



Pathology Practical  
Renal Block  
Med435

Color Coding:  
Very important: RED  
Males Doctor: Blue  
Females Doctor: Purple

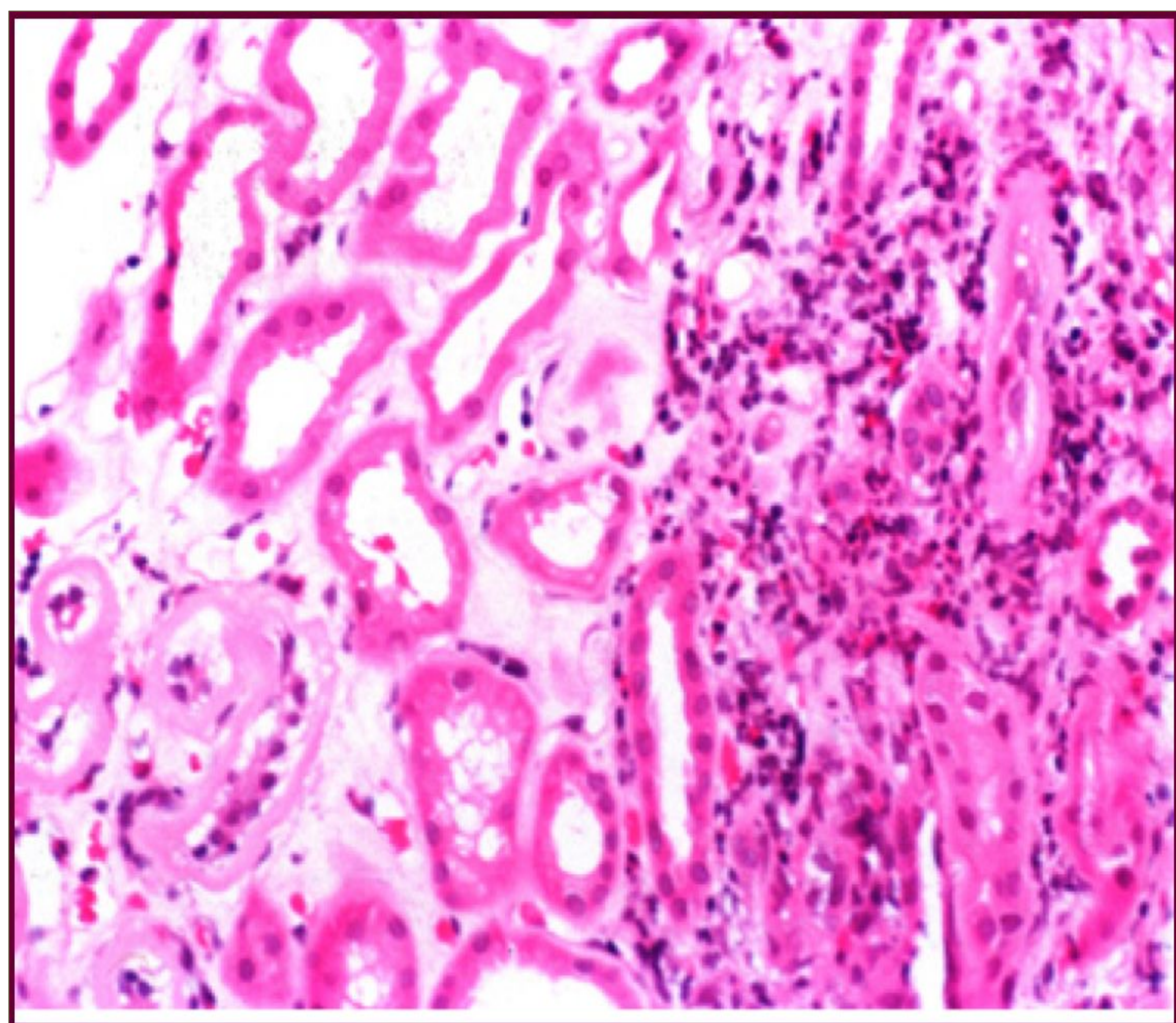
# #1 ACUTE KIDNEY INJURY

Theoretical information: \* YOU NEED TO KNOW 2 CAUSES IT LEAST\*.

Pre-renal	Renal	Post-renal
<ul style="list-style-type: none"> <li>- Low blood volume, low blood pressure, and heart failure.</li> <li>- Renal artery stenosis, and renal vein thrombosis.</li> <li>- Renal ischemia.</li> </ul>	<ul style="list-style-type: none"> <li>- Glomerulonephritis (GN).</li> <li>- Acute tubular necrosis (ATN).</li> <li>- Acute interstitial nephritis (AIN).</li> </ul> <p>four elements:</p> <ol style="list-style-type: none"> <li>1. Glomeruli.</li> <li>2. Tubules.</li> <li>3. Blood Vessels.</li> <li>4. Interstitium</li> </ol>	<ul style="list-style-type: none"> <li>- Benign prostatic hyperplasia.</li> <li>- Kidney stones.</li> <li>- Obstructed urinary catheter.</li> <li>- Bladder stone .</li> <li>- Bladder, ureteral or renal malignancy.</li> </ul>

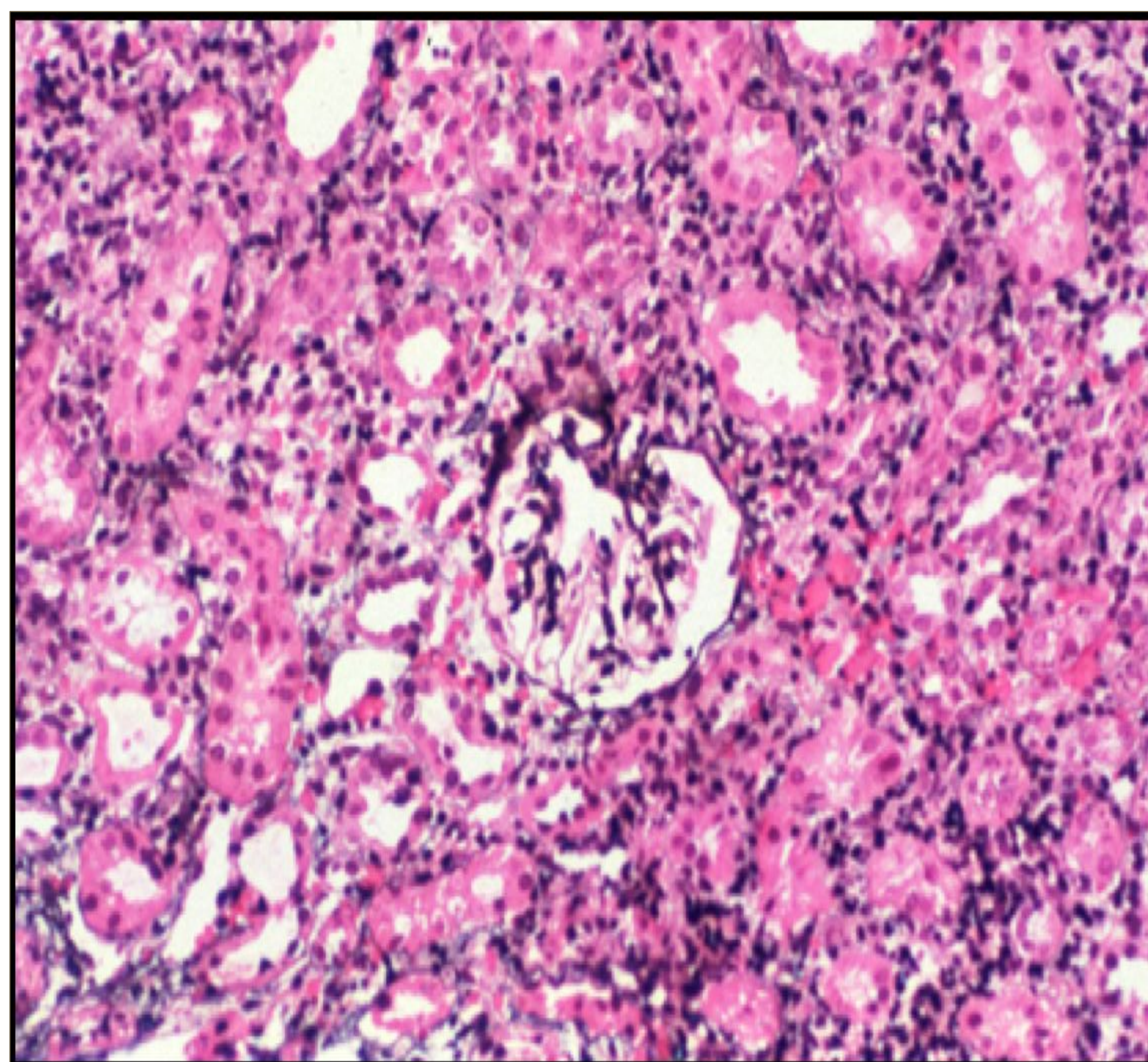
Causes for acute interstitial nephritis: including toxins, viral infections and drug-induced hypersensitivity reactions. The glomeruli are uninvolved, unless there is an associated minimal change disease-type injury caused by NSAIDs

Acute Interstitial Nephritis



- Acute and chronic inflammatory cells in the interstitium.
- In Chronic cases: tubular atrophy + fibrosis.
- AIN induced by drugs: eosinophils

Acute Interstitial Nephritis



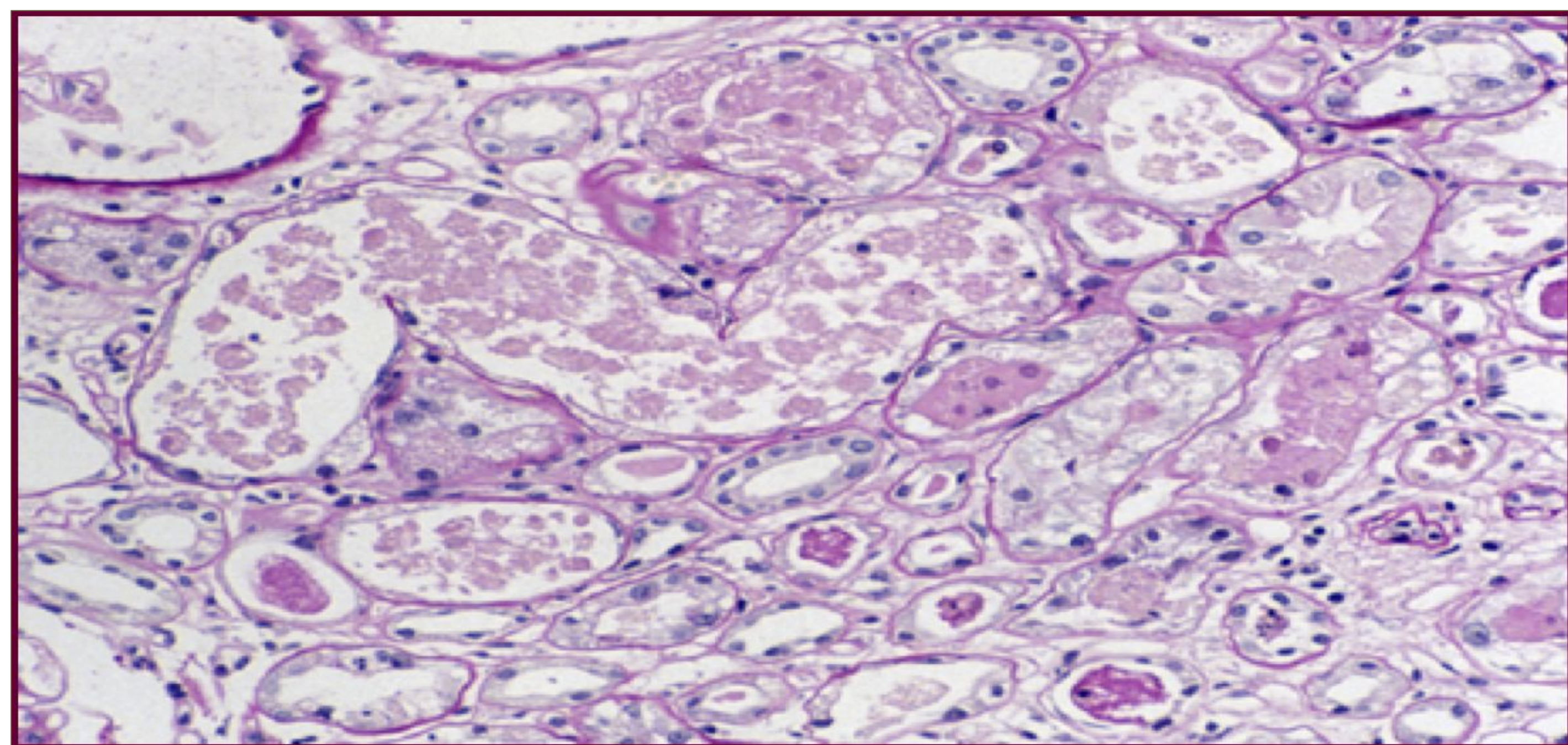
- Acute and chronic inflammatory cells in the interstitium.
- In Chronic cases: tubular atrophy + fibrosis.
- Usually associated with edema

