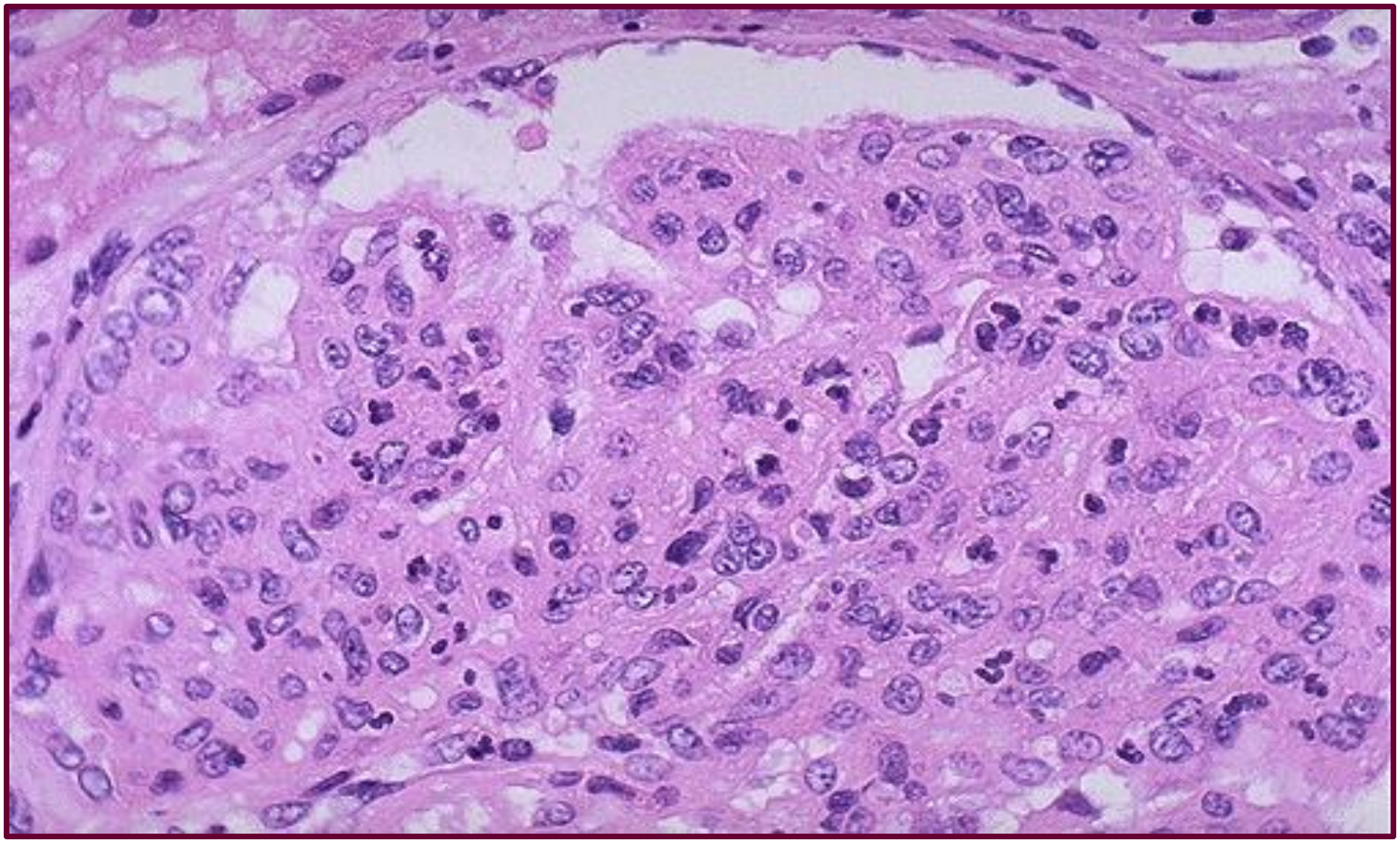
- **The glomeruli are enlarged, lobulated and hypercellular with obliteration of capsular space.**
- **Cellularity is due to proliferation of endothelial and mesangial cells with some neutrophils.**
- **Many capillaries appear obliterated.**
- **Tubules show degenerative changes.**

Acute (Post-streptococcal) Glomerulonephritis



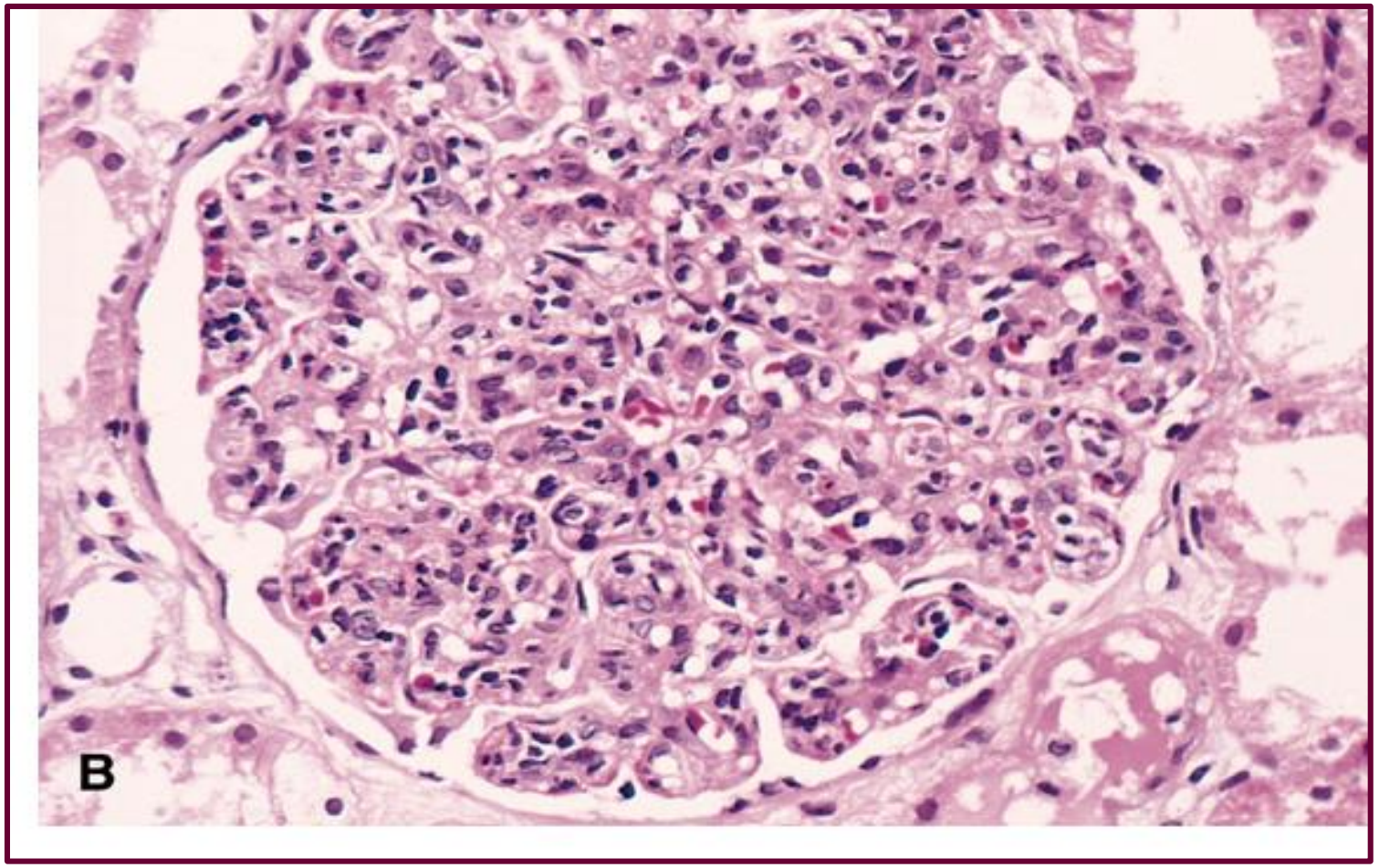
This glomerulus is hypercellular and capillary loops are poorly defined. This is a type of proliferative glomerulonephritis known as post-infectious glomerulonephritis

Acute (Post-streptococcal) Glomerulonephritis



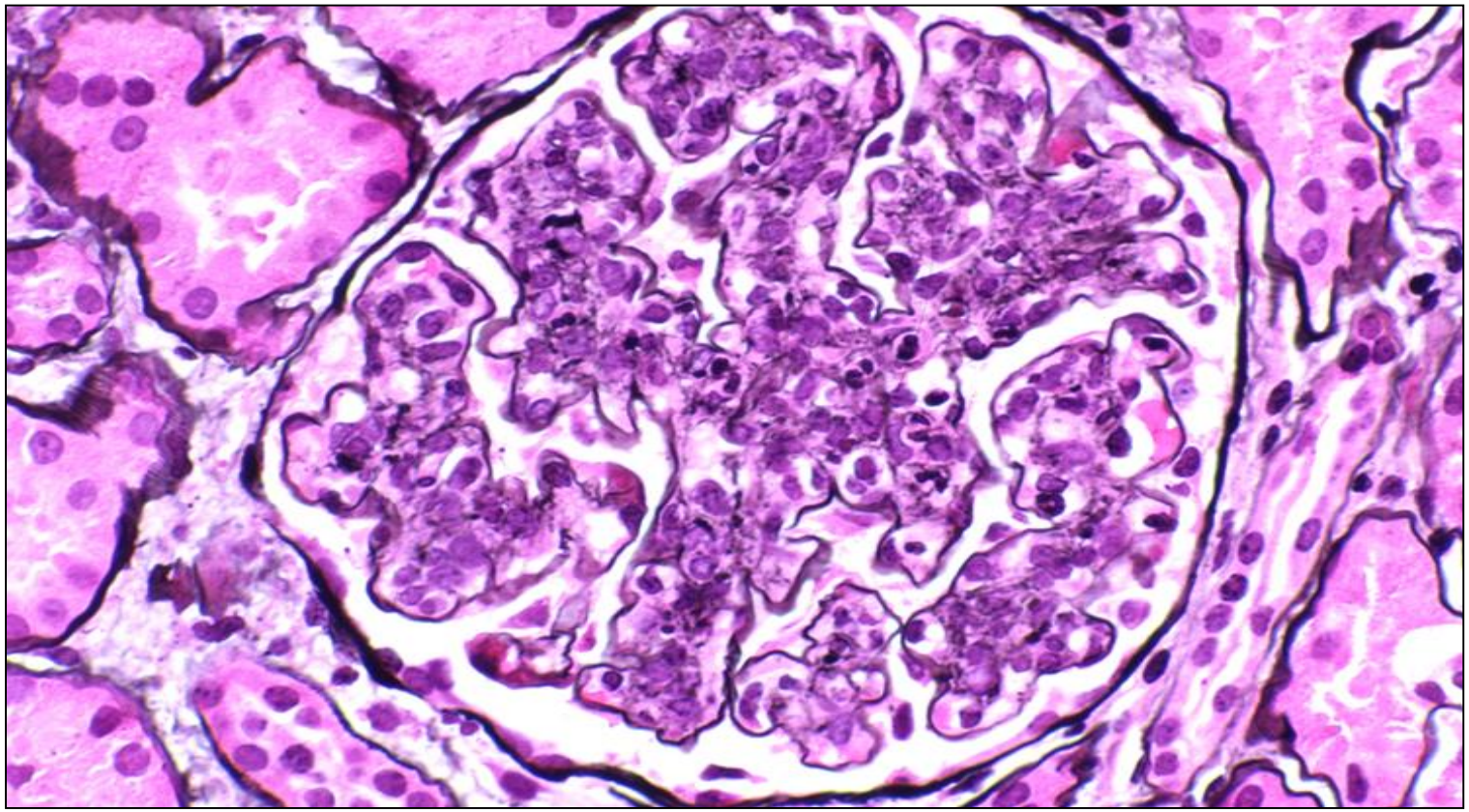
The hypercellularity of post-infectious glomerulonephritis is due to increased numbers of epithelial, endothelial, and mesangial cells as well as neutrophils in and around the glomerular capillary loops

Acute (Post-streptococcal) Glomerulonephritis



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

Acute Post-streptococcal Glomerulonephritis

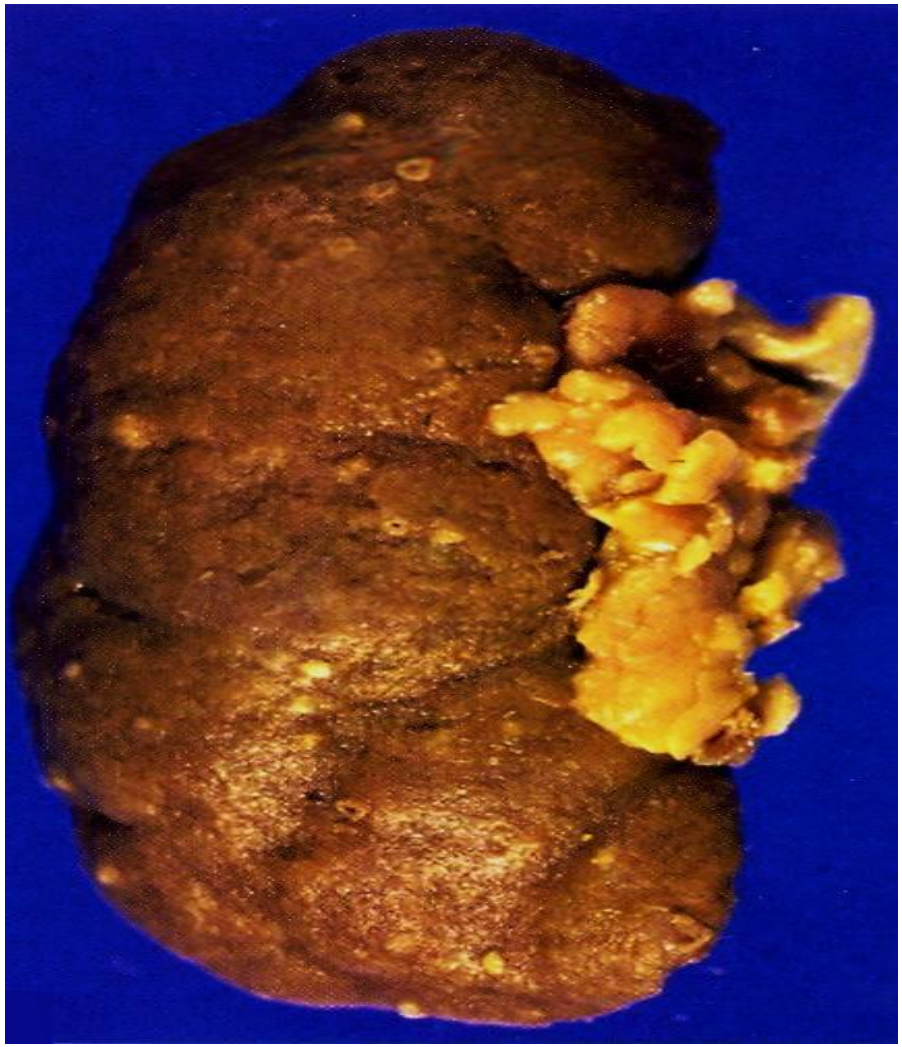


Acute Poststreptococcal Glomerulonephritis is evident in this high-power silver stain with large number of PMNs. The glomerular basement membrane does not show splitting or spikes. There is proliferation of endothelial and mesangial cells and infiltrating cells and filling and distending capillary loops.

ACUTE & CHRONIC PYELONEPHRITIS

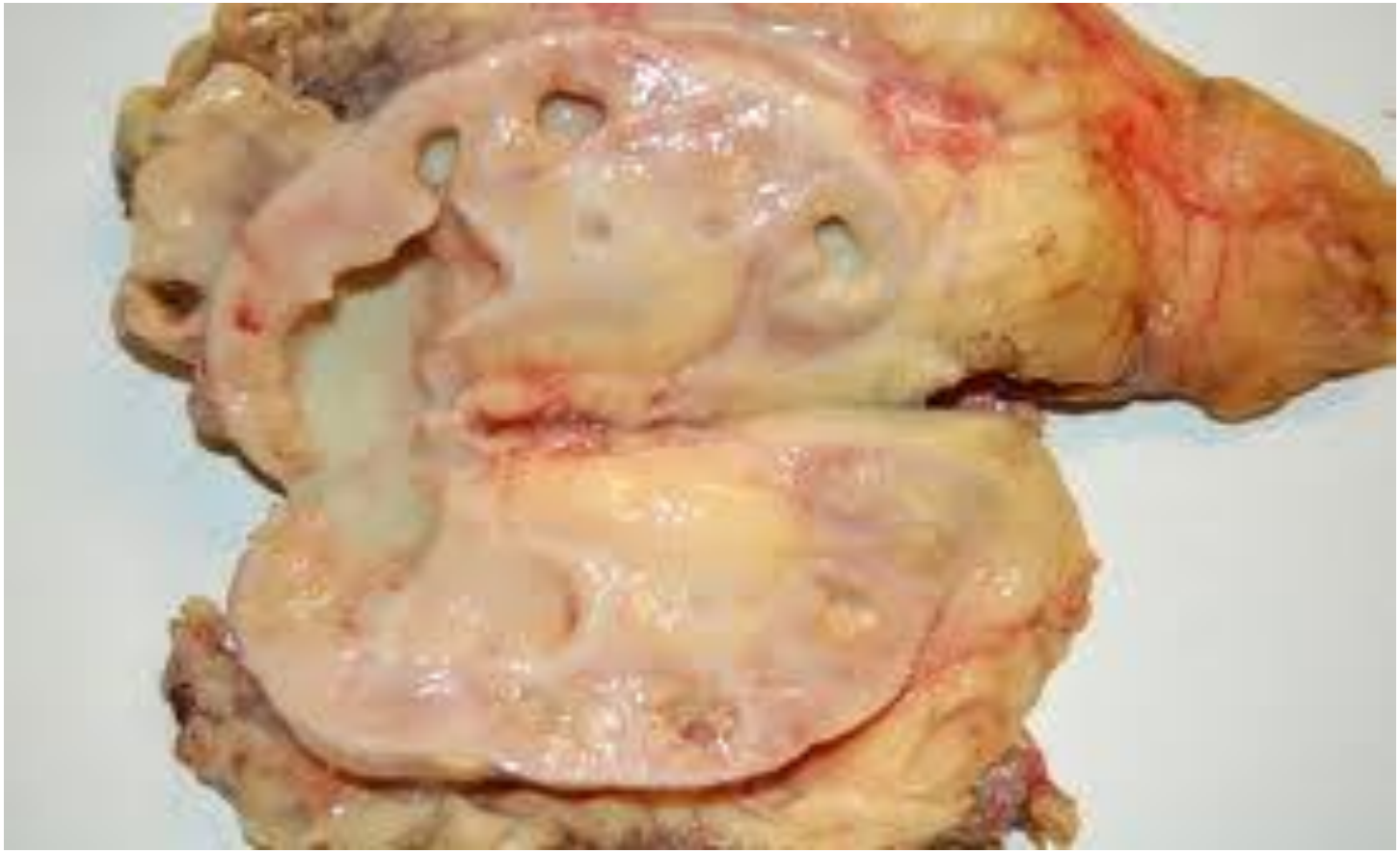
- Acute pyelonephritis is a sudden and severe kidney infection, life-threatening infection that often leads to renal scarring.
- Chronic pyelonephritis is characterized by renal inflammation and fibrosis induced by recurrent or persistent renal infection, vesicoureteral reflux, or other causes of urinary tract obstruction or due to the long-term damage done by recurrent urine infection

Pyelonephritis with small cortical abscesses



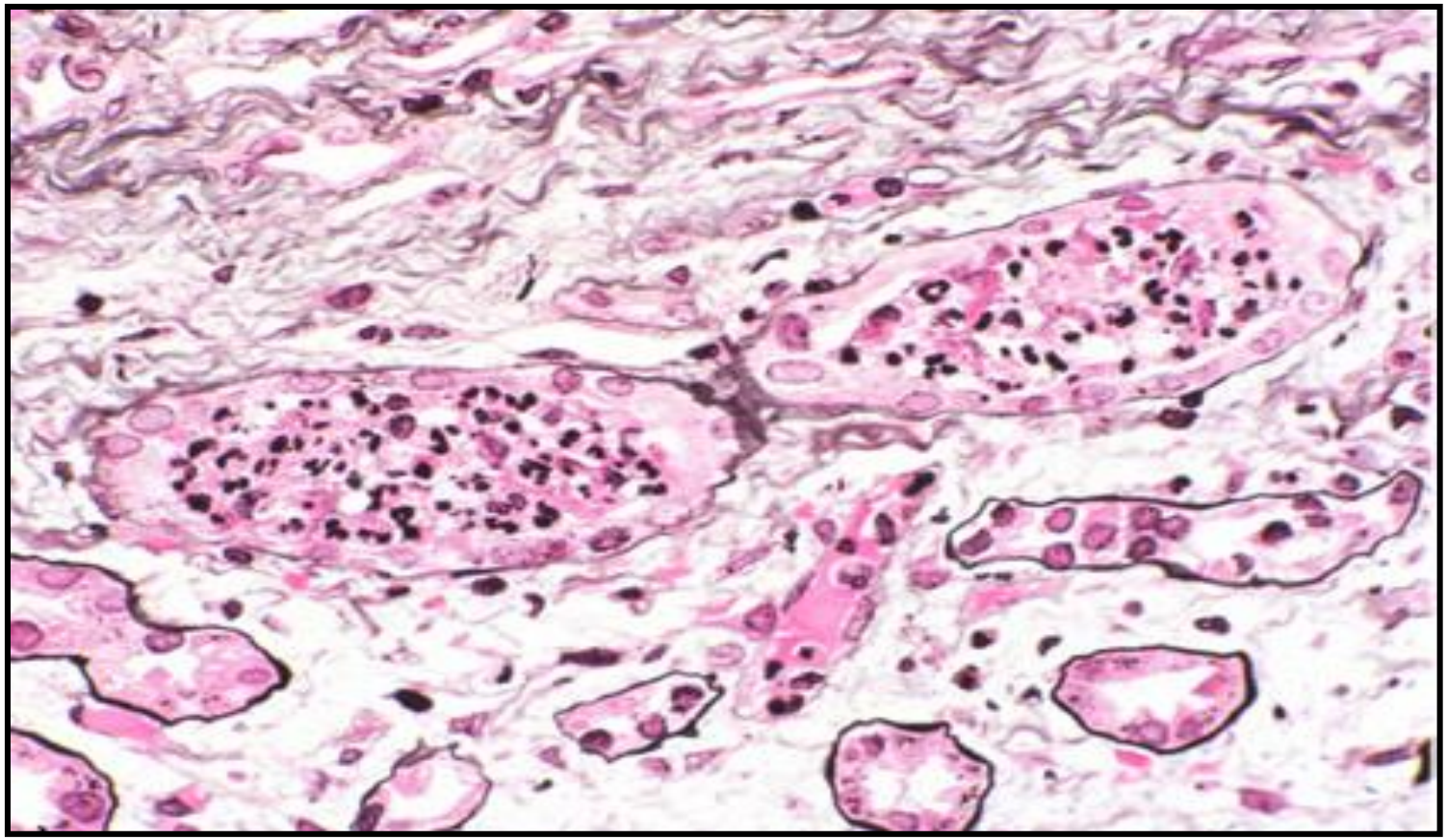
Pyelonephritis with small cortical abscesses

Classic picture of Pyelonephritis



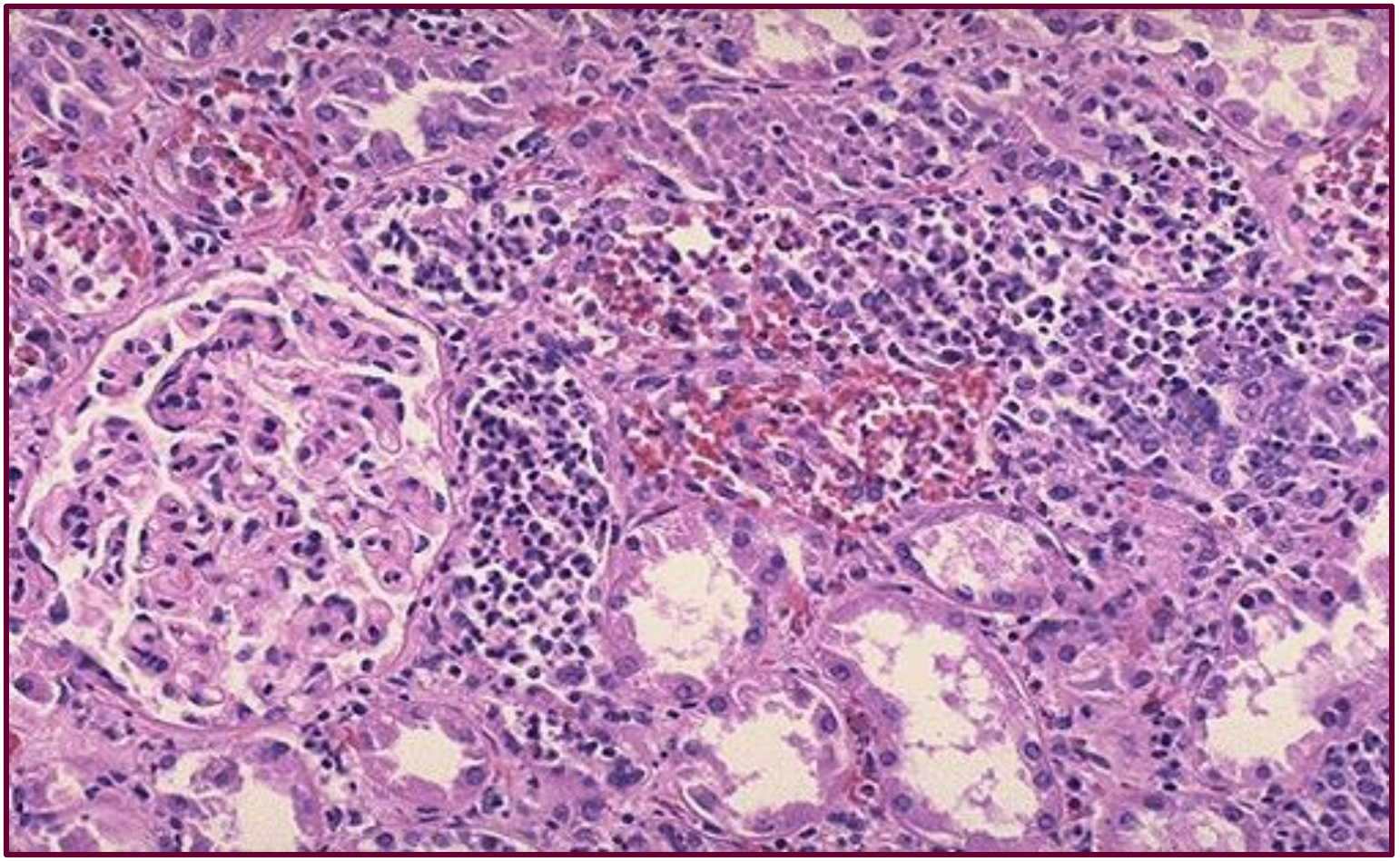
This kidney is bisected to reveal a dilated pelvis and calyxes filled with a yellow-green purulent pus which is consistent with a pyelonephritis. The cortex and medulla are pale and the corticomedullary junction is ill-defined.
No tumors are seen.

