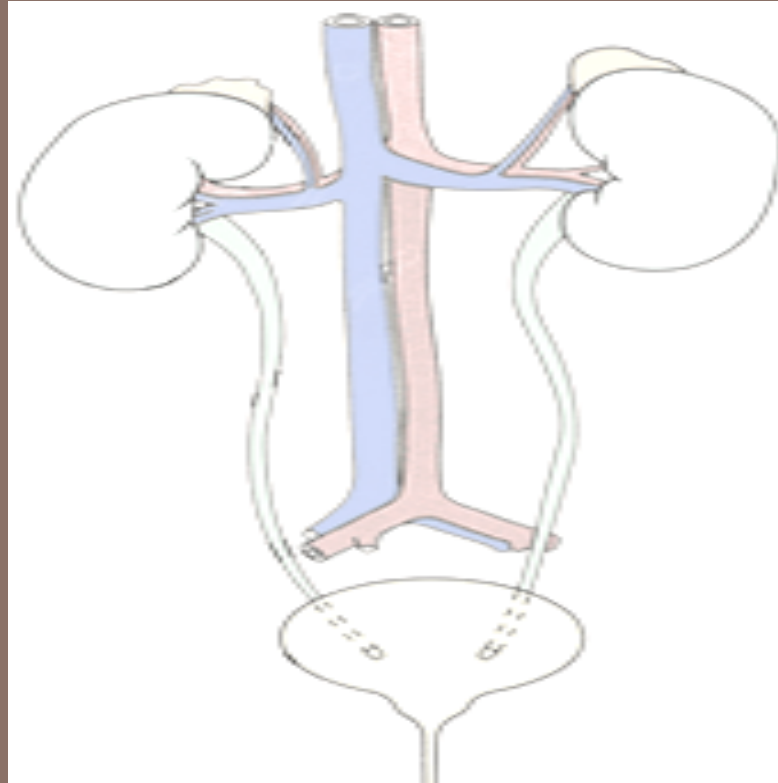


# RENAL BLOCK



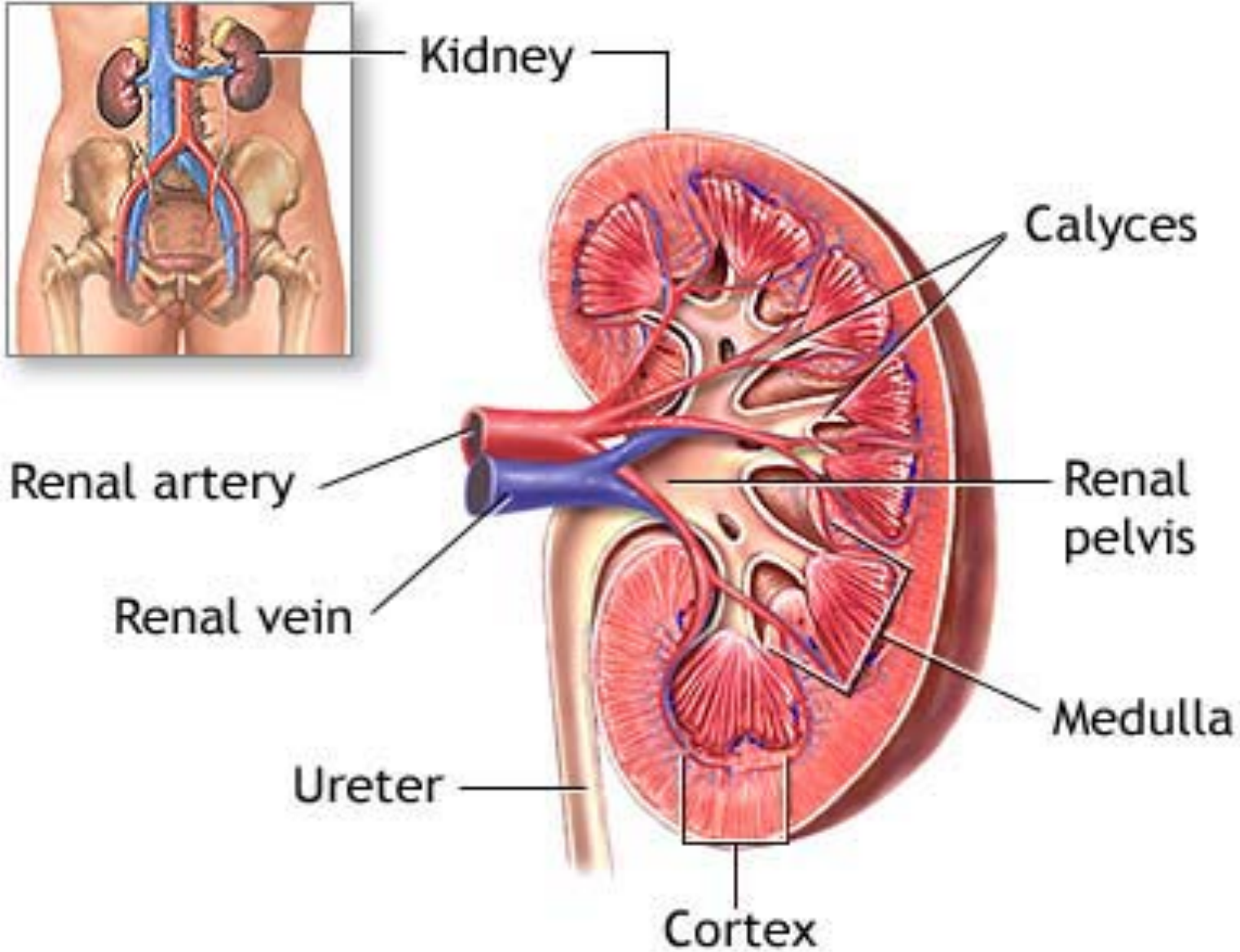
## PATHOLOGY PRACTICAL

Prepared by:

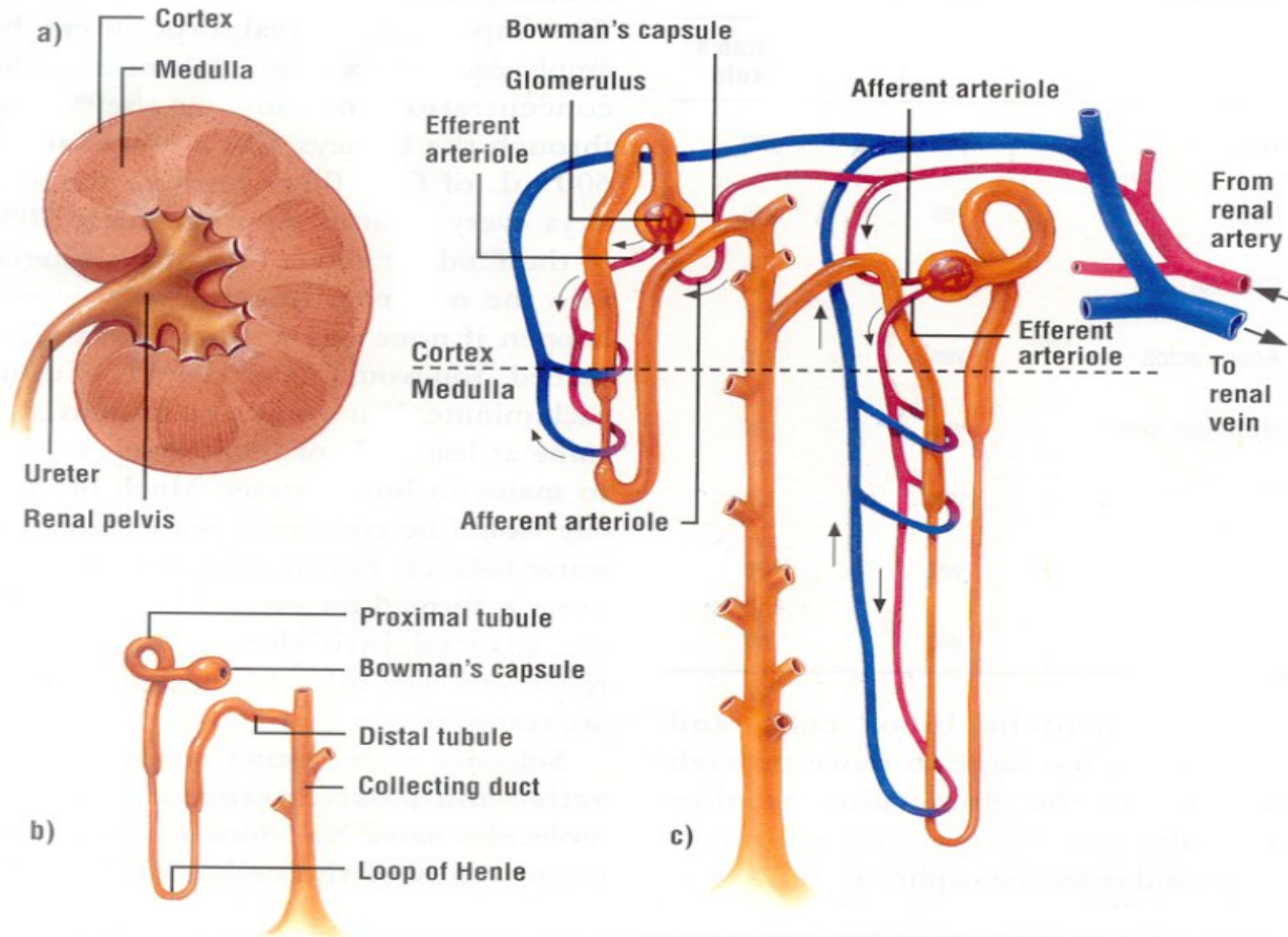
- *Prof. Ammar Al Rikabi*
- *Dr. Sayed Al Esawy*
- *Dr. Shaesta Zaidi*
- *Dr Abdullah Basabein*