Acute kidney injury



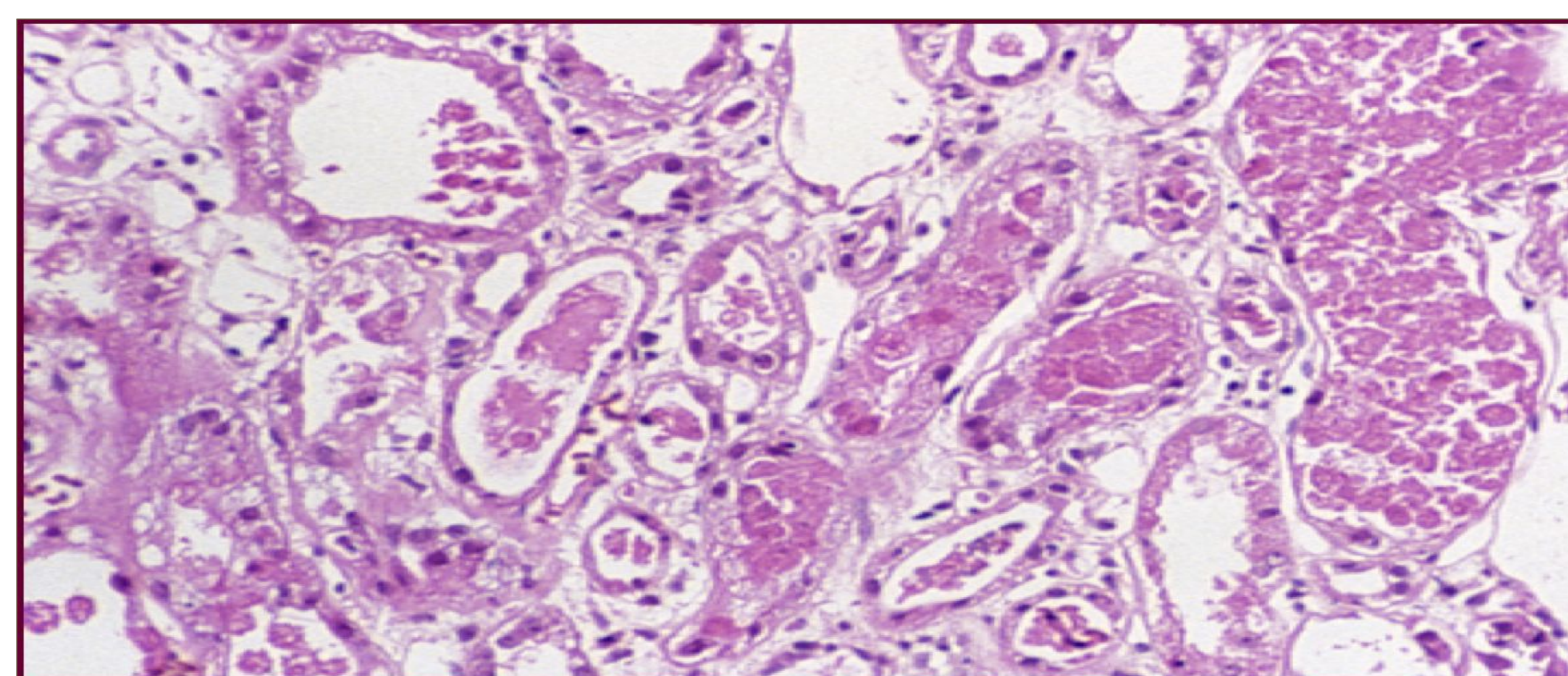
- Marked pallor of the renal cortex.
- Congested renal medulla.

Acute Tubular Necrosis



- Necrosis within the lumen of the tubule. (CASTS).
- Necrotic epithelial cells.
- Loss of brush border.
- Flat and vacuolated epithelium.

Acute Tubular Necrosis



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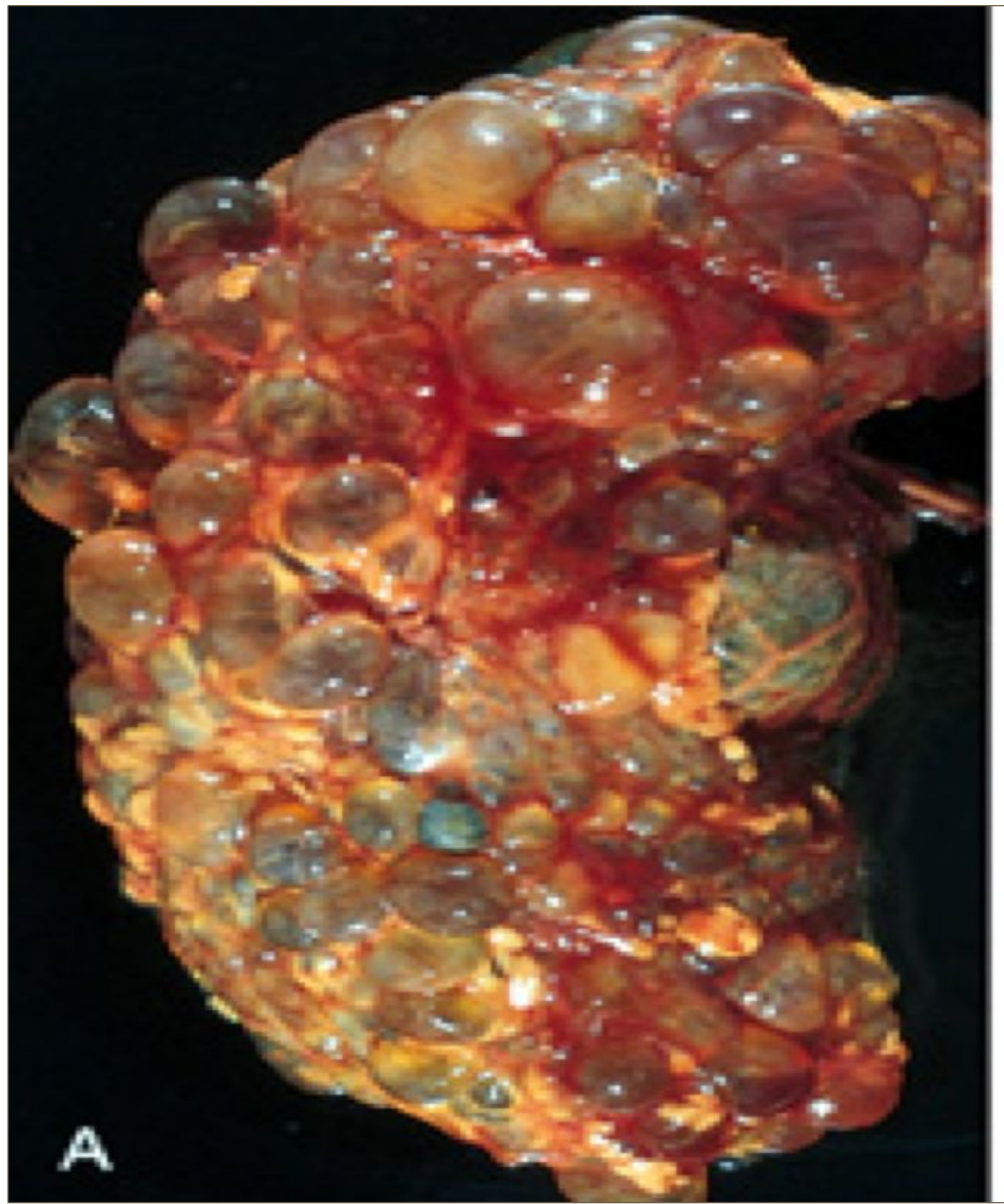
# #2 POLYCYSTIC KIDNEY

Theoretical information:

COMPLICATIONS: Hypertension, Renal failure

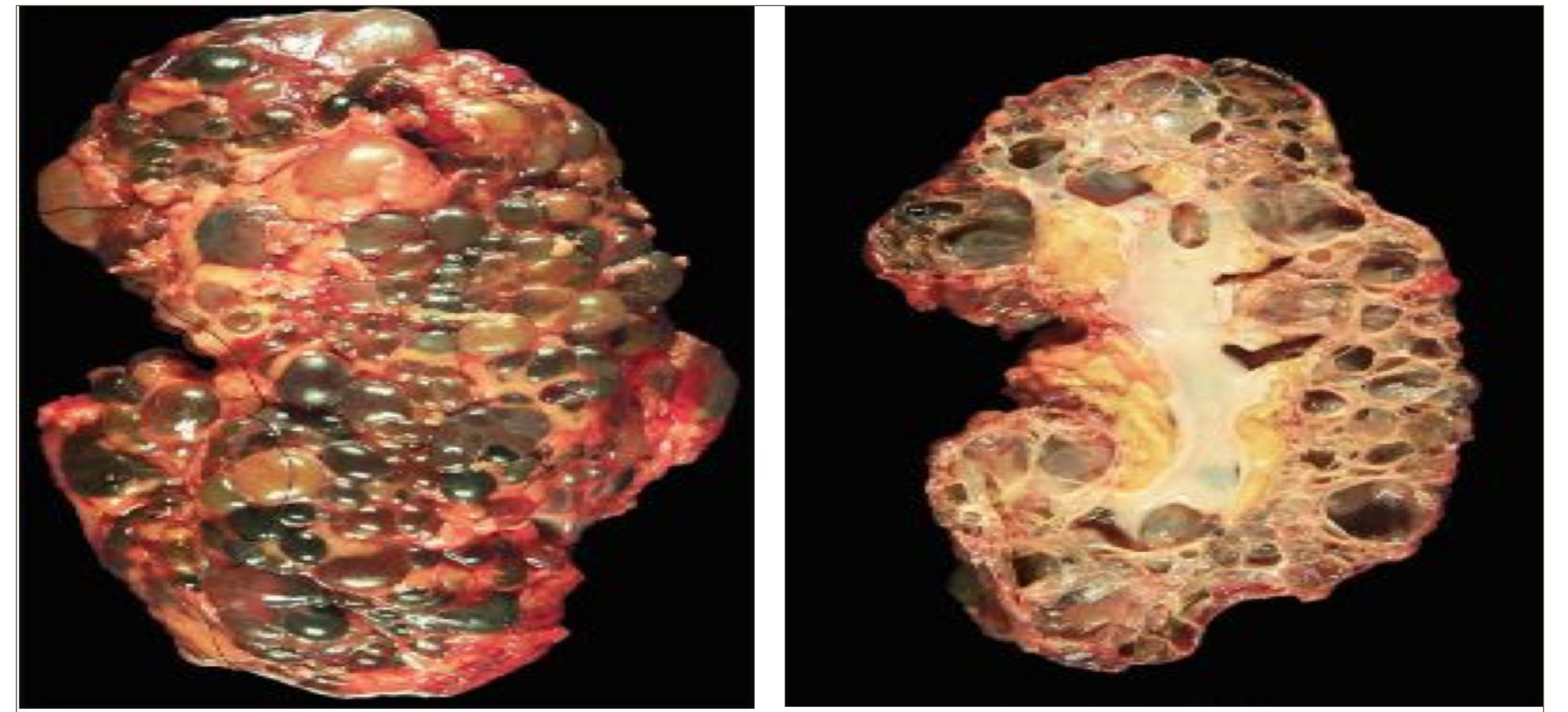
Types: **Gentic**, autosomal recessive -> seen in children. **autosomal dominant** -> **more common, seen in adults.**