Acute Pyelonephritis - Histopathology



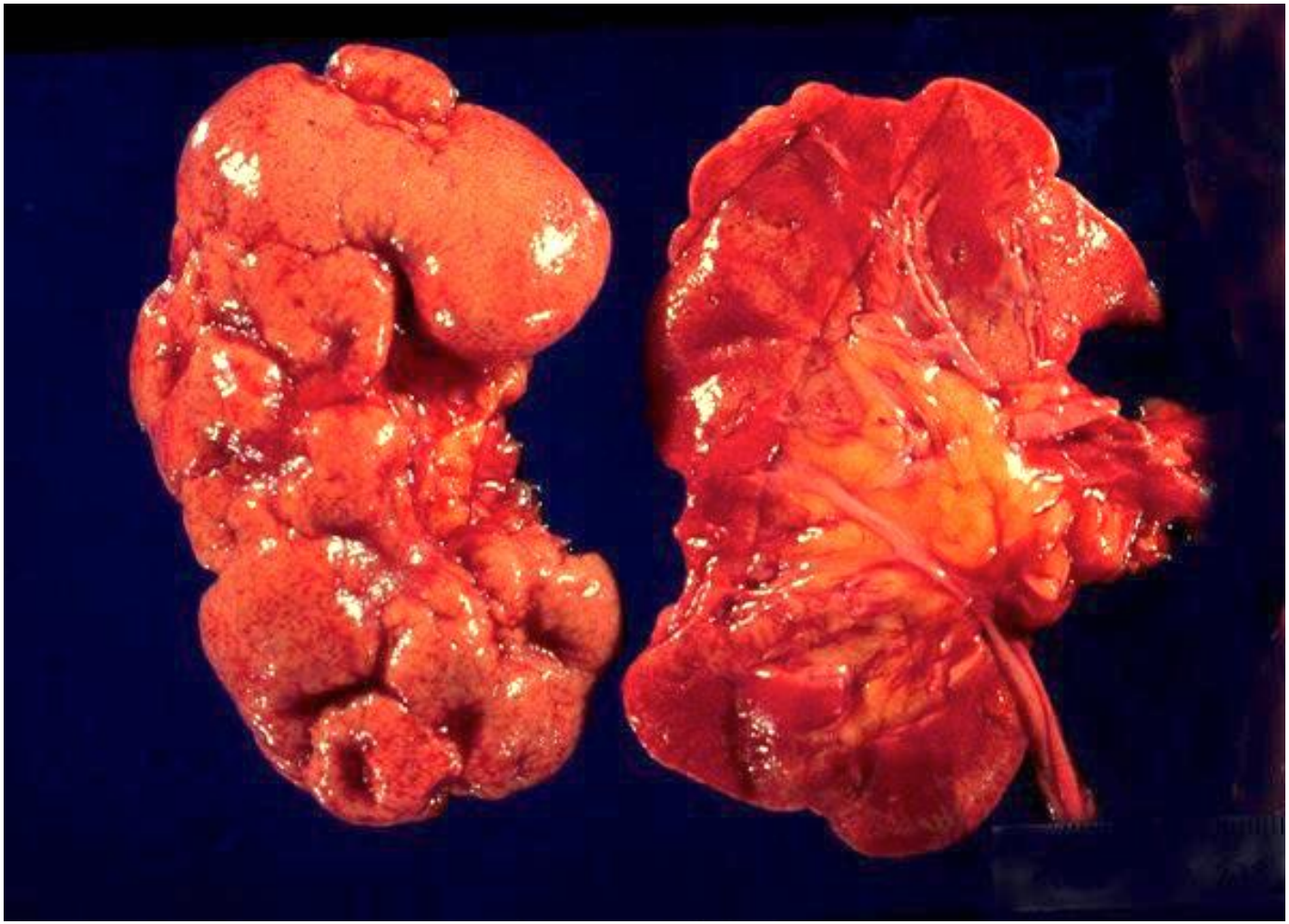
Acute pyelonephritis is diagnosed by intratubular aggregations of polymorphonuclear neutrophils (PMNs). There may be surrounding interstitial inflammation with a mixture of PMNs, lymphocytes, and plasma cells, but the predominant inflammation is within the tubule

Acute Pyelonephritis - Histopathology



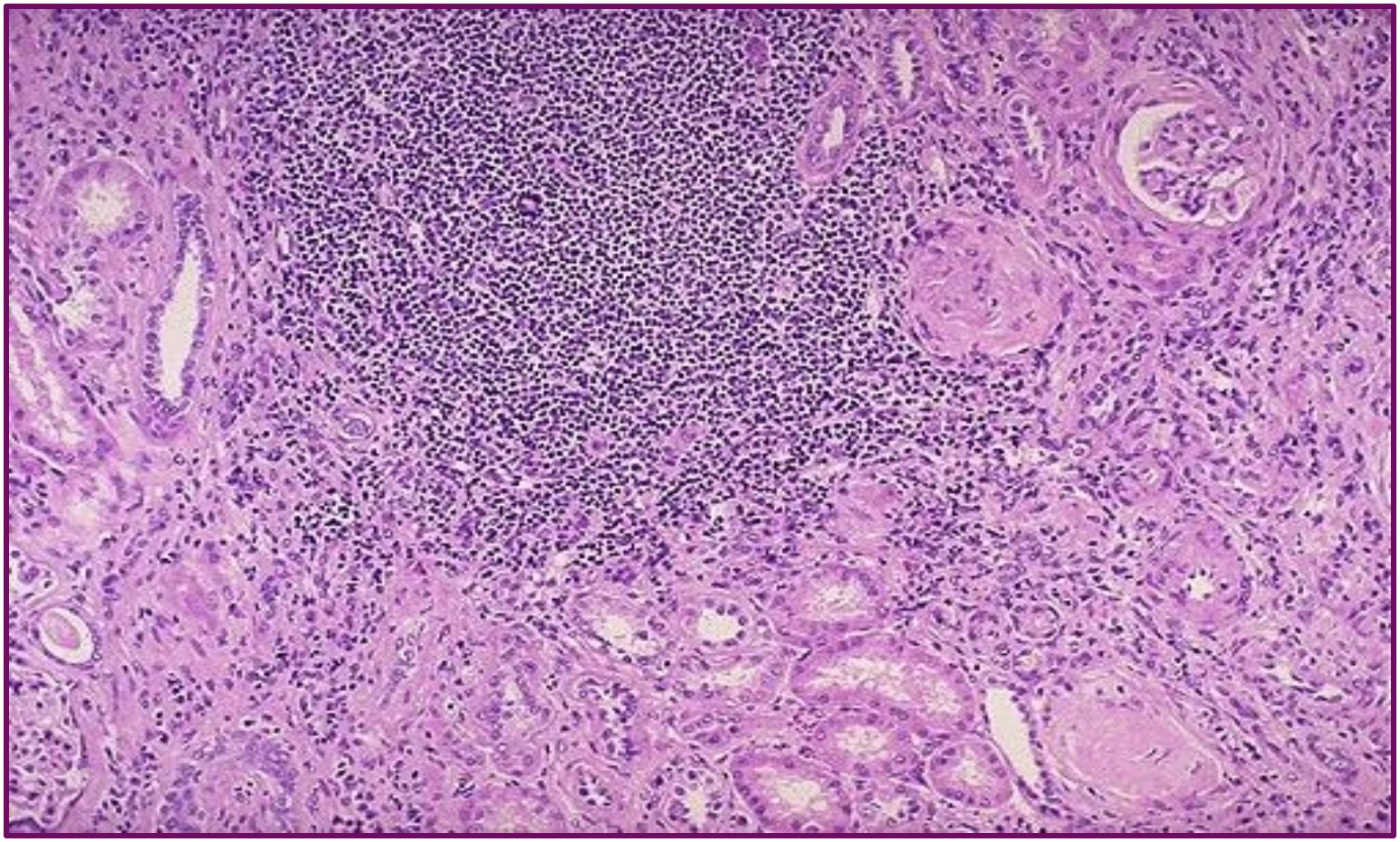
Numerous PMN's are seen filling renal tubules across the center and right of this picture. These leukocytes may form into a cast within the tubule. Casts appearing in the urine originate in the distal renal tubules and collecting ducts

Chronic Pyelonephritis - Gross Pathology



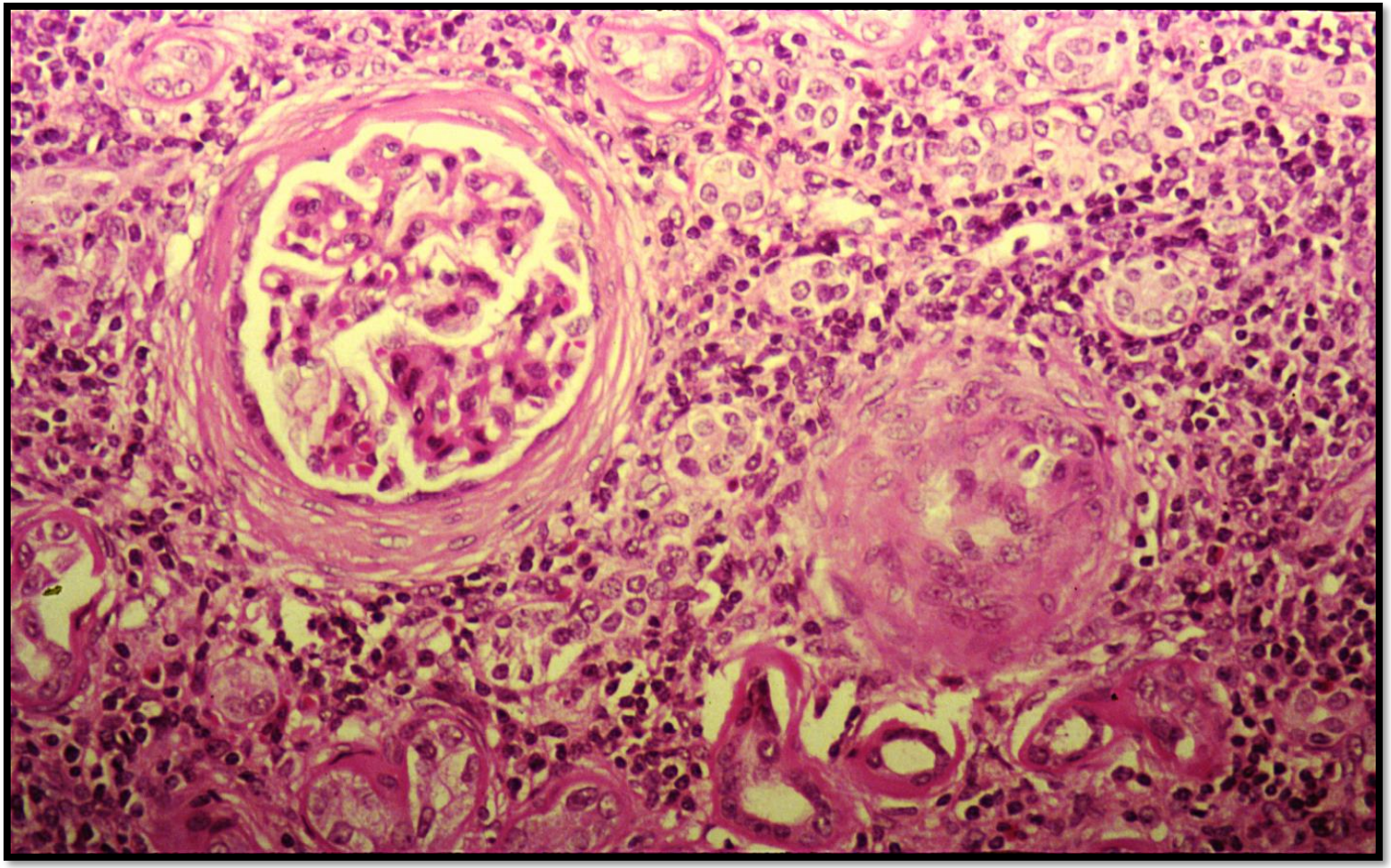
The picture shows slightly atrophic and deformed kidneys with cortical coarse scars .

Chronic Pyelonephritis - Histopathology



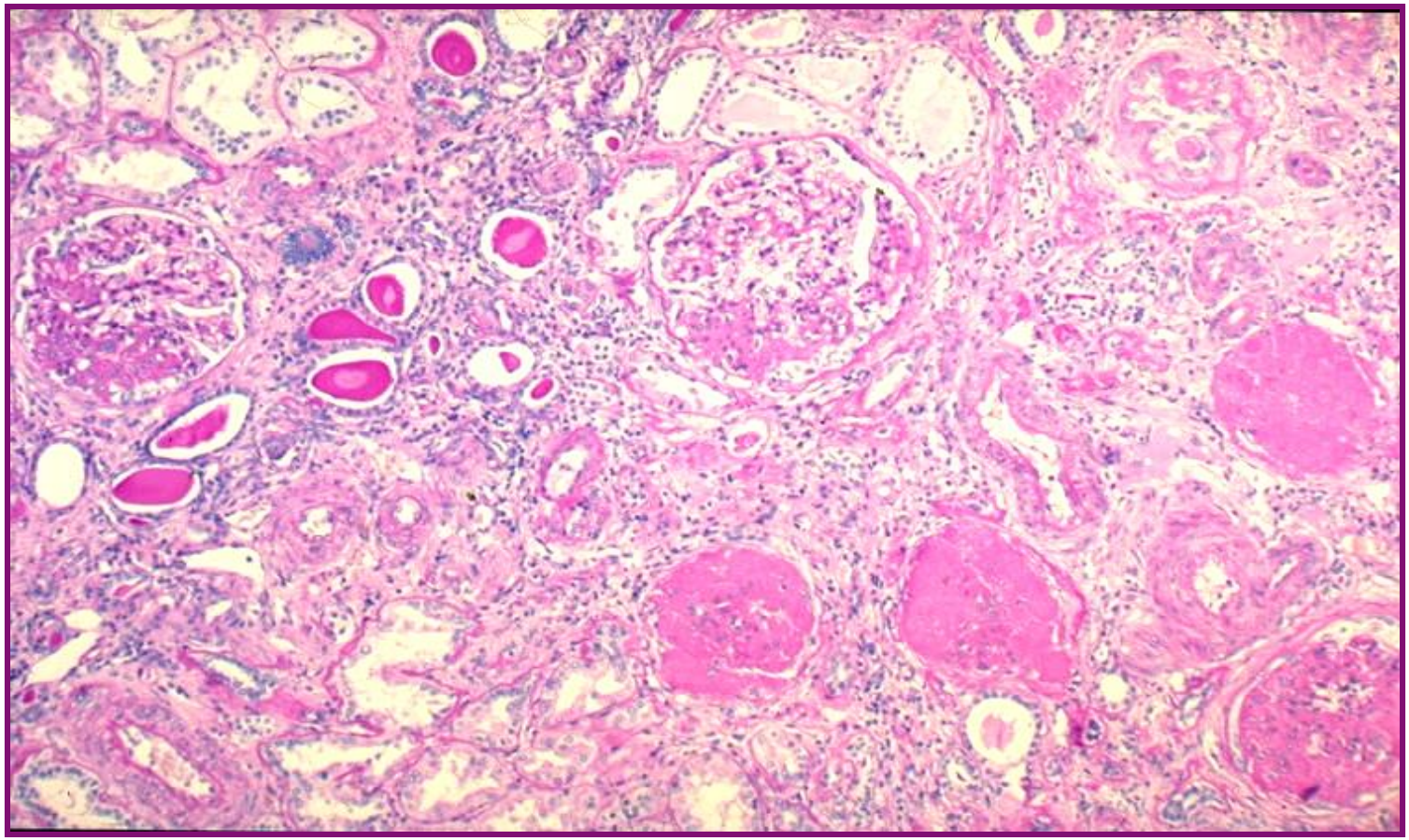
This is chronic pyelonephritis where a large collection of chronic inflammatory cells . The severity of disease depends upon the amount of remaining functional renal parenchyma

Chronic Pyelonephritis - Histopathology



High power shows periglomerular fibrosis , glomerular sclerosis and hyalinization, atrophy of the renal tubules and marked chronic interstitial inflammation .

Chronic Pyelonephritis - Histopathology

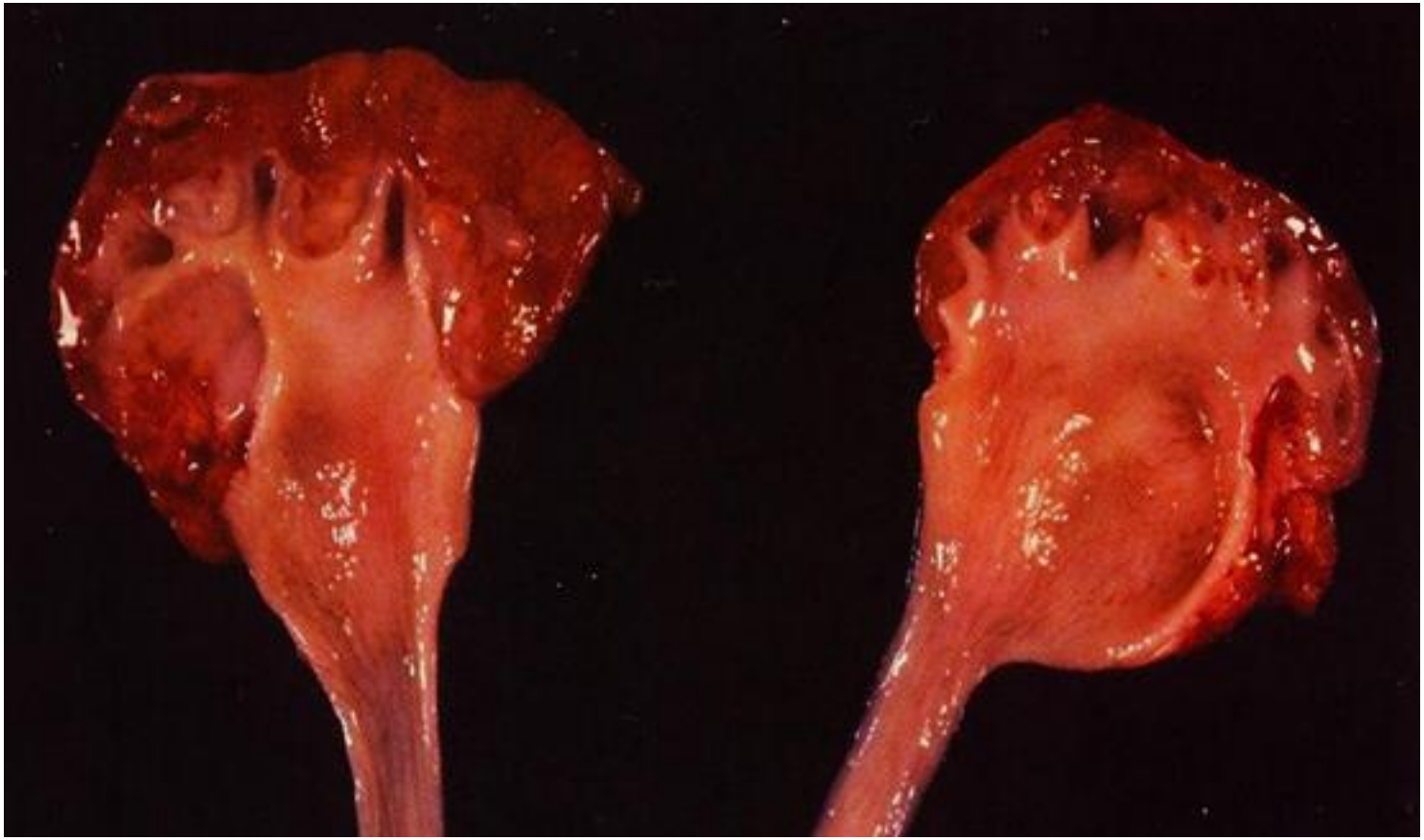


- ***Glomeruli*** *show varying degrees of sclerosis & periglomerular fibrosis.*
- ***Tubules*** *show varying degrees of atrophy, Some tubules are dilated and filled with Eosinophilic hyaline casts resembling colloid (thyroidization).*
- ***Interstitial tissue*** *shows chronic inflammatory cells infiltrate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

HYDRONEPHROSIS

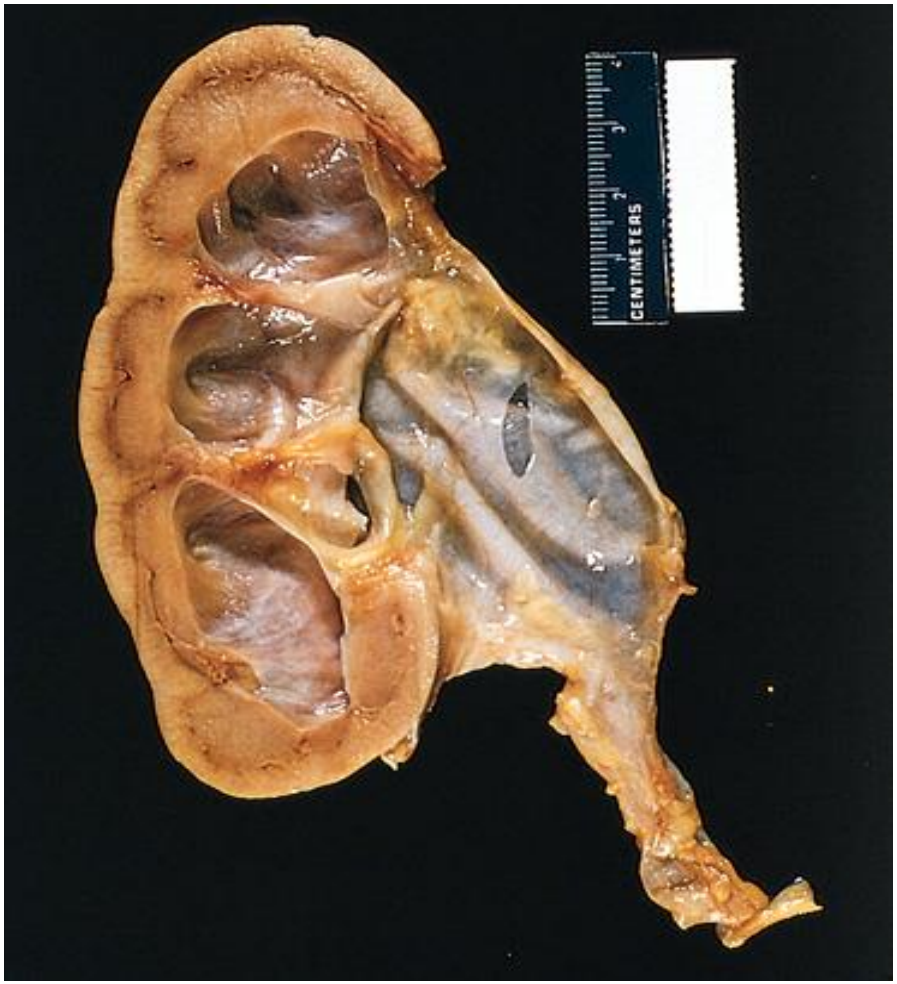
- is the swelling of a kidney due to a back-up of urine. Hydronephrosis occurs with other diseases such as kidney stones, urinary tract infections, or acute or chronic unilateral obstructive uropathy.
- dilatation of renal pelvis and calyceal system with a thinning of the renal cortex.

Hydronephrosis



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

Hydronephrosis

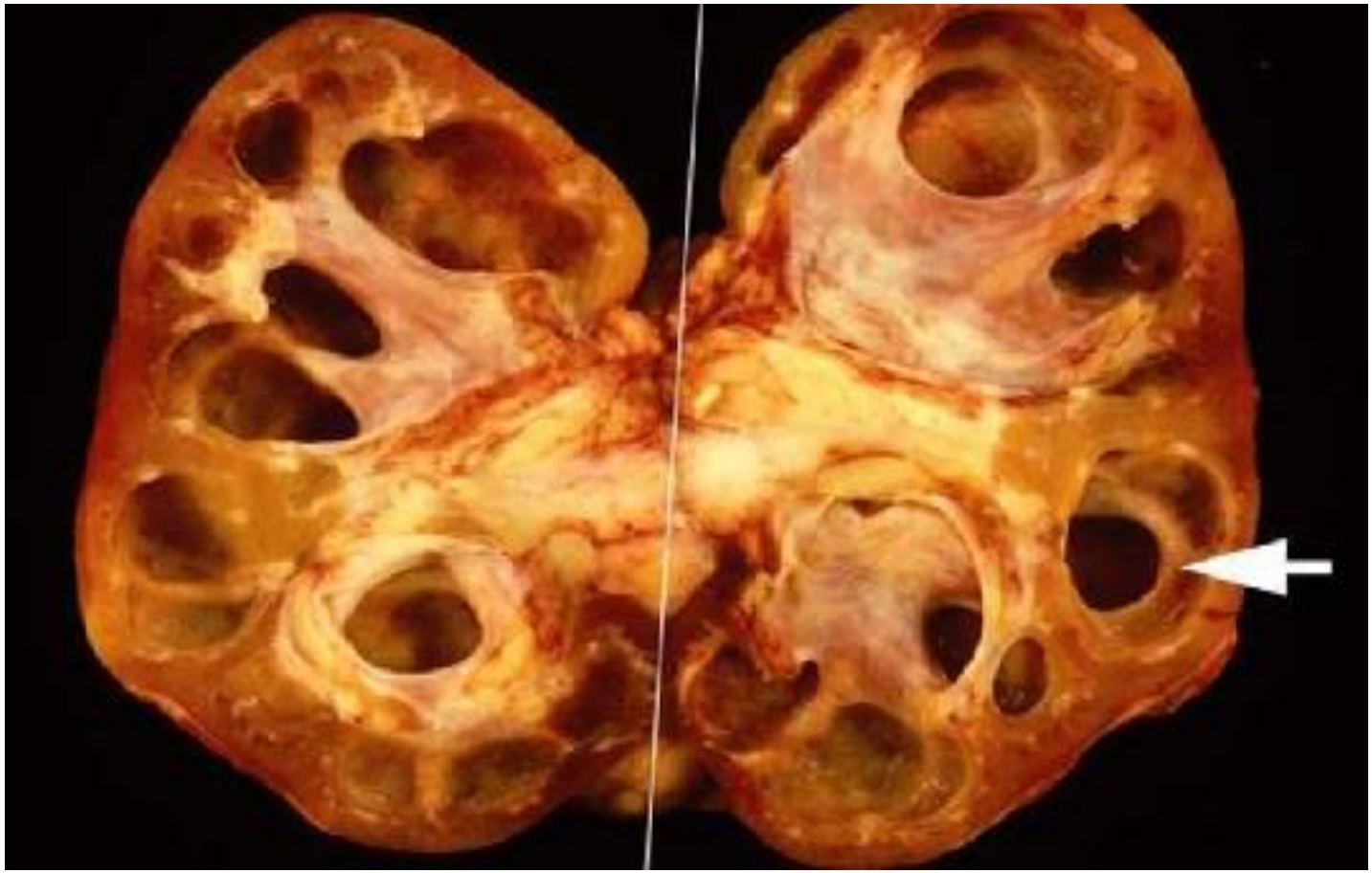


The most common causes are:

- *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 .*

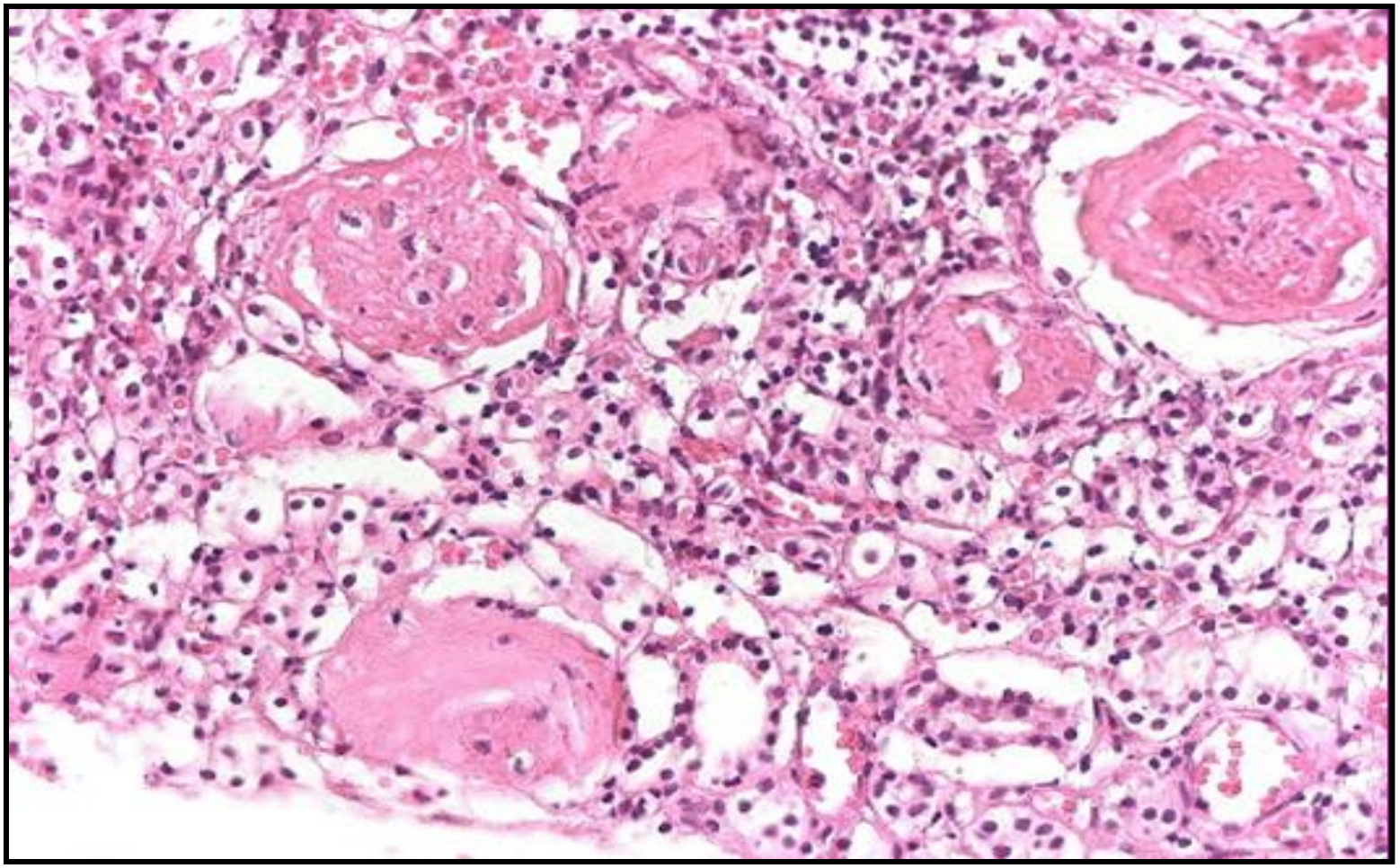
The picture shows markedly dilated renal pelvis and calyces with atrophic and thin renal cortex /parenchyma

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

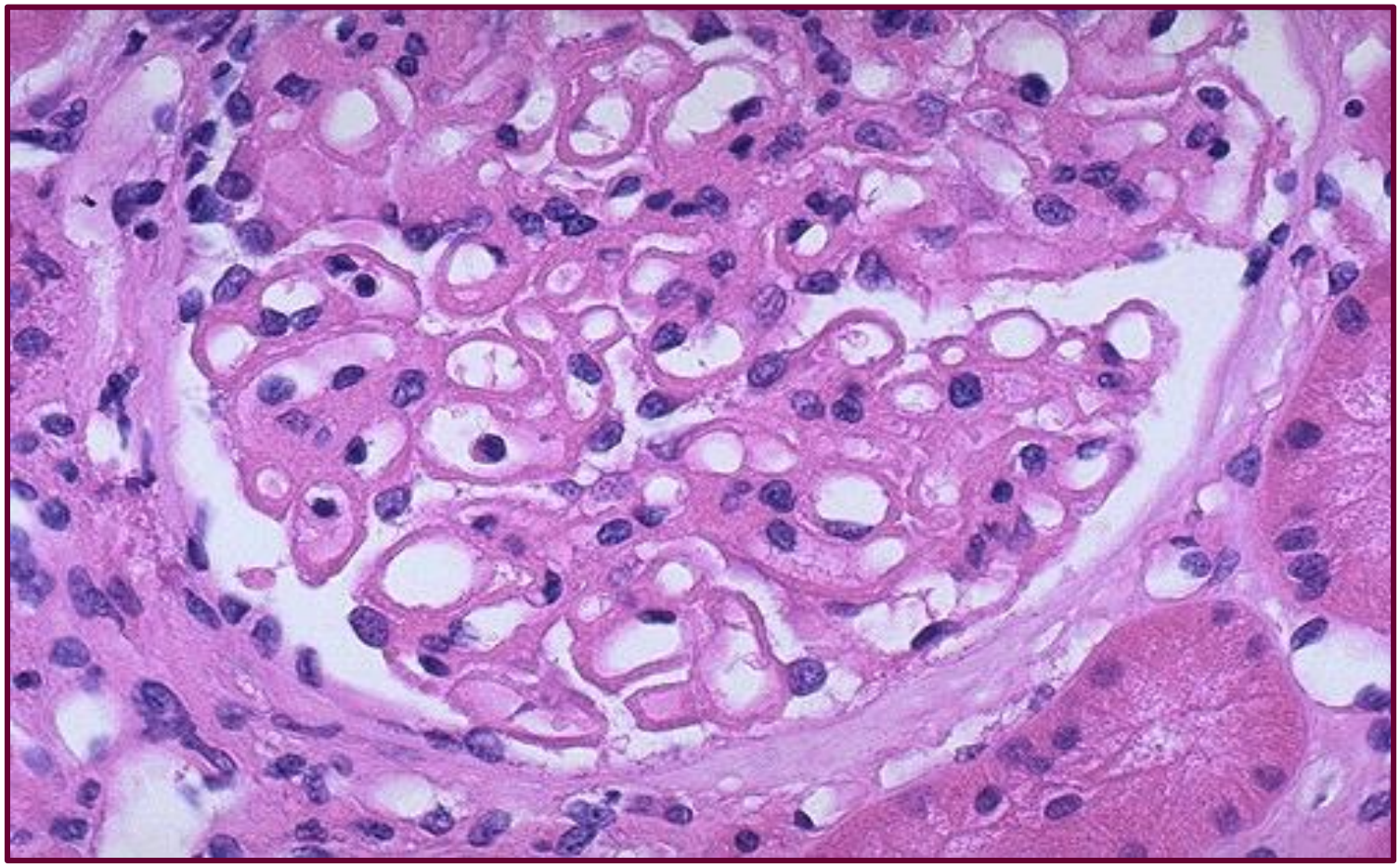
PRACTICAL SESSION : 3

NEPHROTIC SYNDROME

- is a collection of symptoms due to kidney damage. This includes protein in the urine >3.5g in 24hrs, low blood albumin levels, high blood lipids, and significant swelling.
- Common primary causes : minimal-change nephropathy, membranous nephropathy, and focal glomerulosclerosis.

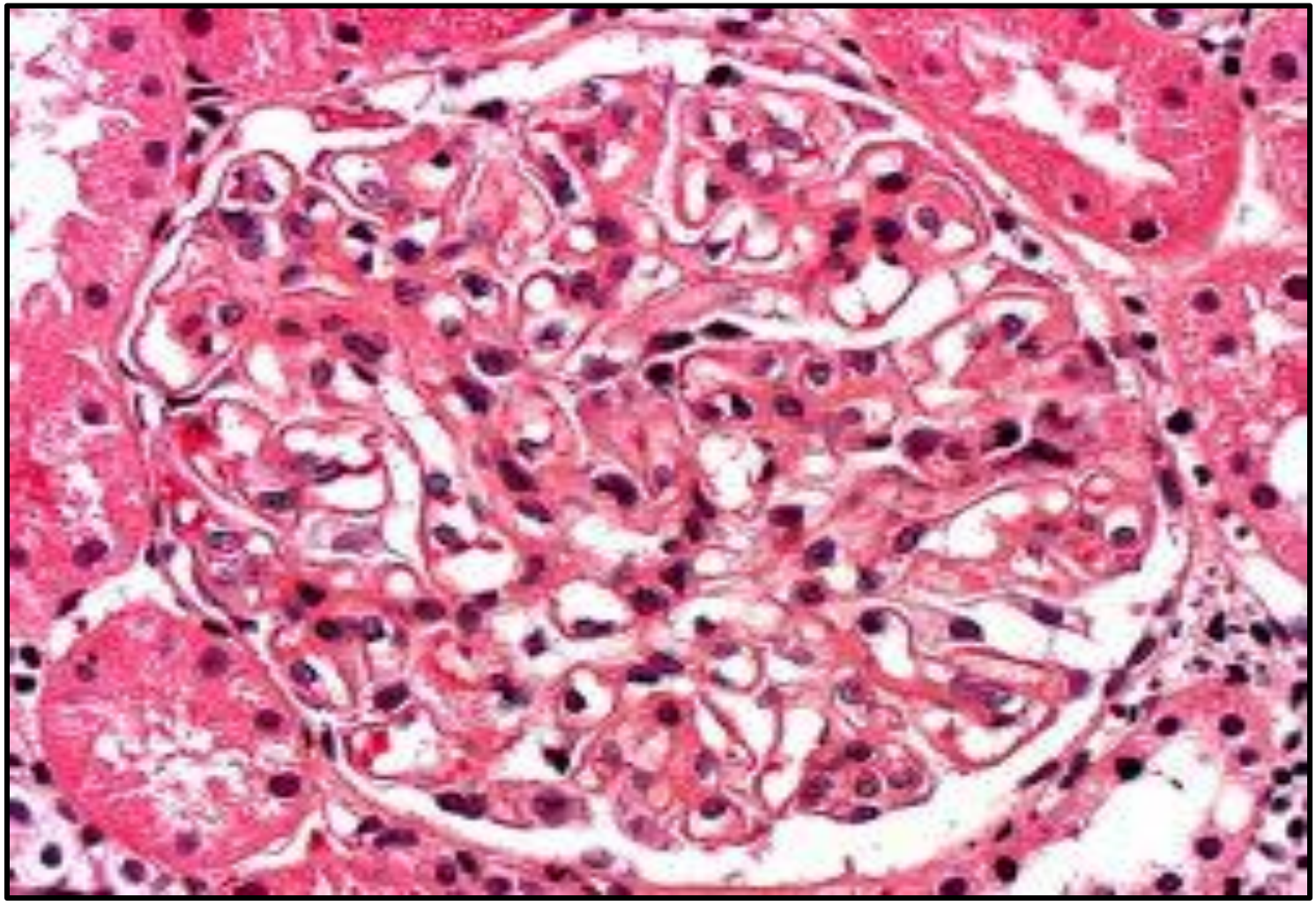
- Secondary causes :systemic diseases such as diabetes mellitus, lupus erythematosus, myloidosis, infection and drugs.

Membranous Glomerulonephritis



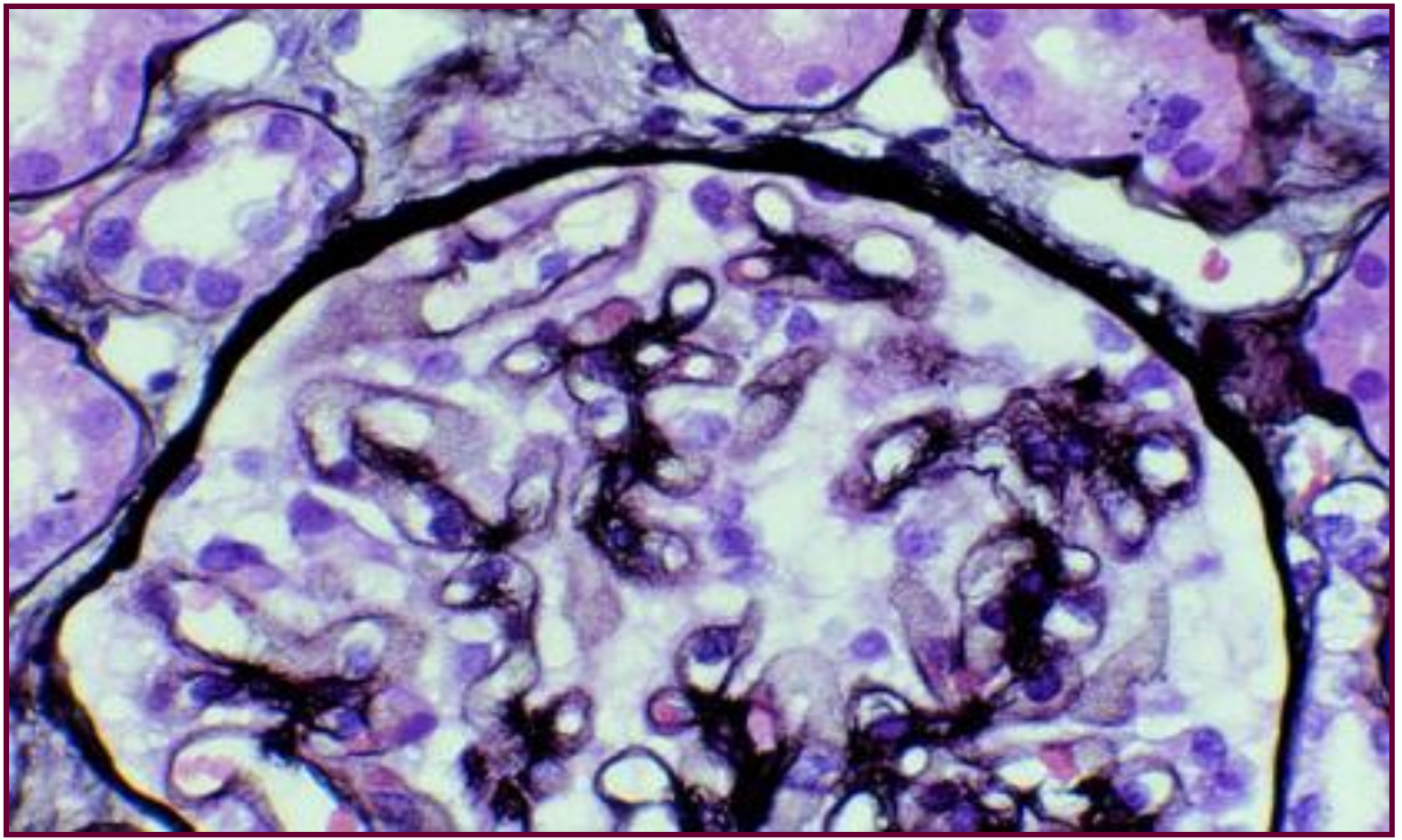
Membranous glomerulonephritis (The common cause of Nephrotic syndrome in adults): the capillary loops are thickened and prominent, but the cellularity is not increased.

Membranous Glomerulonephritis



Close-up of glomerulus illustrating rigid, uniformly-thickened capillary walls (H&E stain, 400x original magnification).

Membranous Glomerulonephritis

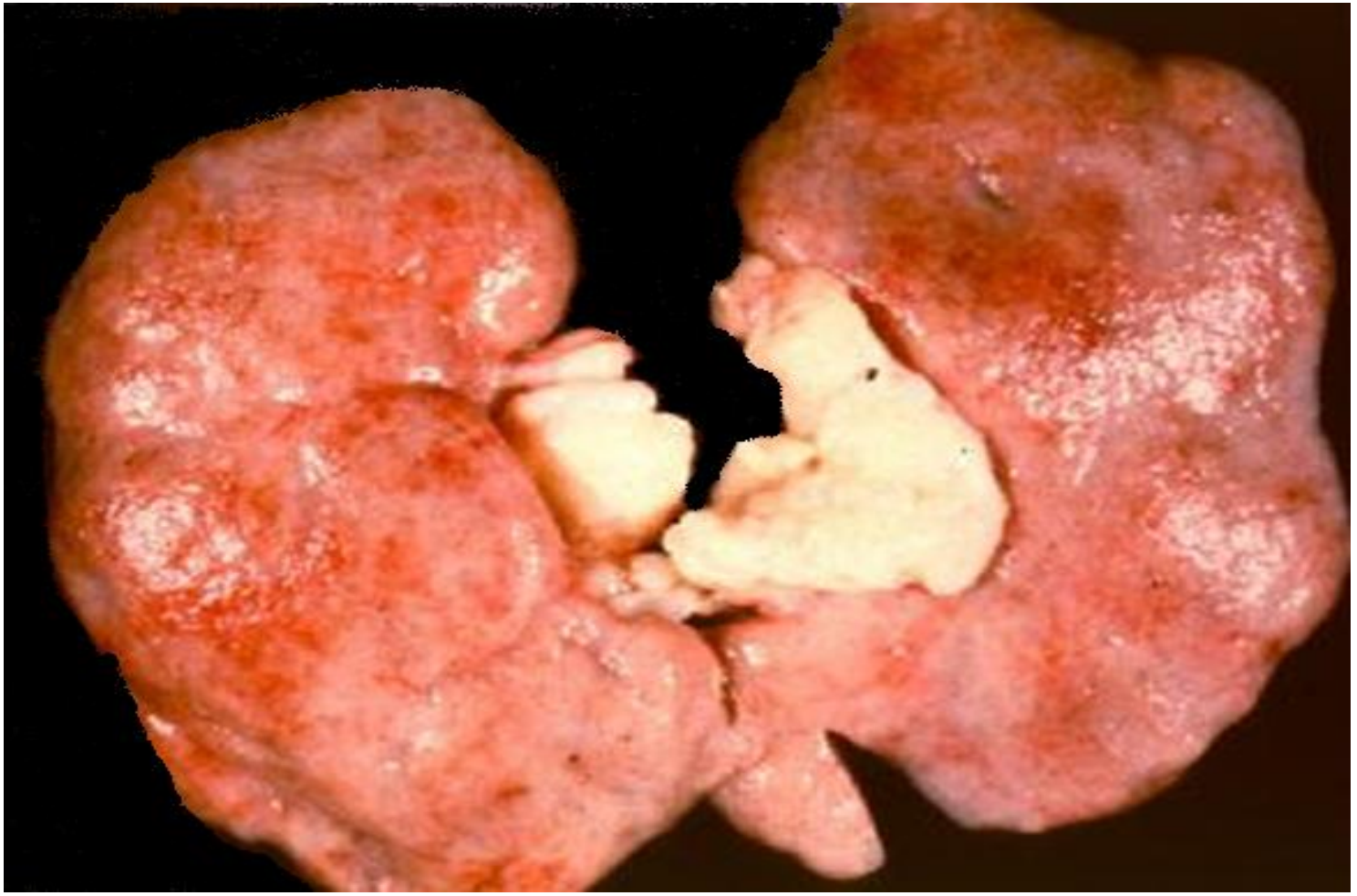


Early stage II membranous glomerulonephritis: The thickened capillary wall shows numerous "holes" in tangential sections, indicating deposits. (Deposits do not take up the silver stain.) Well-developed spikes around the deposits are not present here.

NEPHRITIC SYNDROME

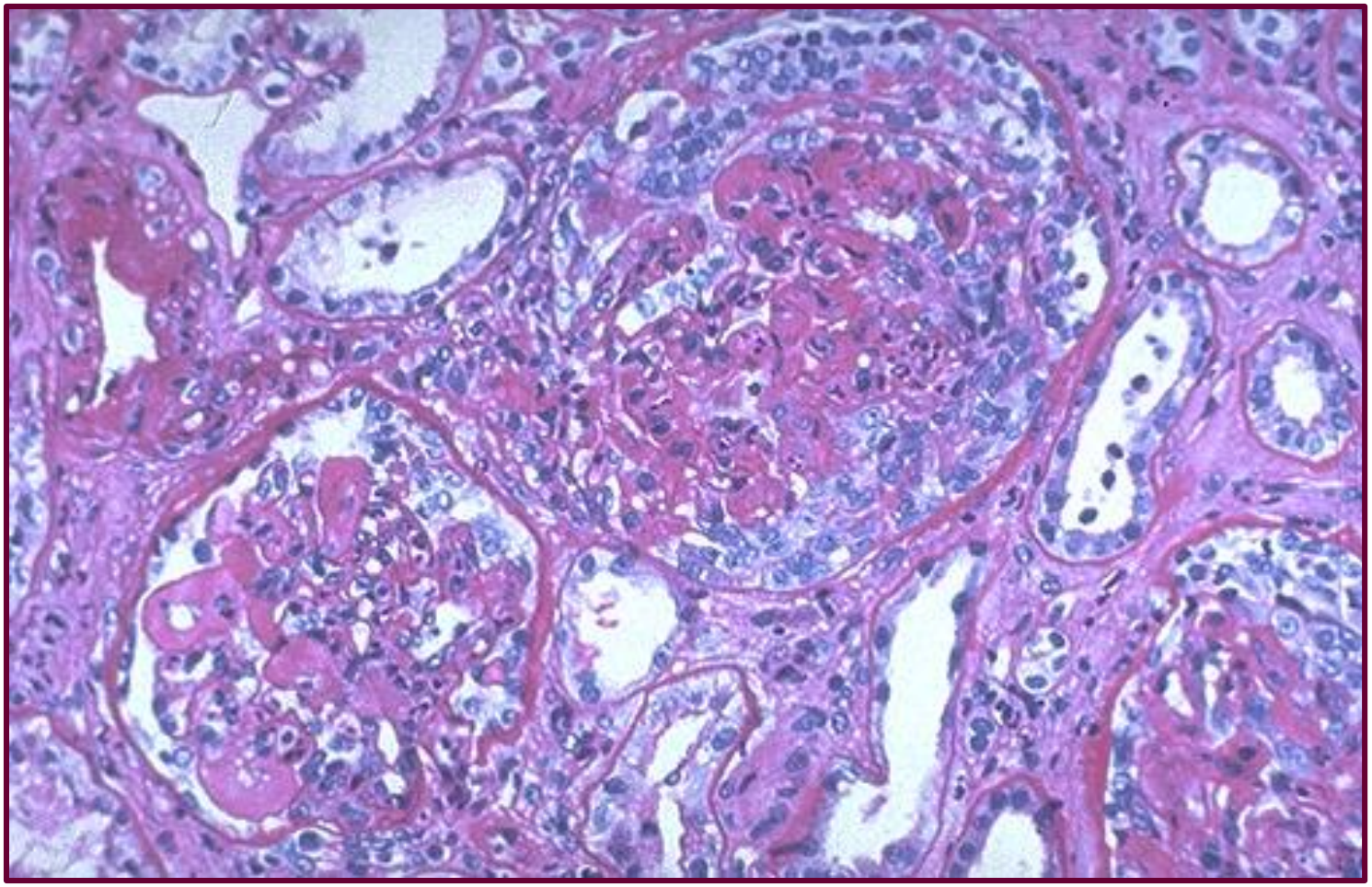
- is a manifestation of glomerular inflammation (glomerulonephritis)
- Haematuria, Proteinuria
- Low urine volume <300ml/day

Rapid Progressive Glomerulonephritis (RPGN)



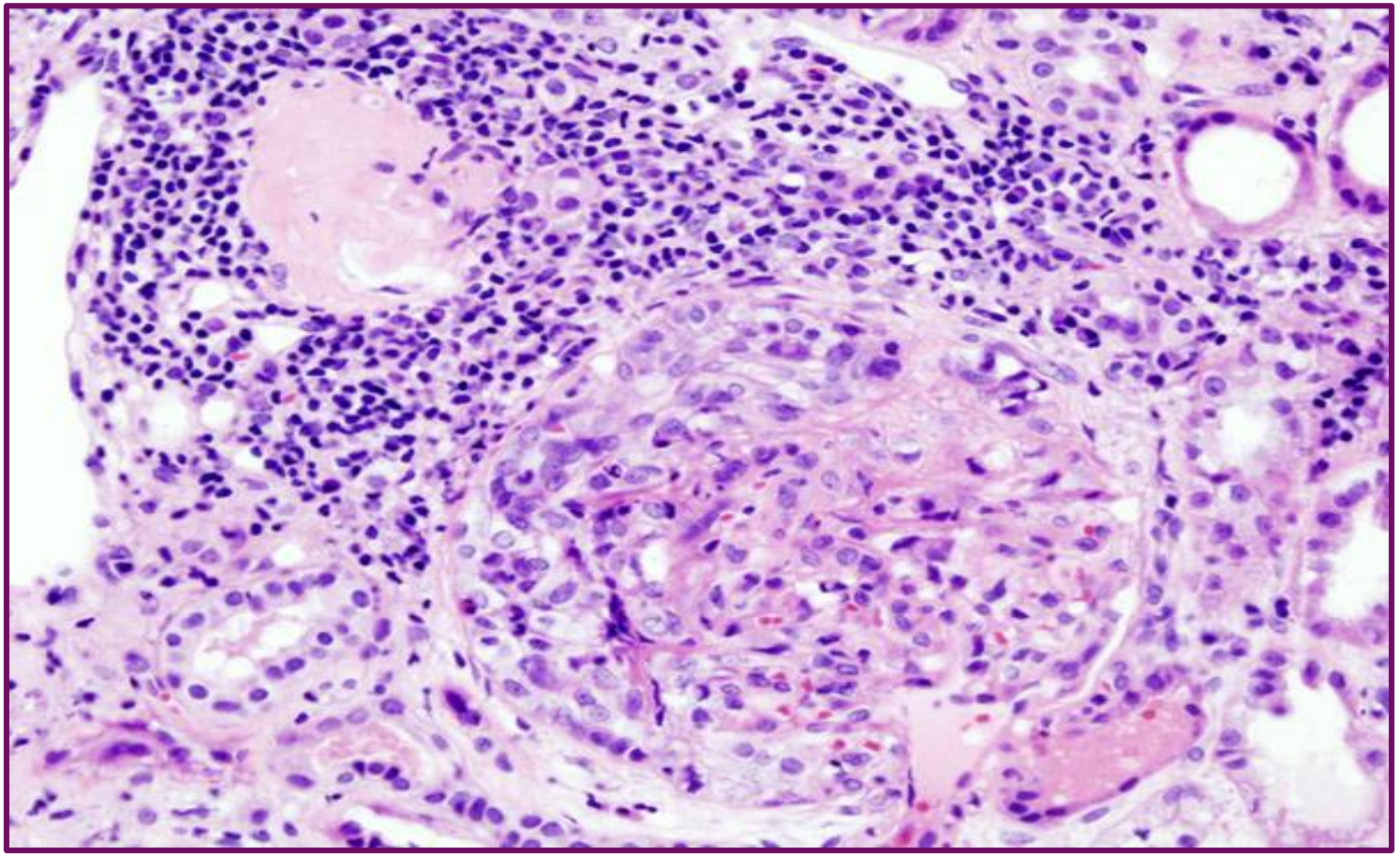
Gross appearance of RPGN - note the flea beaten appearance

Rapid Progressive Glomerulonephritis (RPGN)



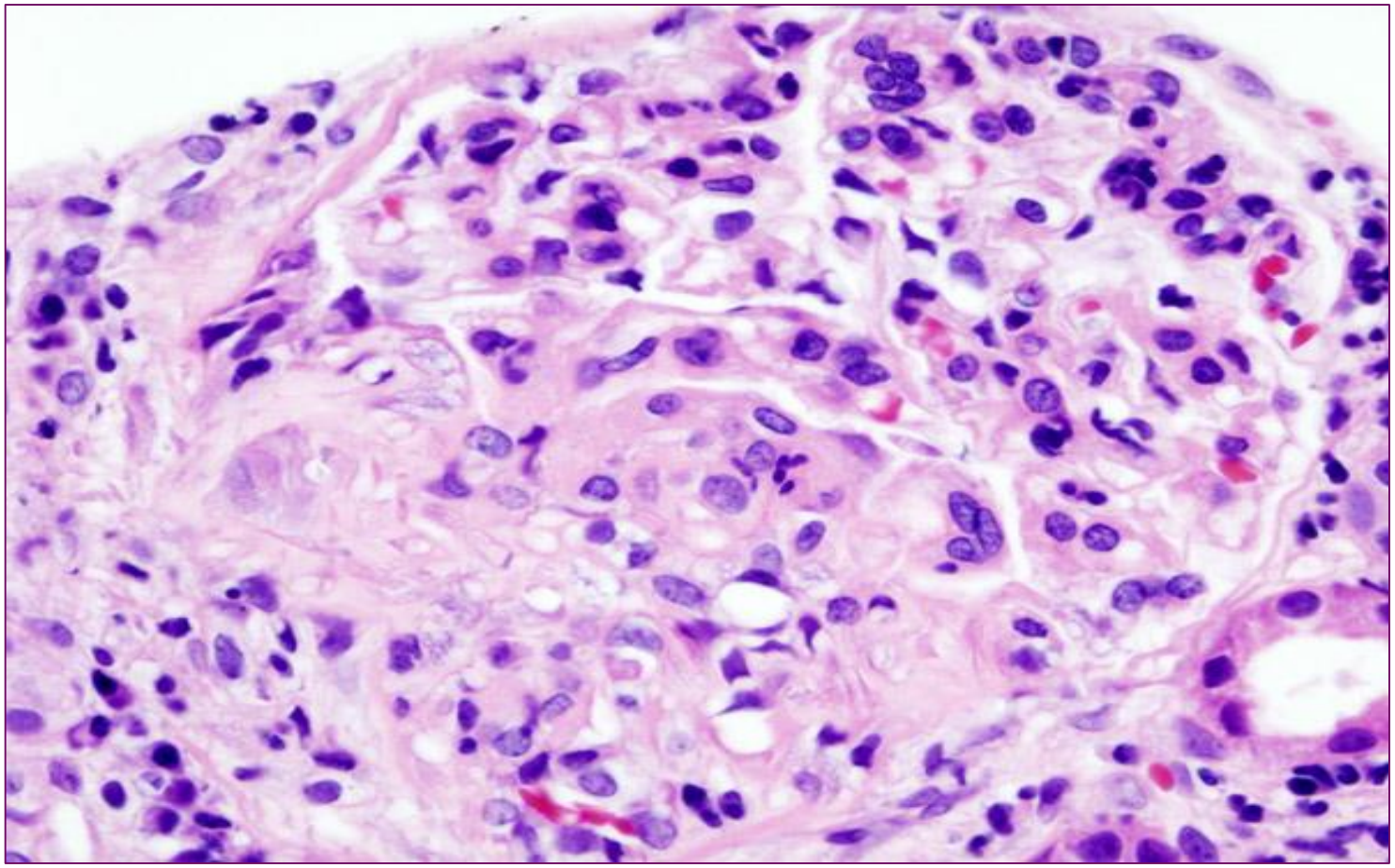
Seen here within the glomeruli are crescents composed of proliferating epithelial cells. Crescentic glomerulonephritis is known as rapidly progressive glomerulonephritis (RPGN) because this disease is very progressive