# ***NORMAL ANATOMY AND HISTOLOGY***

# Anatomy of the Kidney

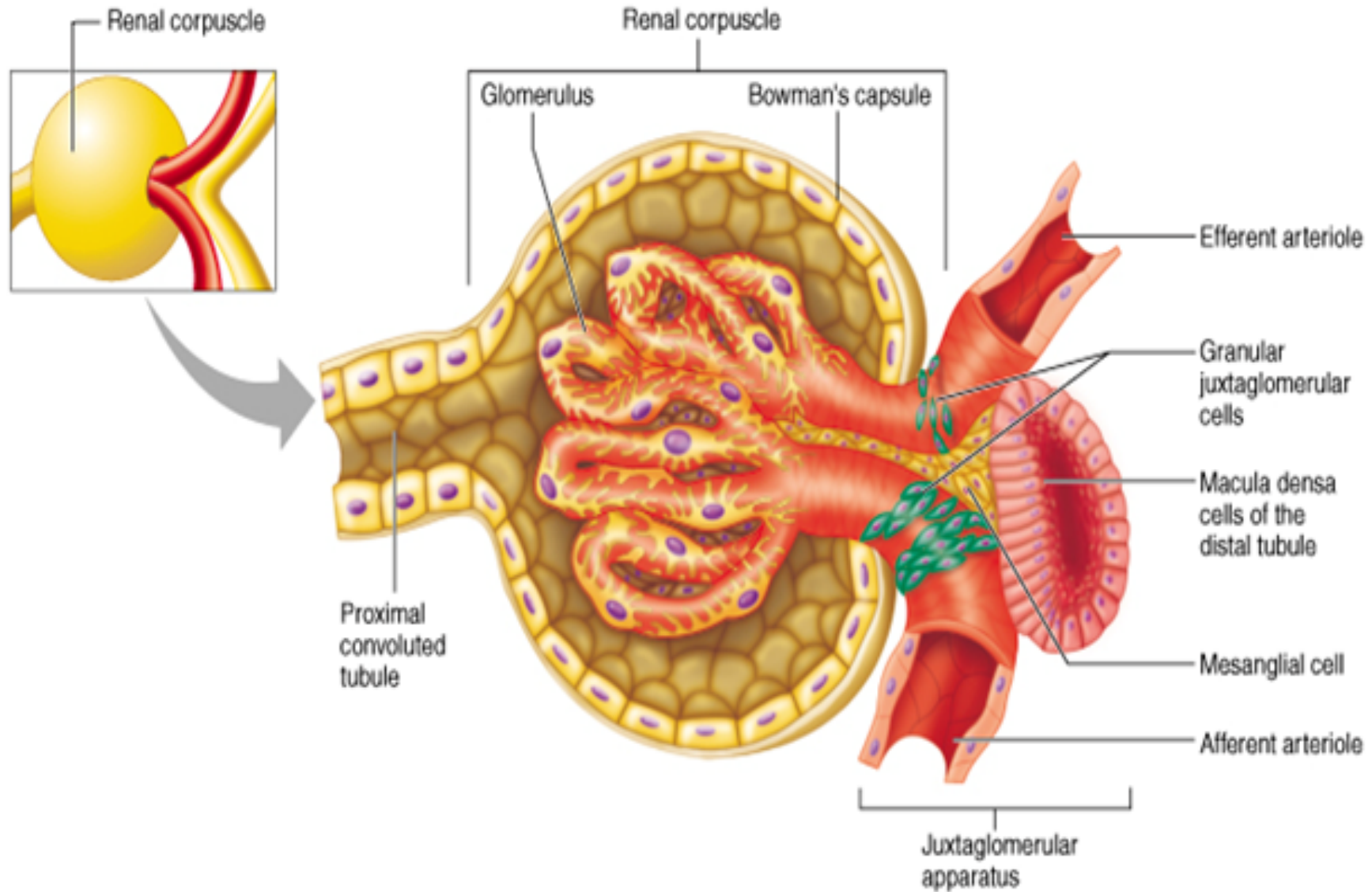


# NEPHRON STRUCTURE





# Renal Corpuscle



## **KIDNEY ANATOMY : NEPHRONS**

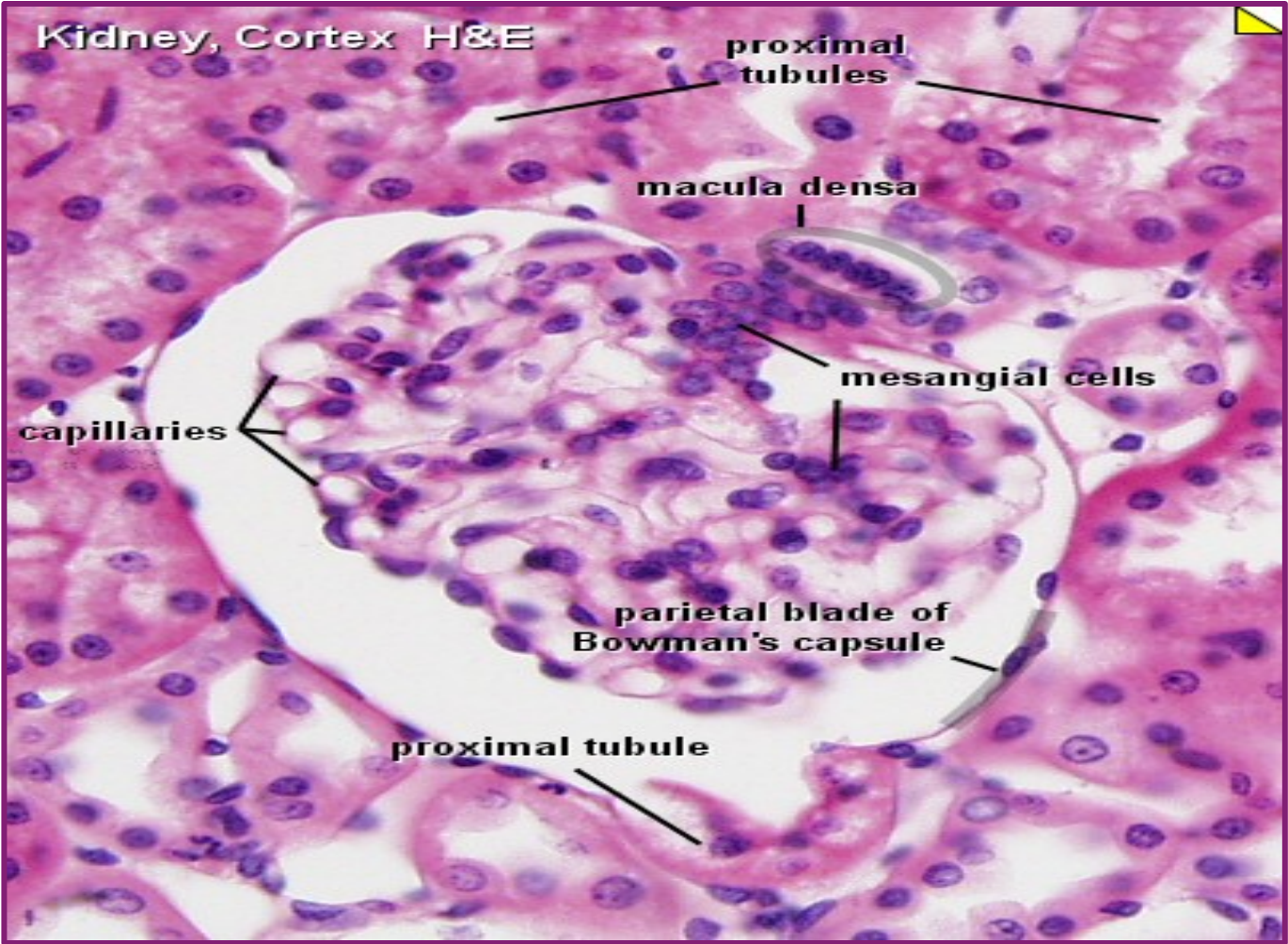
- ***Nephron is the functional unit of the kidney.***
- ***Each kidney contains about 1,000,000 to 1,300,000 nephrons.***
- ***The nephron is composed of glomerulus and renal tubules .***
- ***The nephron performs its function by ultra filtration at glomerulus and secretion and reabsorption at renal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

## Normal Kidney - Gross



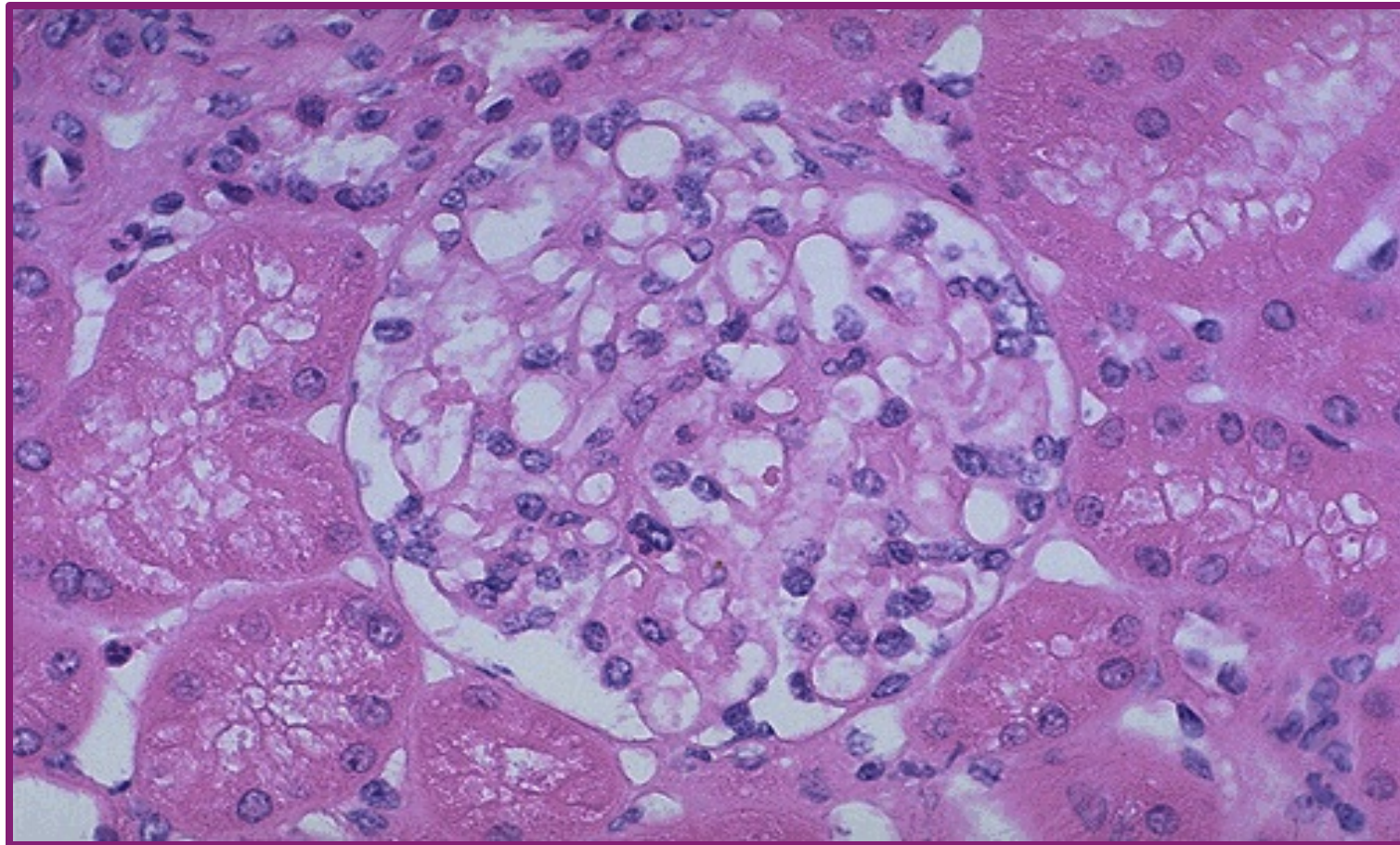
***In cross section, this normal adult kidney demonstrates the lighter outer cortex and the darker medulla, with the renal pyramids into which the collecting ducts coalesce and drain into the calyces and central pelvis.***

# Renal Corpuscle – Normal Histology





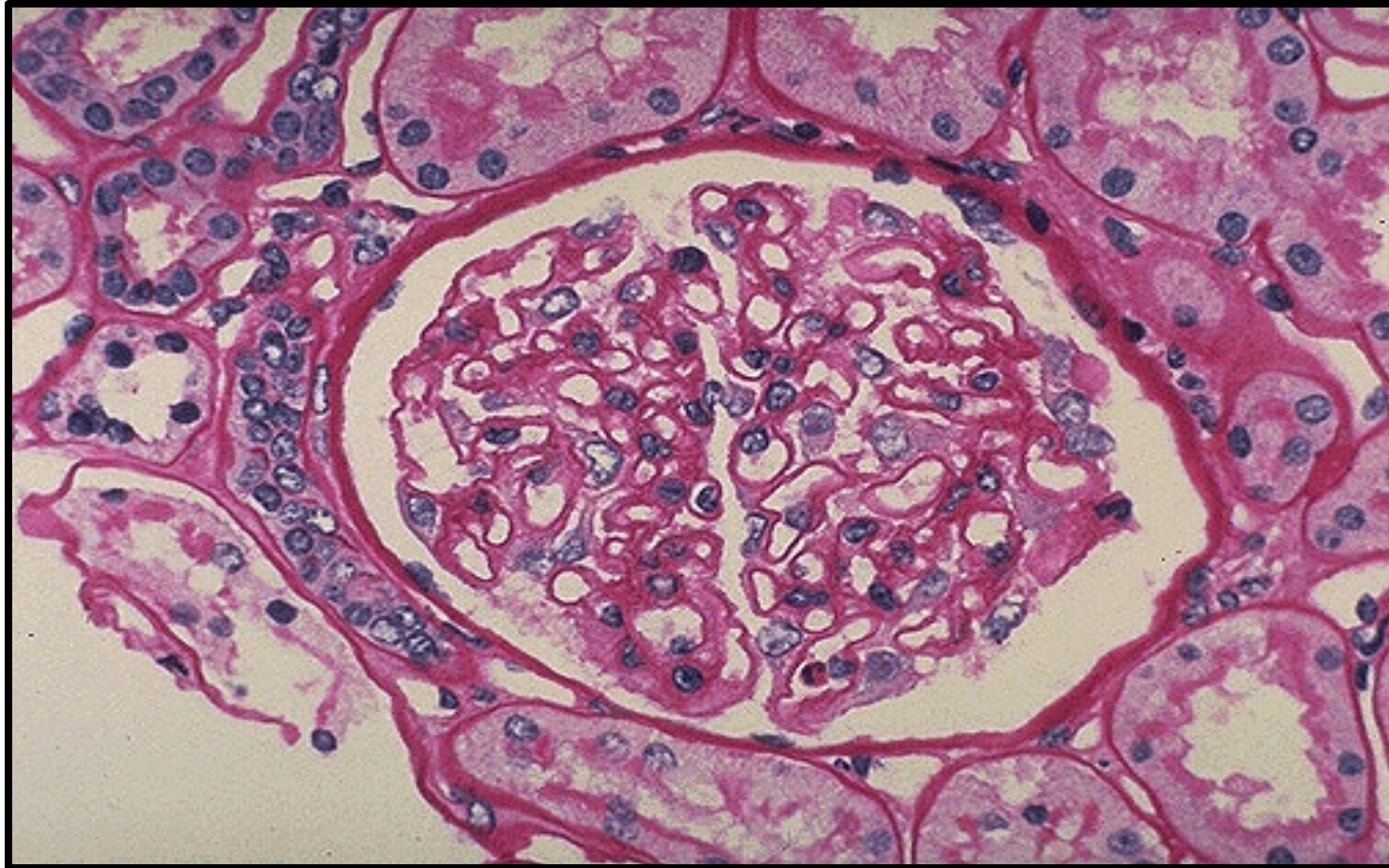
## **Renal Corpuscle – Normal Histology**