## Polycystic kidney – Gross Anatomy



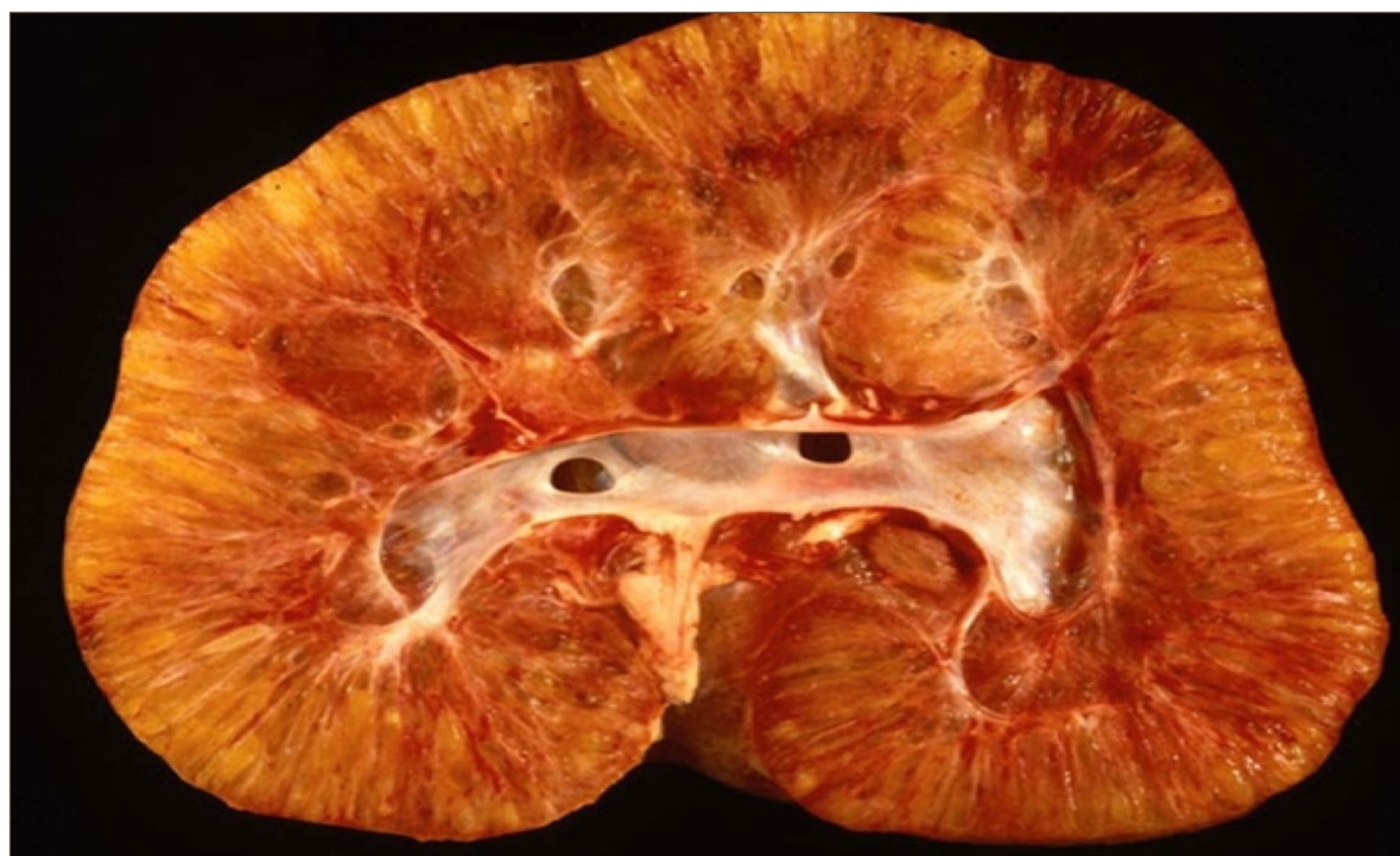
Markedly **enlarged** kidney and replacement of the renal parenchyma by **numerous cysts of variable sizes in entire cortex**  
**Filled with fluid**

## Polycystic kidney – Gross Anatomy and cut section



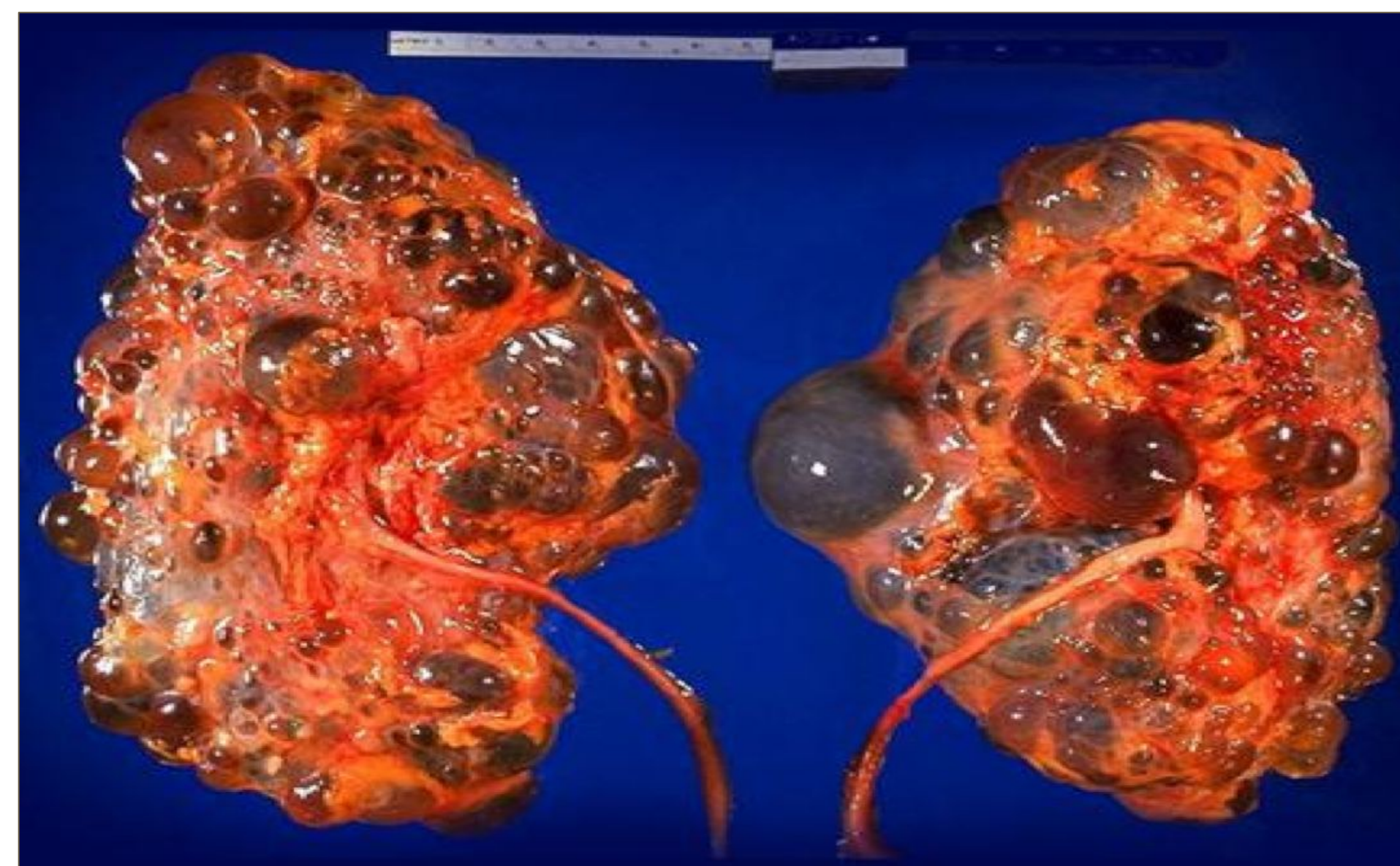
- 1- Massively enlarged kidney disrupted by numerous cysts
- 2- Cut surface of the kidney, showing extensive cortical destruction by cysts

## Infantile Polycystic kidney – Gross



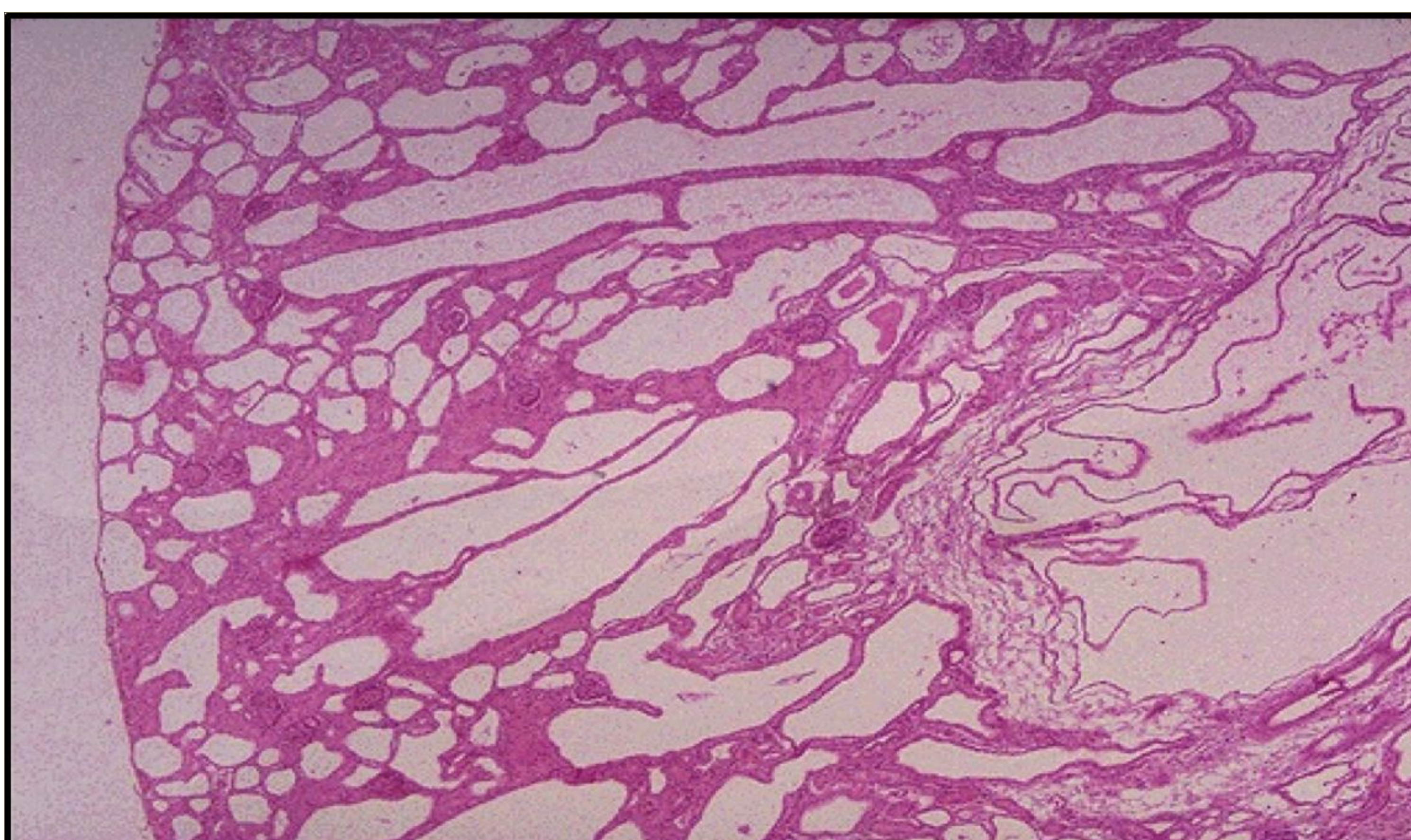
Coronal section of an **infantile** polycystic kidney