Rapid Progressive Glomerulonephritis (RPGN)



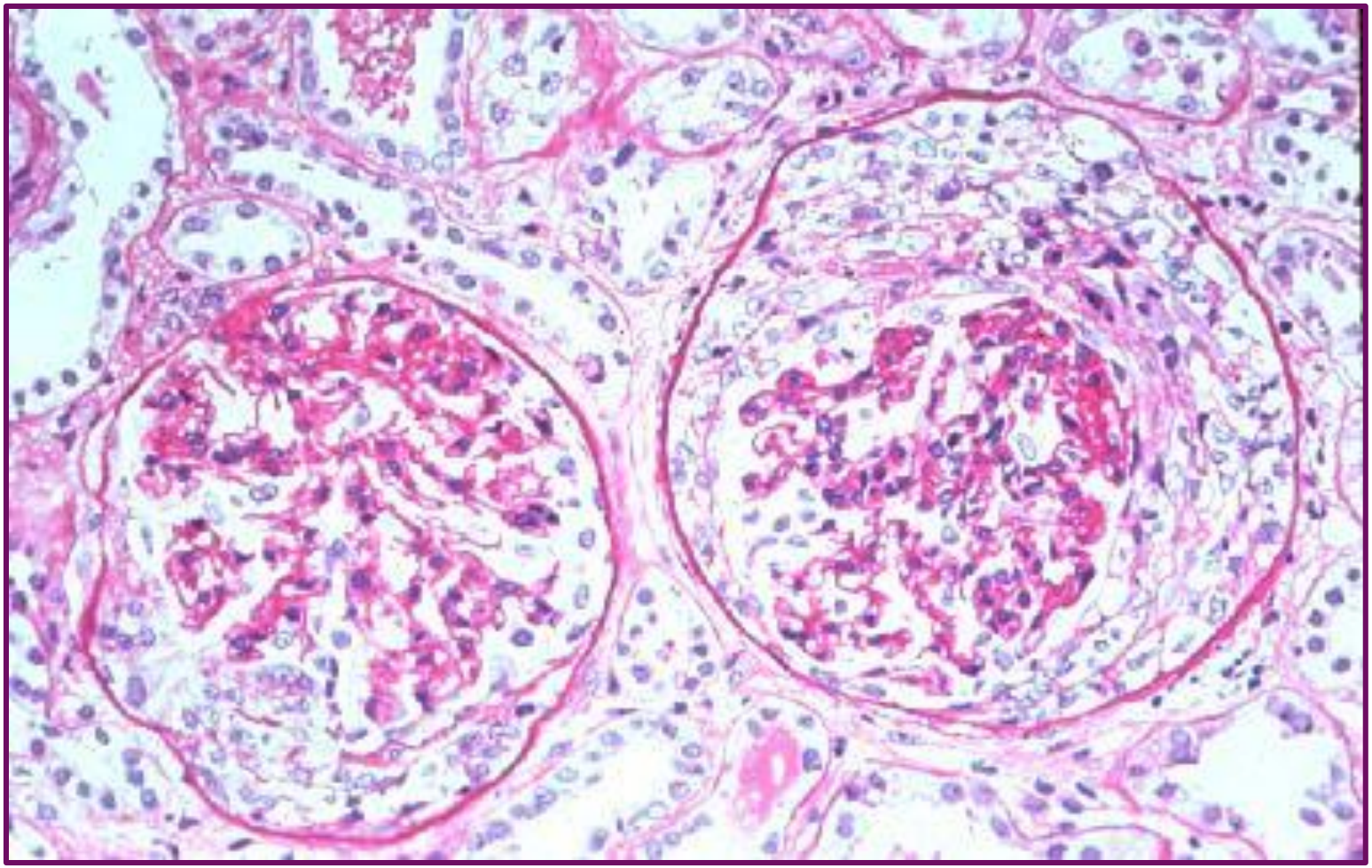
Crescentic glomerulonephritis in a patient with Rapid Progressive Glomerulonephritis (RPGN) . All types of RPGN are characterized by glomerular injury and formation of crescents with monocytes and macrophages proliferation compressing the glomerulus

Rapid Progressive Glomerulonephritis (RPGN)



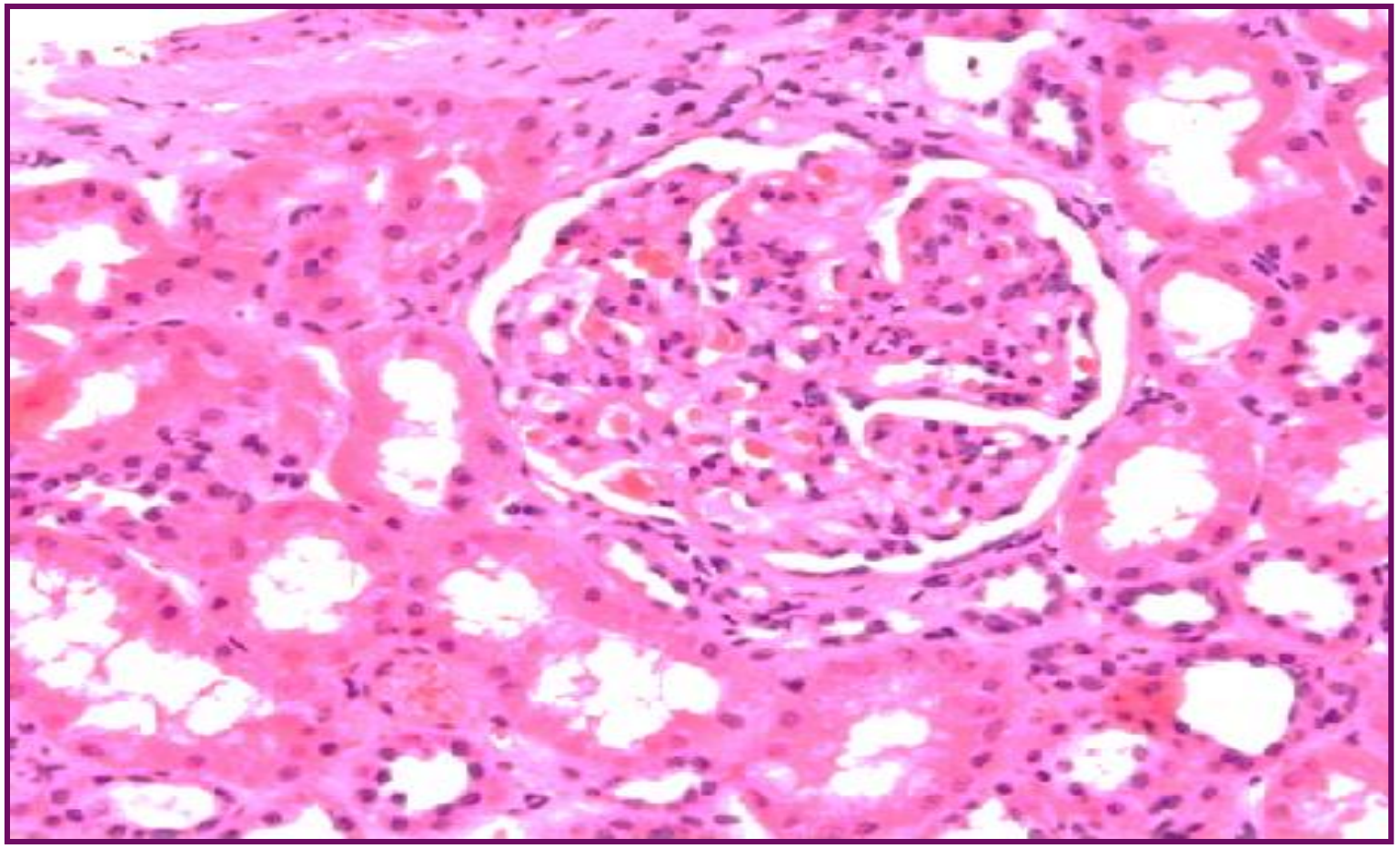
In severe injury, fibrin contribute most strongly to crescent formation. Epithelial cells of Bowman capsule are proliferated . Infiltrating WBCs such as monocytes and macrophages also proliferate compressing the glomerulus, forming a crescent-shaped scar

Rapid Progressive Glomerulonephritis (RPGN)



Epithelial cells of Bowman capsule are proliferated . Infiltrating WBCs such as monocytes and macrophages also proliferate compressing the glomerulus, forming a crescent-shaped scar

Nephropathy : Nephritic / Nephrotic Syndrome



The glomeruli showed mesangial proliferation.
The glomerular basement membrane was normal.
The interstitium and blood vessels were unremarkable

RENAL TUMORS

BENIGN RENAL TUMORS

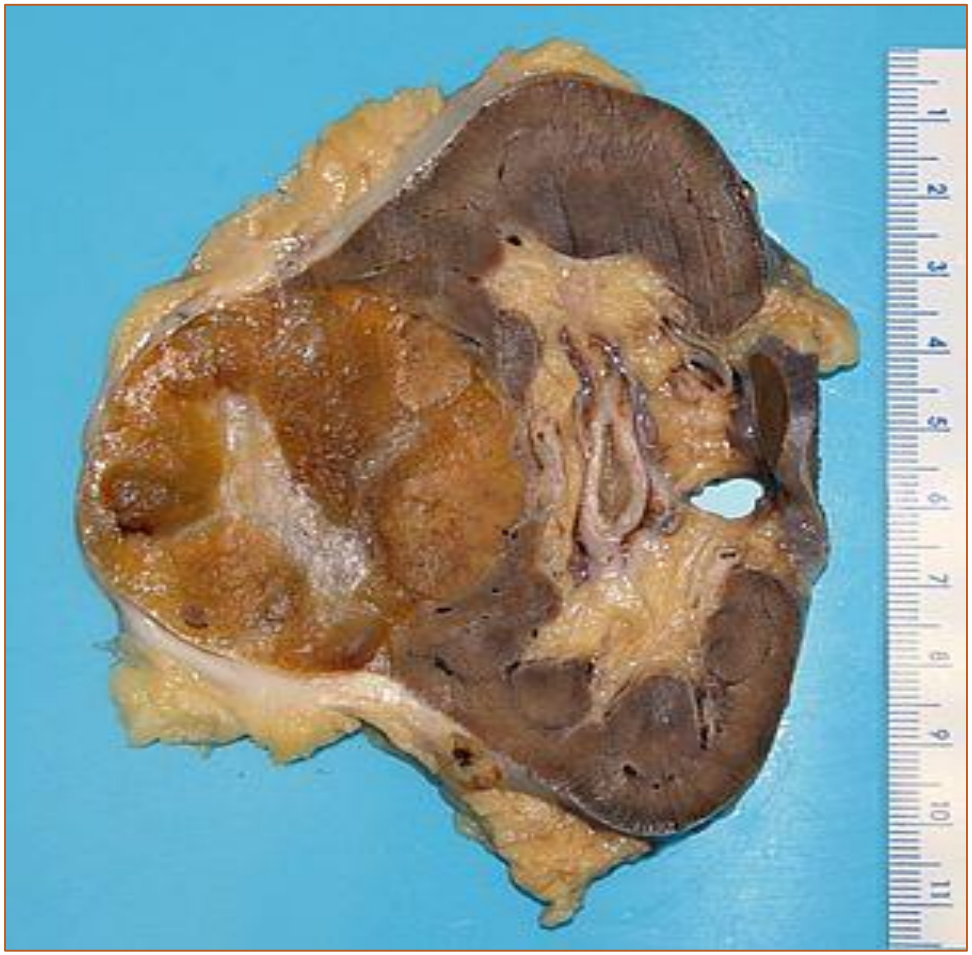
RARE Tumors

- *Papillary Adenoma (SIZE very important)*
- *Fibroma/ Hamartoma*
- *Angiomyolipoma*
- *Oncocytoma (very red, granular, mitochondria)*

Benign Renal Oncocytoma

- Benign tumor made of oncocytes; uniform round / polygonal cells with abundant, intensely eosinophilic and granular cytoplasm, characterized by an excessive amount of mitochondria with with uniform small, round and central nuclei with evenly dispersed chromatin.
- 4% - 7% of adult renal epithelial tumors
- May coexist with renal cell carcinoma
- Arises from intercalated cells of collecting duct

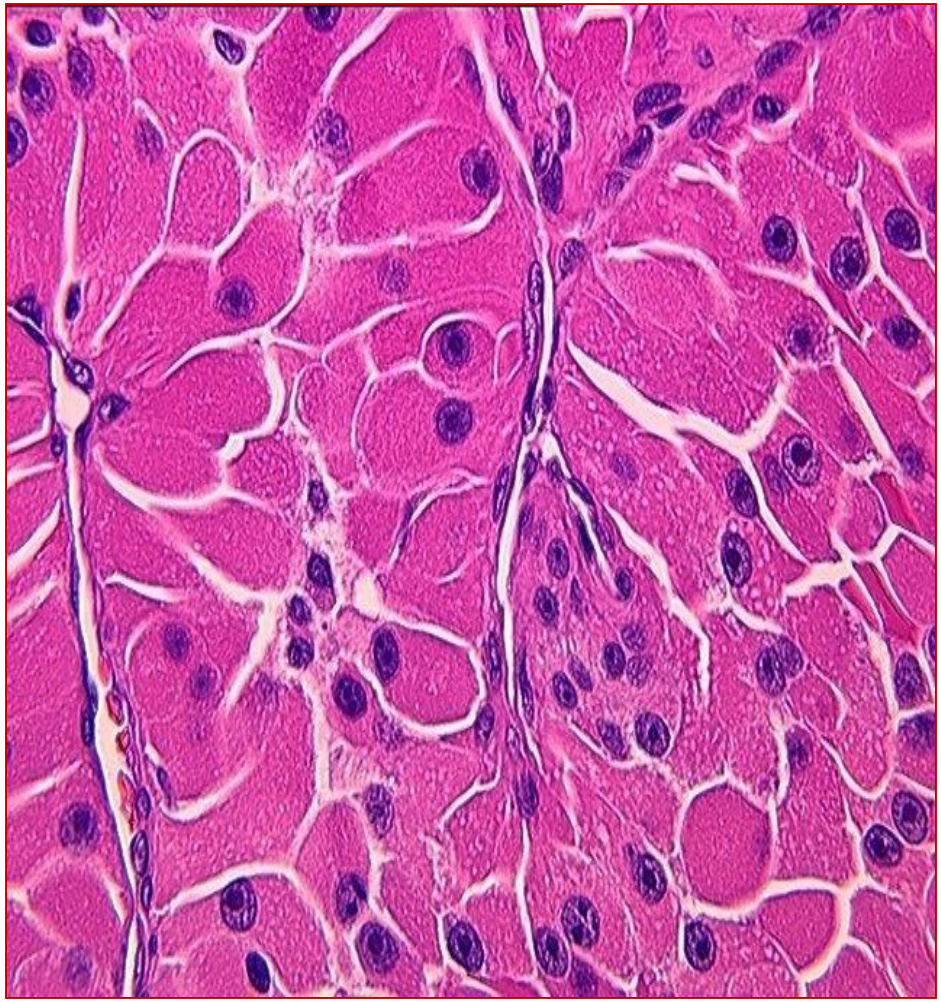
Oncocytoma - Gross



Gross appearance of a renal oncocytoma (left of image) and a slice of a normal kidney (right of image).

- ***rounded contour (***well circumscribed)***, the mahogany colour***
- ***and the central scar***

Benign renal oncocytoma



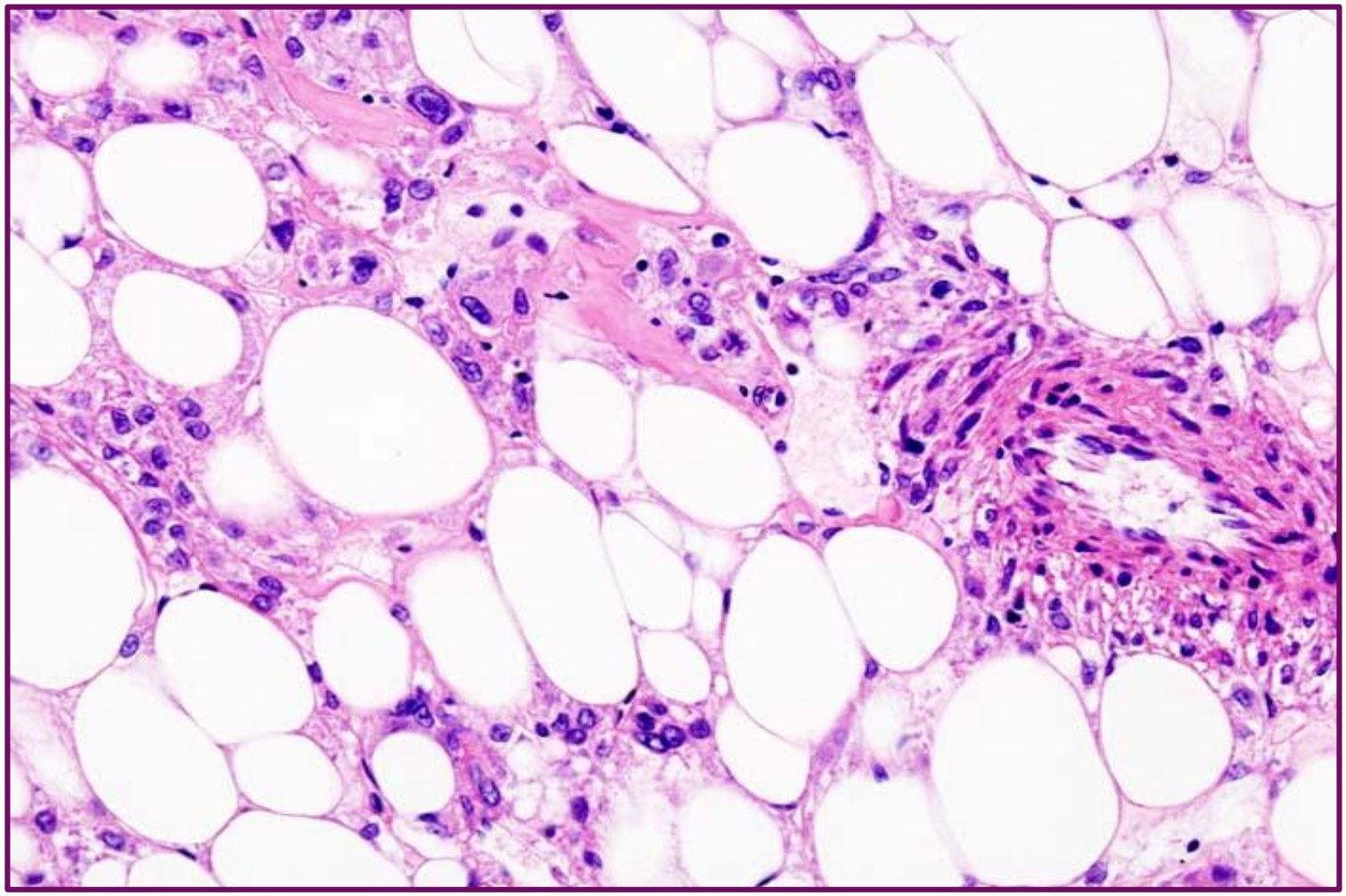
- Nesting, alveolar or tubular patterns of uniform round / polygonal cells with abundant, intensely eosinophilic and granular cytoplasm, uniform small, round and central nuclei

Oncocytes are very RED and granular cytoplasm with vesicular nuclei and prominent nucleoli.

Angiomyolipma

- benign neoplasm composed of admixture of blood vessels, smooth muscle and adipose tissue
- Apart from kidney, can occur in extrarenal sites such as liver, lungs, retroperitoneal soft tissue
- May be sporadic but is associated with tuberous sclerosis
- Usually diagnosed in adults
- < 1% of all renal tumors
- grossly: well Circumscribed, non encapsulated with pushing border, Tumors are usually unilateral and unifocal

Angiomyolipoma



Benign tumor composed of vessels (thick walled hyalinized vessels)***, smooth muscle component (***originate from vessel walls) ***and fat (***mature adipose tissue)

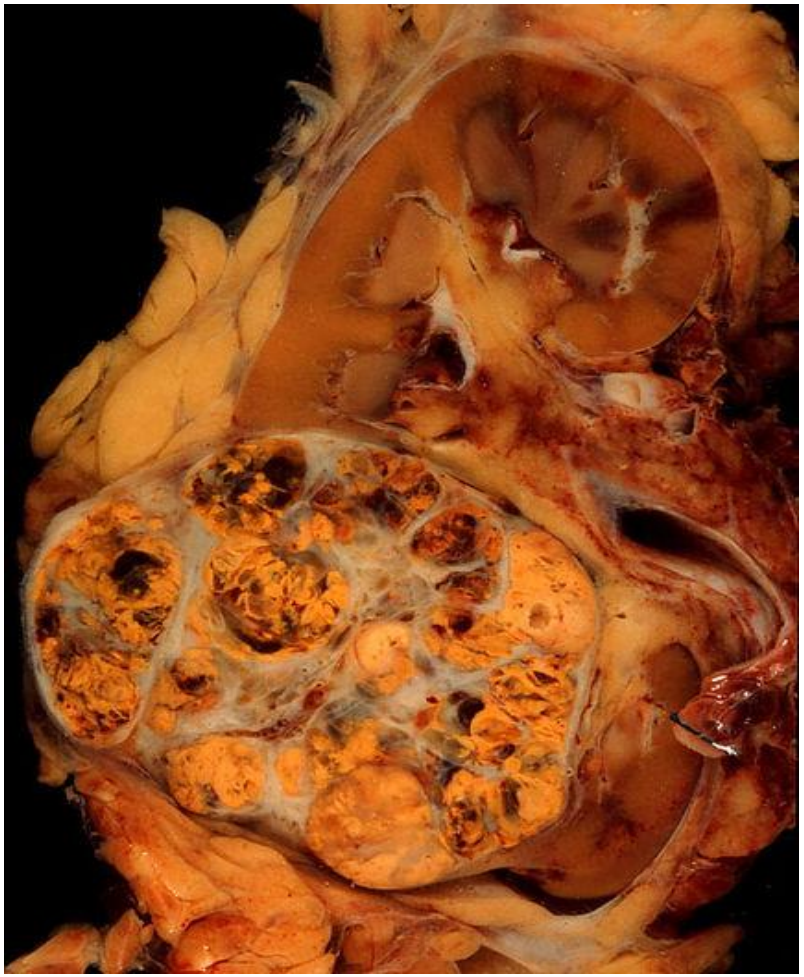
MALIGNANT RENAL TUMORS

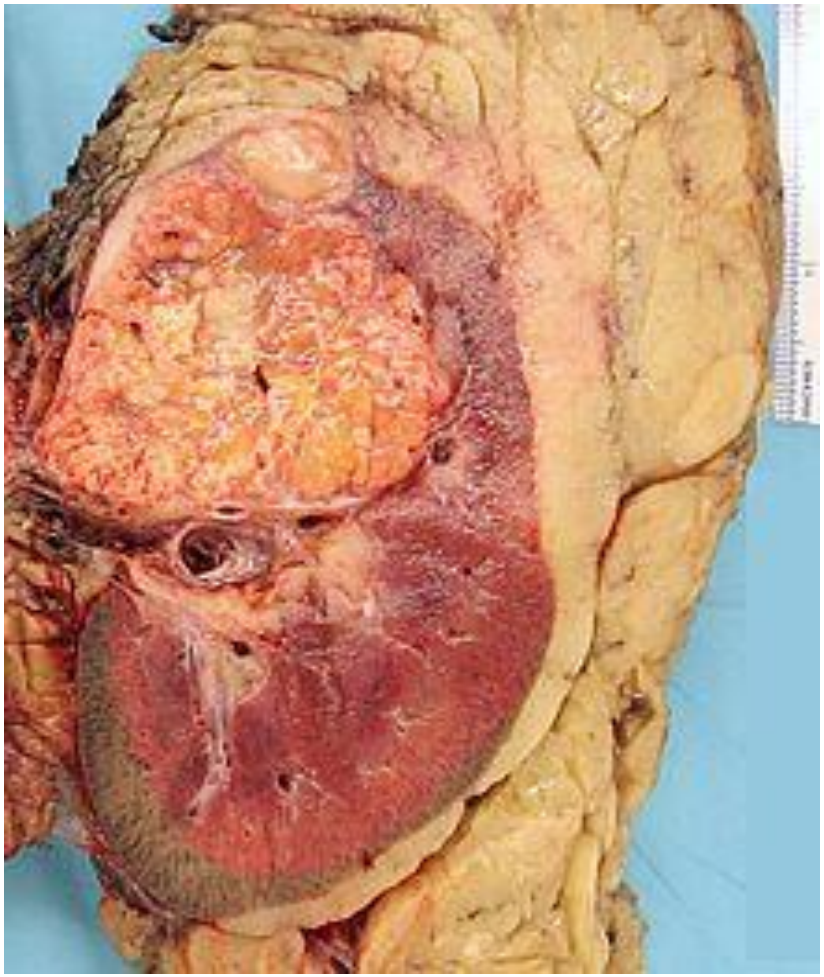
- *Renal Cell Carcinoma :*
  - *Clear Cell Carcinoma*
  - *Adenocarcinoma*
  - *Hypernephroma*
- *Urothelial (Transitional)*

Renal Clear Cell Carcinoma

- 65% - 70% of adult renal cancers (WHO 2016)
- Approximately 2:1 male: female ratio
- Usually > 50 years old
- Risk factors: obesity, smoking, hypertension, acquired cystic kidney disease due to end stage renal disease, occupational exposure to trichloroethylene, treated neuroblastoma
- Genetic susceptibilities estimated to account for 2 - 4% (VHL)

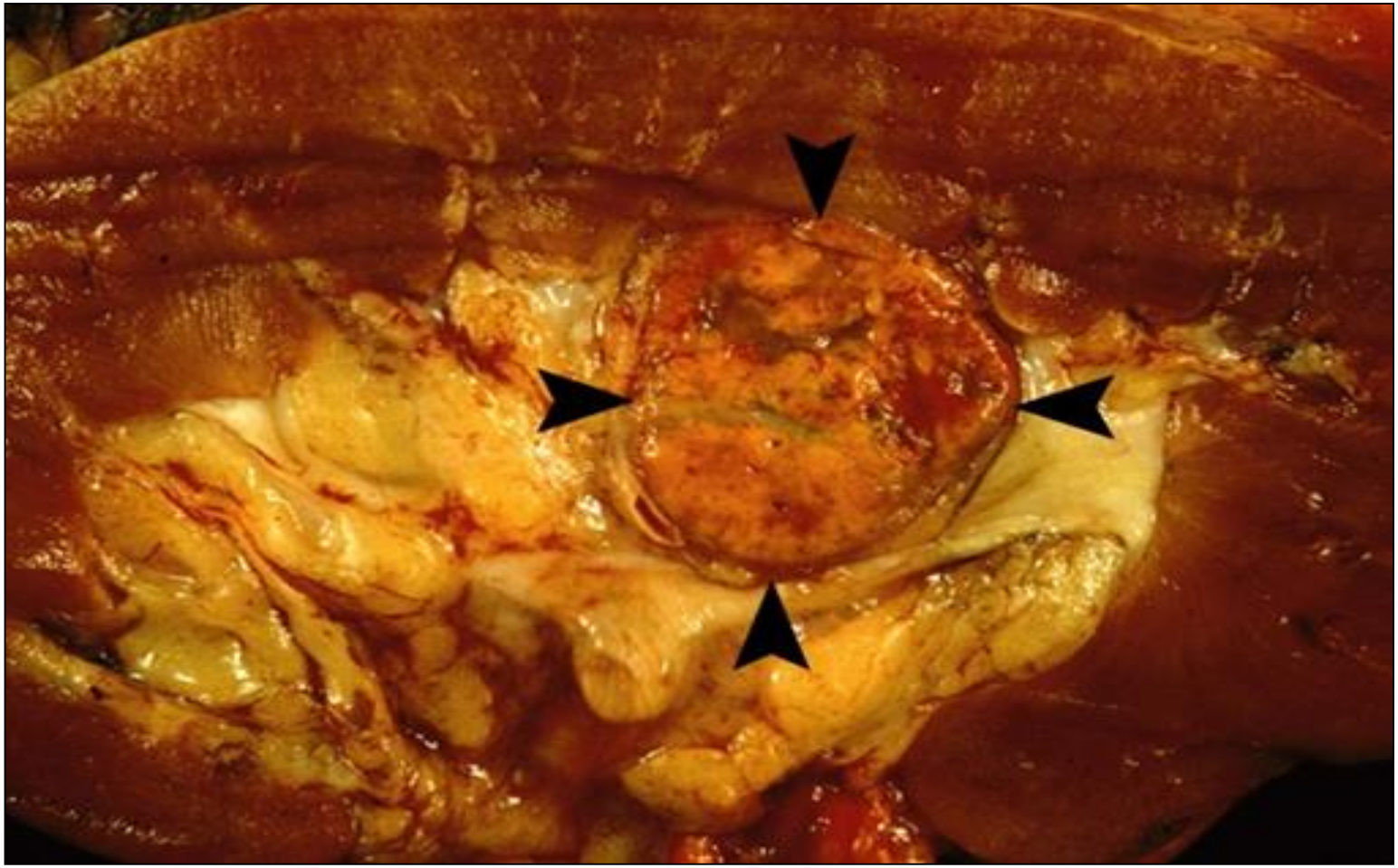
Renal Clear Cell Carcinoma – Gross pathology





A well circumscribed renal cortical mass which is partly yellow due to presence of fat and partly hemorrhagic with lobulated cut surface .