**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**

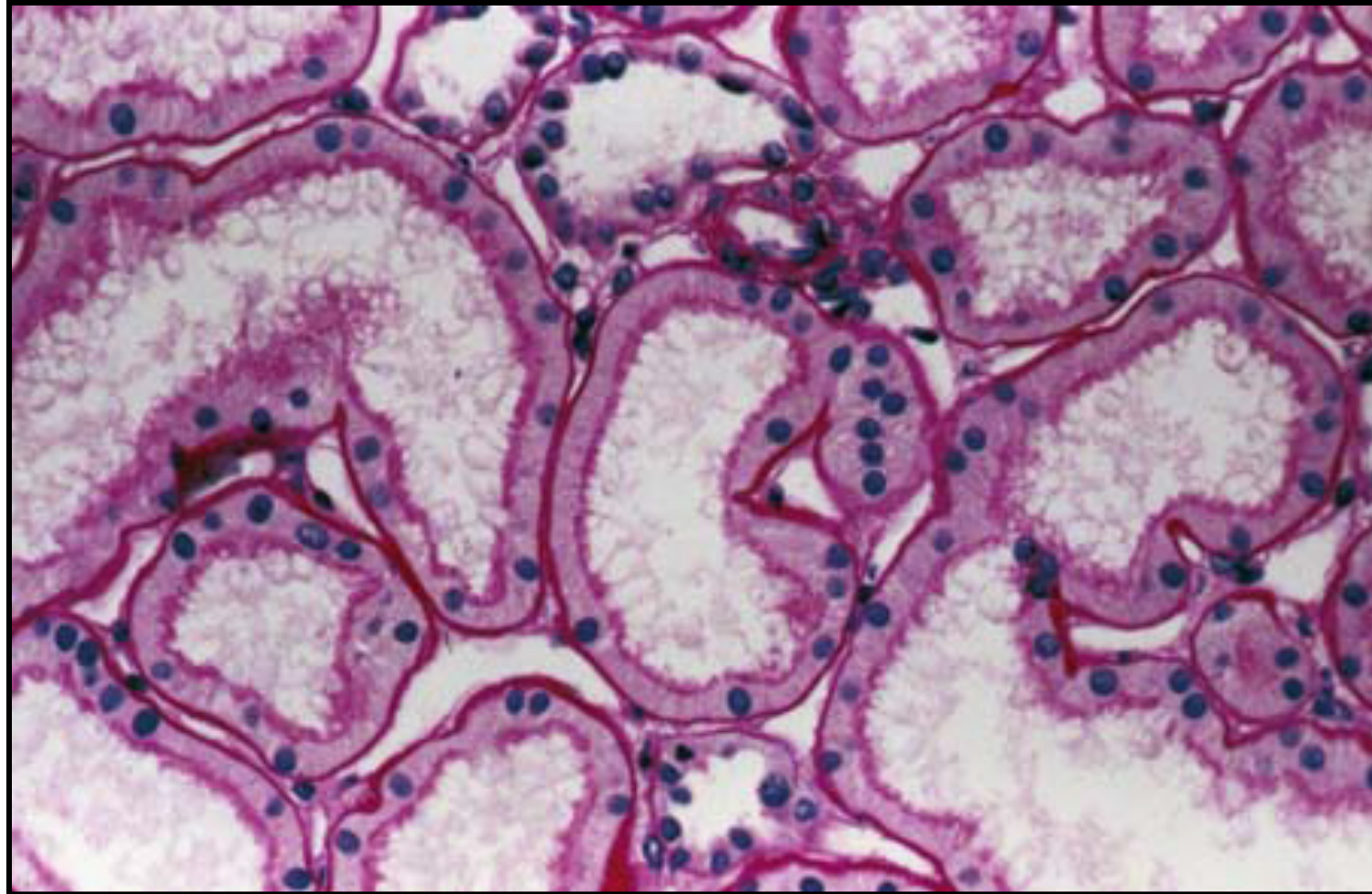


## Renal Corpuscle – Normal Histology



***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.*

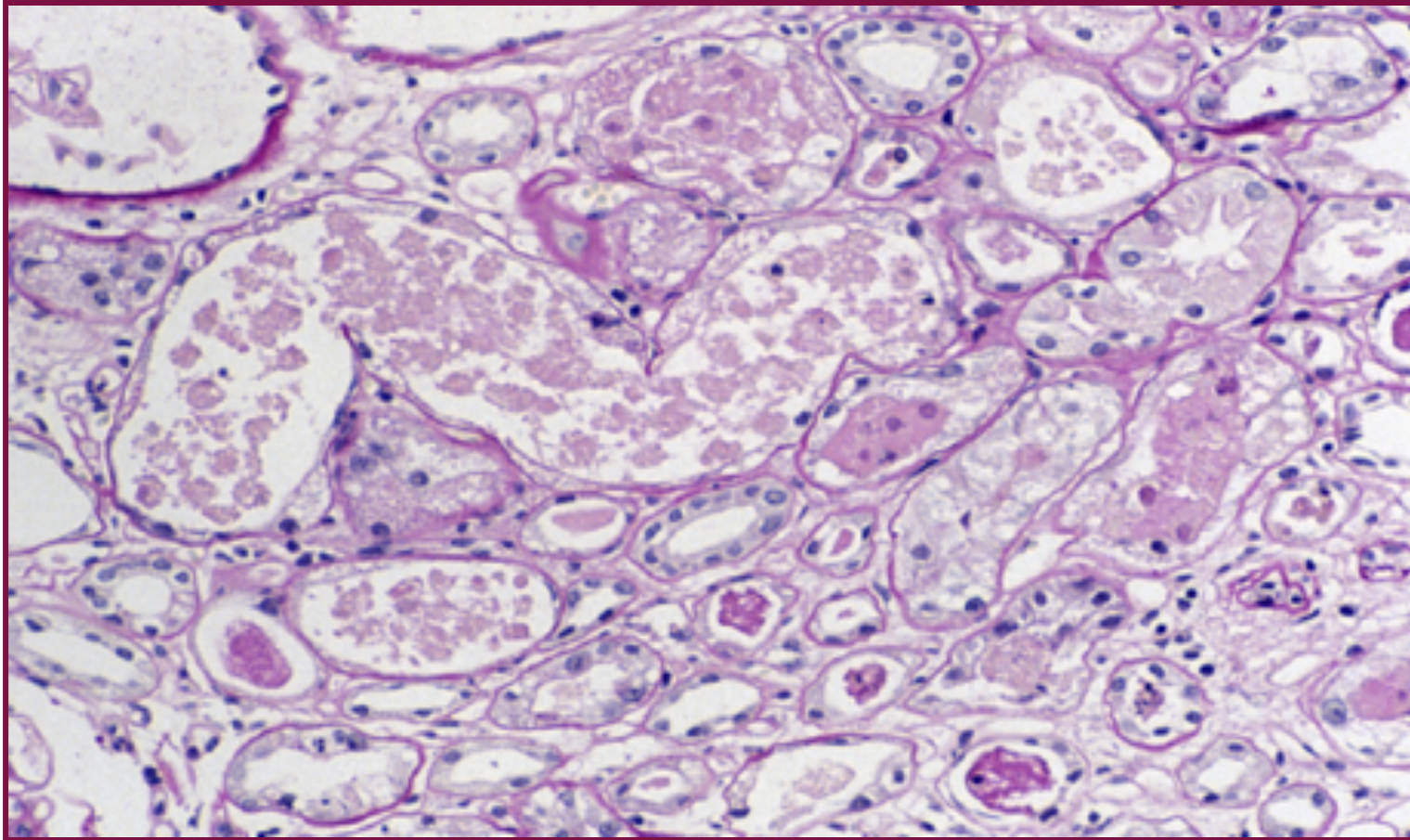
## Acute Kidney Injury



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

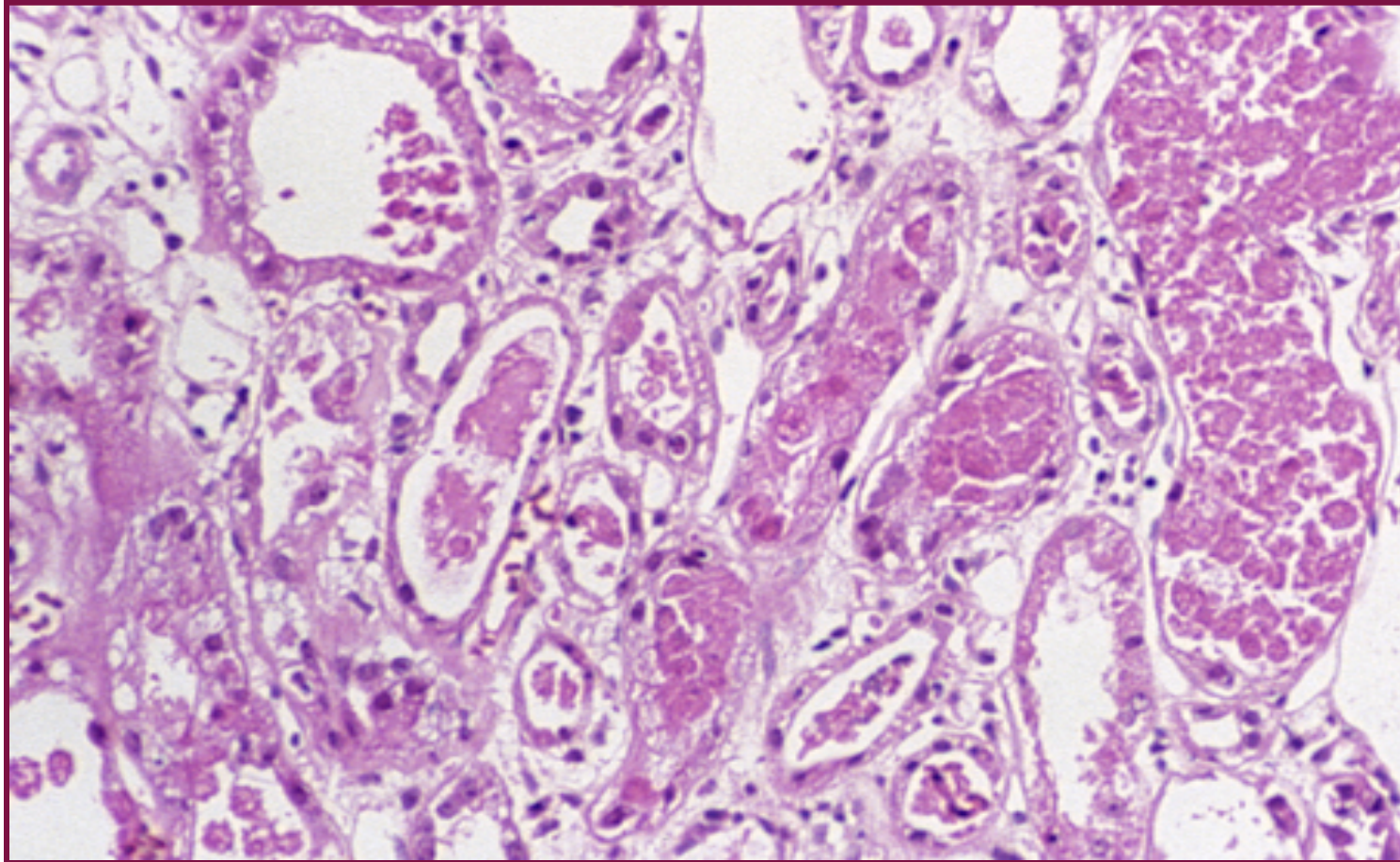


## Acute Tubular Necrosis



***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***

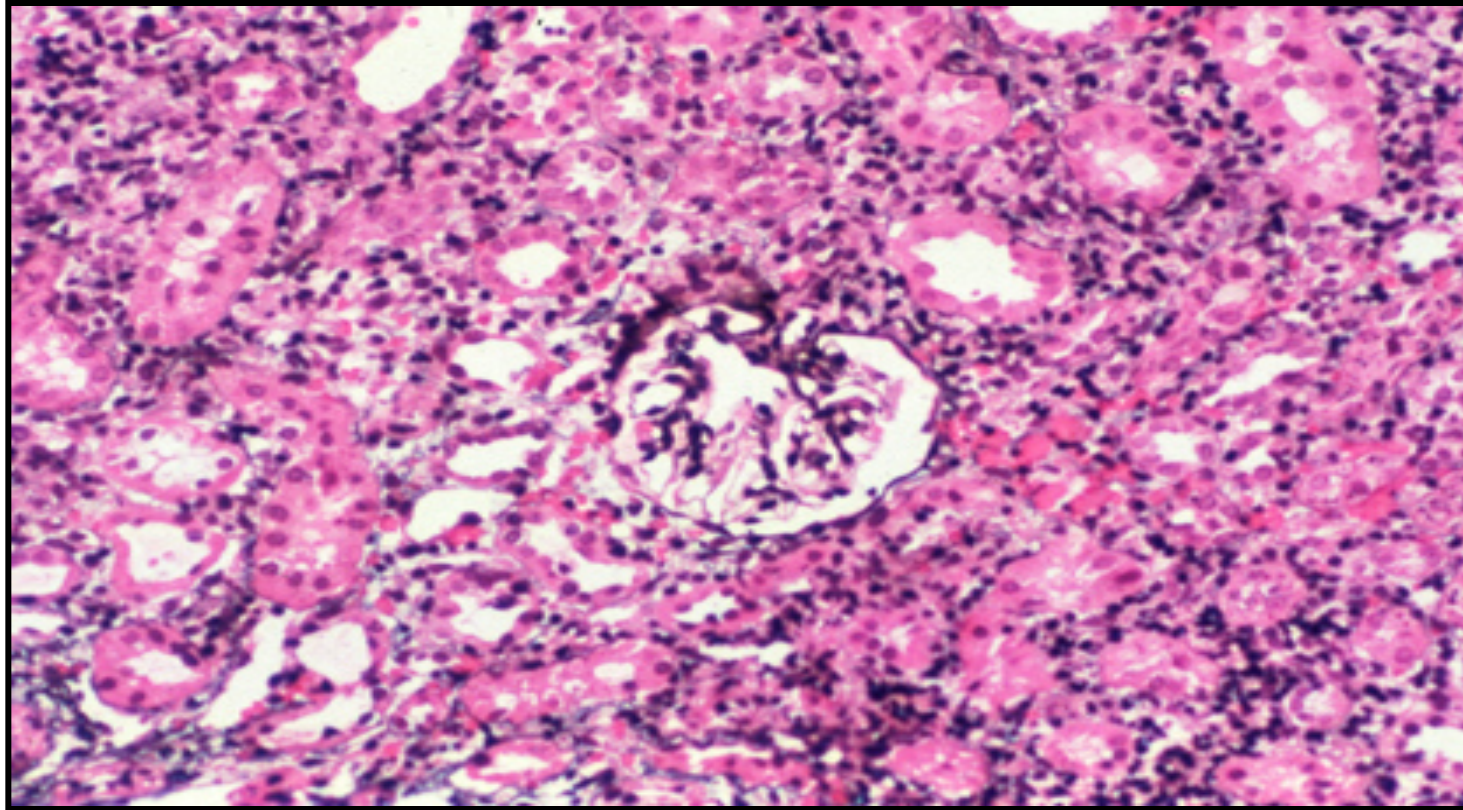
## Acute Tubular Necrosis



*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)*

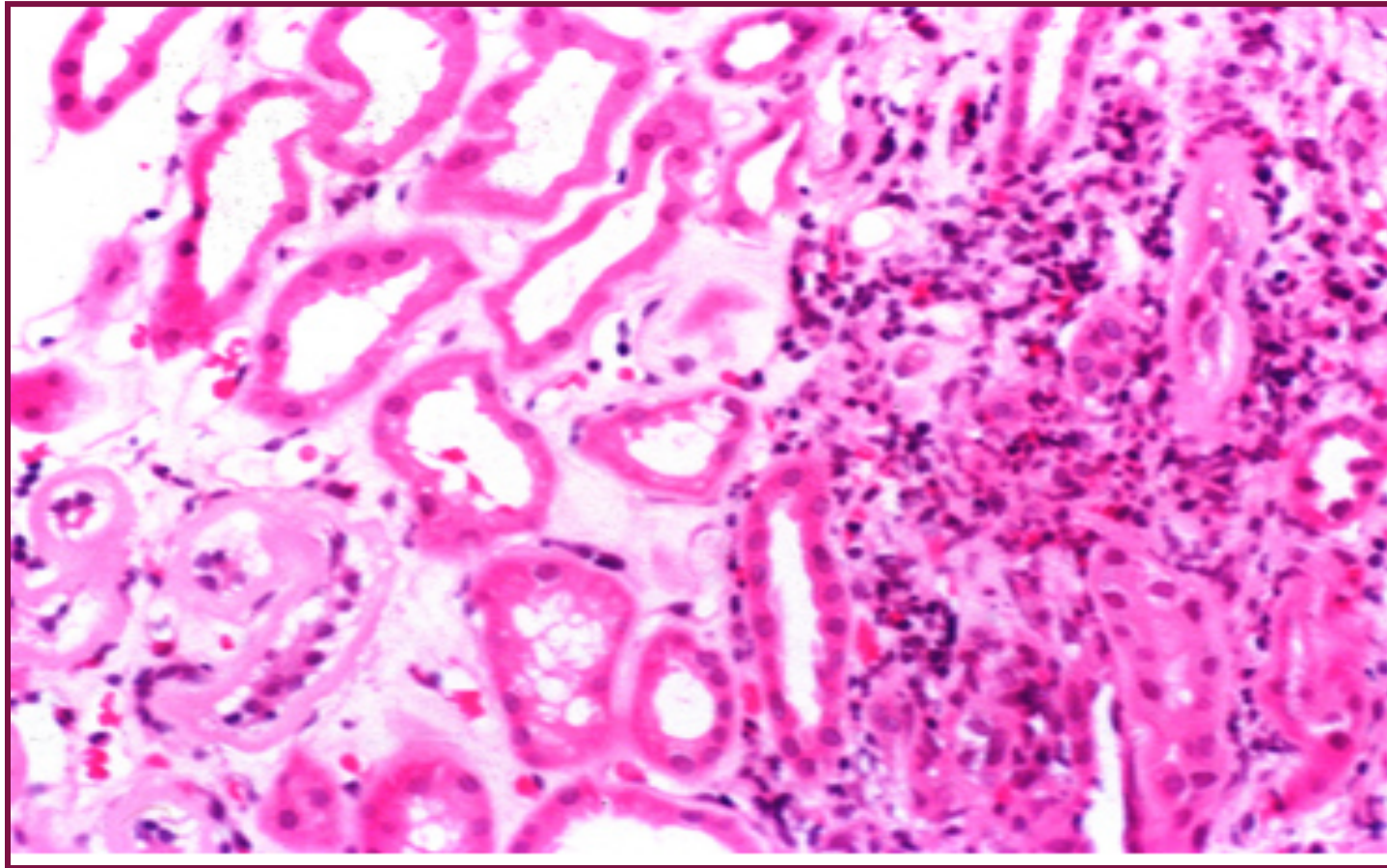