## Polycystic kidney – Gross



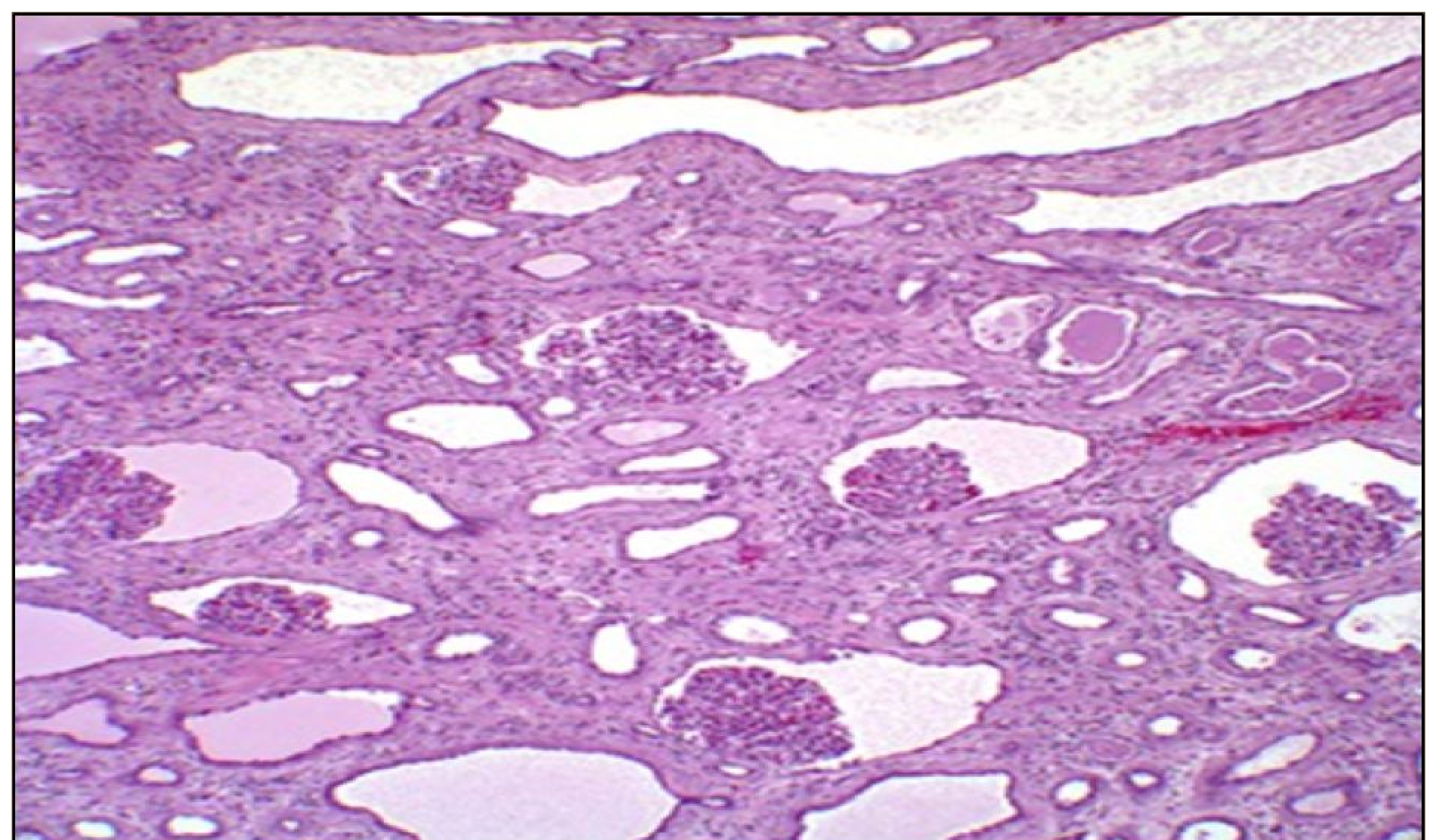
**Bilateral** autosomal dominant polycystic kidney disease

## Polycystic Kidney Disease



- Cysts fill most of the parenchyma
- Cystic formation in the glomeruli and interstitium and bowman's space.

## Polycystic kidney – Histopathology



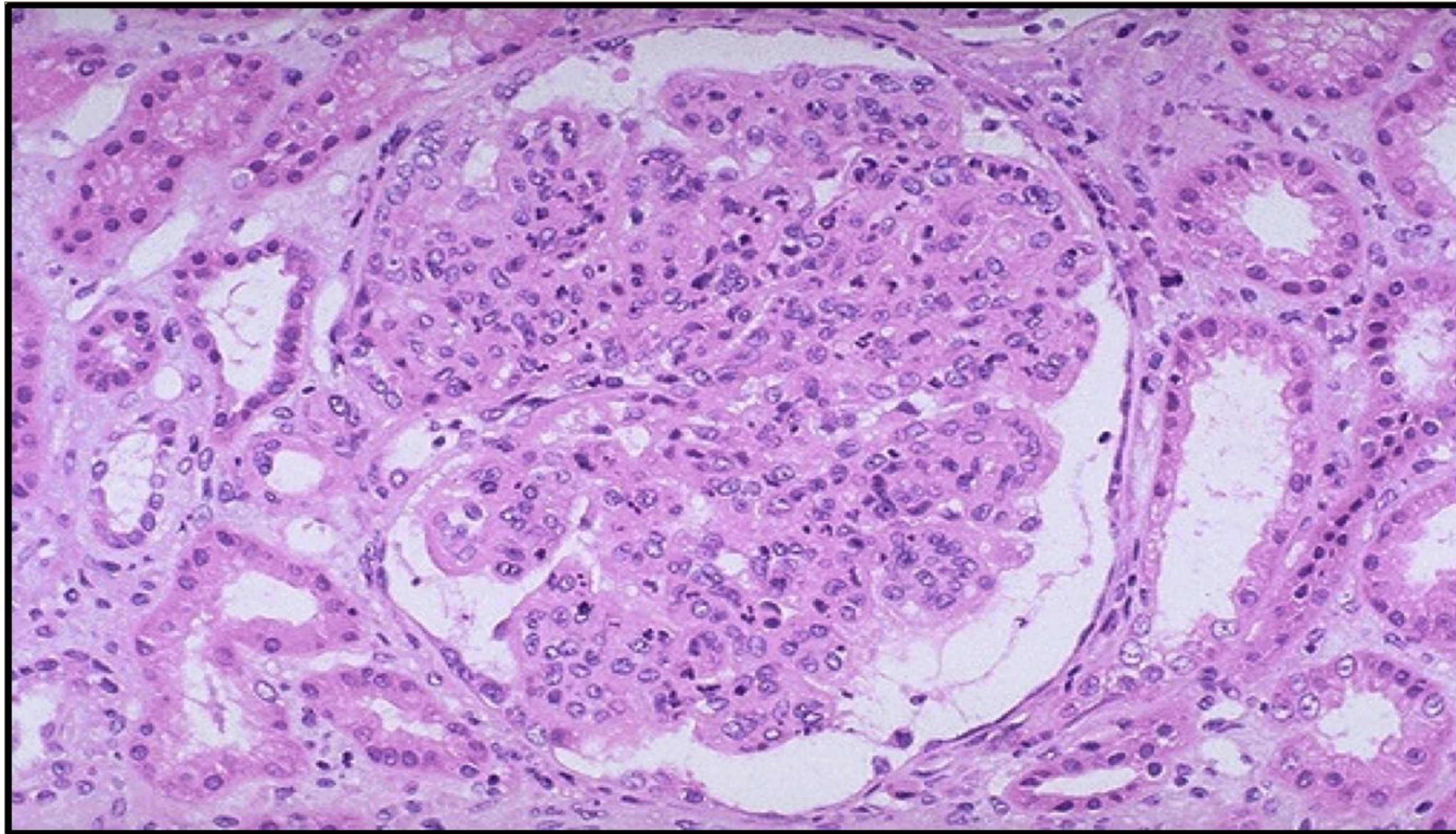
- Cysts fill most of the parenchyma.
- Cystic formation in the glomeruli and interstitium and bowman's space.

# #3 ACUTE (POST-STREPTOCOCCAL) GLOMERULONEPHRITIS

Theoretical information:

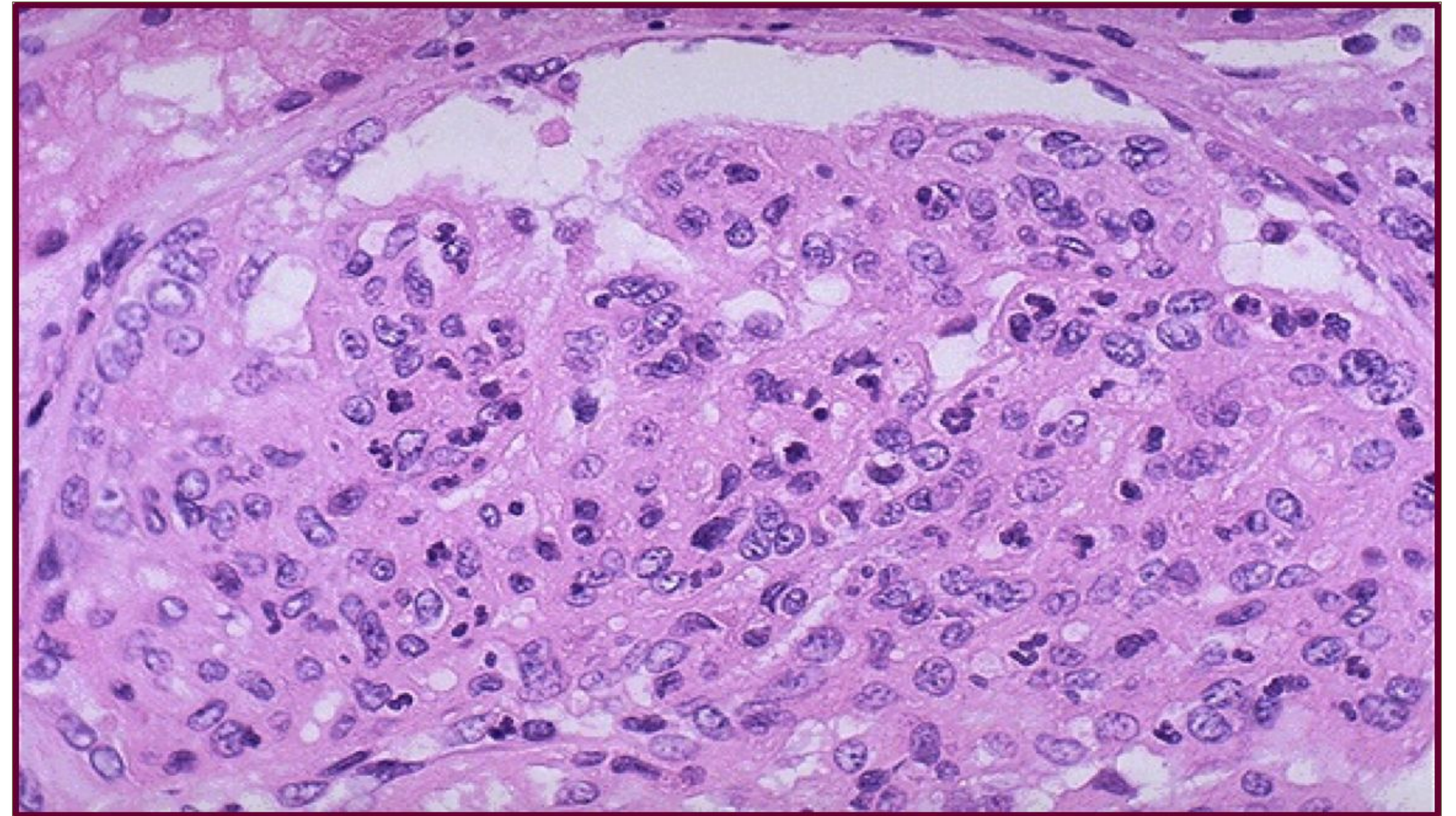
- The usual scenario : streptococcal pharyngitis , 2wks later kidney is affected , glomerulonephritis + smoky urine
- **Nephritic syndrome.**