Renal Clear Cell Carcinoma – Gross

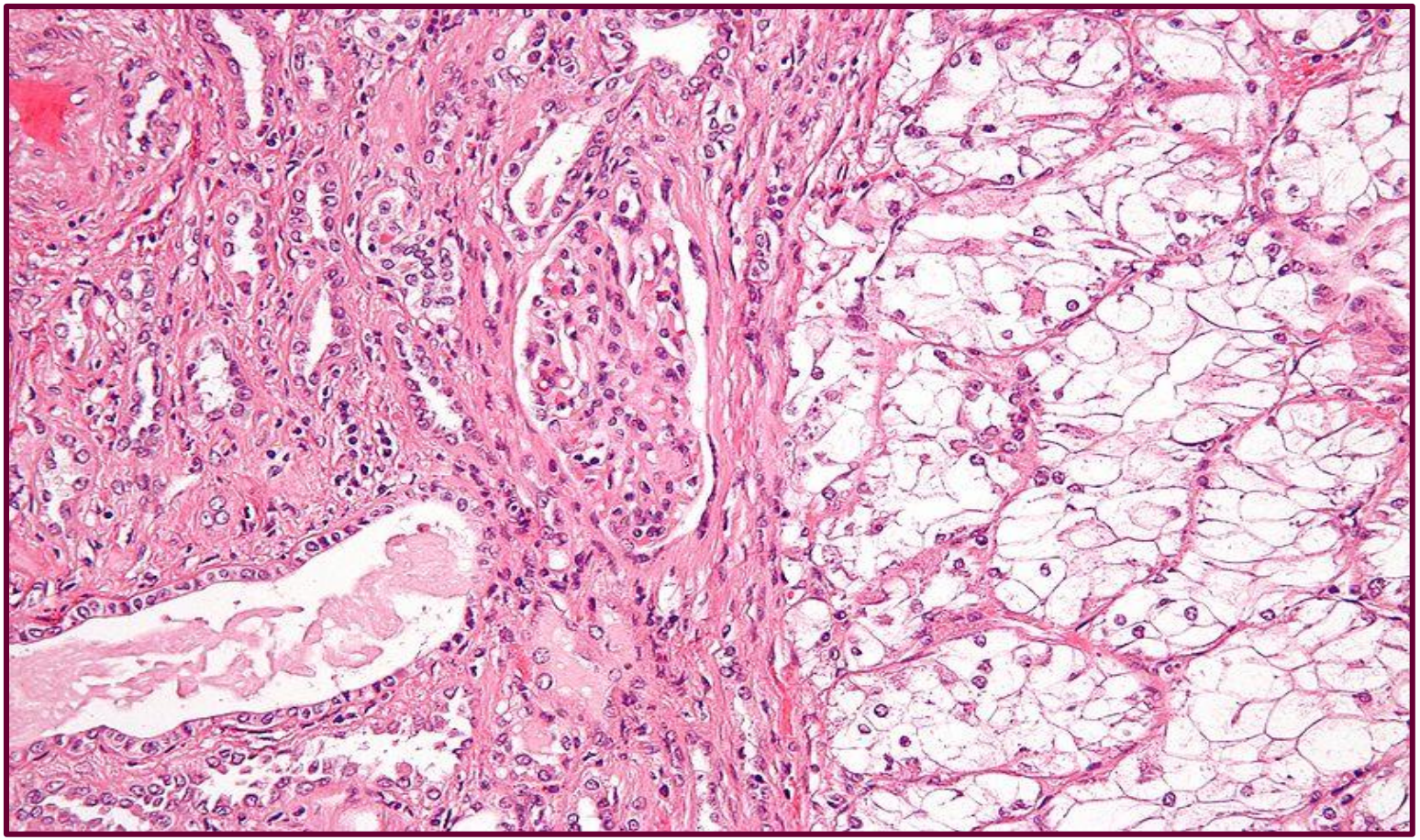


Renal clear cell carcinoma. ***The tumor is well demarcated from the surrounding non-neoplastic renal parenchyma by a pseudocapsule***

Renal Clear Cell Carcinoma - Histopathology

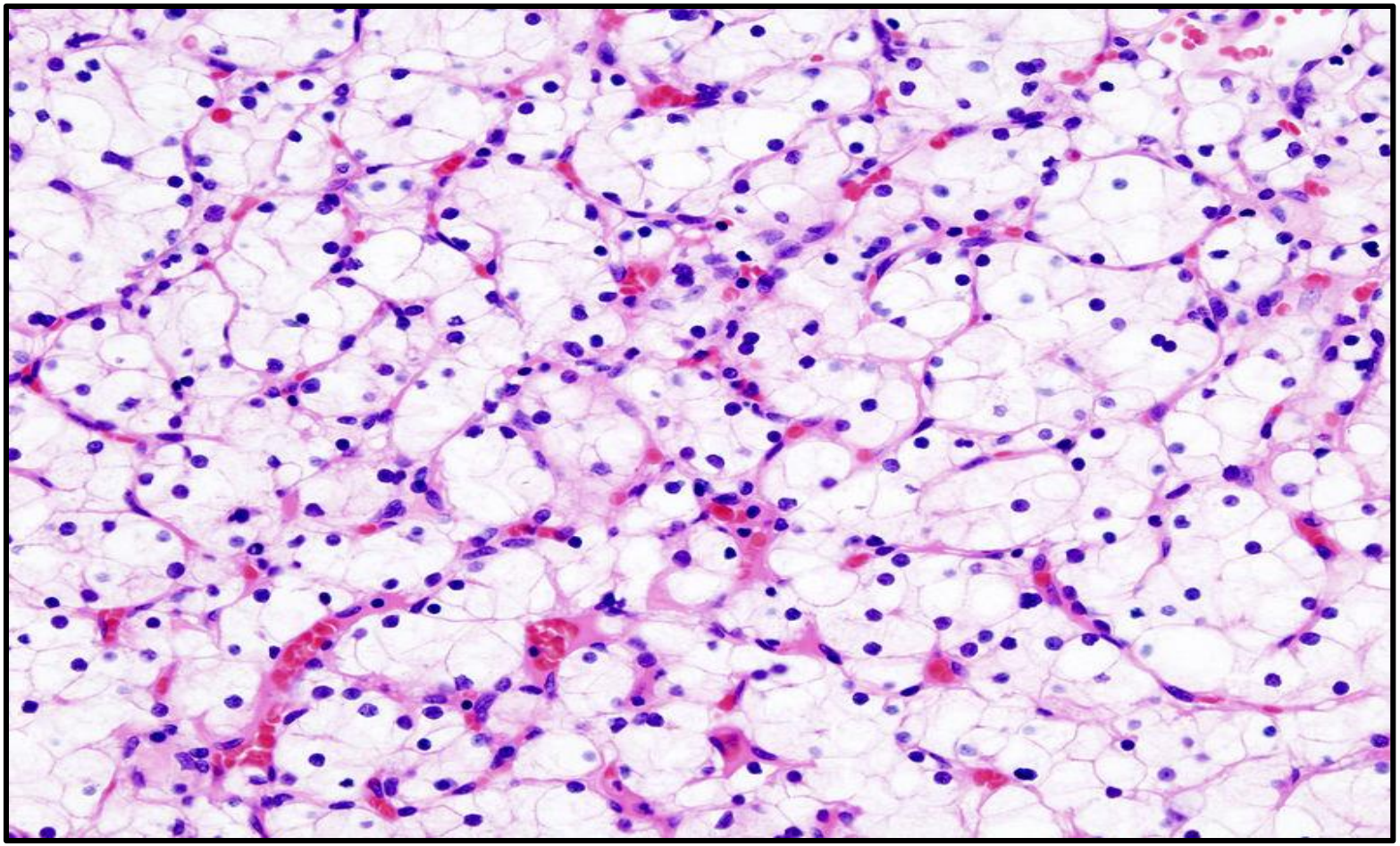
- *Tumor cells are large polygonal with clear cytoplasm (dissolved glycogen and lipid) and piknotic nuclei.*
- *Cells are arranged as alveolar groups or tubules with papillary formations separated by thin fibrovascular septae.*
- *Cells show pleomorphism and mitosis.*
- *Areas of haemorrhage and necrosis are present.*

Renal Clear Cell Carcinoma - Histopathology



The most common type of renal cell carcinoma (clear cell) - on right of the image : Cells with clear cytoplasm, typically arranged in nests and Nuclear atypia is common. Non-tumour kidney is on the left of the image

Renal Clear Cell Carcinoma - Histopathology



The most common type of renal cell carcinoma (clear cell) .
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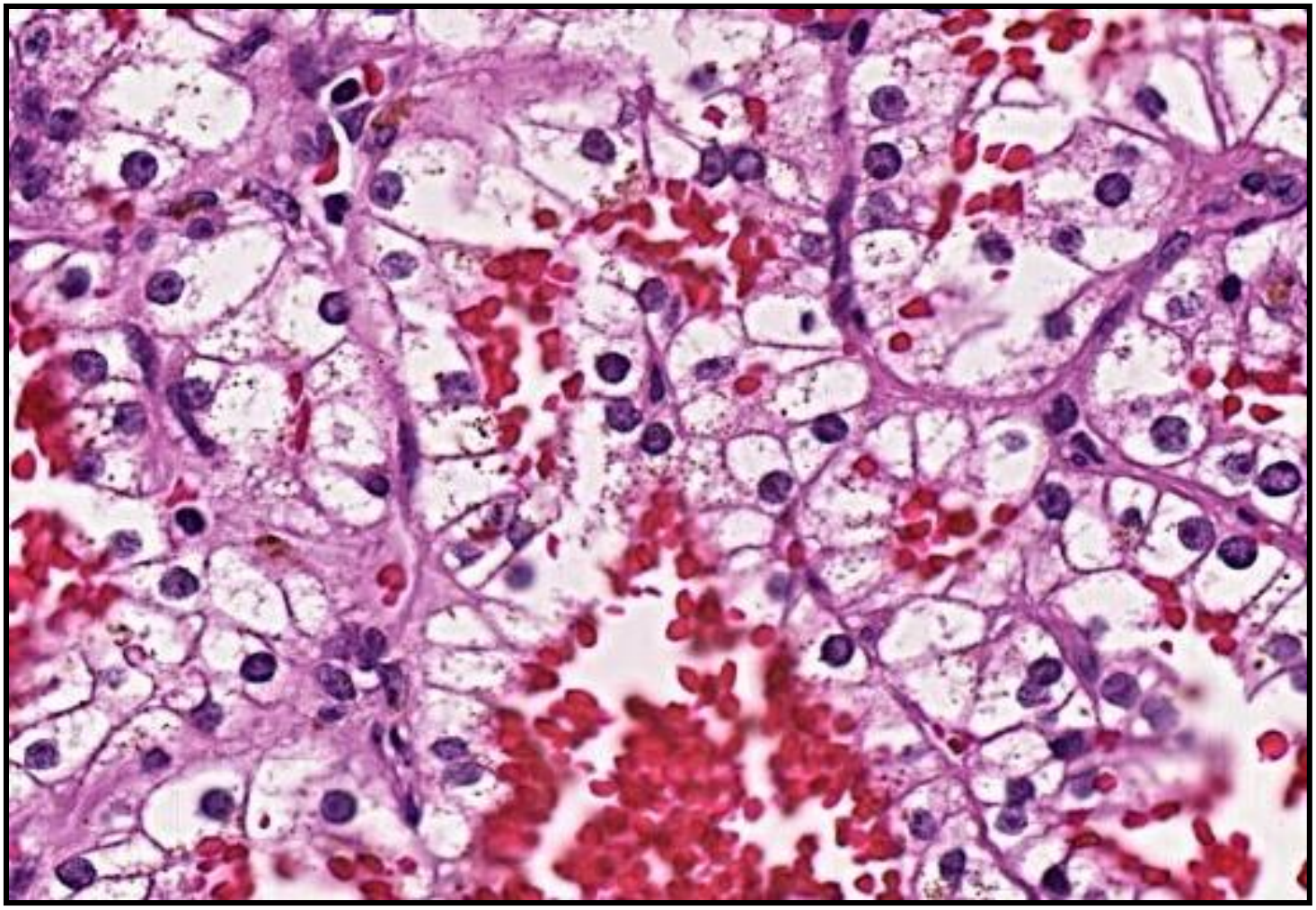
Renal Clear Cell Carcinoma - Histopathology

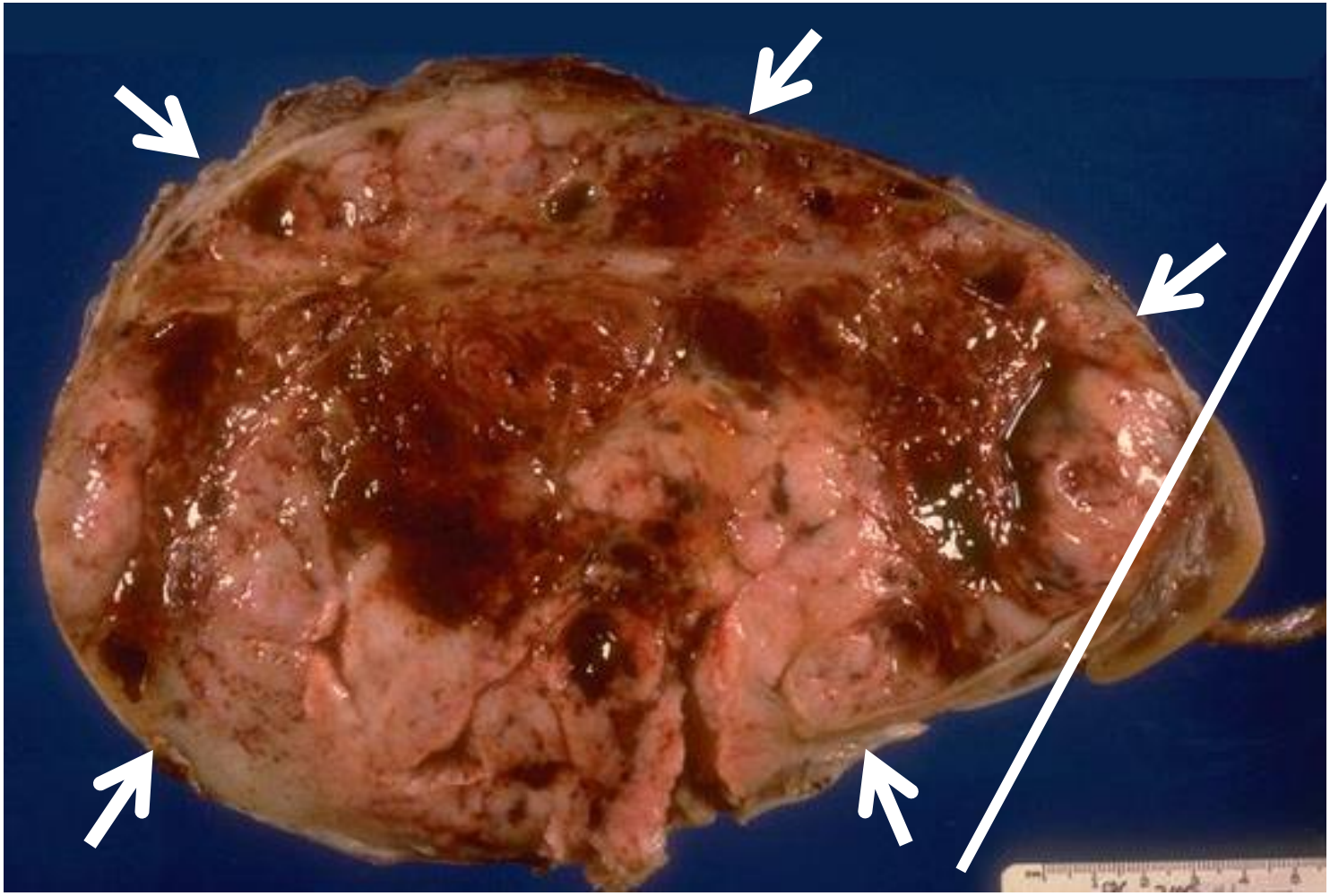


Section shows clear tumor cells with pleomorphic nuclei and areas of hemorrhage .

WILM'S TUMOR

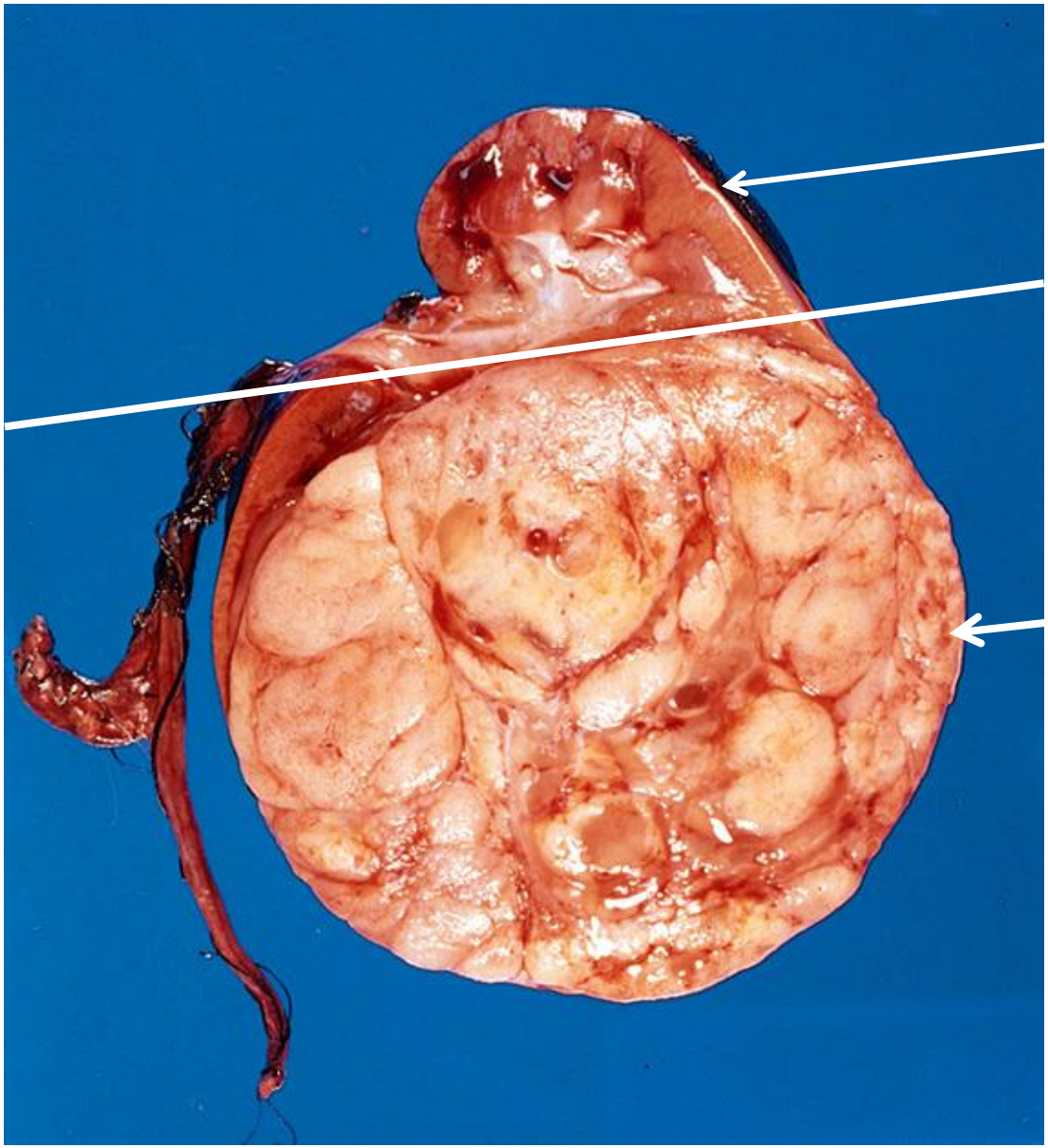
- is a rare kidney cancer that primarily affects children. Also known as nephroblastoma,
- it's the most common cancer of the kidneys in children.
- Wilms' tumor most often affects children ages 3 to 4
- is a malignant mixed tumor containing metanephric blastema, stromal and epithelial derivatives

Wilm's Tumor – Gross Pathology

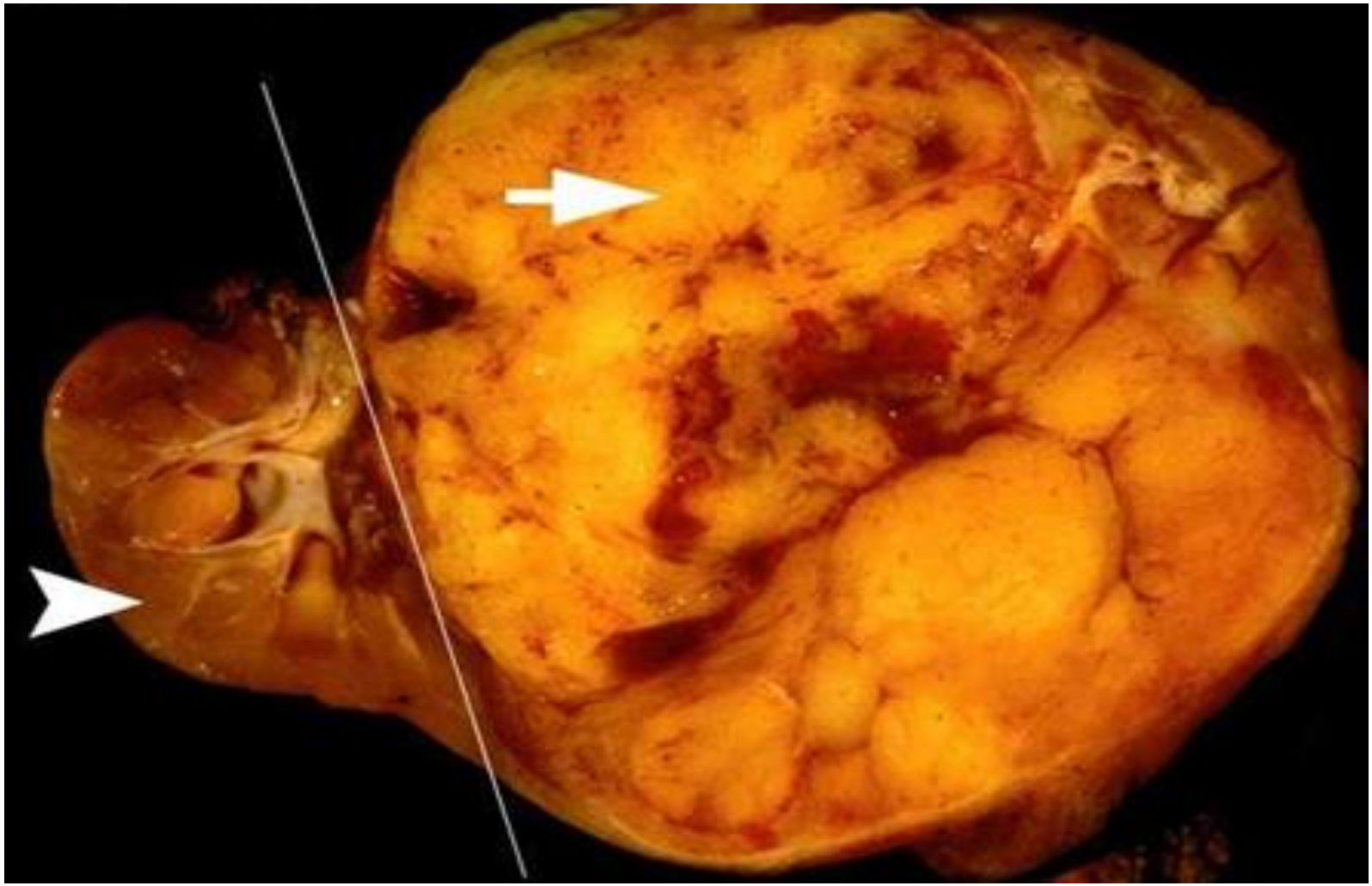


Gross picture shows partly pale and partly hemorrhagic solid tumor replacing almost the entire renal parenchyma and areas of necrosis also seen .