## Acute Interstitial Nephritis



*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*

# ***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***

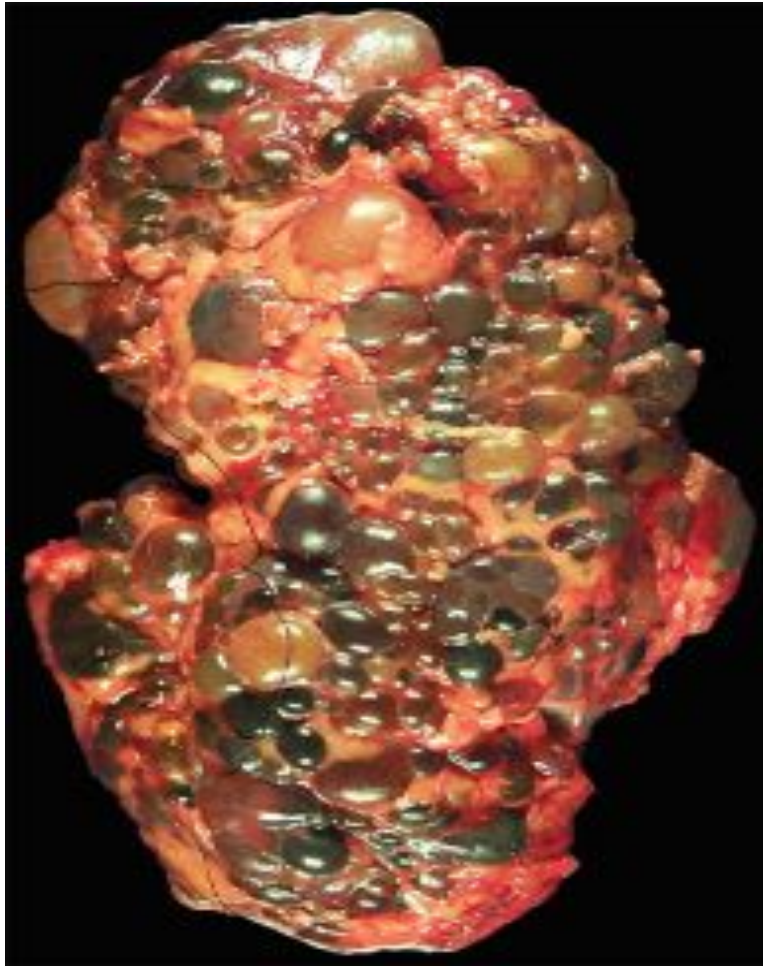
## *Polycystic kidney – Gross Anatomy*



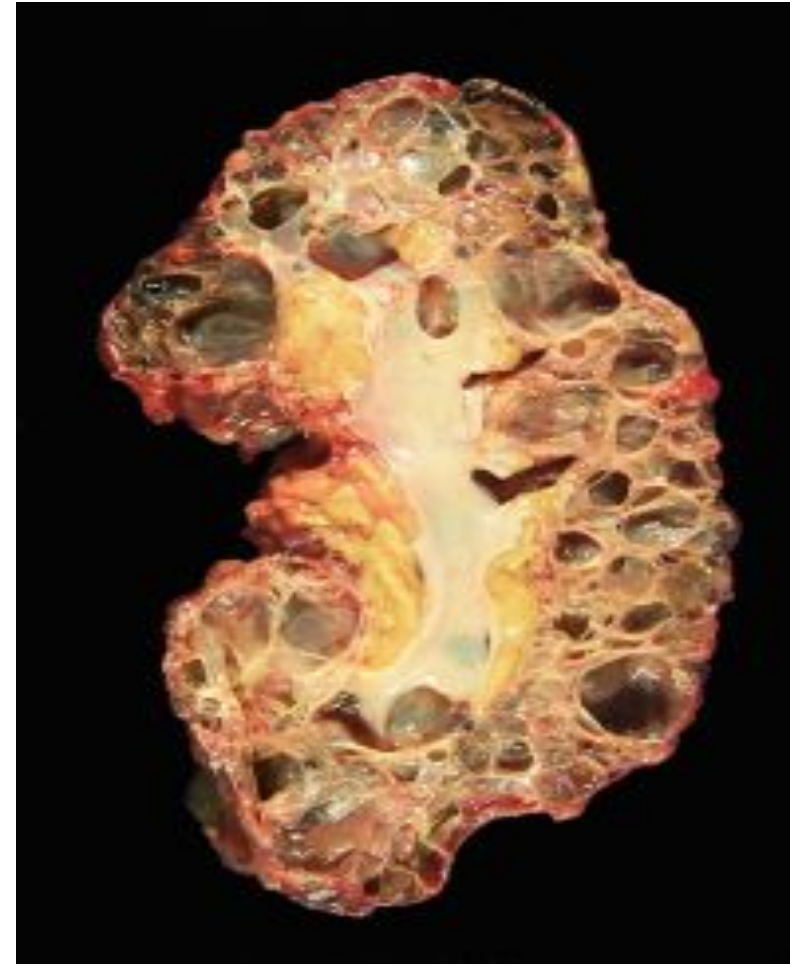
***Bilateral autosomal dominant polycystic kidney disease***



## *Gross Polycystic kidney and its Cut Section*

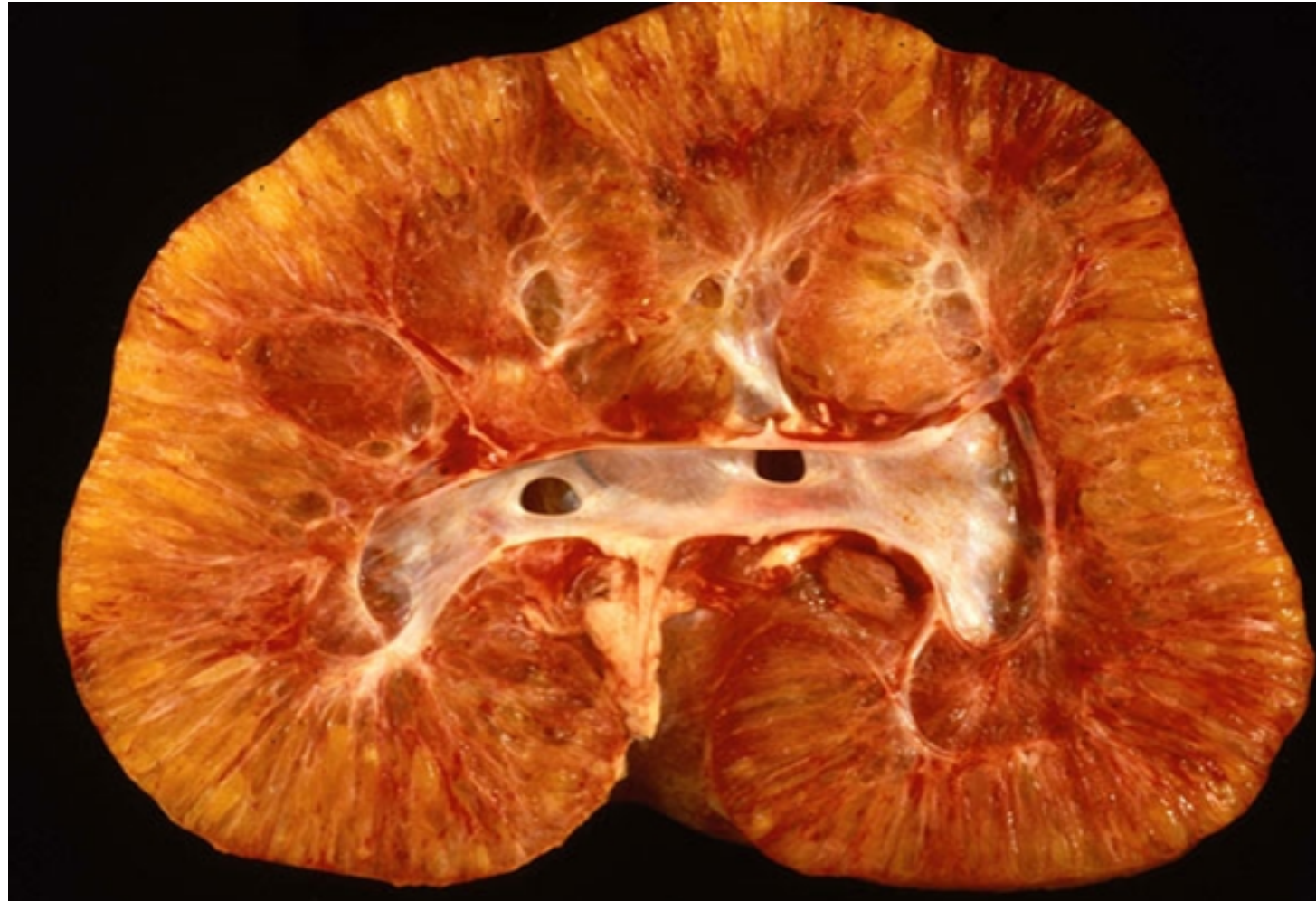


***Massively enlarged kidney disrupted by numerous cysts***



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

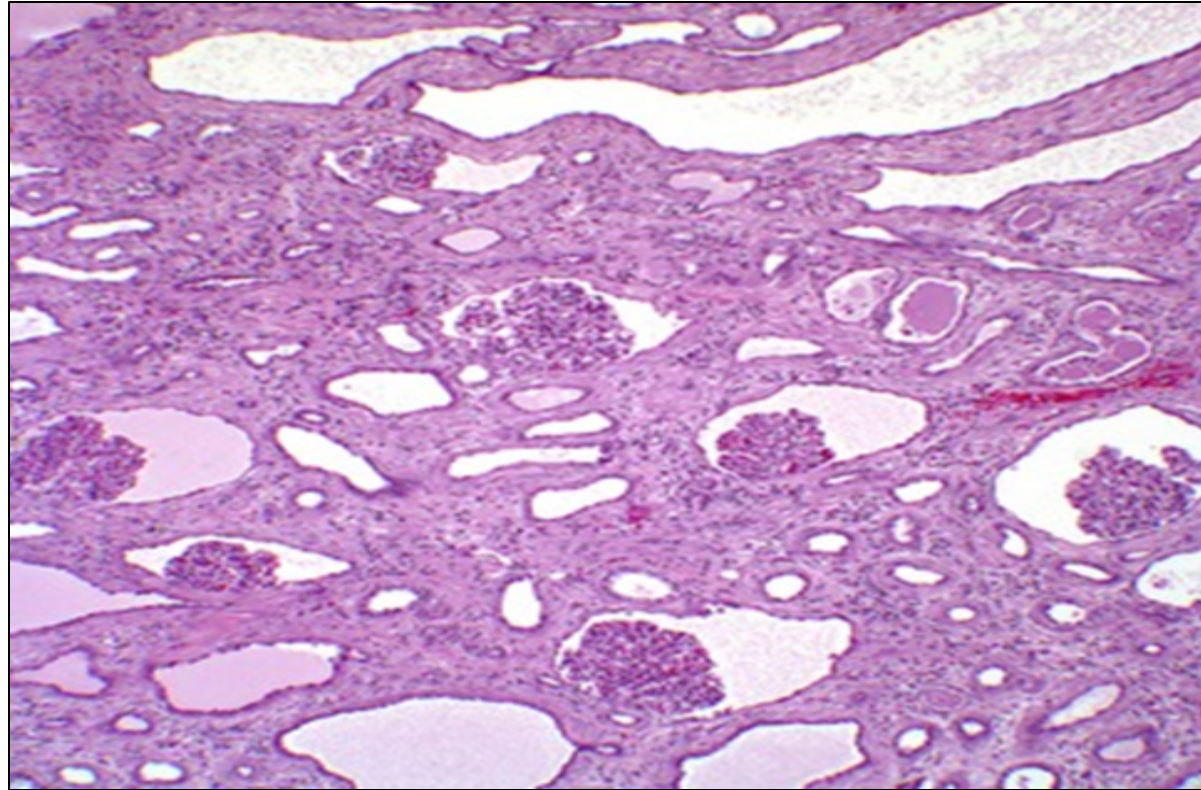
## *Infantile Polycystic kidney – Gross*