Acute (Post-streptococcal) Glomerulonephritis



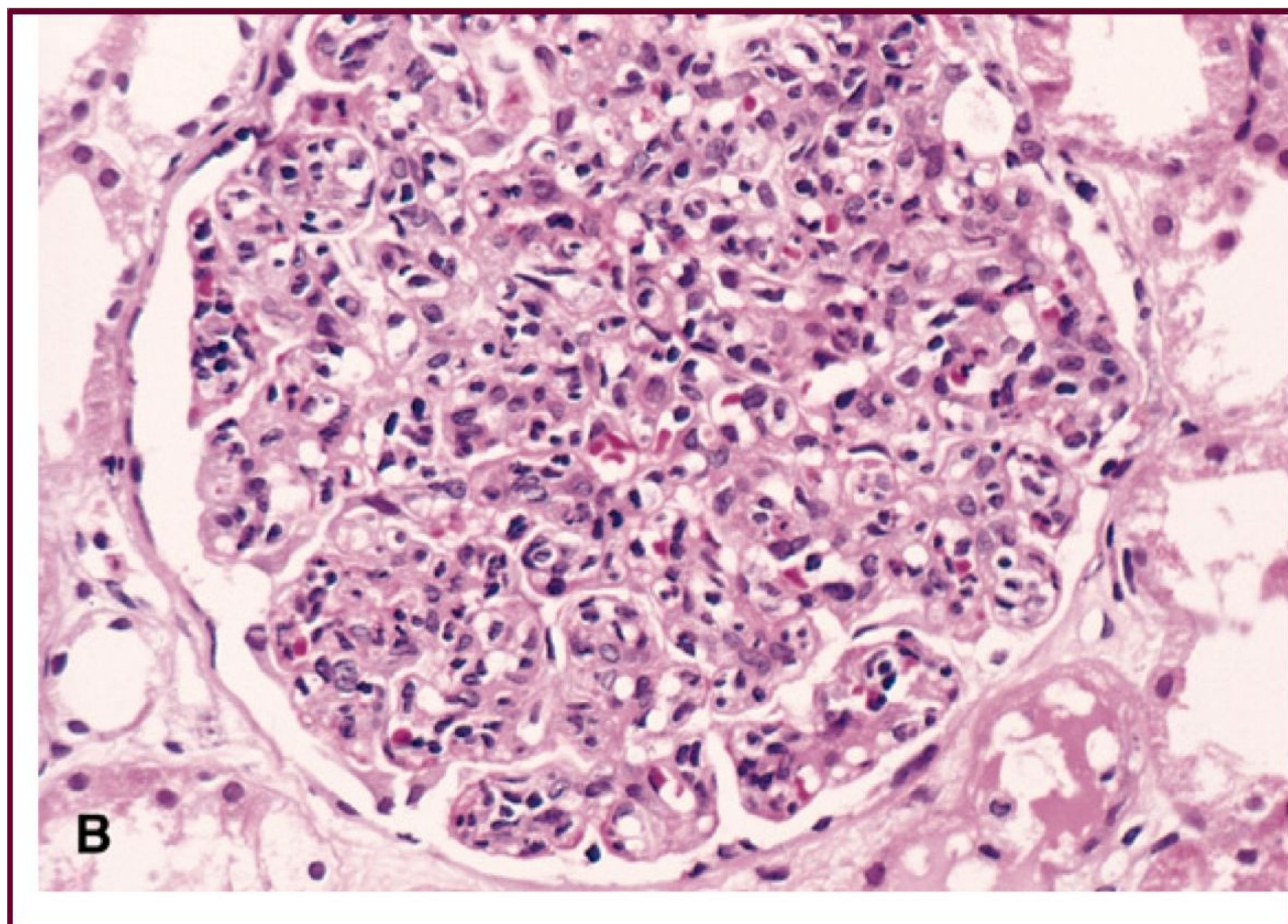
- hypercellularity is due to increased numbers of epithelial, endothelial , mesangial cells.
- neutrophils in and around the glomerular capillary loops

Acute (Post-streptococcal) Glomerulonephritis



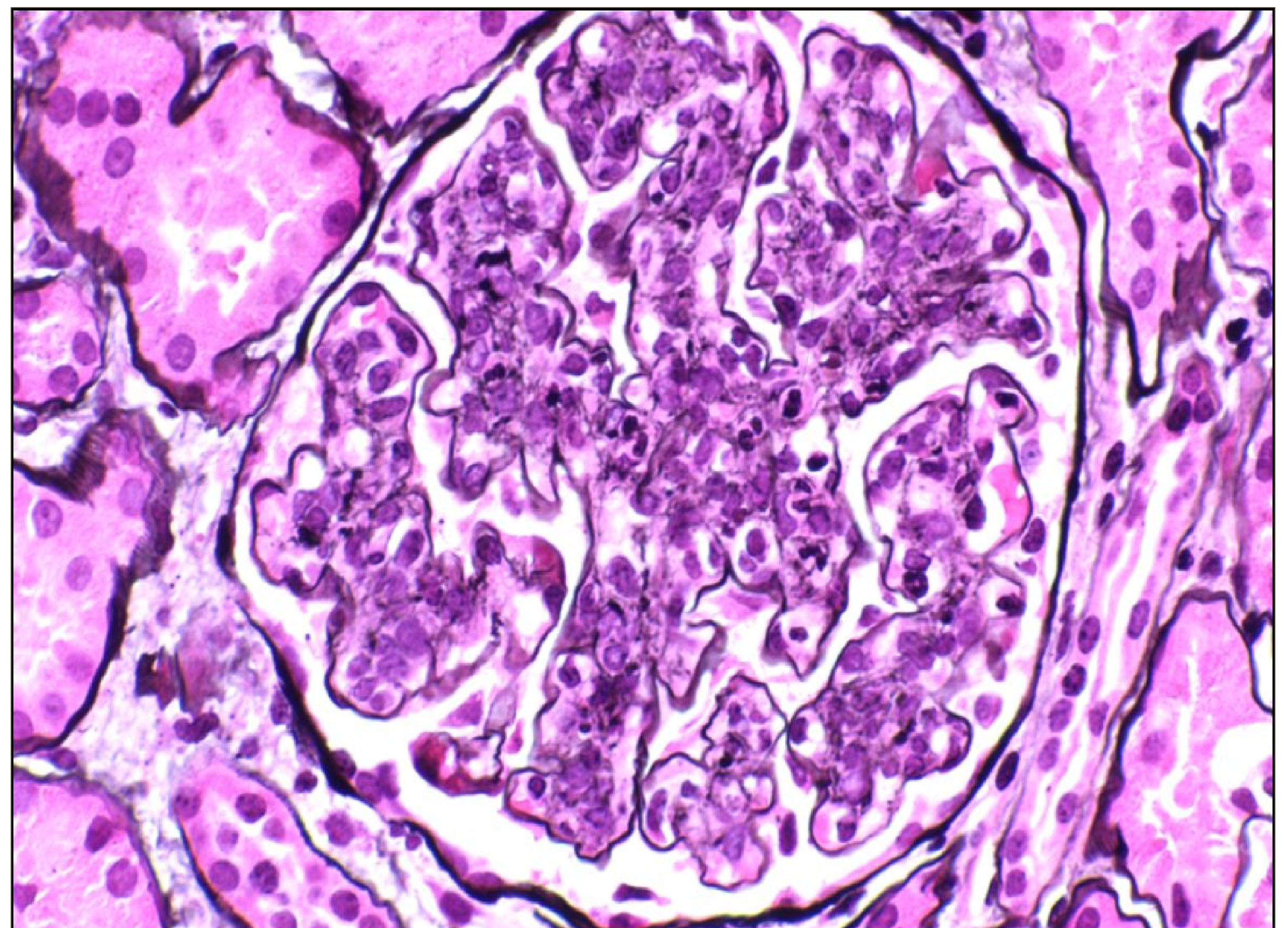
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Acute (Post-streptococcal) Glomerulonephritis



- hypercellularity is due to increased numbers of epithelial, endothelial , mesangial cells.
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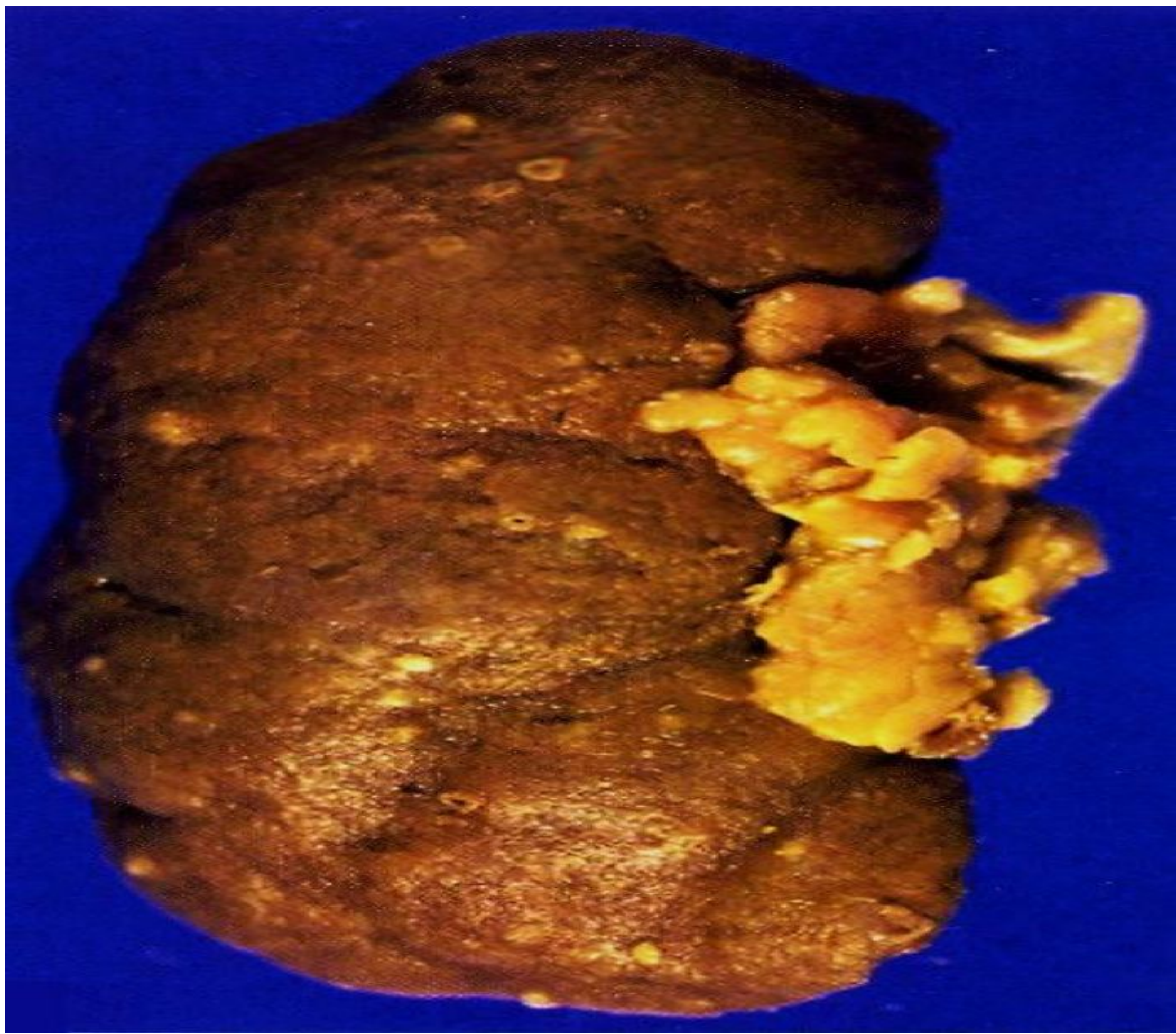
Acute (Post-streptococcal) Glomerulonephritis