Wilm's Tumor – Gross Pathology



Wilm's Tumor – Gross Pathology



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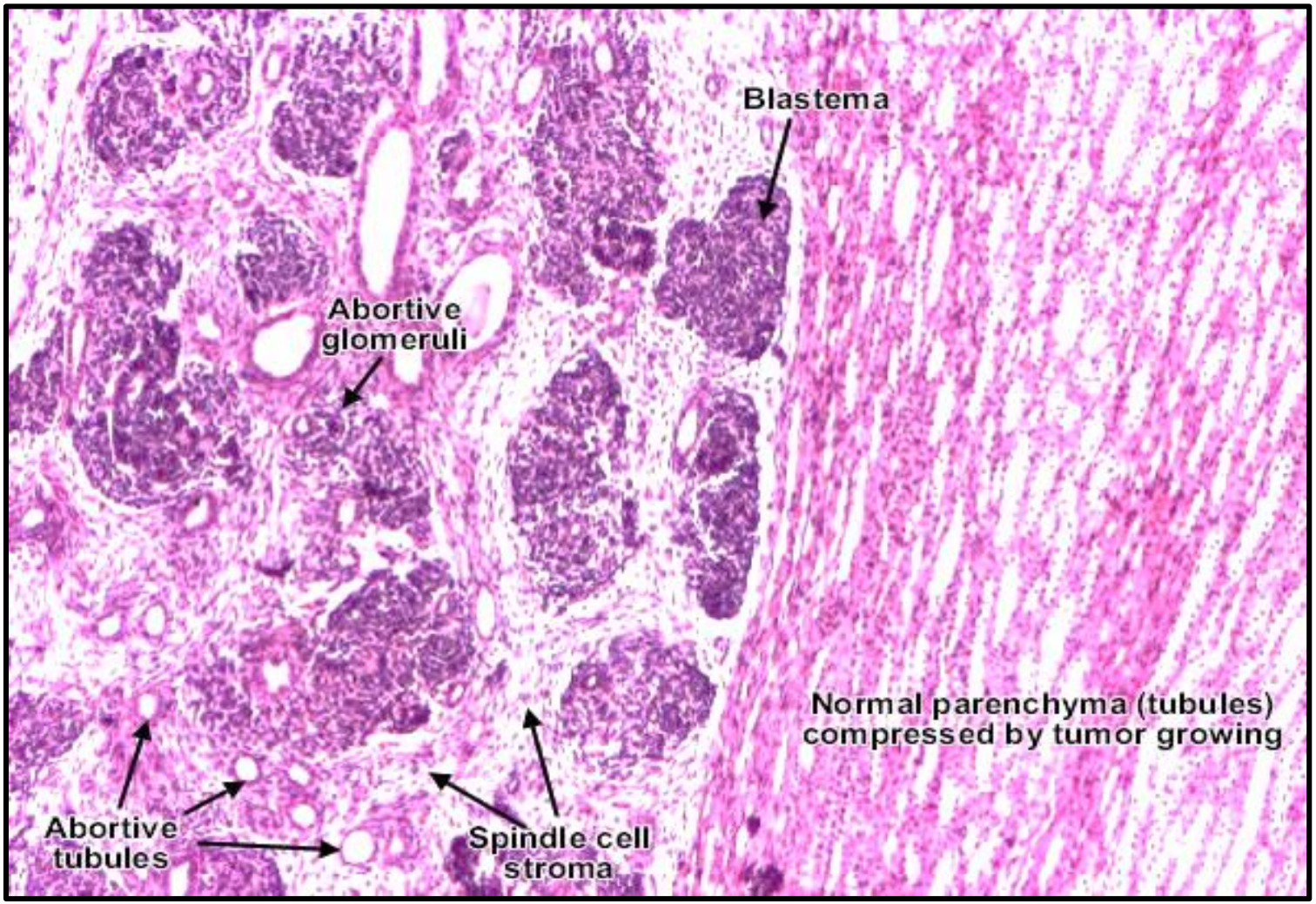
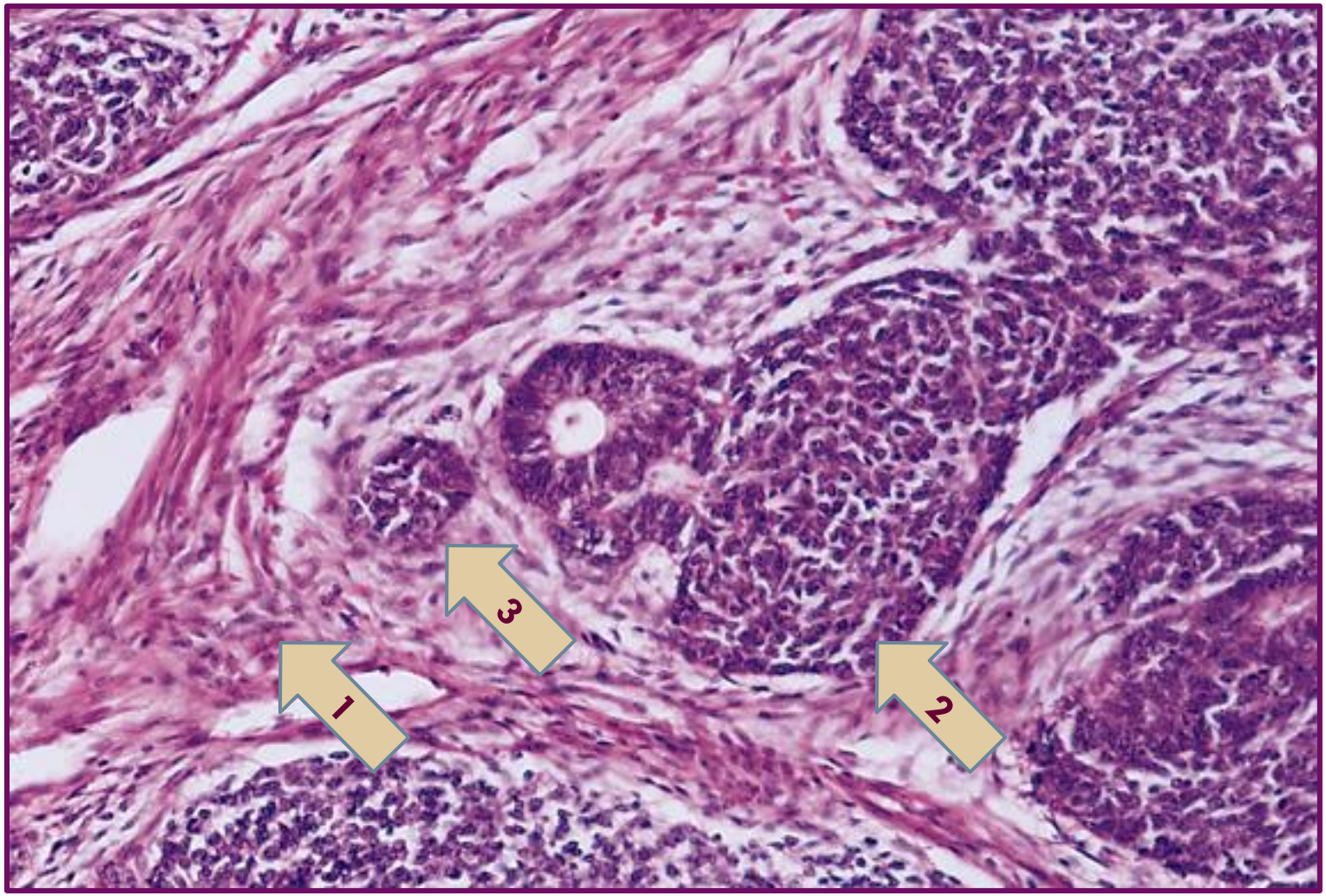
Wilm's Tumor – Histopathology



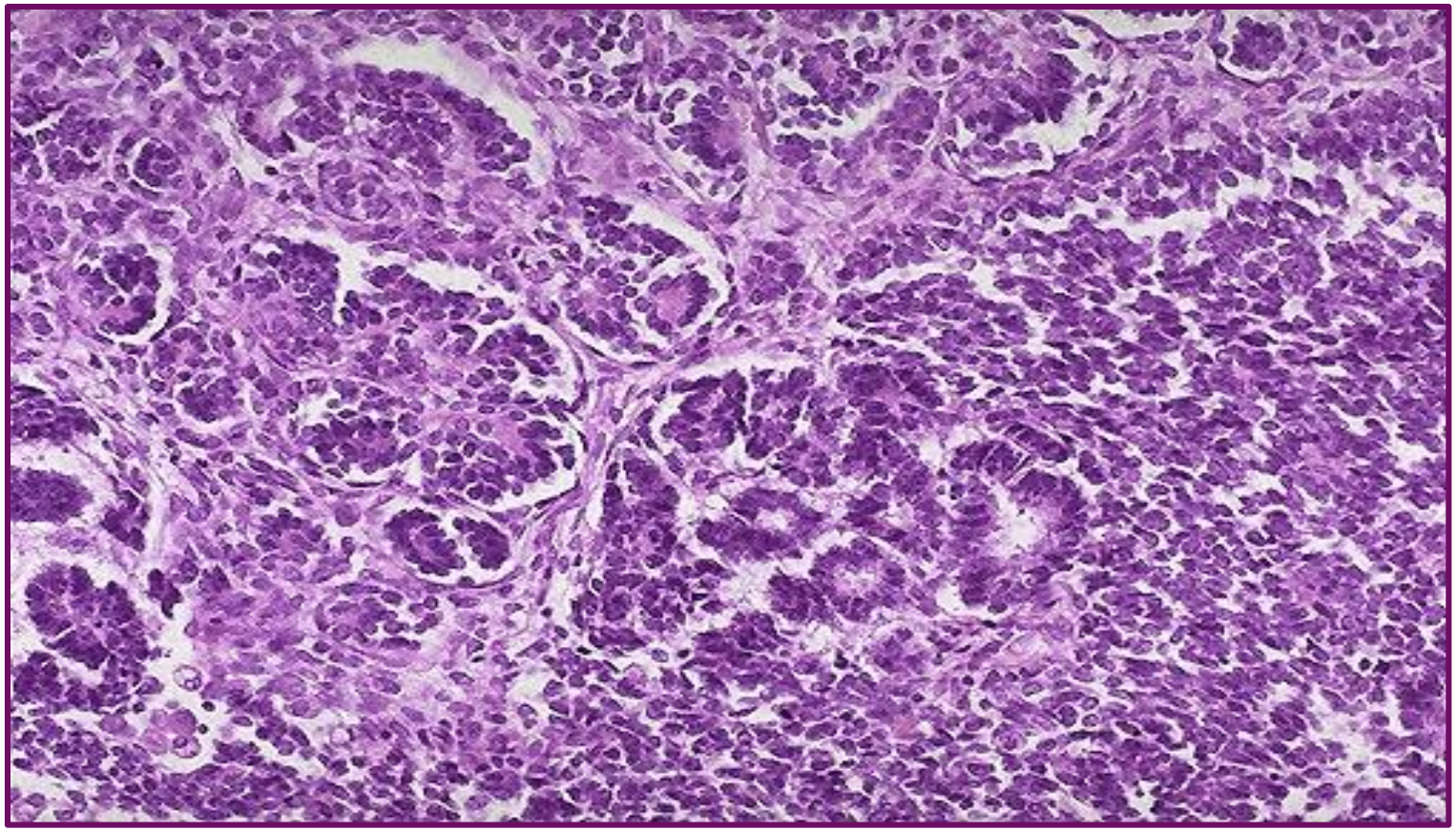
Blastema in WT consists of sheets of densely packed small blue cells with hyperchromatic nuclei, little cytoplasm and conspicuous mitotic activity.

Wilm's Tumor – Histopathology



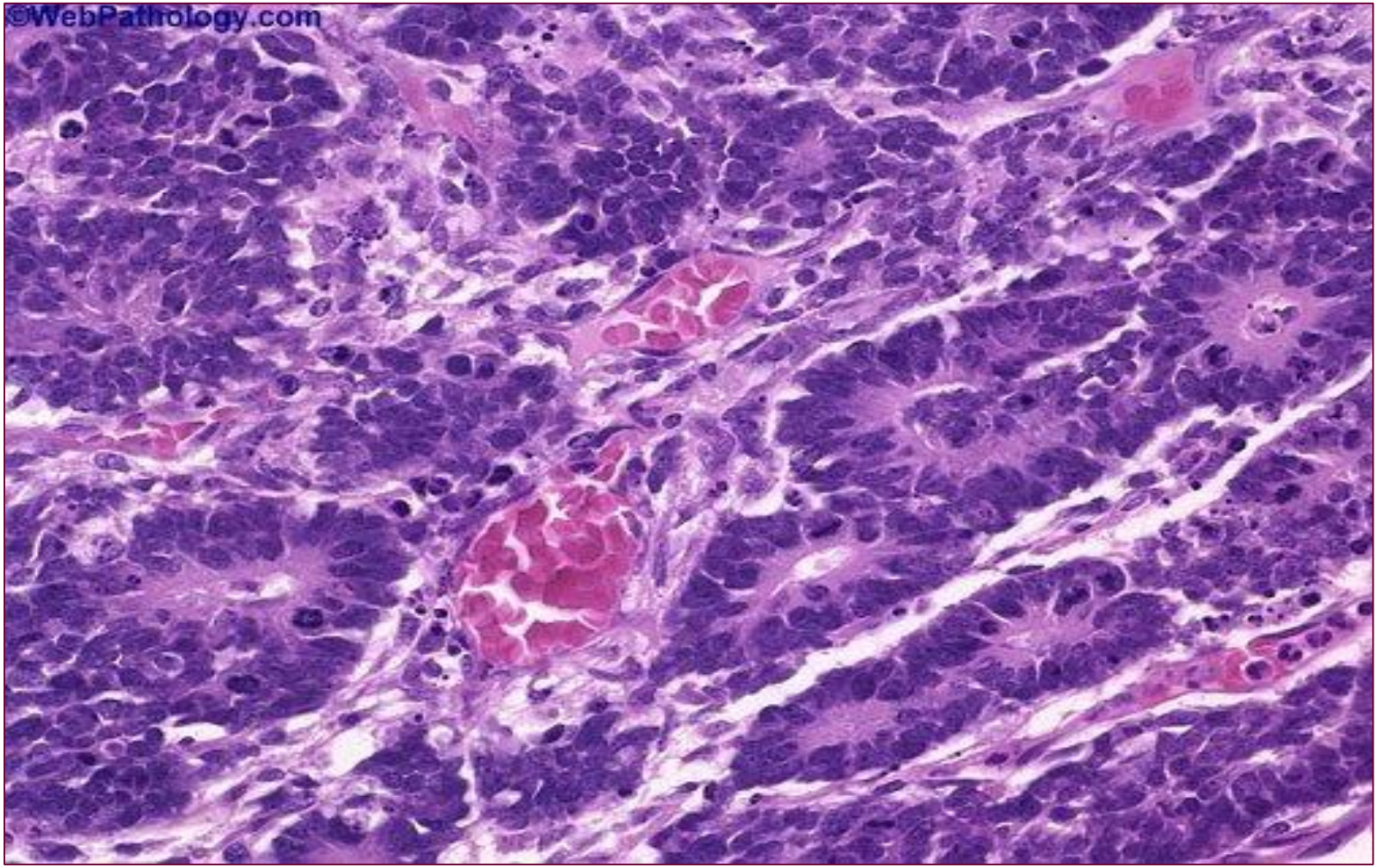
1. Spindle cell stroma.
2. Blastema.
3. Abortive glomeruli.

Wilm's Tumor – Histopathology



Wilm's tumor resembles the fetal nephrogenic zone of the kidney.
Three major components: Undifferentiated blastema cells , epithelial tissue which shows attempts to form primitive glomerular & tubular structures and mesenchymal (stromal) tissue

Wilm's Tumor – Histopathology



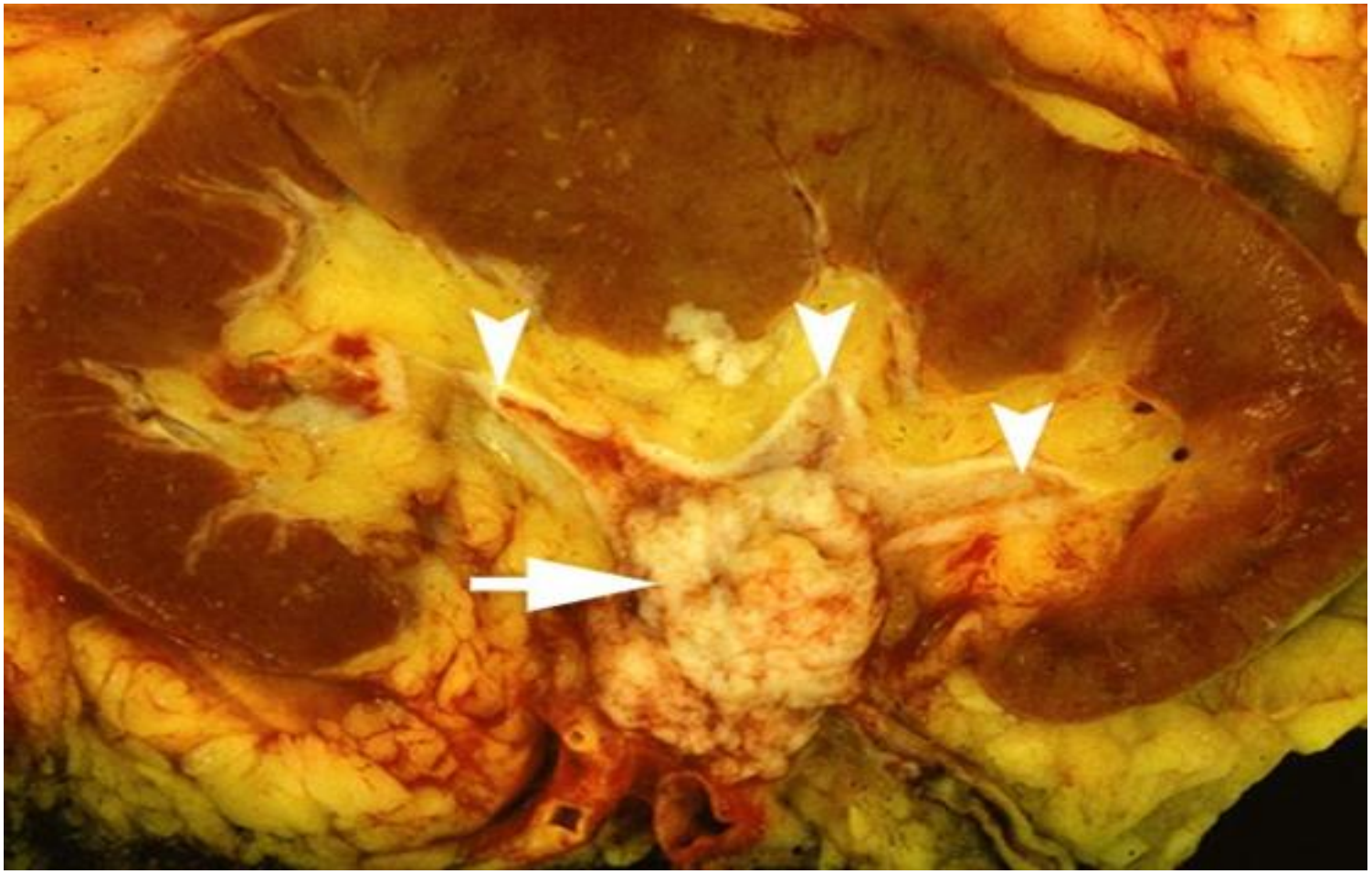
The epithelial component in this Wilm's tumor consists of primitive cuboidal cells forming tubular structures and rosettes.

CARCINOMA OF RENAL PELVIS AND URETER

Urothelial neoplasm in the renal pelvis and ureter

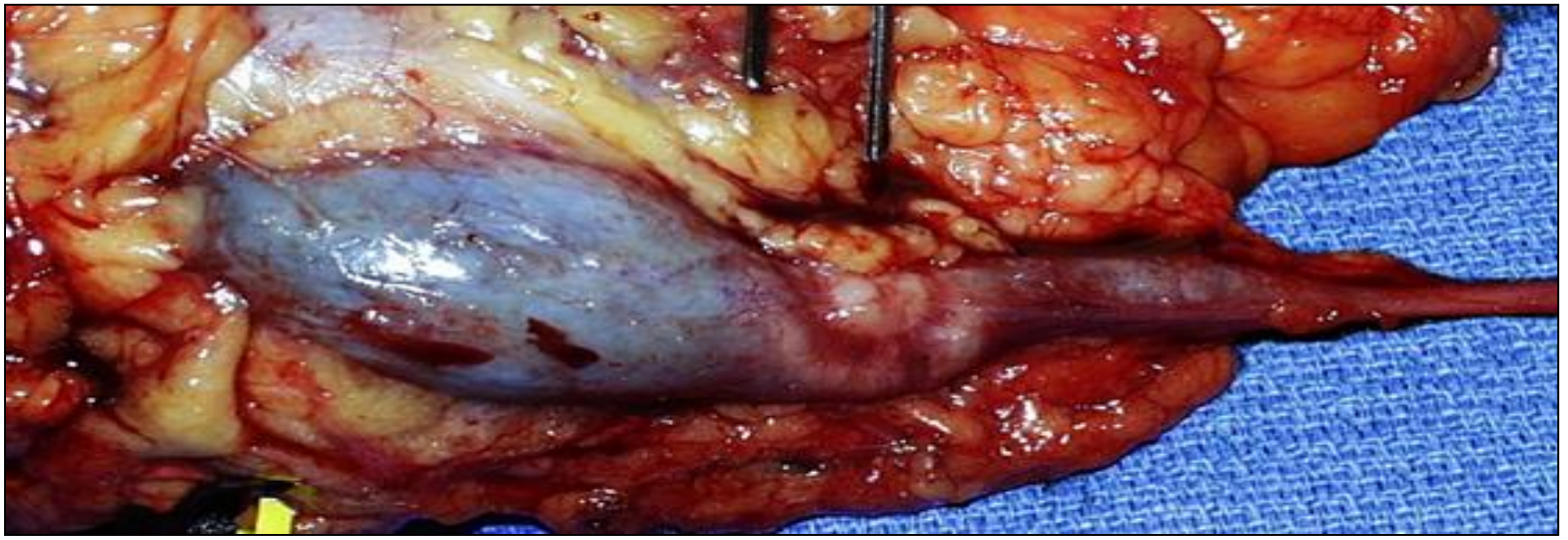
- 85% are papillary; 65% of these are high grade
- Vast majority (93%) of low grade papillary neoplasms are noninvasive
- 64% men, mean age 67 years
- Risk factors: tobacco use, phenacetin use, industrial carcinogen exposure (coal, asphalt, petrochemicals, tar), thorium containing radiologic contrast material,

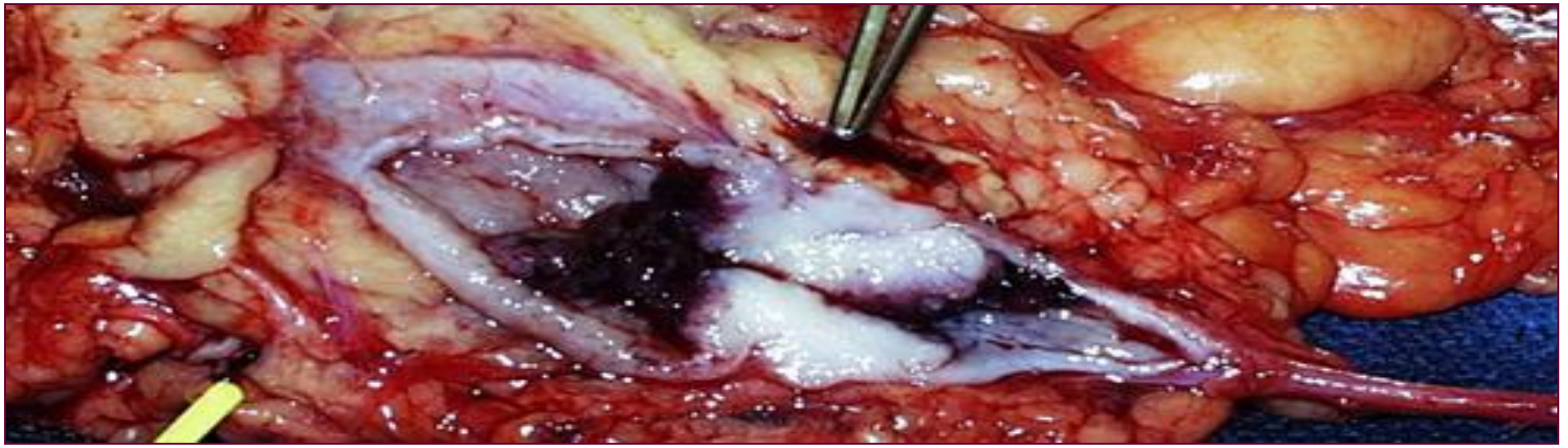
Urothelial (Transitional) Carcinoma of Renal Pelvis



More commonly infiltrative and prognosis is more worse than urothelial carcinoma of the bladder

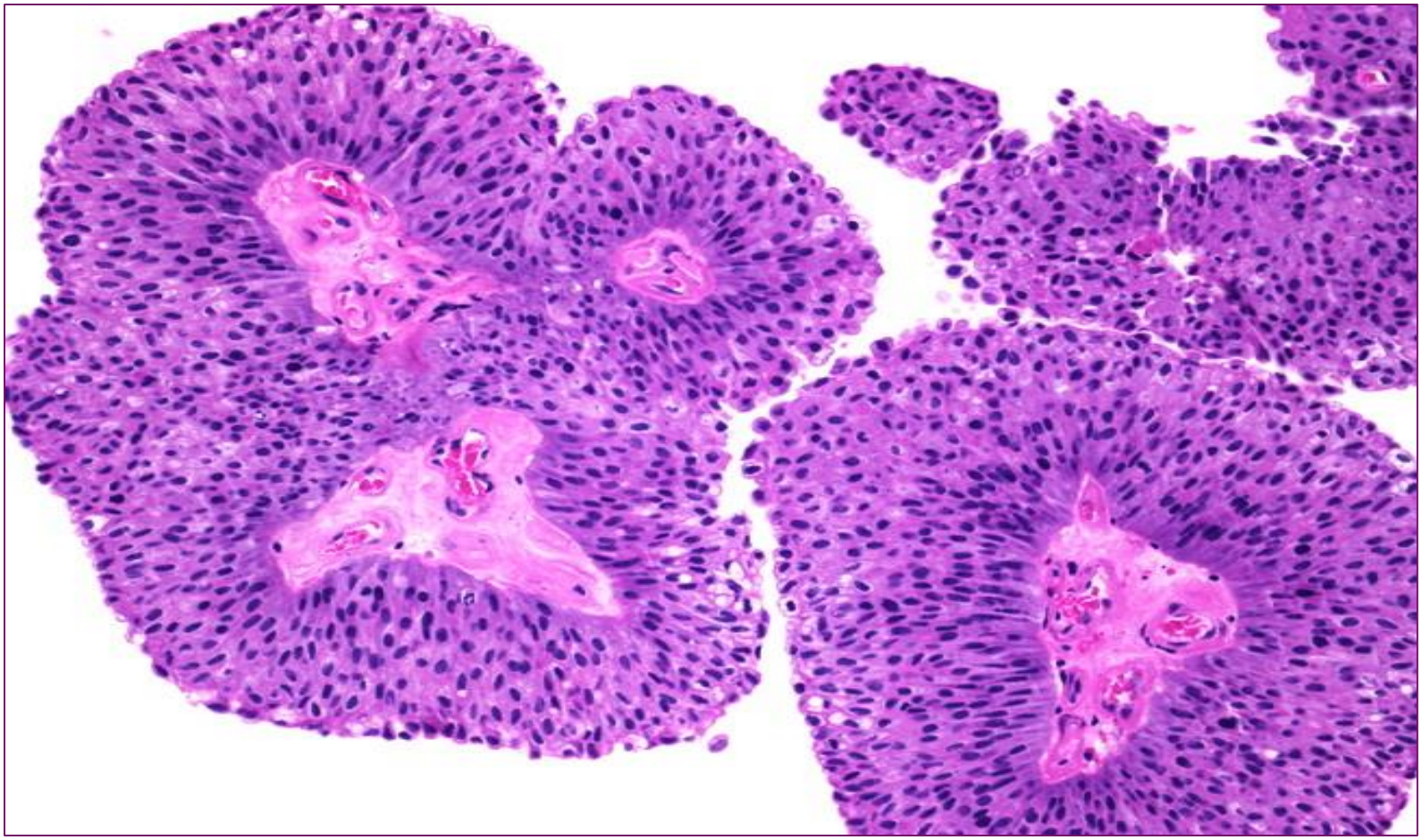
Urothelial Carcinoma involving Ureter - Gross





A nephroureterectomy specimen showing bulbous expansion of proximal ureter near the renal pelvis caused by papillary urothelial carcinoma