***Coronal section of an infantile polycystic kidney***

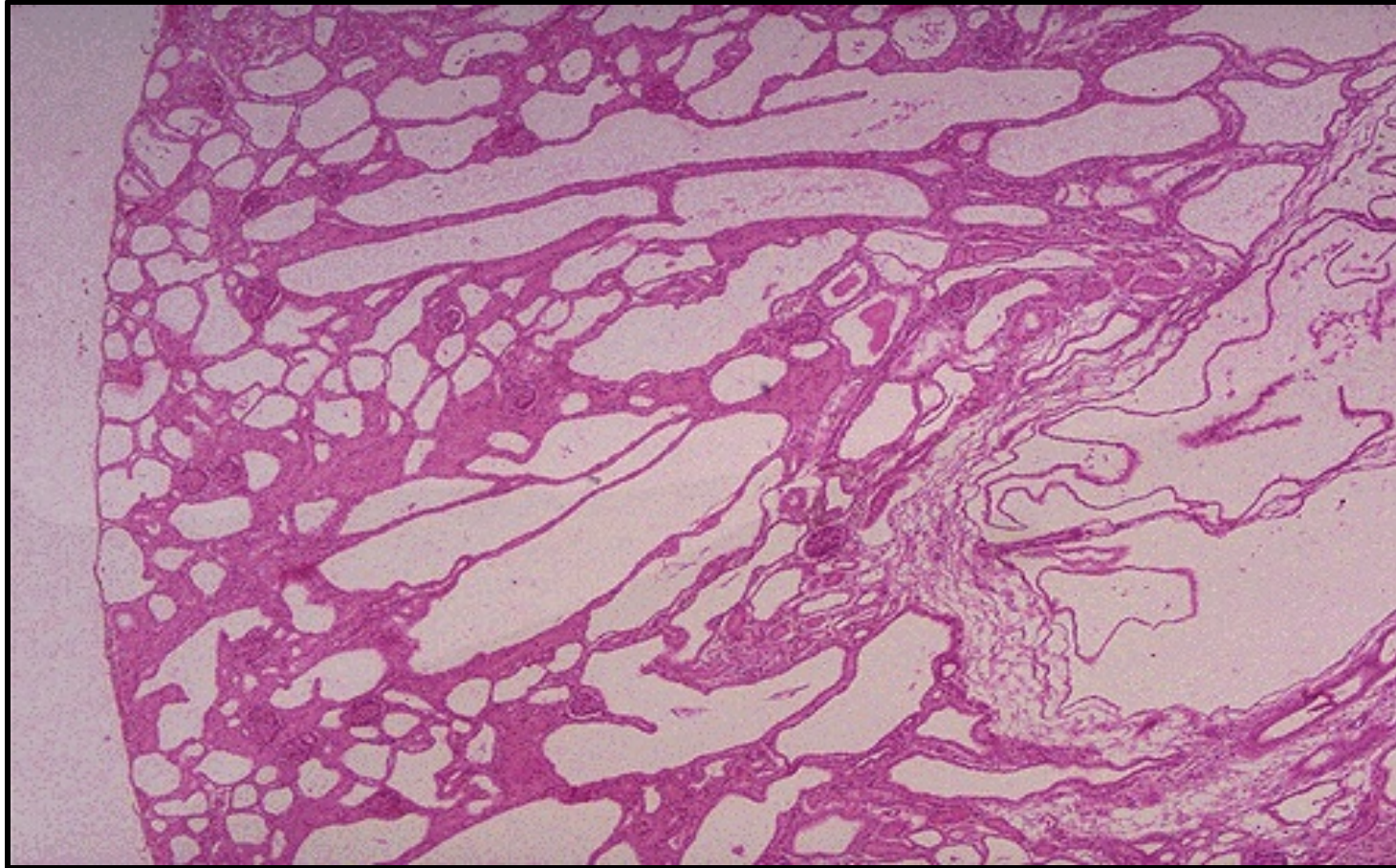


## *Polycystic kidney – Histopathology*



***Kidney of child with autosomal dominant PCKD.  
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.***

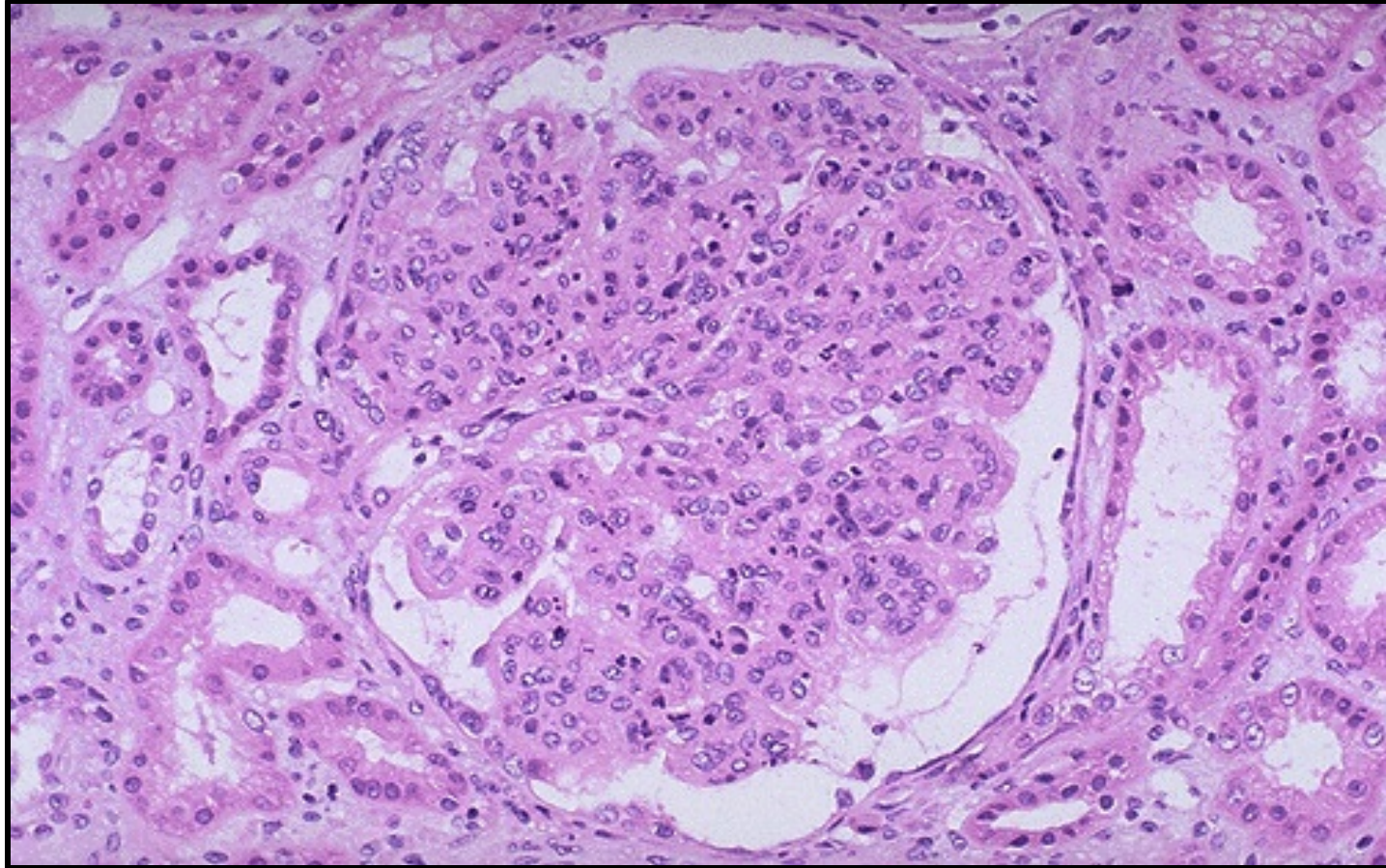
## ***PRACTICAL SESSION : 2***

# ***INFECTION OF THE URINARY TRACT***



# ***ACUTE (POST-STREPTOCOCCAL) GLOMERULONEPHRITIS***

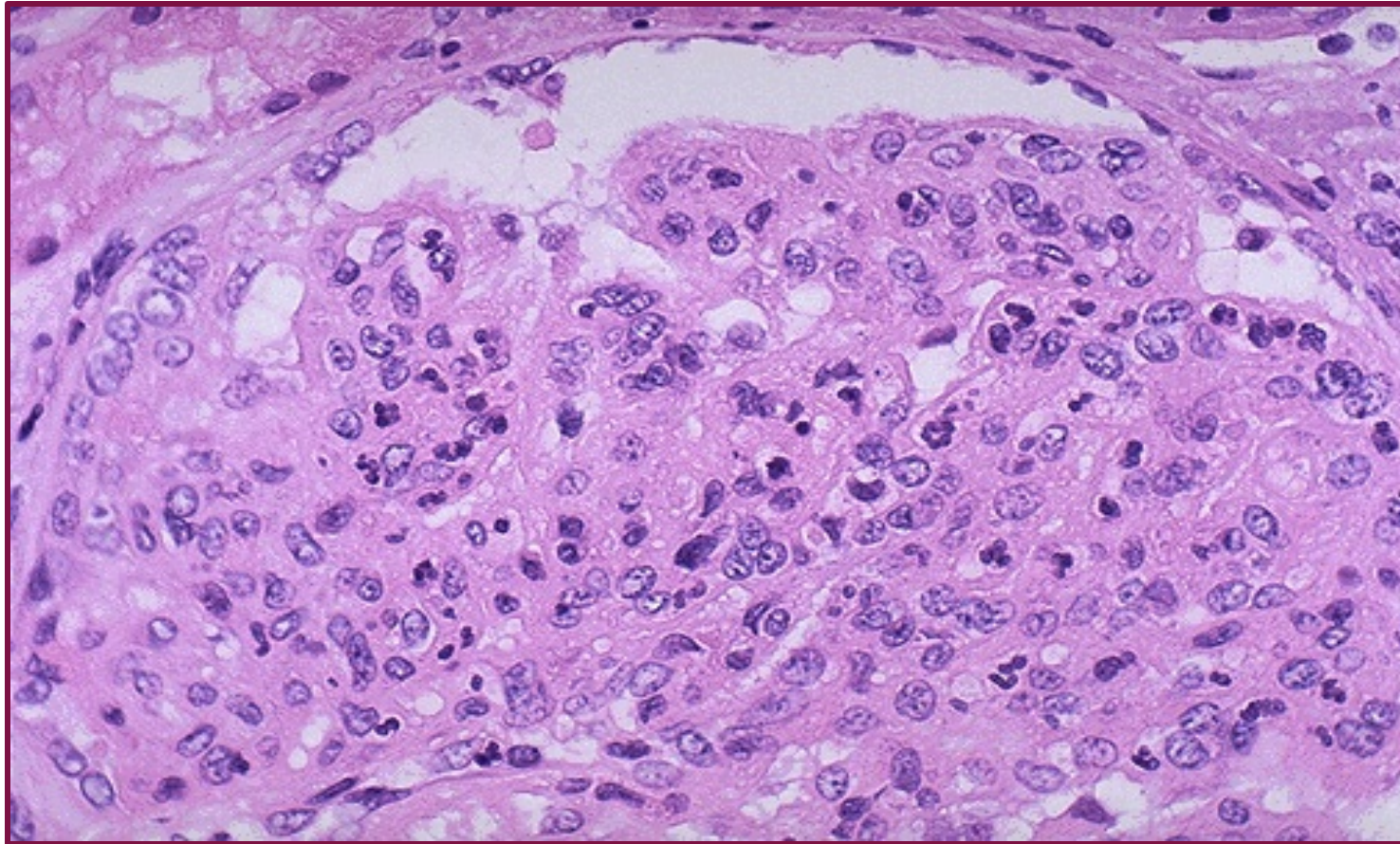
## ***Acute (Post-streptococcal) Glomerulonephritis***



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



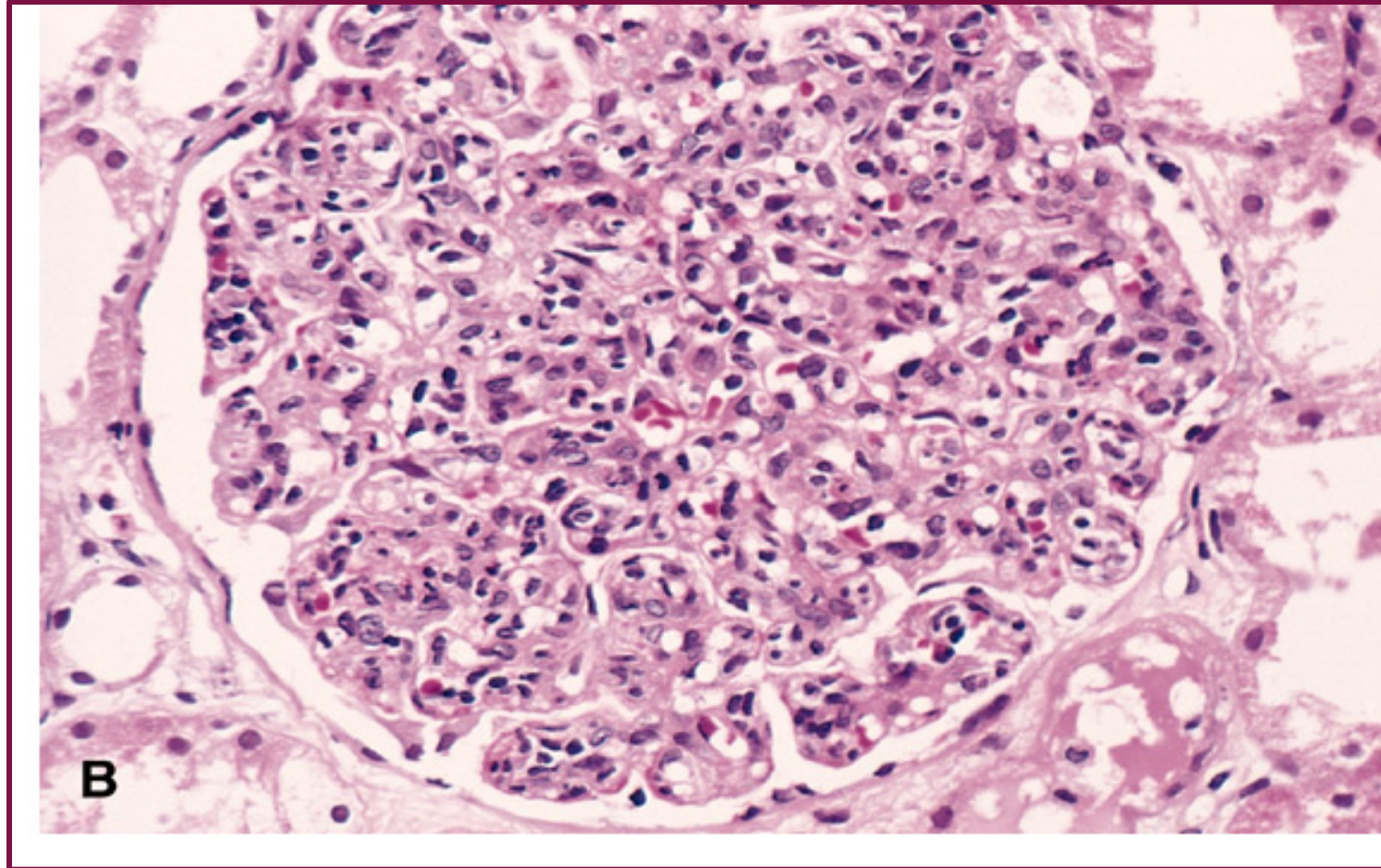
## Acute (Post-streptococcal) Glomerulonephritis