- large number of PMNs.
- There is proliferation of endothelial and mesangial cells, infiltrating cells and filling and distending capillary loops.
- **The glomerular basement membrane does not show splitting or spikes.**

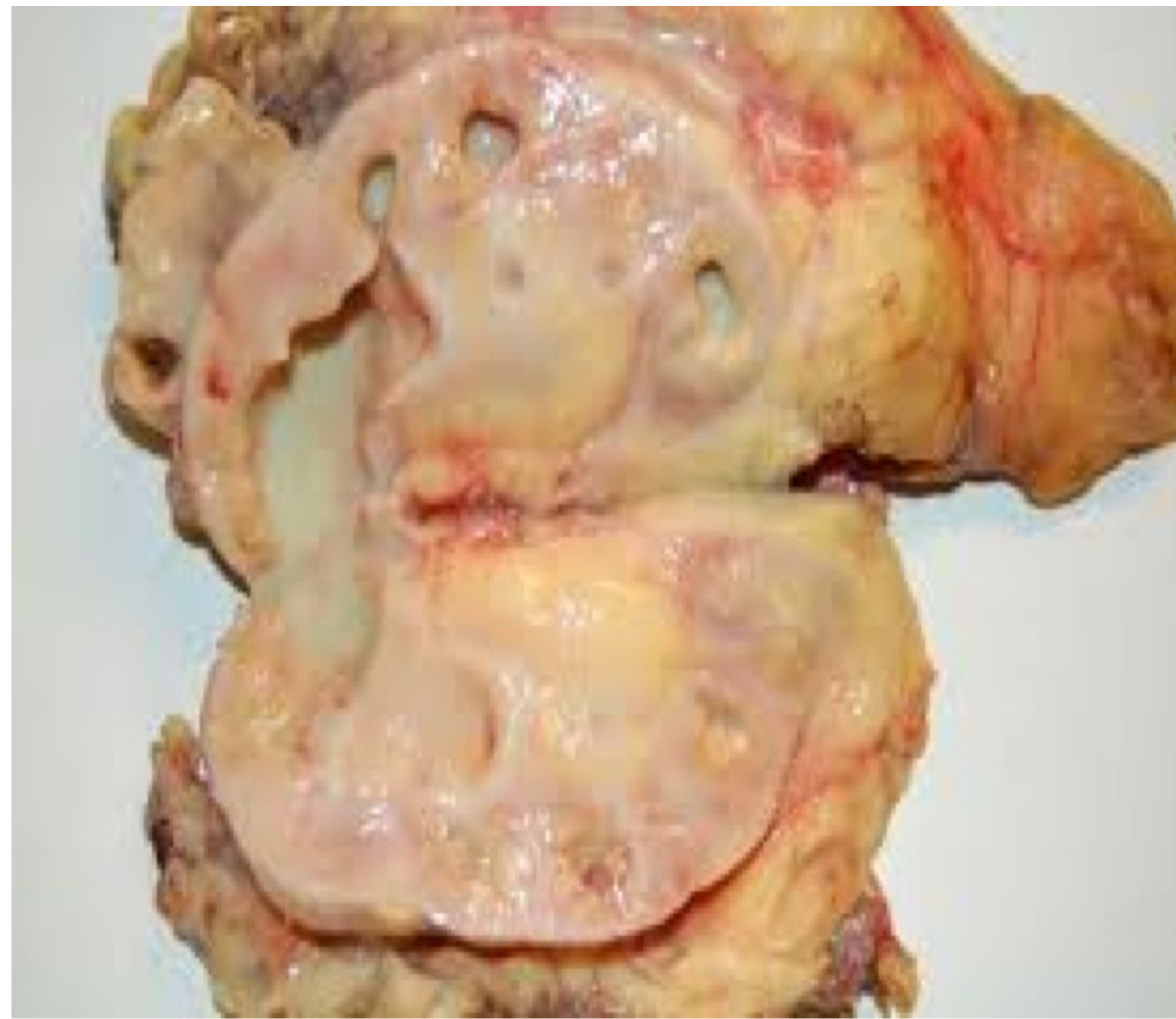
# #4 ACUTE PYELONEPHRITIS

Pyelonephritis with small cortical abscesses



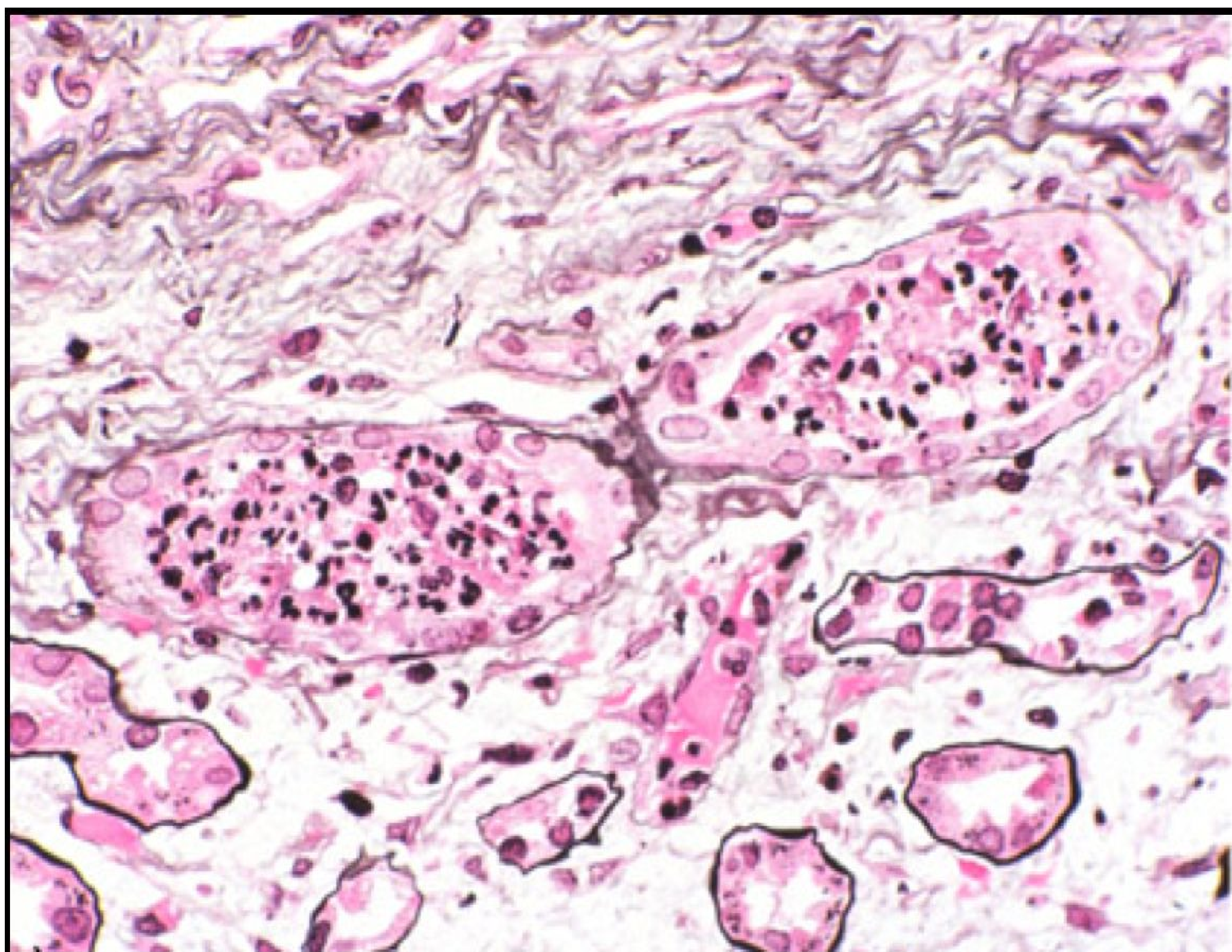
Pyelonephritis with small cortical abscesses  
Pyelonephritis = inflammation within the kidney.

Classic picture of Pyelonephritis



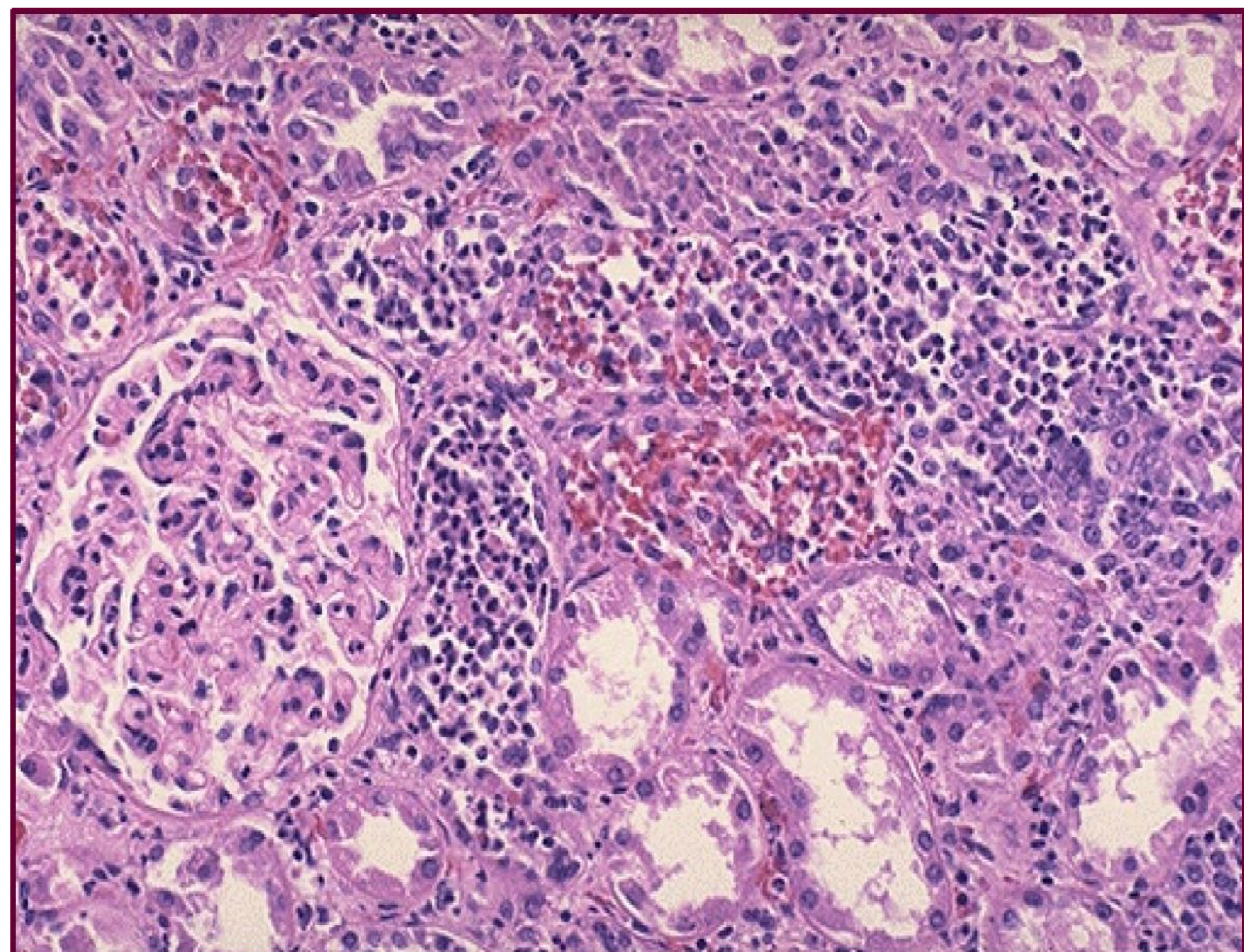
pelvis and calyces filled with a pus

Acute Pyelonephritis - Histopathology



- Necrotic epithelial tubules
- Collection of the neutrophils

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.