Papillary Urothelial carcinoma of the renal pelvis – Low Grade



Low-grade papillary urothelial carcinoma shows minimal cytologic and architectural atypia. Adjacent papillary fronds may fuse, as seen in this image

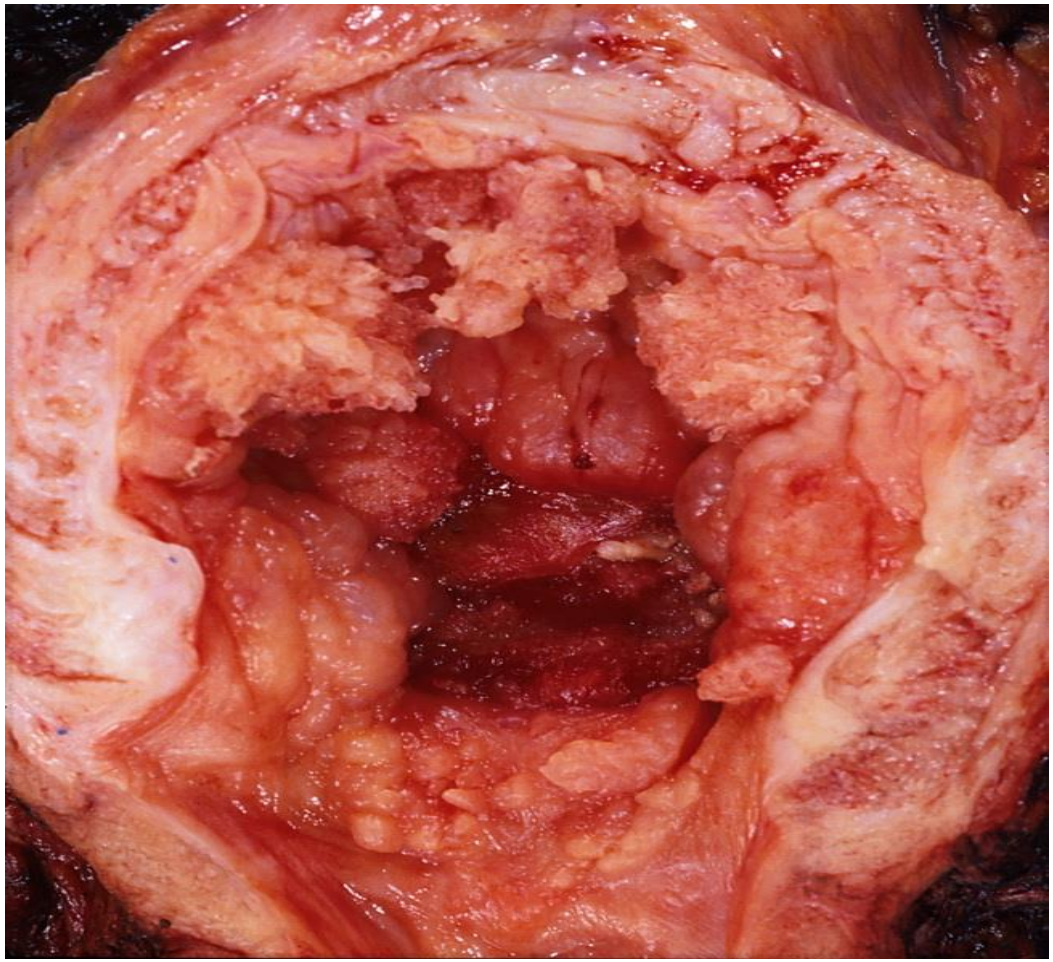
CARCINOMA OF THE URINARY BLADDER

• Cigarette smoking is major risk factor (50-80% of cancers, risk associated with duration and intensity).
Other risk factors: *Exposure to aniline and Azo dyes* and *Cyclophosphamide*

• In developing countries, *Schistosoma haematobium* ova are deposited in bladder wall and cause chronic inflammation, squamous metaplasia, dysplasia; 70% of tumors are squamous cell carcinoma

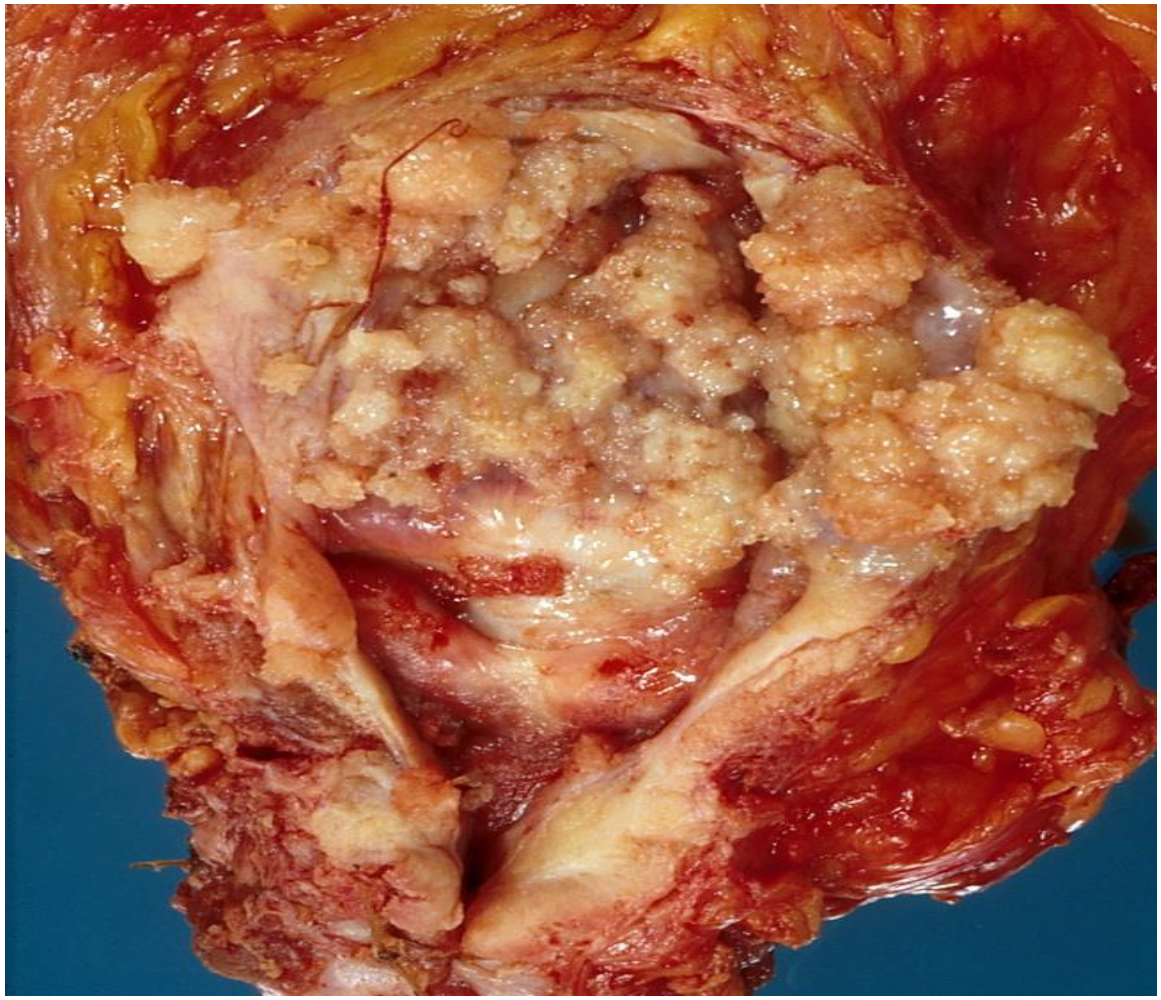
- Typically age 60+ years
- Initial symptoms are painless hematuria, infection, obstruction if near ureteral orifices
- The World Health Organization classifies bladder cancers as low grade (grade 1 and 2) or high grade (grade 3)

Urinary Bladder Carcinoma - Urothelial (Transitional cell) papillary Carcinoma - Gross



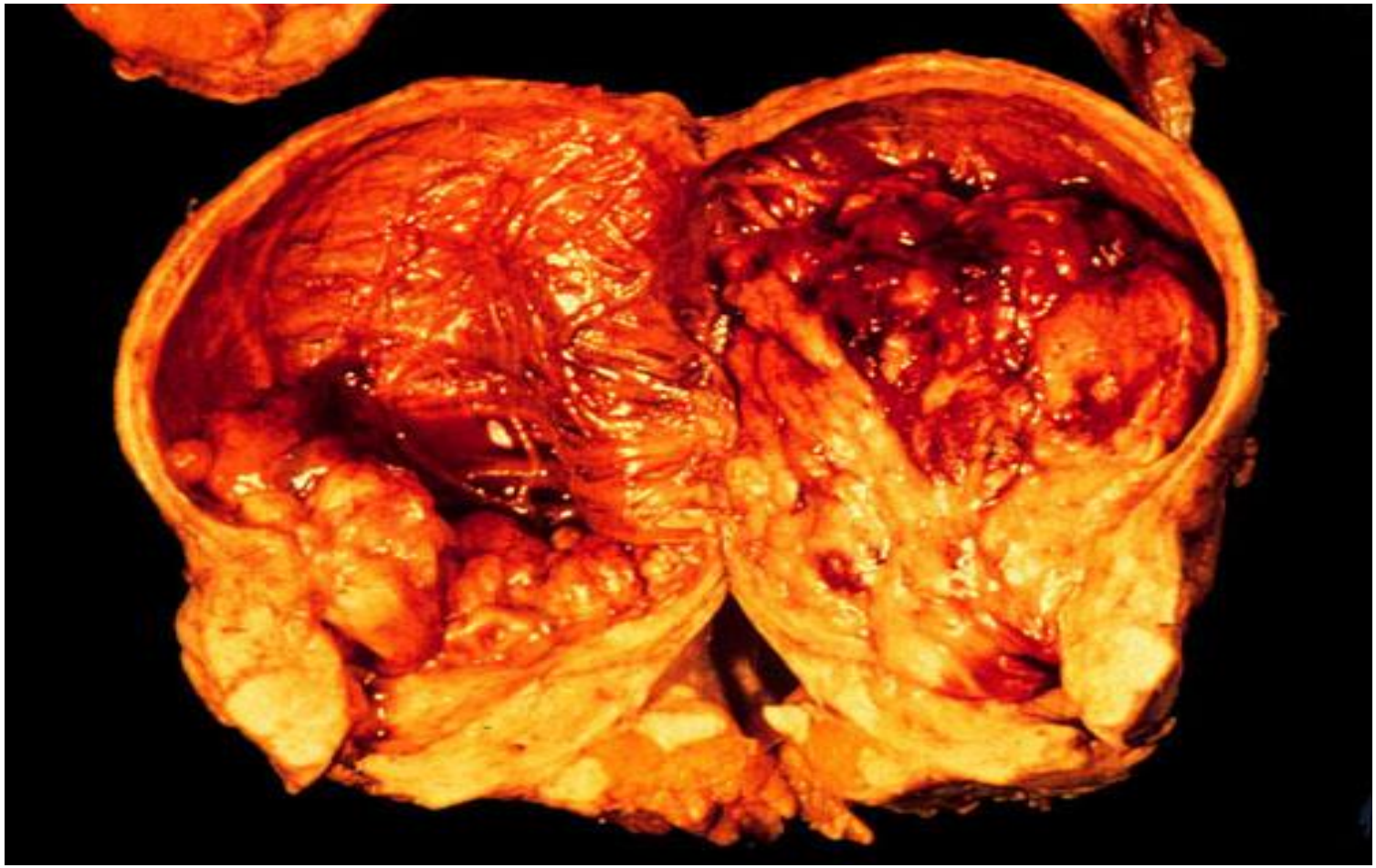
90% of bladder cancers are transitional cell carcinoma. The other 10% are squamous cell carcinoma, adenocarcinoma, sarcoma, small cell carcinoma, and secondary metastases

Papillary Urothelial Carcinoma of Bladder - Gross



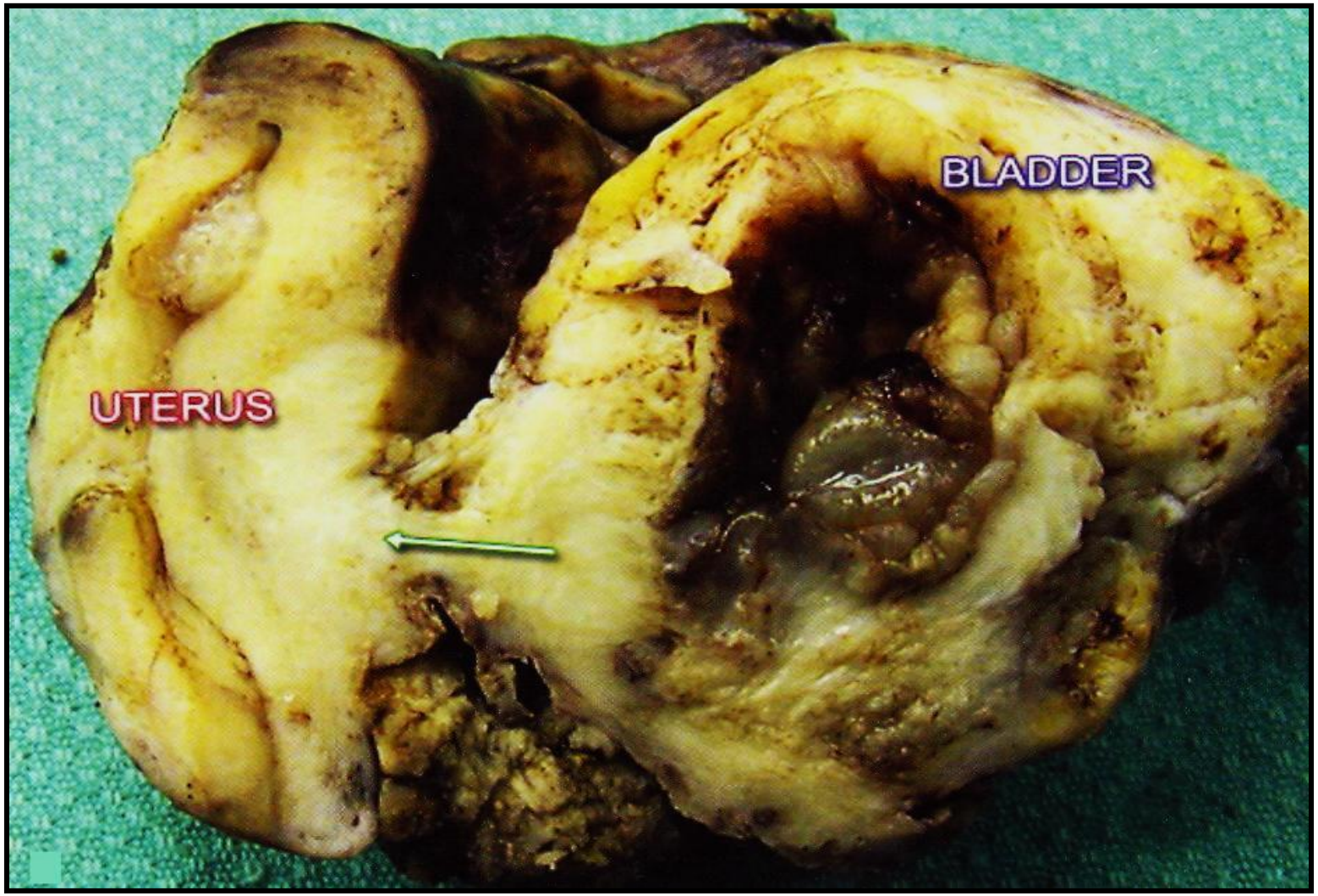
Radical cystectomy specimen showing multifocal papillary urothelial carcinoma..

Transitional Carcinoma of Bladder - Gross



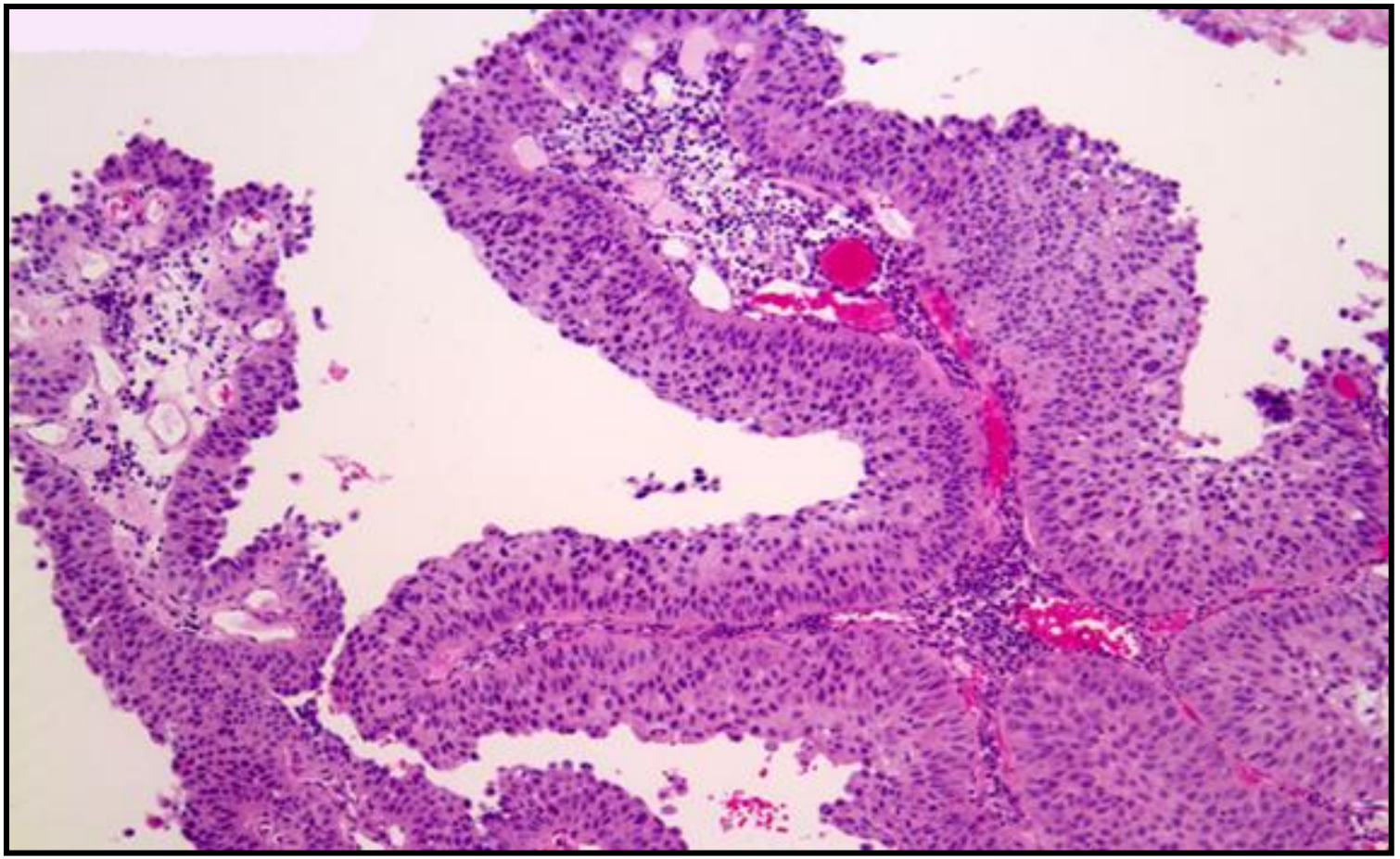
The mucosa of the open urinary bladder appears edematous. There are several whitish or red nodules and patches indicative of a multi-focal nature of this tumor

Bladder Tumor invading the Uterus – Gross



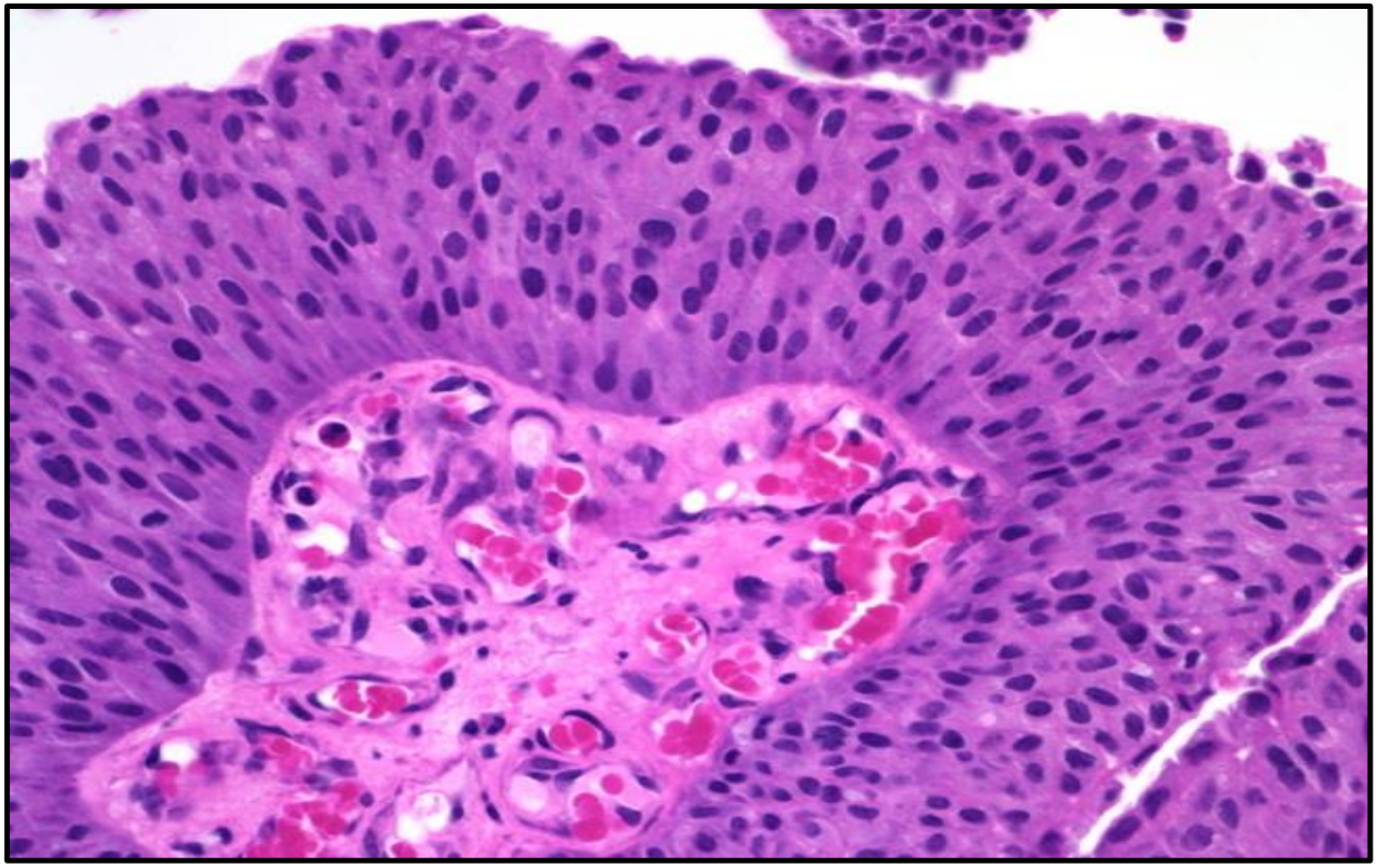
Urinary bladder carcinoma infiltrating the urinary bladder wall with extension to the uterus .

Papillary Urothelial carcinoma – Low Grade



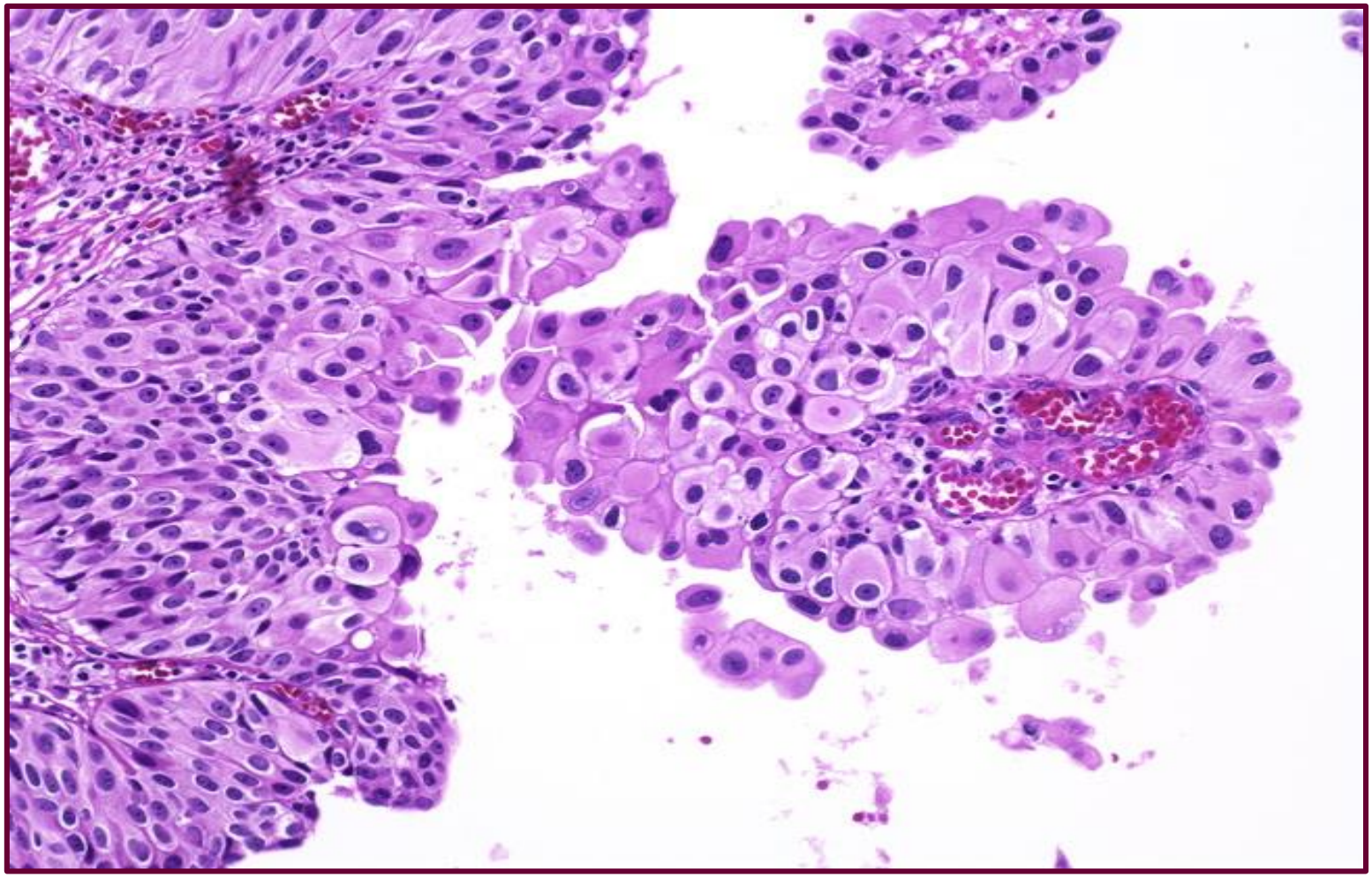
The low grade tumors show overall preservation of cell polarity, few mitoses, and lack of significant morphologic atypia. This exophytic papillary tumor shows multiple finger-like projections lined by multiple layers of urothelium (transitional epithelium)