***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***

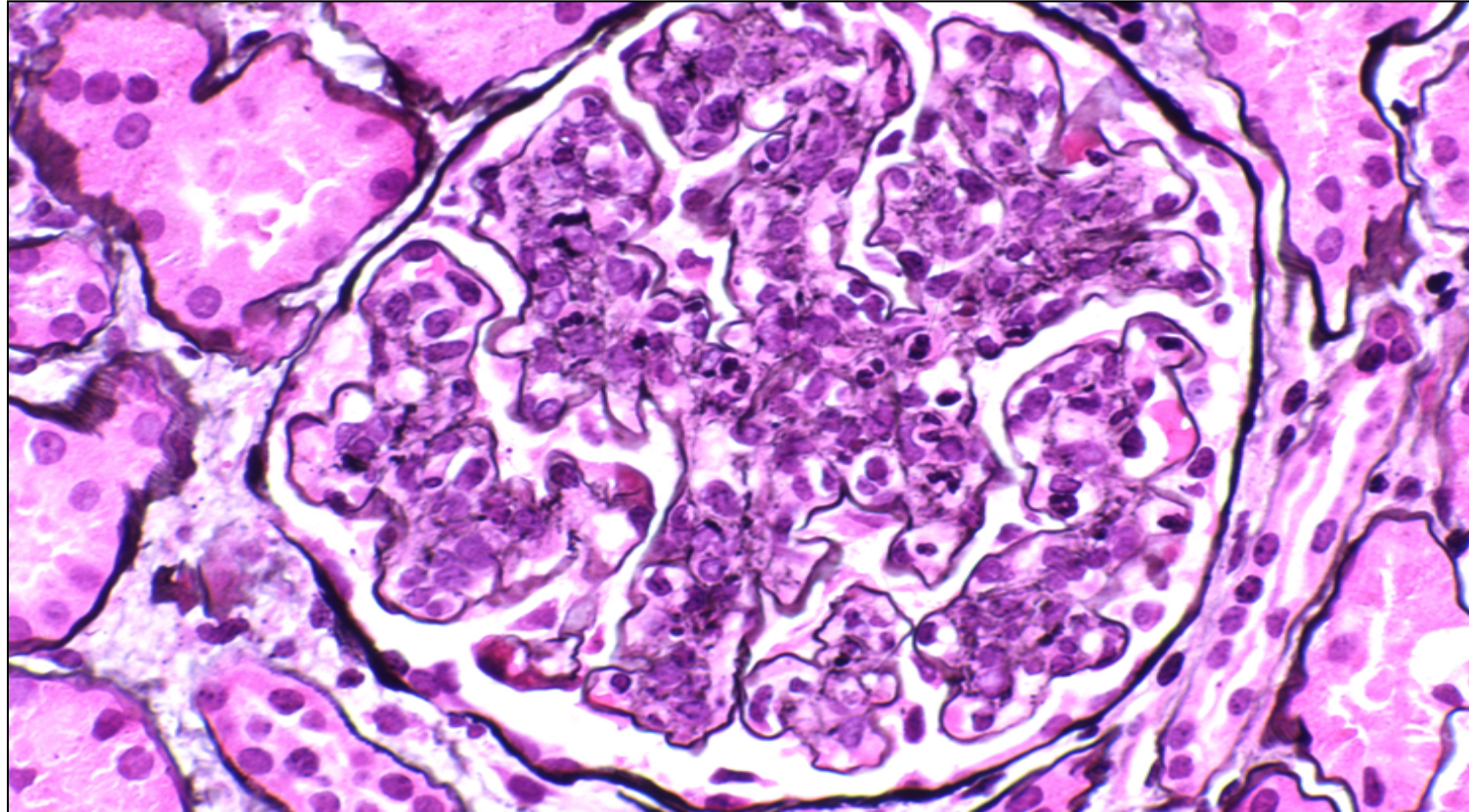


## Acute (Post-streptococcal) Glomerulonephritis



***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 (Post-streptococcal) Glomerulonephritis**

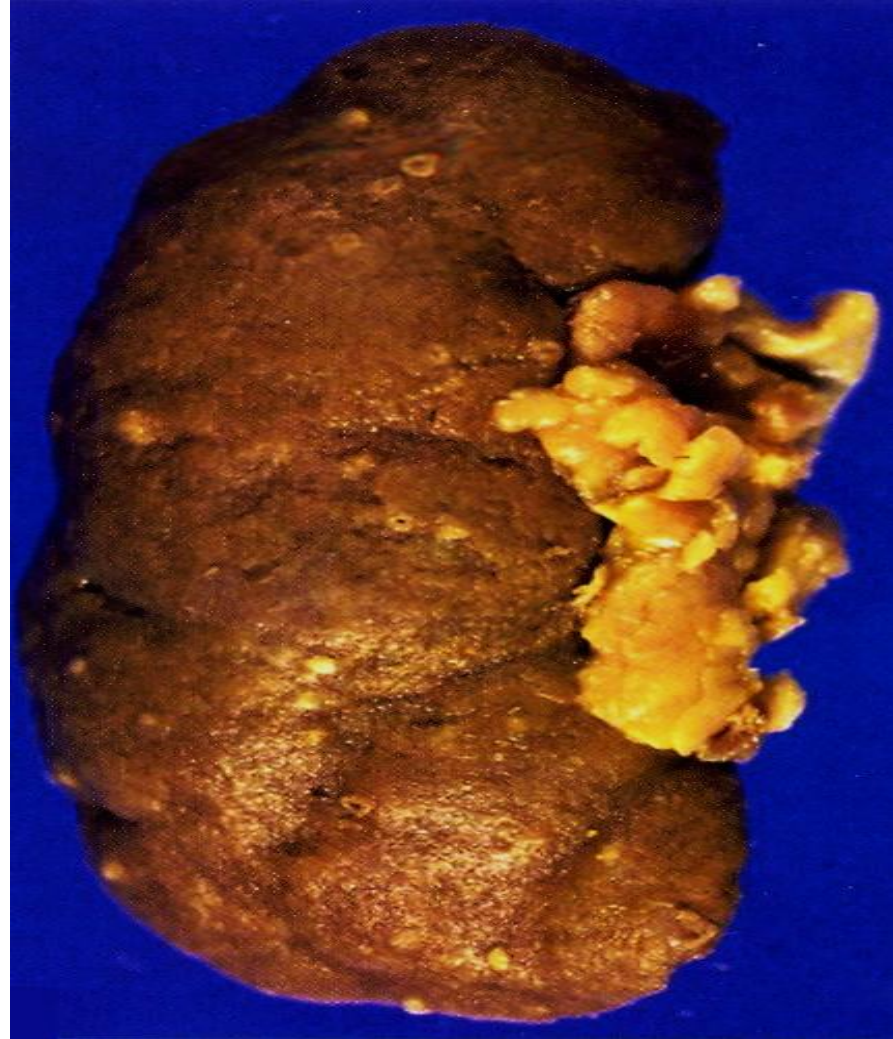
### **Section of the kidney shows:**

- **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 & CHRONIC PYELONEPHRITIS***

***Pyelonephritis with small cortical abscesses***



***Pyelonephritis with small cortical abscesses***

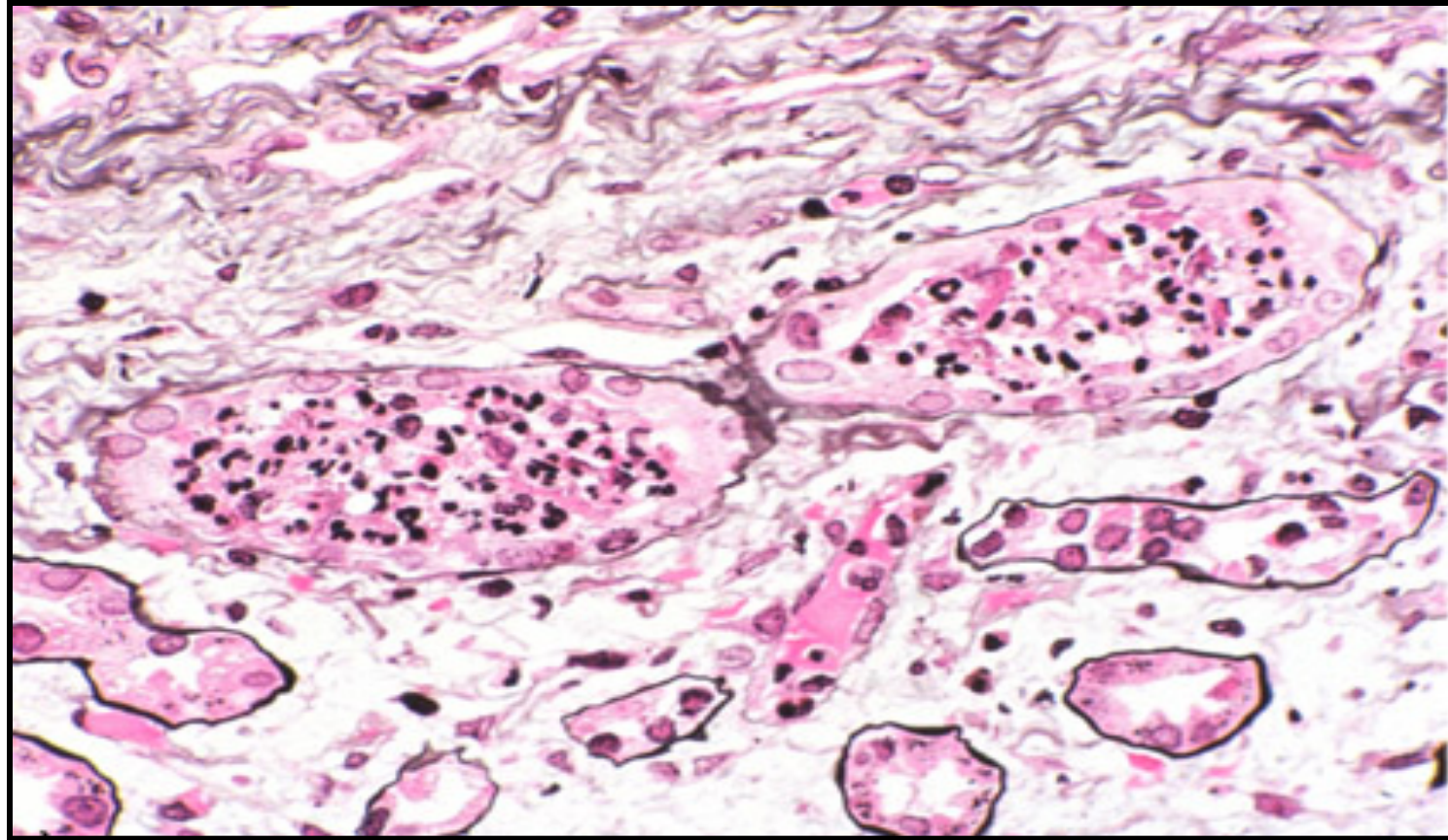
## Classic picture of Pyelonephritis



***This kidney is bisected to reveal a dilated pelvis and calyces 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.***



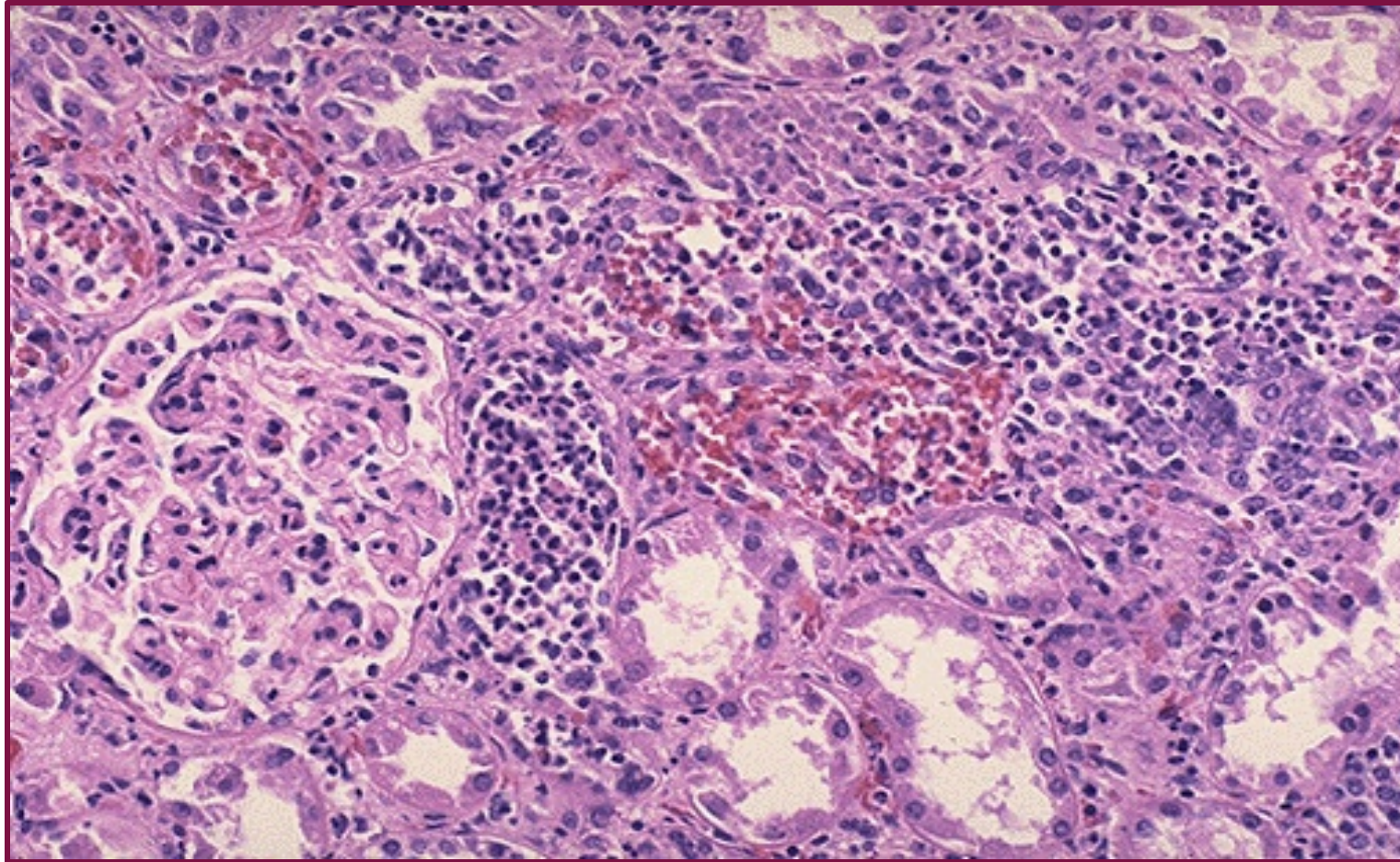
## Acute Pyelonephritis - Histopathology