# #4 CHRONIC PYELONEPHRITIS

Theoretical information:

**Causes:**

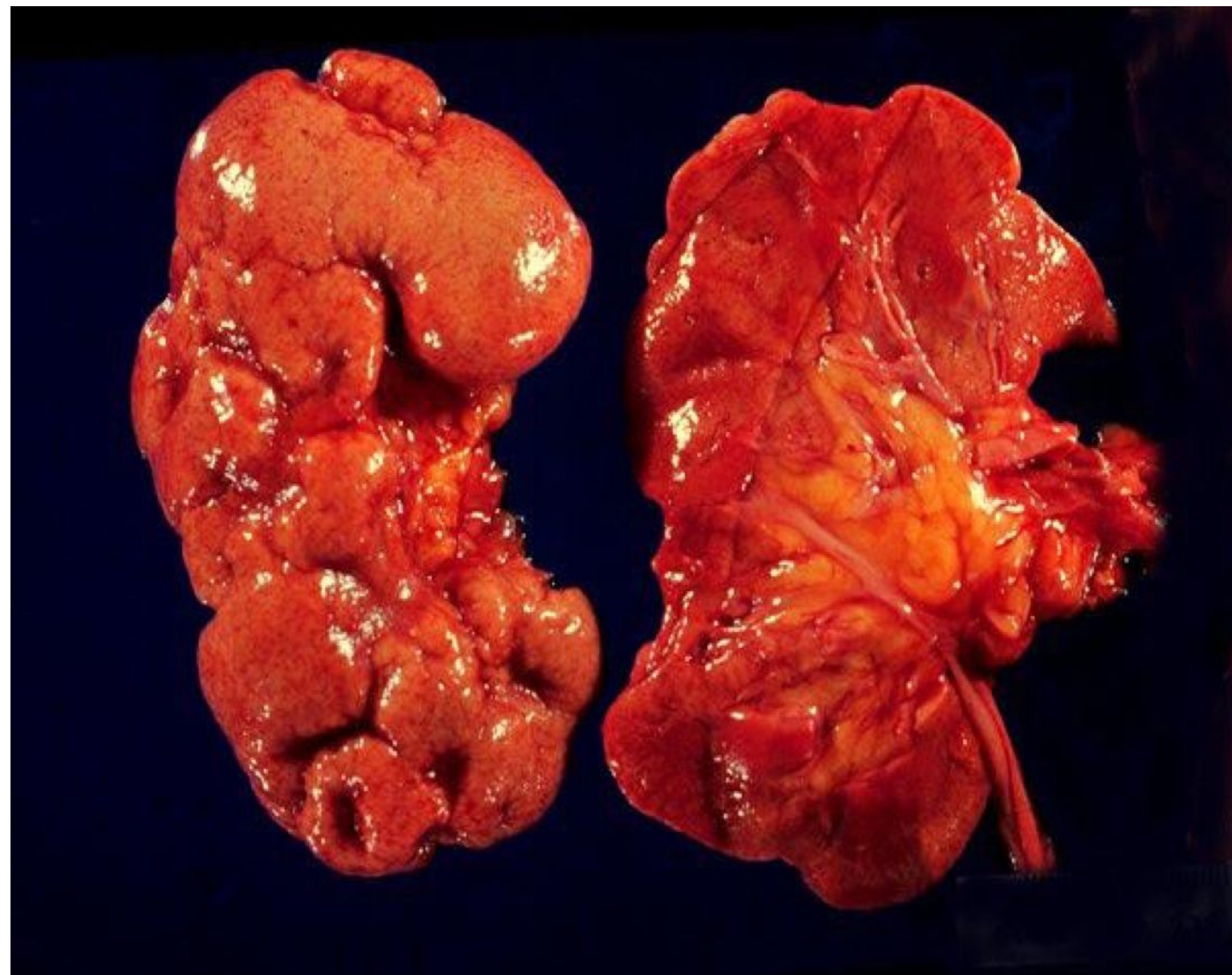
Acute Pyelonephritis

- Hematogenous spread

Chronic Pyelonephritis

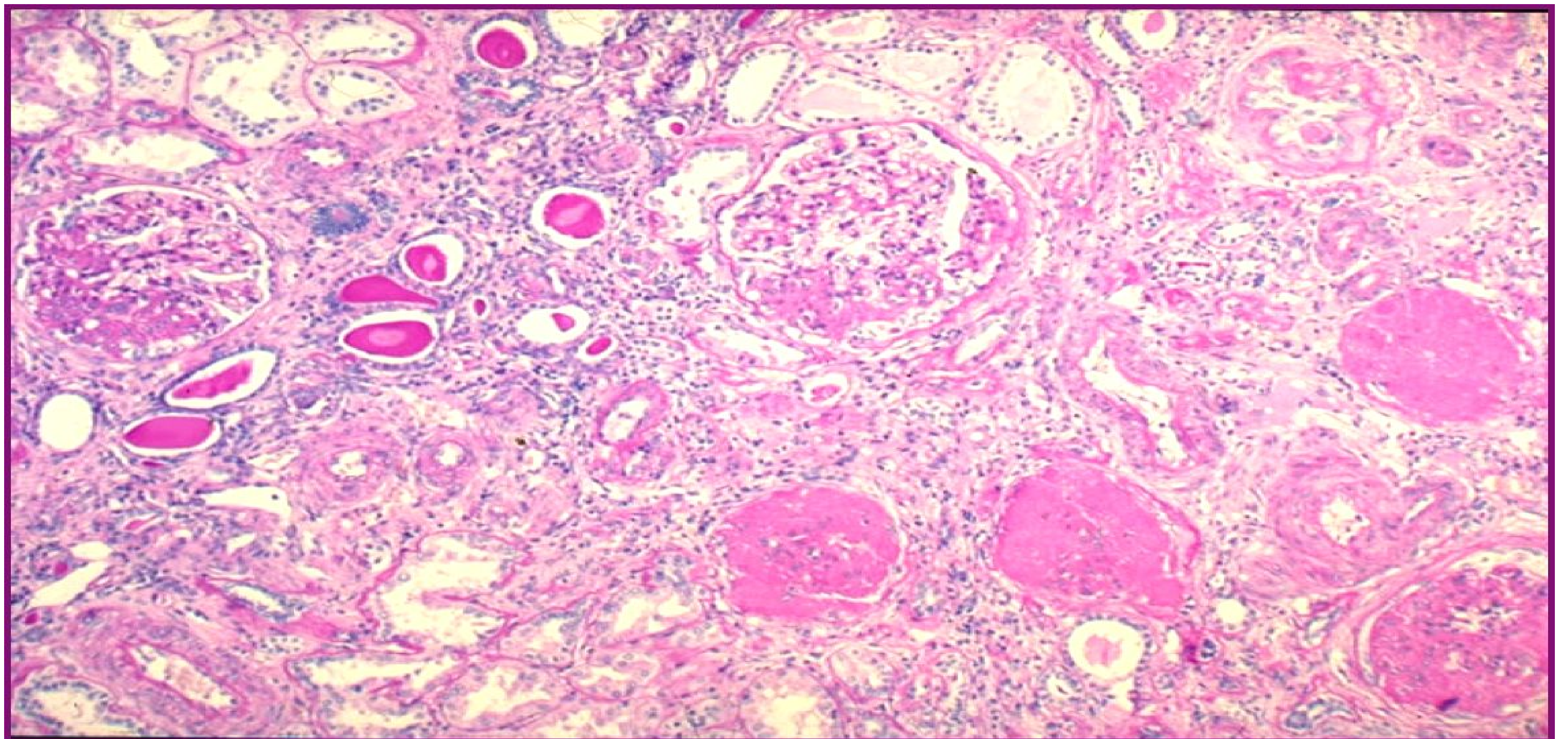
- Recurrent attacks of acute pyelonephritis.
- Drug-induced interstitial nephritis.
- Urinary tract obstruction or reflux.

Chronic Pyelonephritis - Gross Pathology



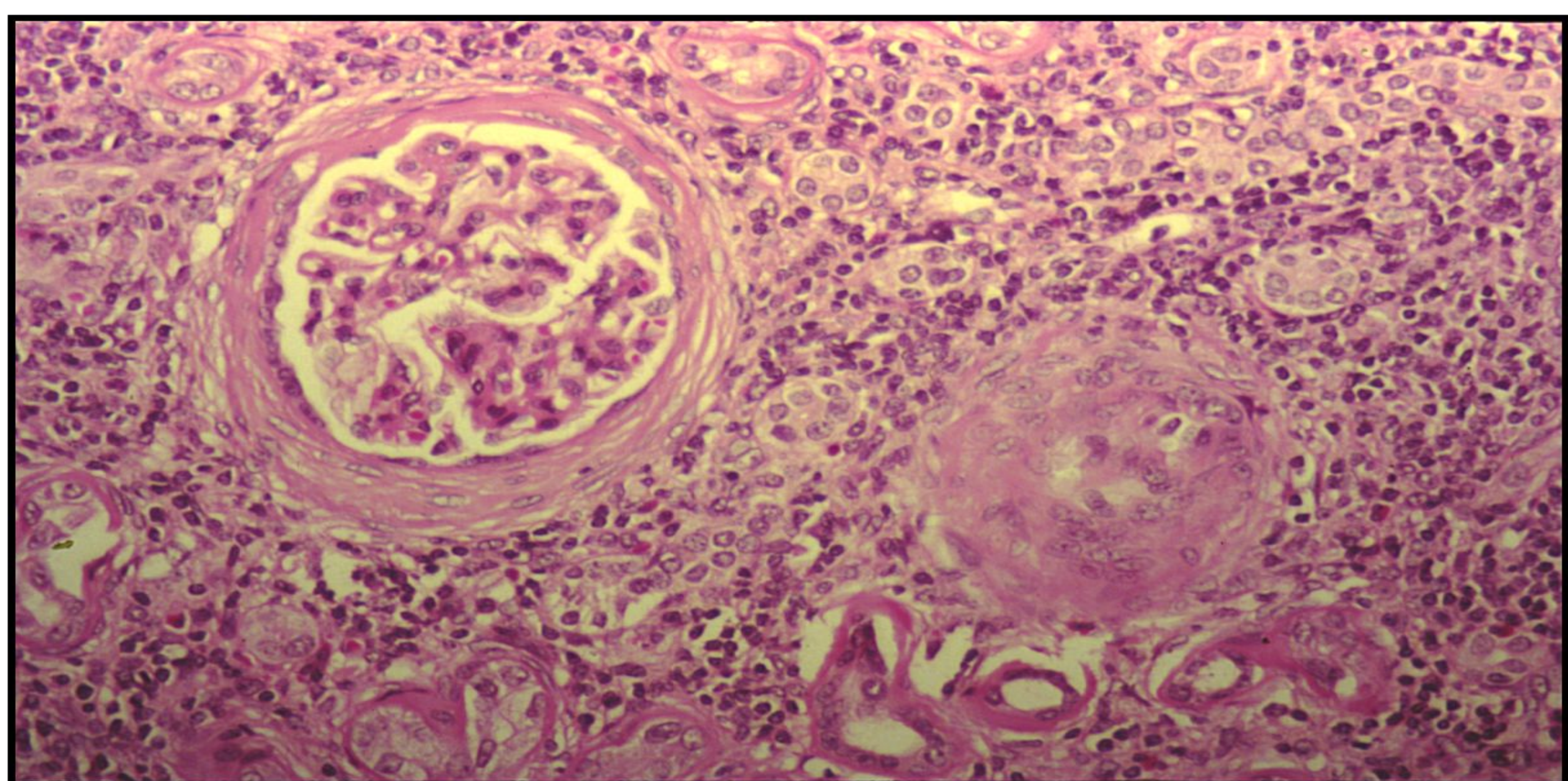
- Deformity
- atrophic kidneys.
- Deep cortical scars.