Papillary Urothelial Carcinoma – Low Grade



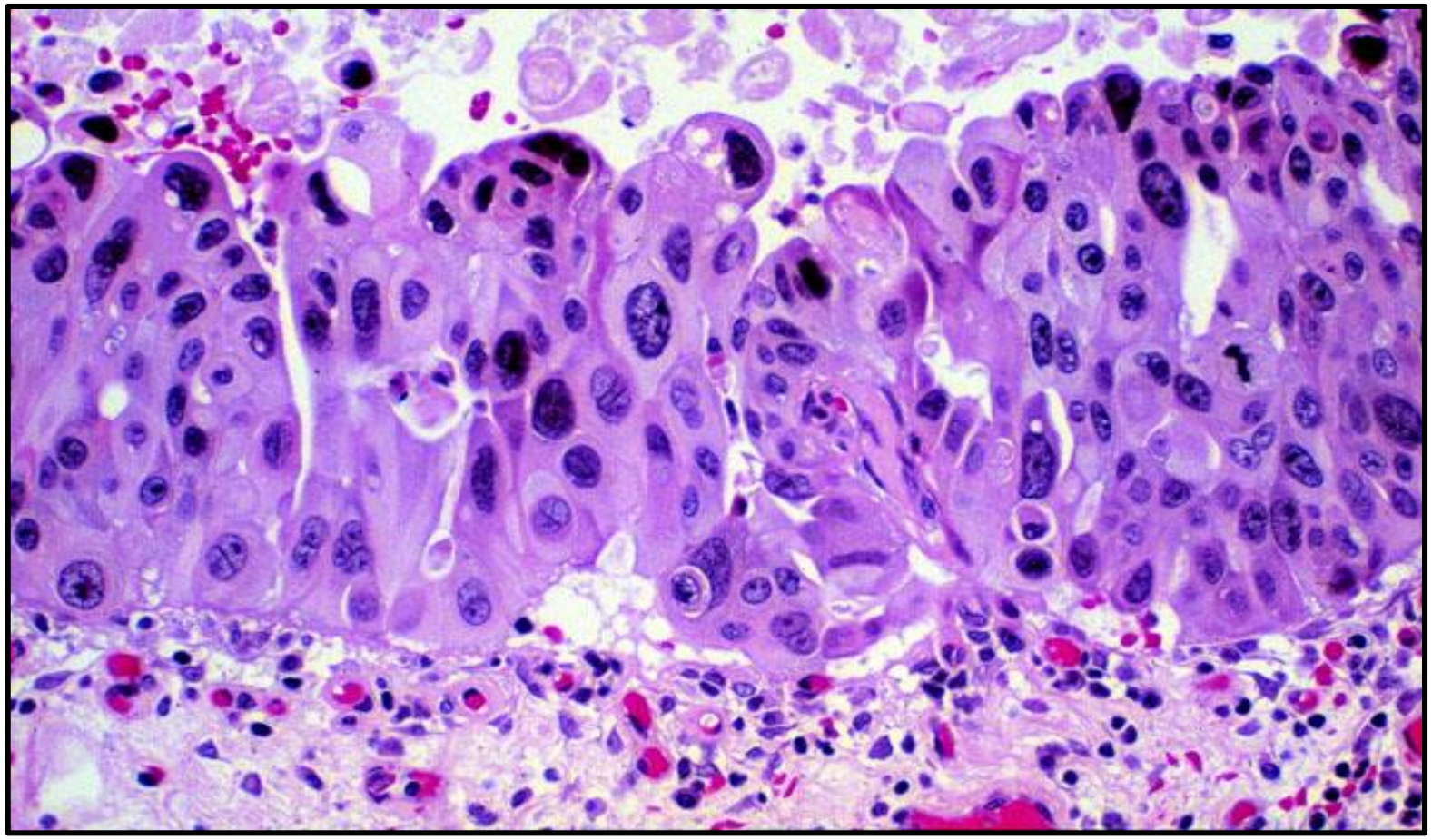
High power view of a low-grade papillary urothelial carcinoma. There are scattered hyperchromatic nuclei and typical mitotic figures

Papillary Urothelial carcinoma – High Grade



This high-grade papillary urothelial carcinoma shows highly pleomorphic cells with voluminous cytoplasm

Urothelial (Transitional) carcinoma – HPF



Almost all cases of Bladder carcinomas are originating from the transitional epithelium. Bladder carcinoma might be squamous cell in nature. Chronic inflammation of the bladder mucosa, caused by stones or schistosomiasis may lead to it . Rarely, it presents as adenocarcinoma

PATHOLOGY OF RENAL ALLOGRAFT

- Allograft: The transplant of an organ or tissue from one individual to another of the same species with a different genotype
- Transplant rejection is a process in which a transplant recipient's immune system attacks the transplanted organ or tissue
- On the basis of the morphology and the underlying mechanism, **rejection reactions are classified as hyperacute, acute, and chronic.**

Classification of renal rejection

- ***Hyperacute Rejection:***

- occurs within minutes or hours after transplantation.
- Immunoglobulin and complement are deposited in the vessel wall, causing endothelial injury and fibrin-platelet thrombi
- Neutrophils rapidly accumulate within arterioles, glomeruli, and peritubular capillaries

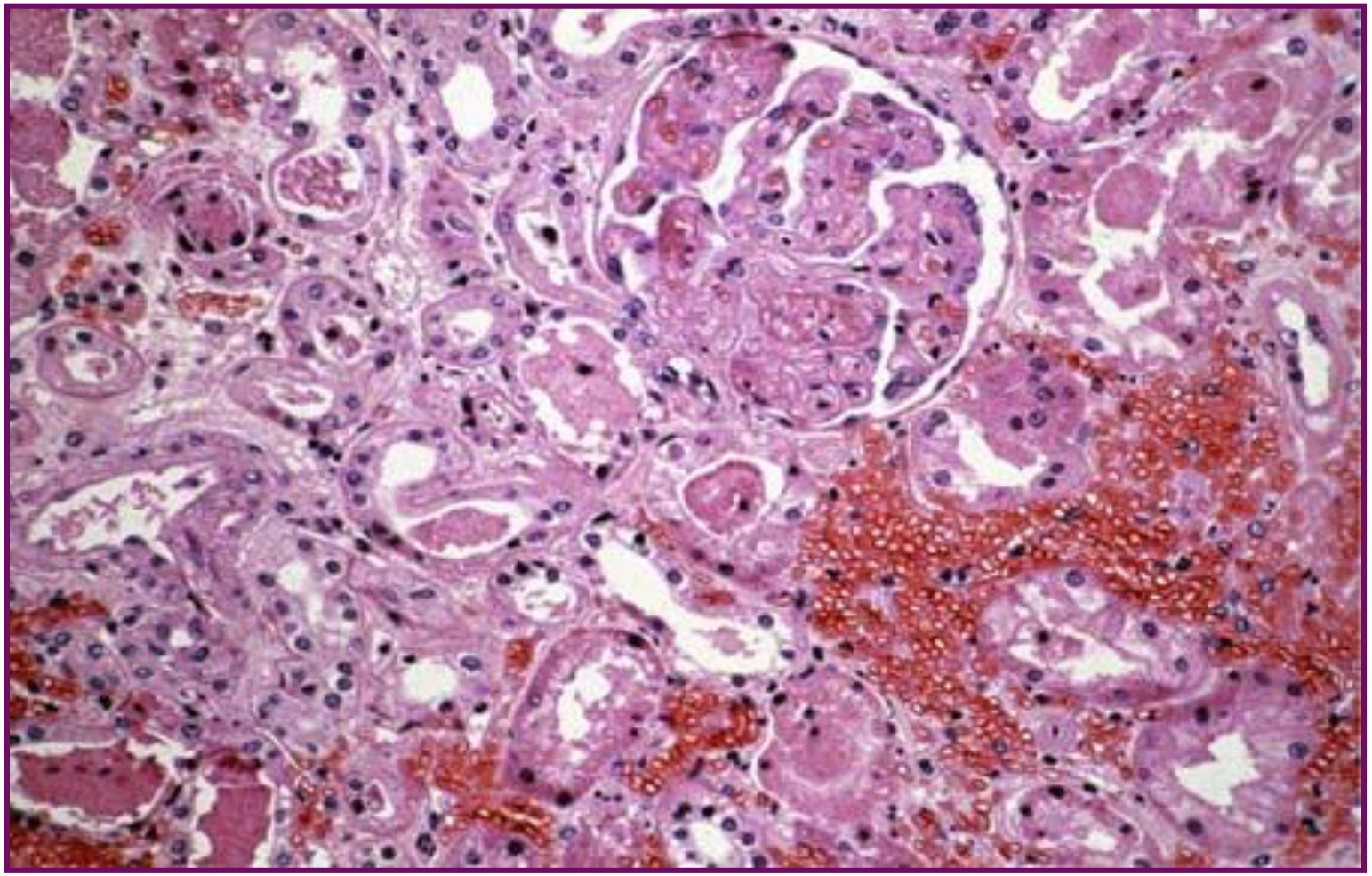
- ***Acute Rejection:***

- within days of transplantation in the untreated recipient or may appear suddenly months or even years later, after immunosuppression has been used and terminated.
- cellular or humoral immune

- ***Chronic Rejection:***

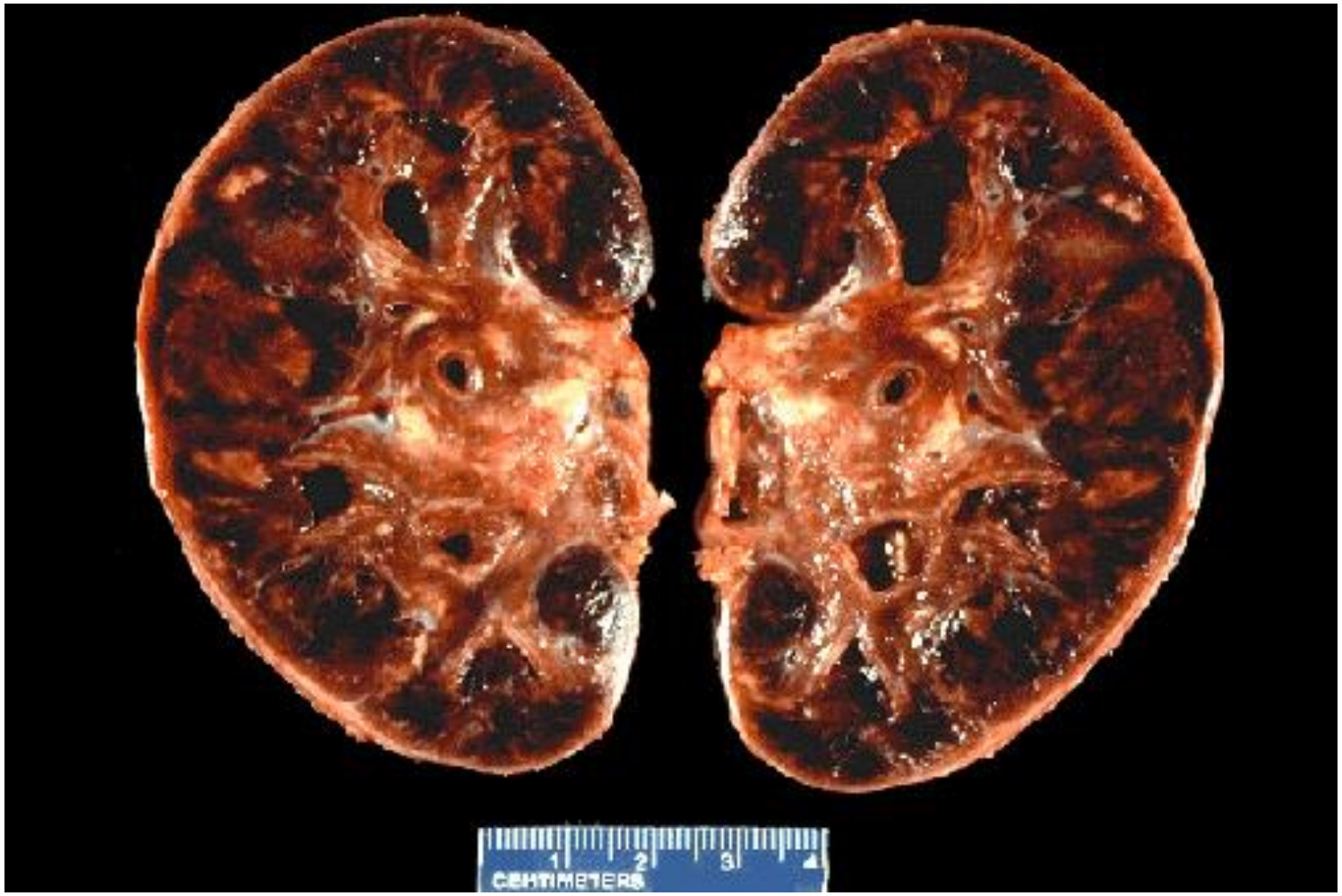
- Chronic rejection is dominated by vascular changes, interstitial fibrosis, and tubular atrophy with loss of renal parenchyma

Hyperacute Allograft Rejection



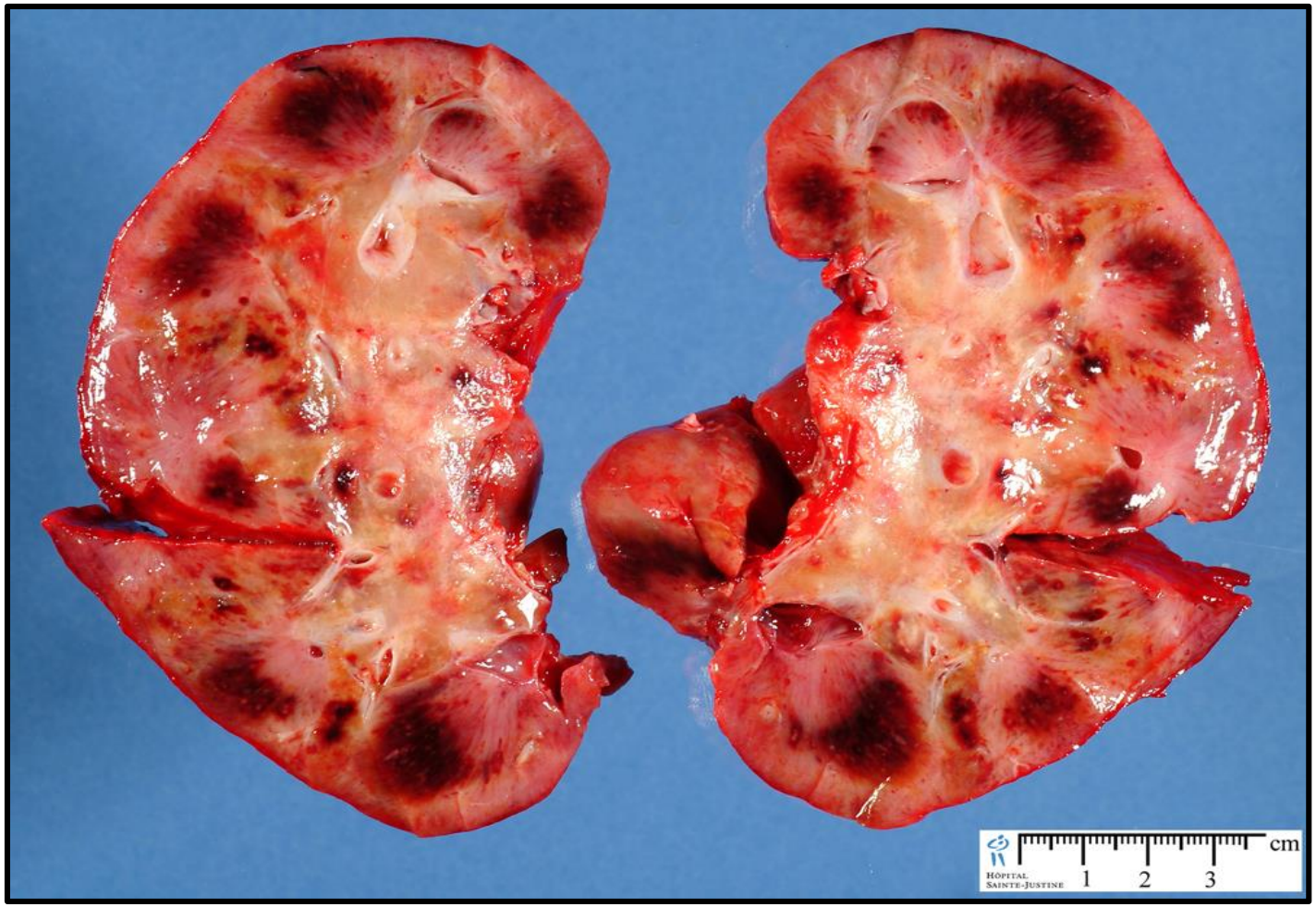
Hyperacute rejection. The cortex shows diffuse hemorrhage and neutrophils in peritubular capillaries with prominent glomerular thrombi 1 day after transplantation

Acute Cellular Allograft Rejection



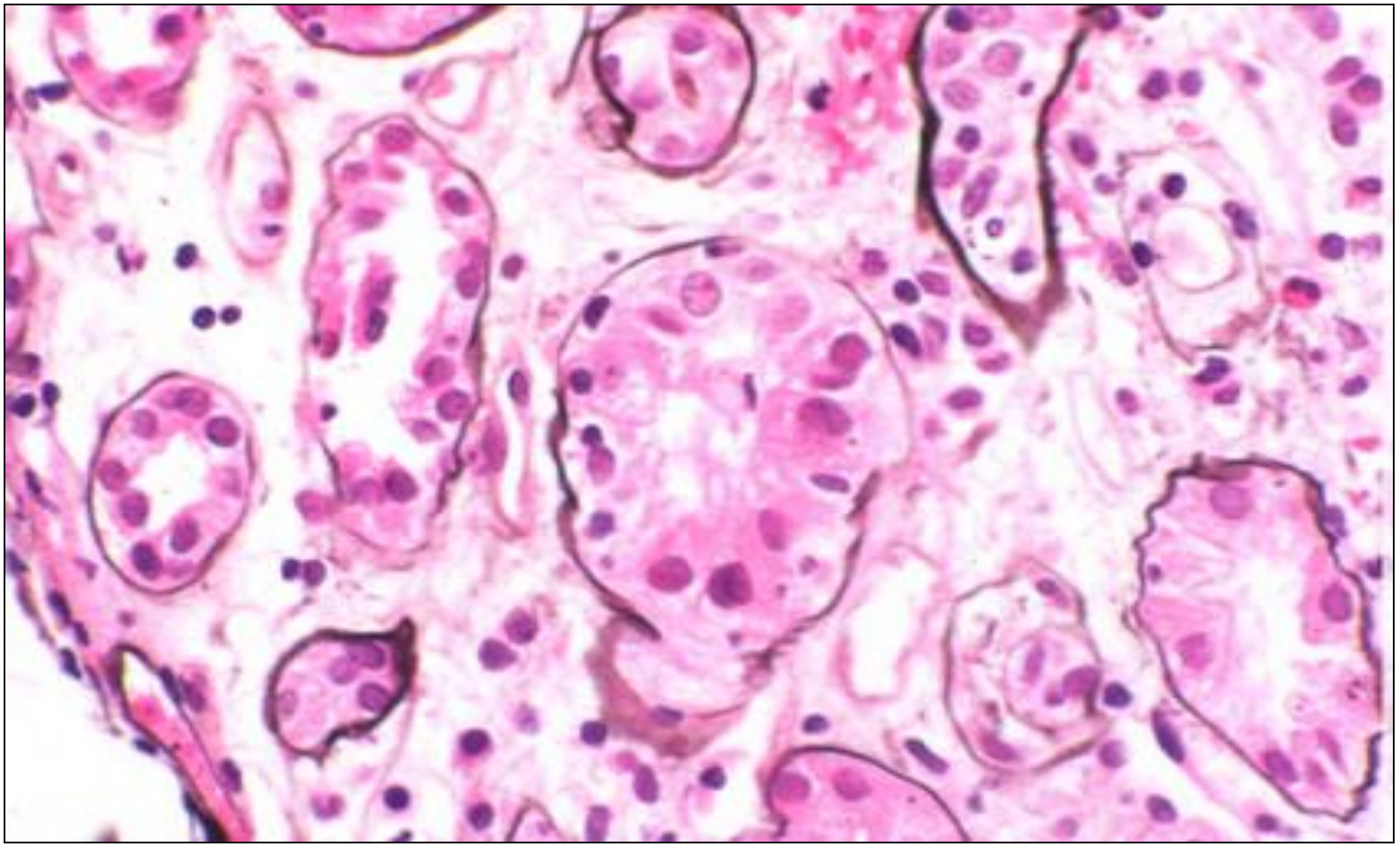
This kidney was removed because of acute transplant rejection. Note the swollen and hemorrhagic appearance of this entire kidney.

Acute Cellular Allograft Rejection



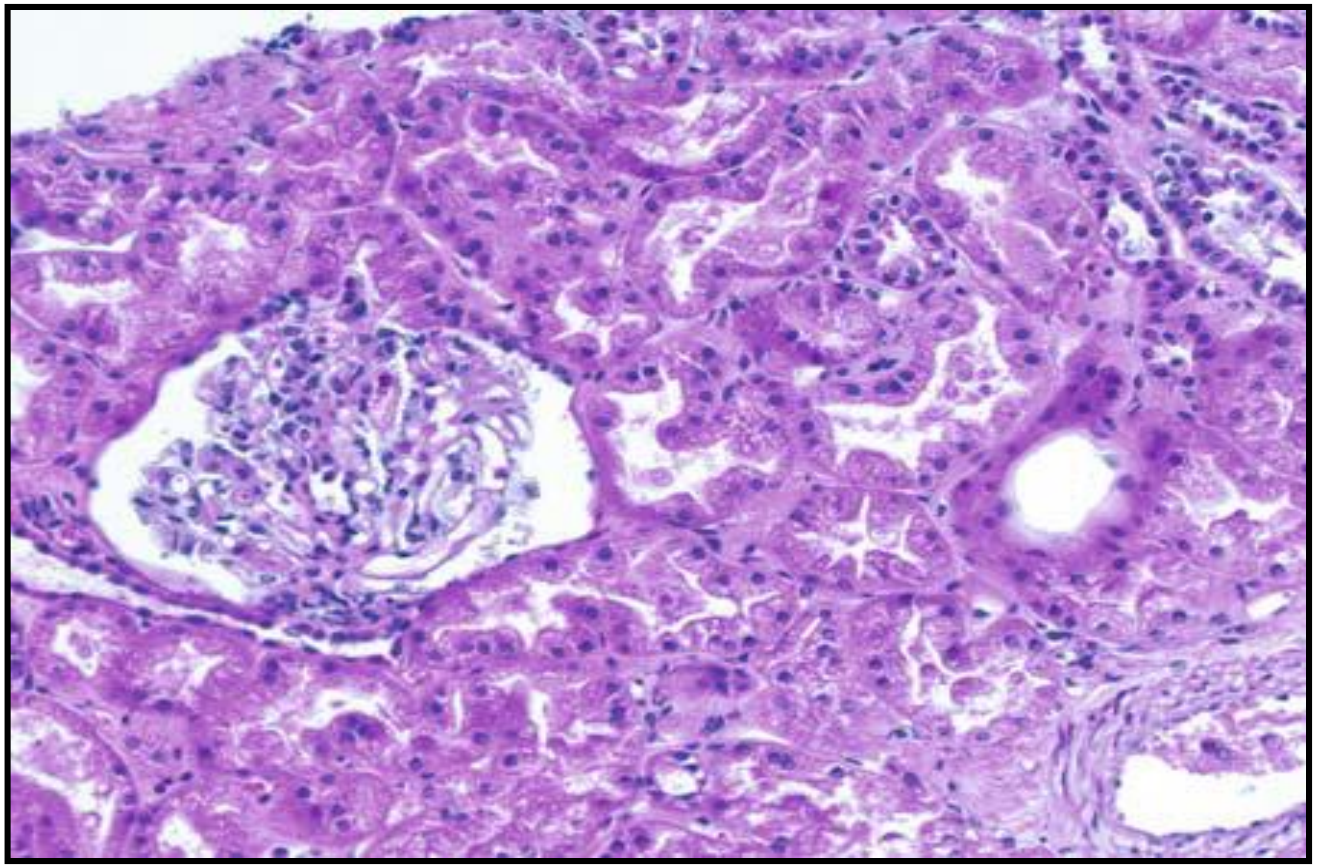
Swollen and hemorrhagic appearance of acutely rejected renal allograft

Acute Cellular Allograft Rejection – Type I



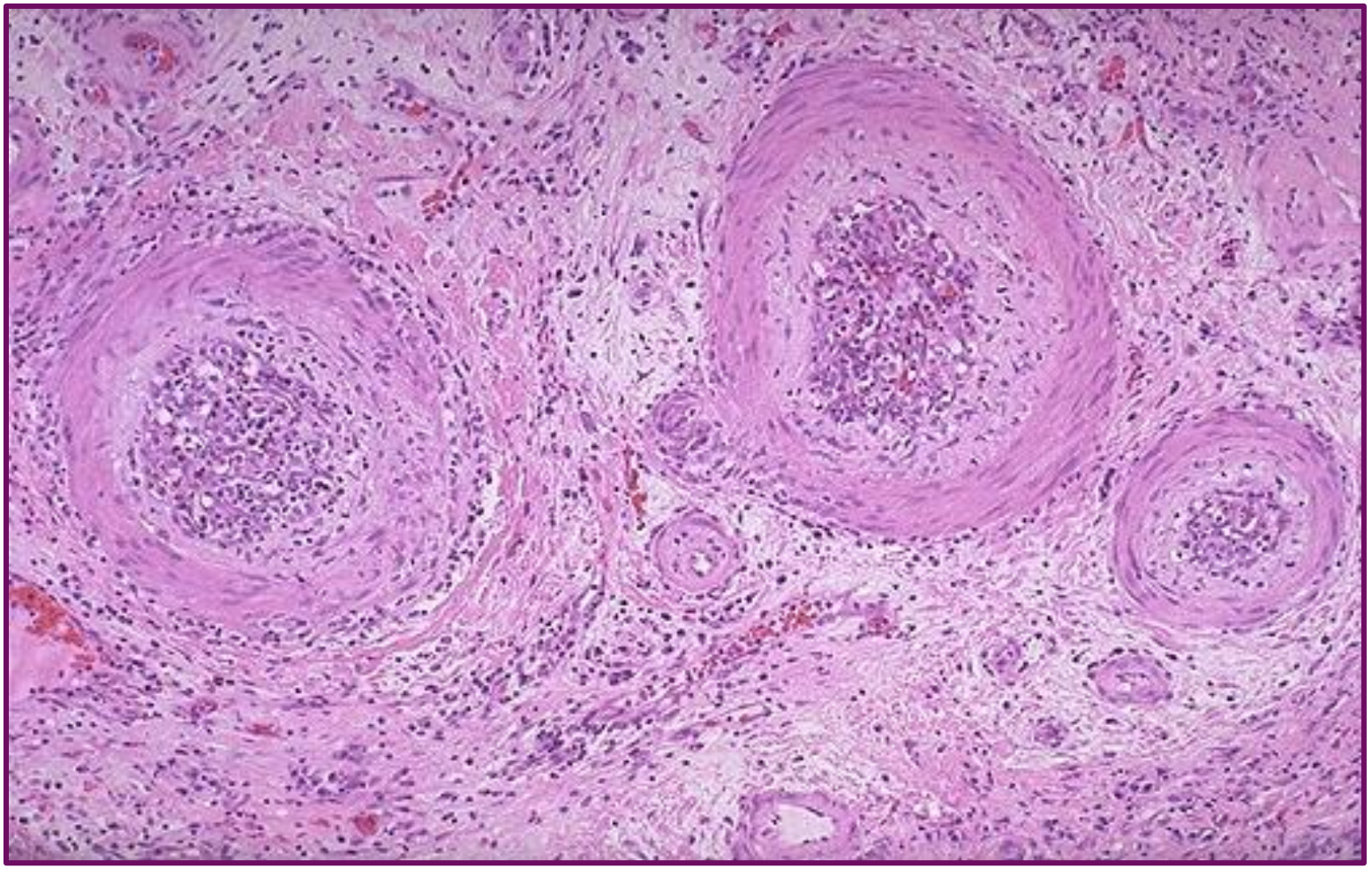
Tubulitis, ie, infiltration of tubular epithelium by lymphocytes, is the hallmark of type I interstitial acute rejection

Acute Humoral Rejection (AHR) – Type I



Humoral (Antibody-mediated) rejection, type I. Acute tubular injury is evident, without neutrophils in capillaries. Peritubular and glomerular capillary inflammation with neutrophils, and necrosis of arteries

Chronic Allograft Rejection



Chronic vascular rejection of a renal transplant, which has a poor prognosis. Note the thickened arteries with intimal fibrosis and also chronic inflammation.

THE END