***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***

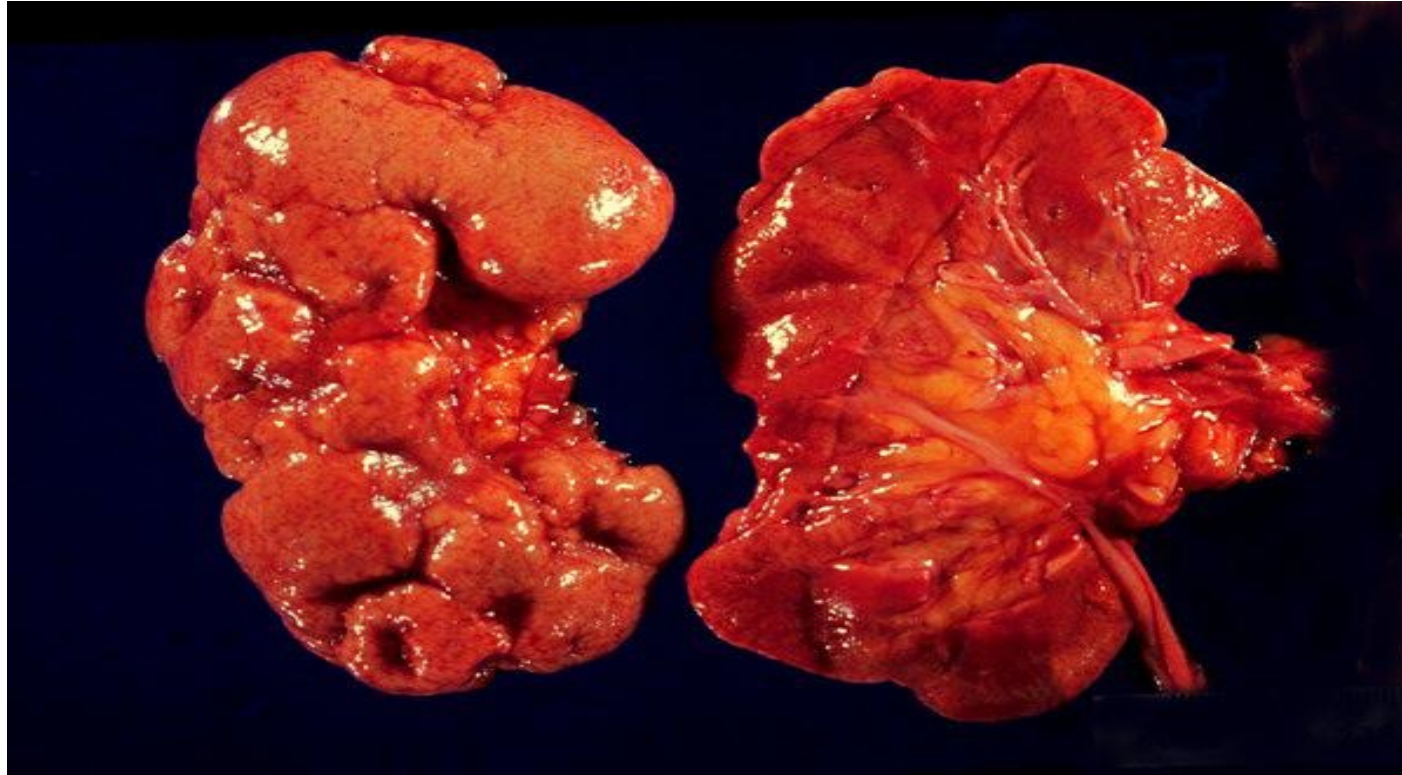


## Acute Pyelonephritis - Histopathology



***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***

## *Renal failure secondary to chronic pyelonephritis*



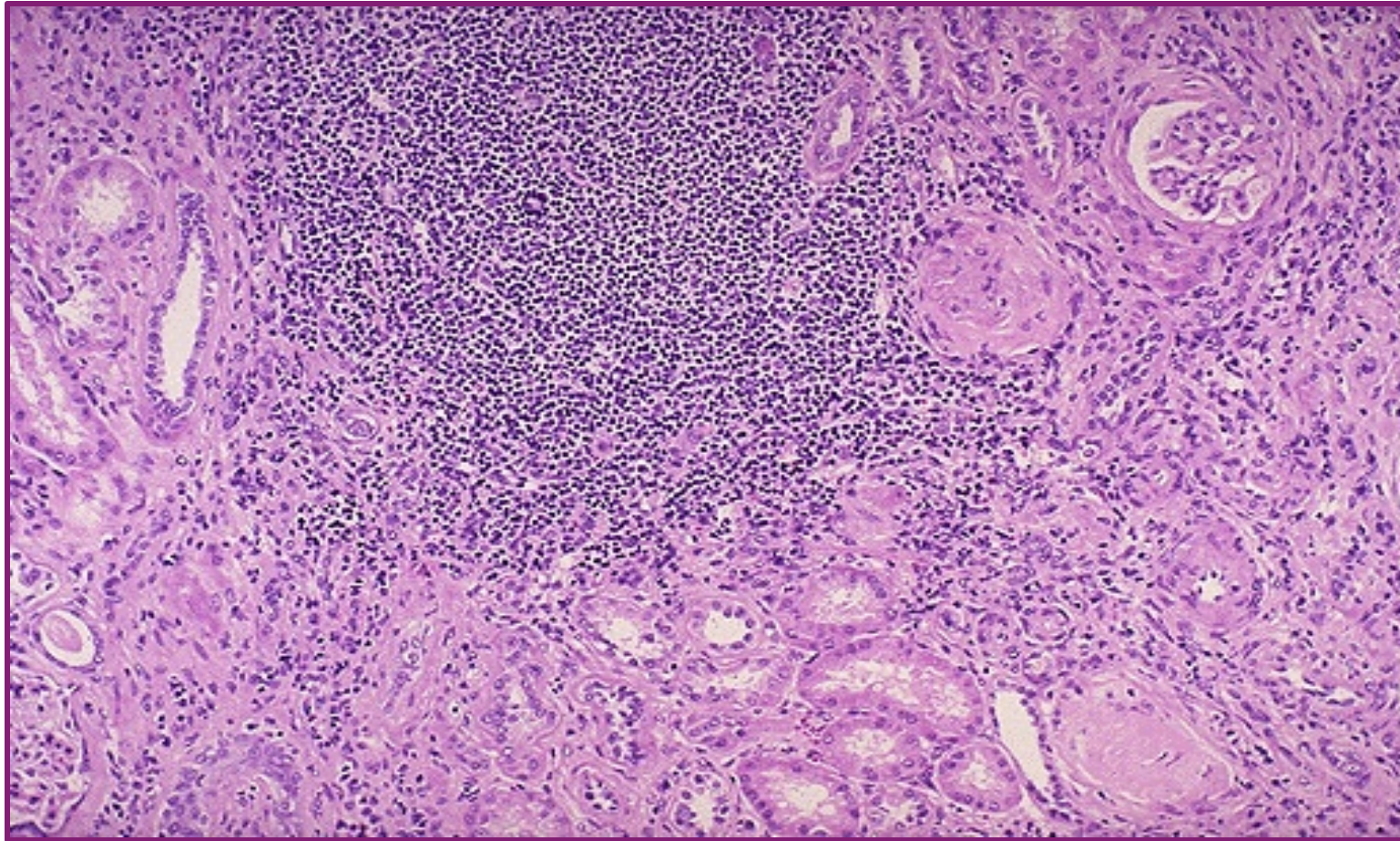
### **Gross pathological hallmark:**

- *Uneven cortical scarring.*
- *Scarring of the pelvis and calyces.*
- *Papillary blunting.*

**Most common cause of this condition in children - *Reflux nephropathy***



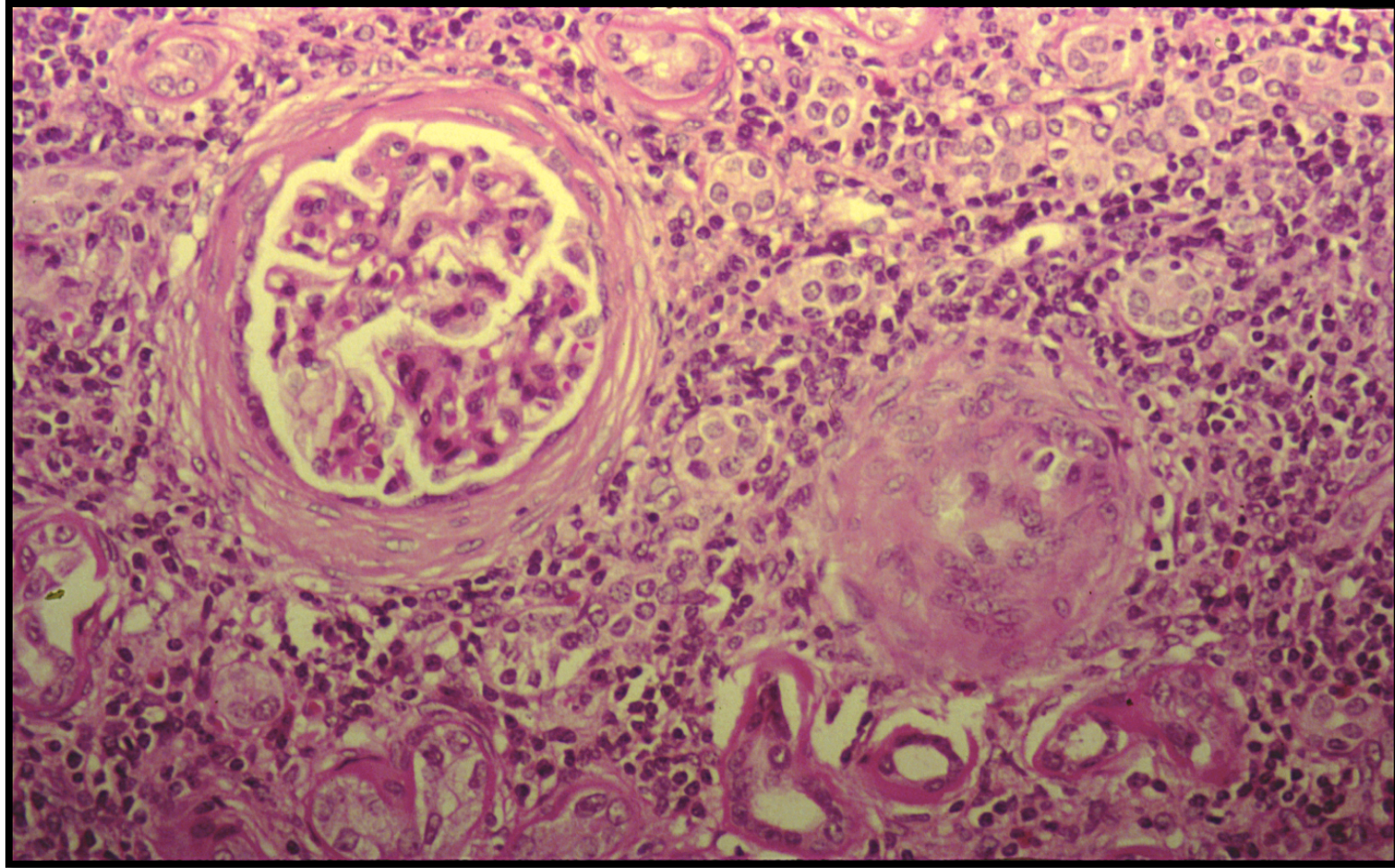
## Chronic Pyelonephritis - Histopathology



- ***This is chronic pyelonephritis where a large collection of chronic inflammatory cells .***
- ***When eosinophilic infiltration is present in the interstitium, the most likely cause of nephritis in such cases - Drug induced interstitial nephritis.***



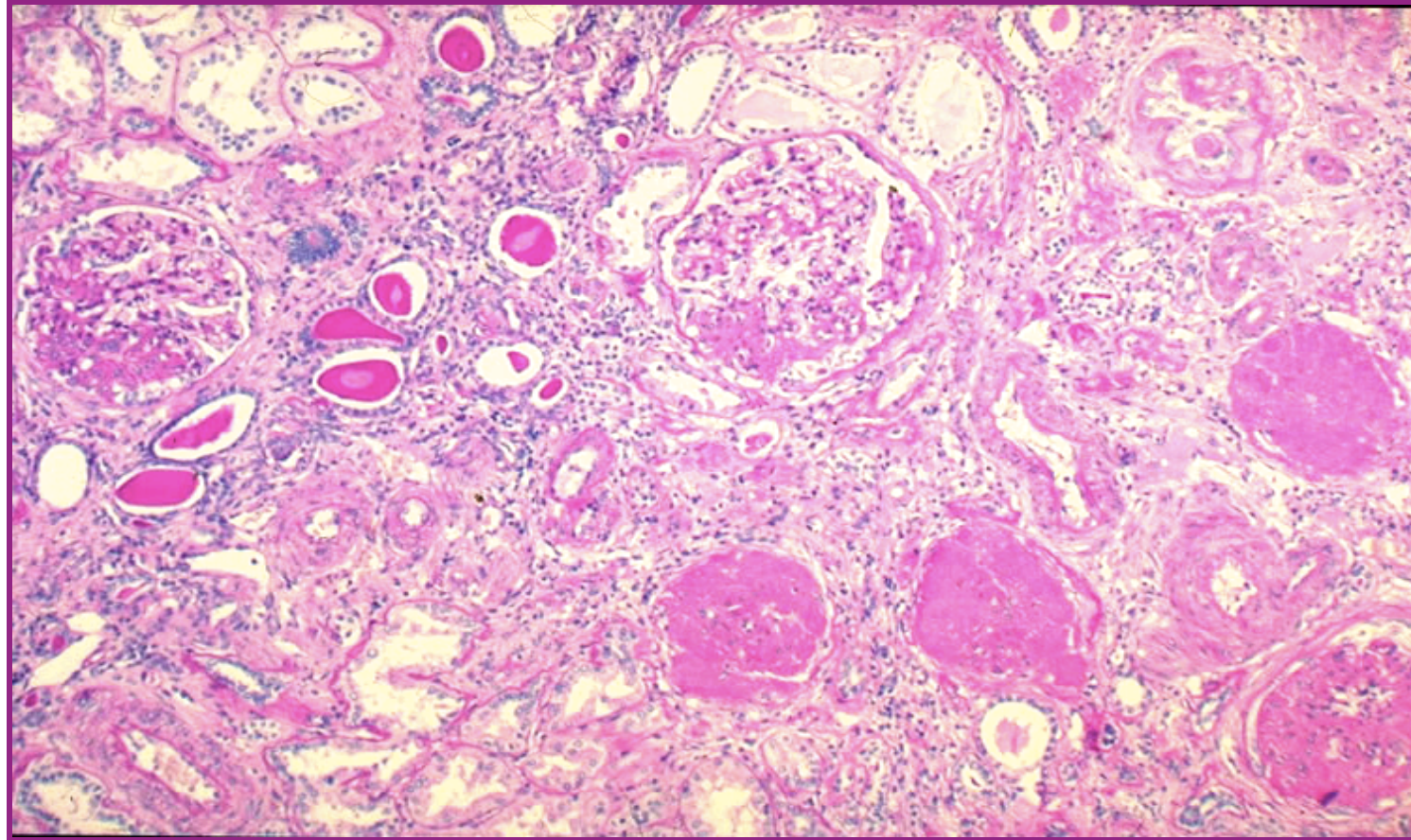
## Chronic Pyelonephritis - Histopathology



- a- Peri-glomerular fibrosis.**
- b- Interstitial inflammation.**
- c- Hyalinized /fibrotic glomeruli.**



## Chronic Pyelonephritis - Histopathology

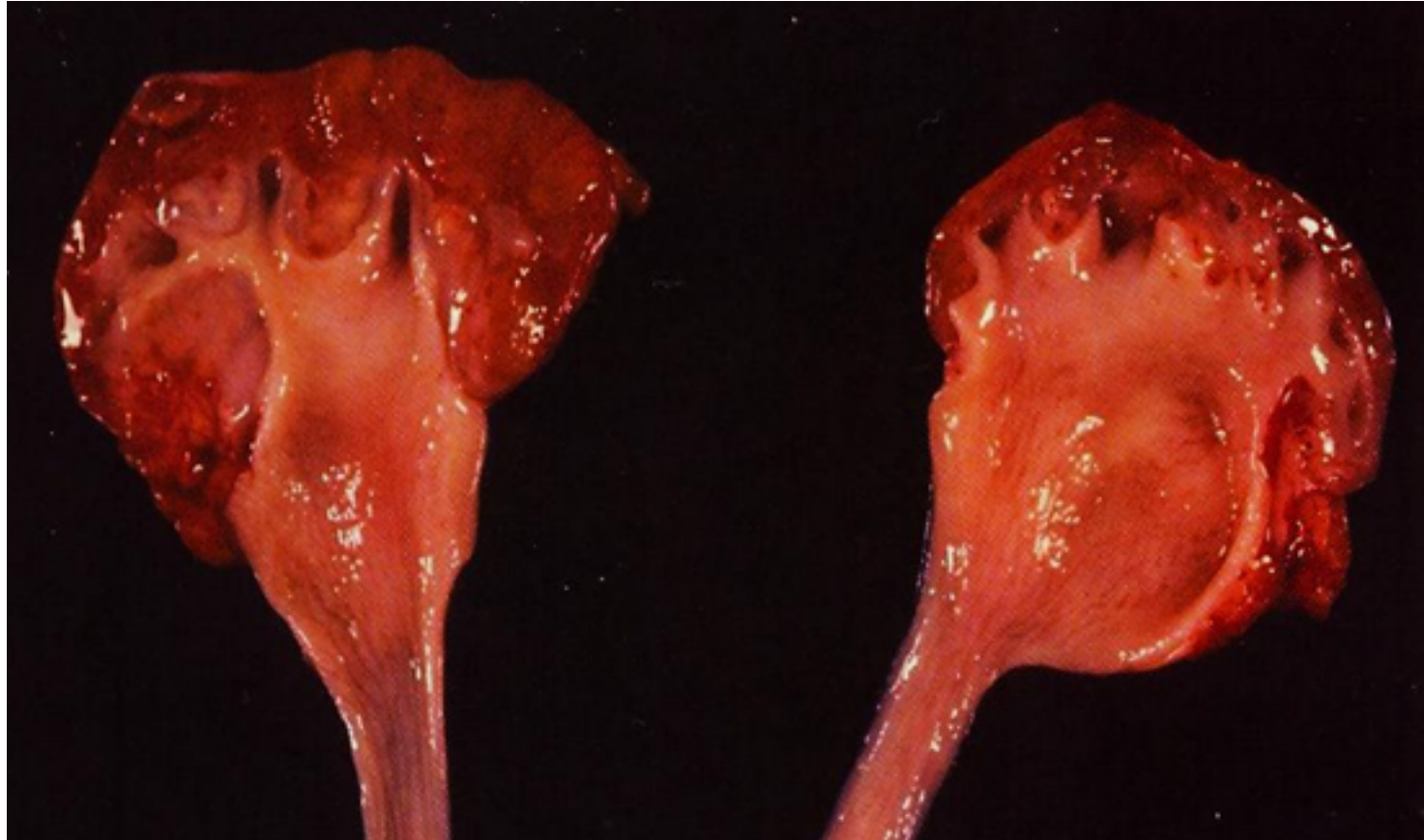


- **Glomeruli** show varying degrees of sclerosis & periglomerular fibrosis.
- **Tubules** show varying degrees of atrophy, Some tubules are dilated and filled with Eosinophilic hyaline casts resembling colloid (thyroidization).
- **Interstitial tissue** shows chronic inflammatory cells infiltrate and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



# ***HYDRONEPHROSIS***

## Hydronephrosis



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

## Hydronephrosis



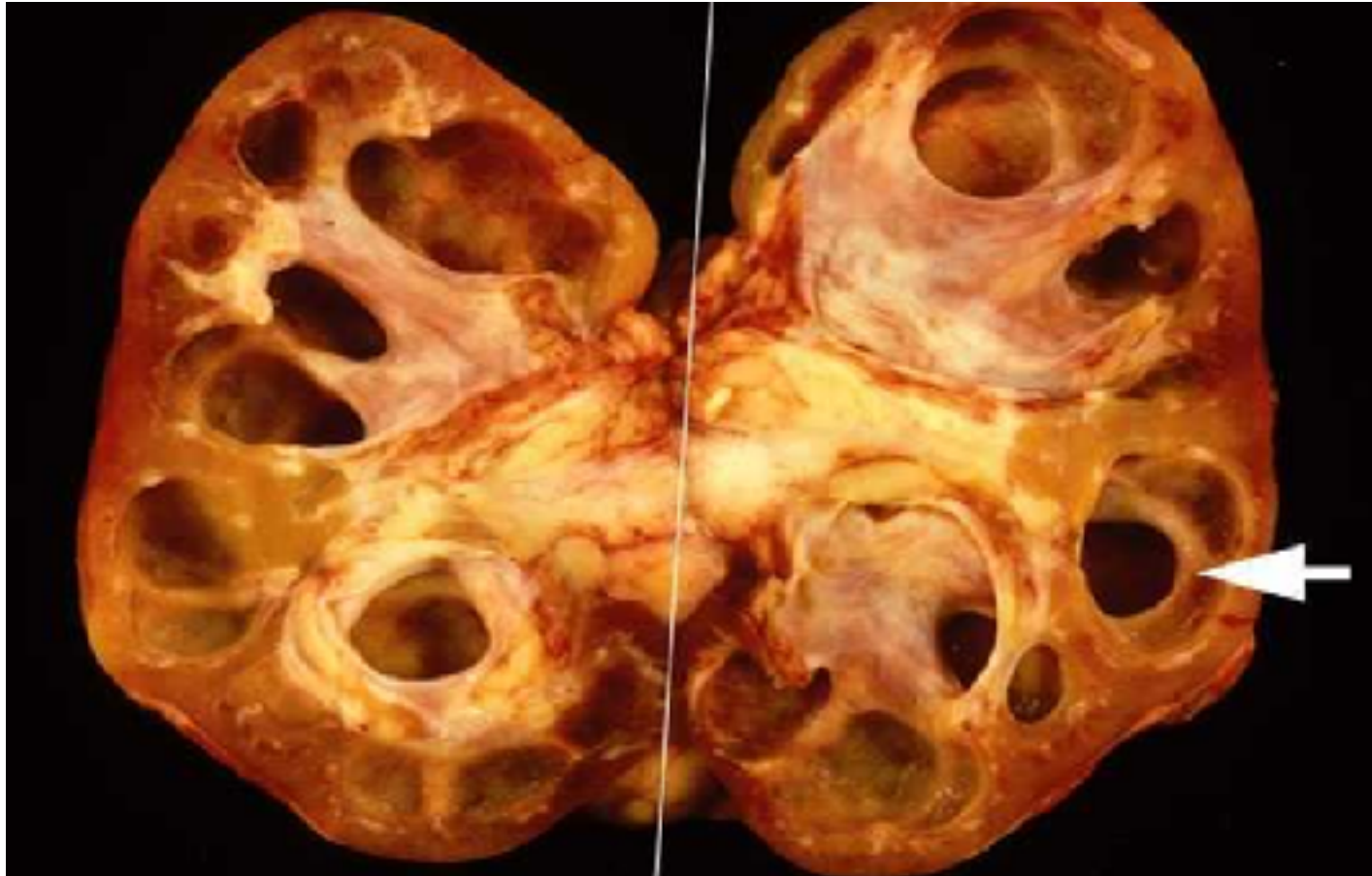
### **The most common causes are:**

- 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**

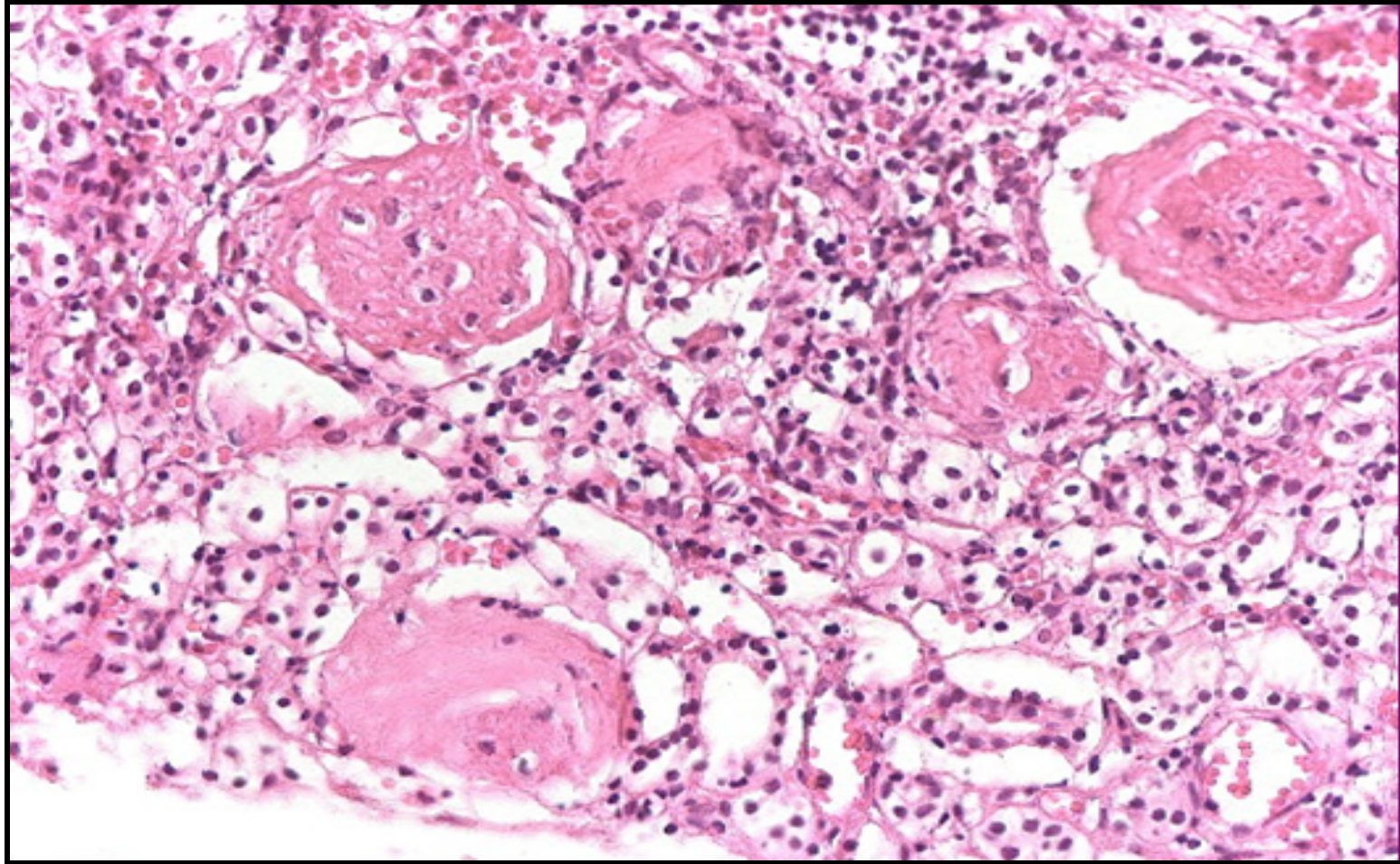


## Hydronephrosis



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

***Chronic Pyelonephritis presenting as  
complication to Hydronephrosis***



***Sclerosis of glomeruli with atrophic tubules***