Chronic Pyelonephritis - Histopathology



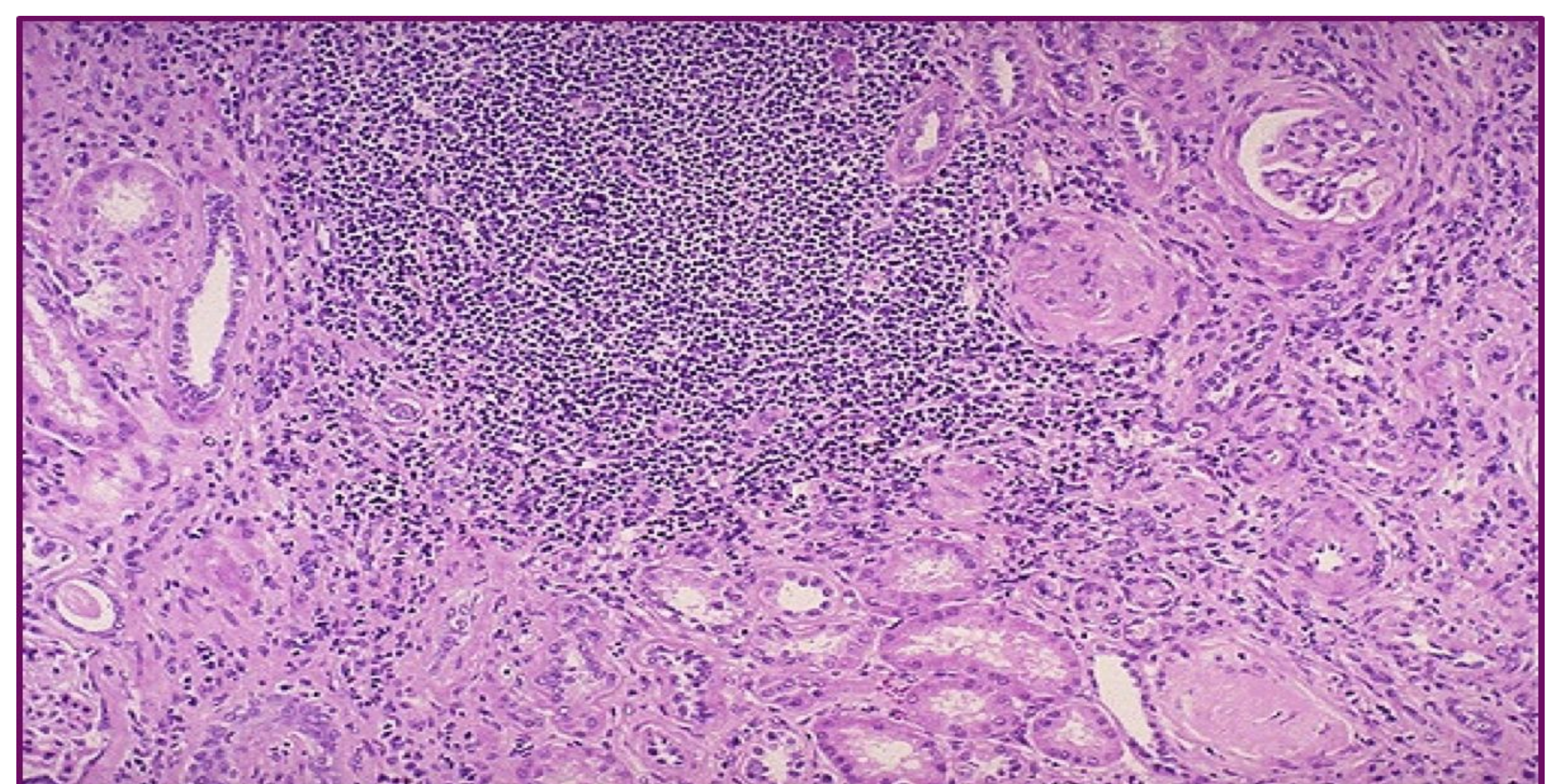
- Chronic interstitial inflammation.
- Atrophy of renal tubules.
- thyroidization of renal tubules.
- Hyalinization of glomeruli.
- interstitial fibrosis.

Chronic Pyelonephritis - Histopathology



- periglomerular fibrosis ,
- glomerular sclerosis and
- hyalinization with marked chronic interstitial inflammation .

Acute (Post-streptococcal) Glomerulonephritis



- 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

# #5 HYDRONEPHROSIS

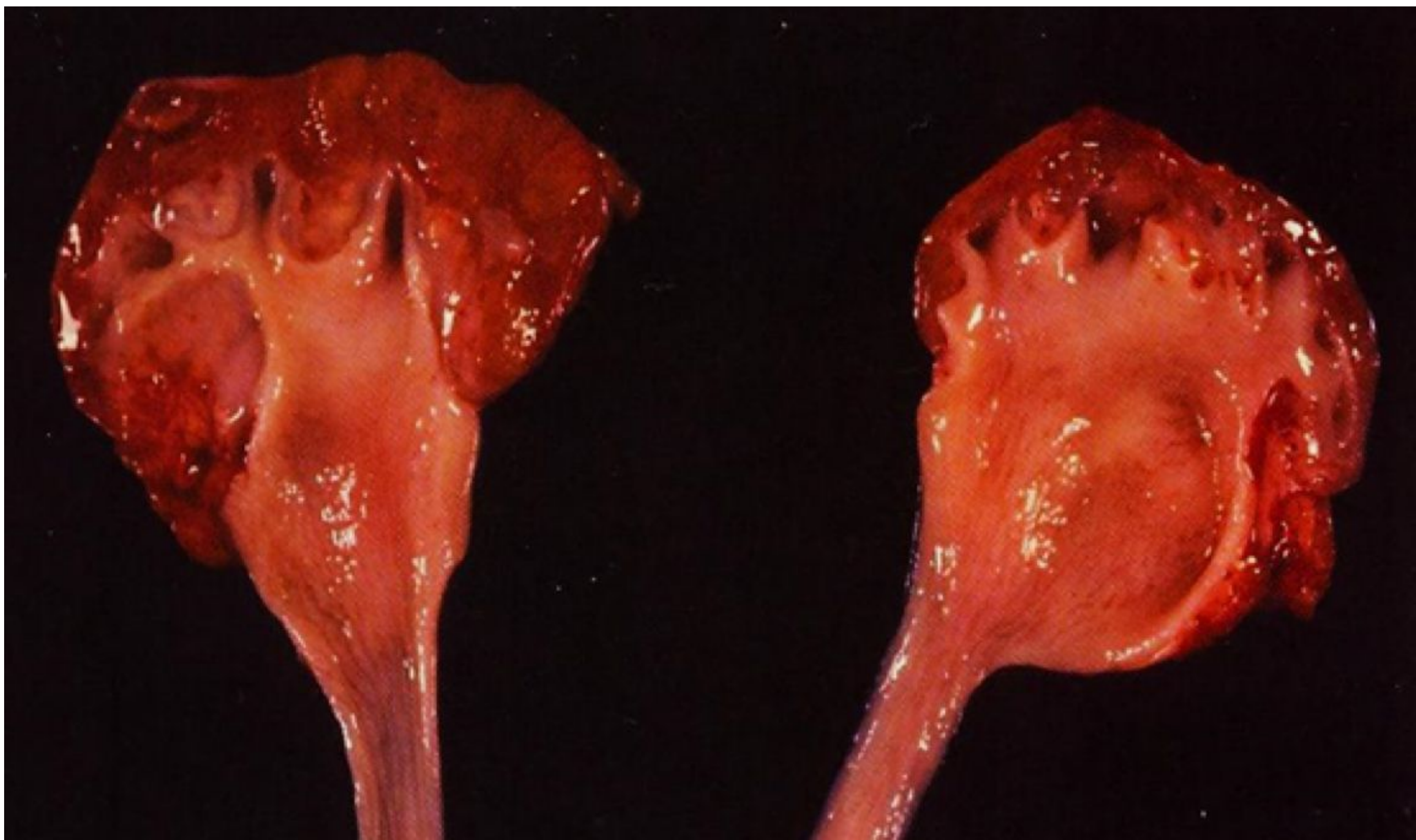
Theoretical information:

The most common causes are:

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

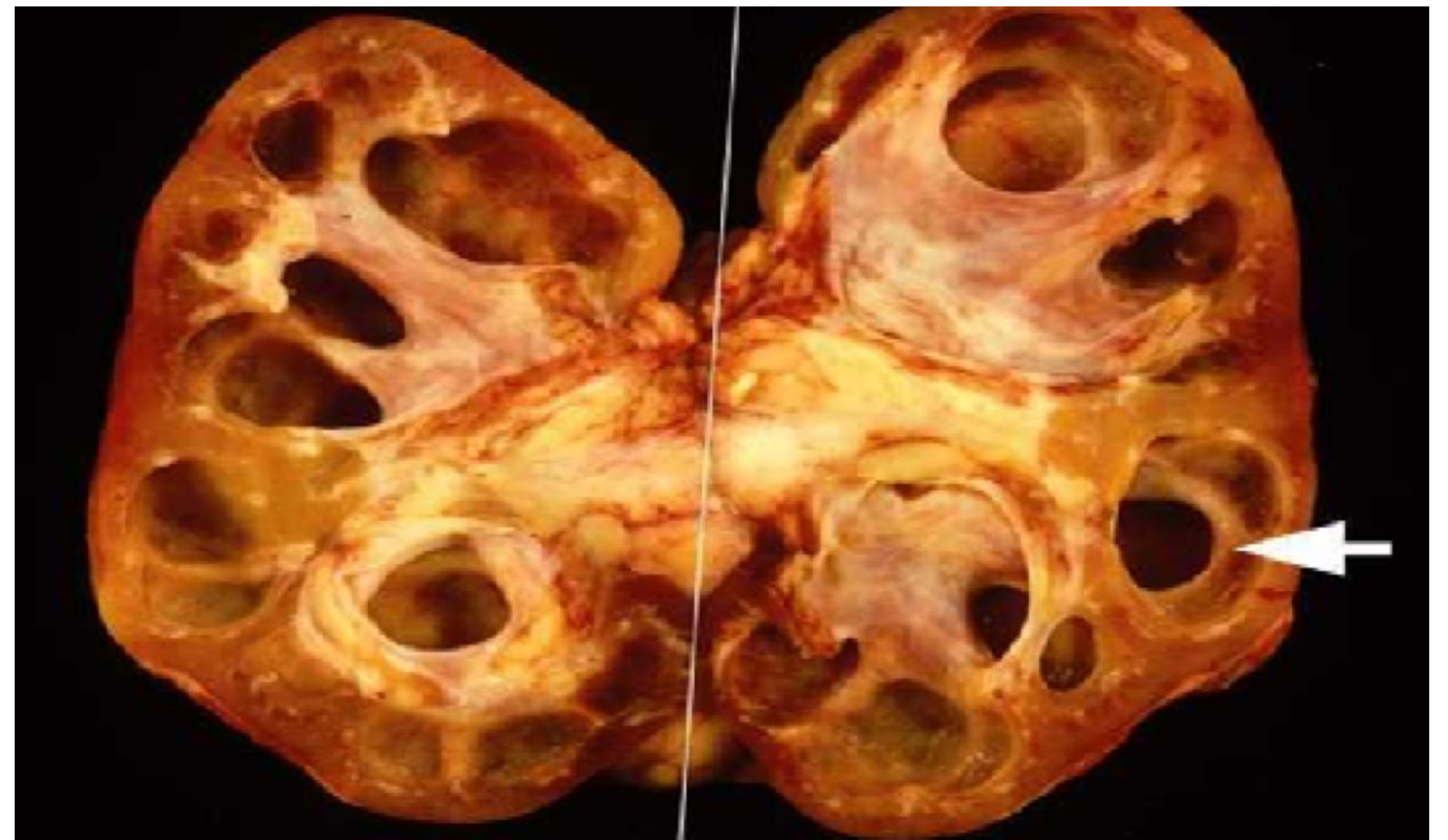
Complication: Chronic Pyelonephritis

Hydronephrosis



- markedly **dilated** renal pelvis and
- calyces with **atrophic** and thin renal cortex

Hydronephrosis



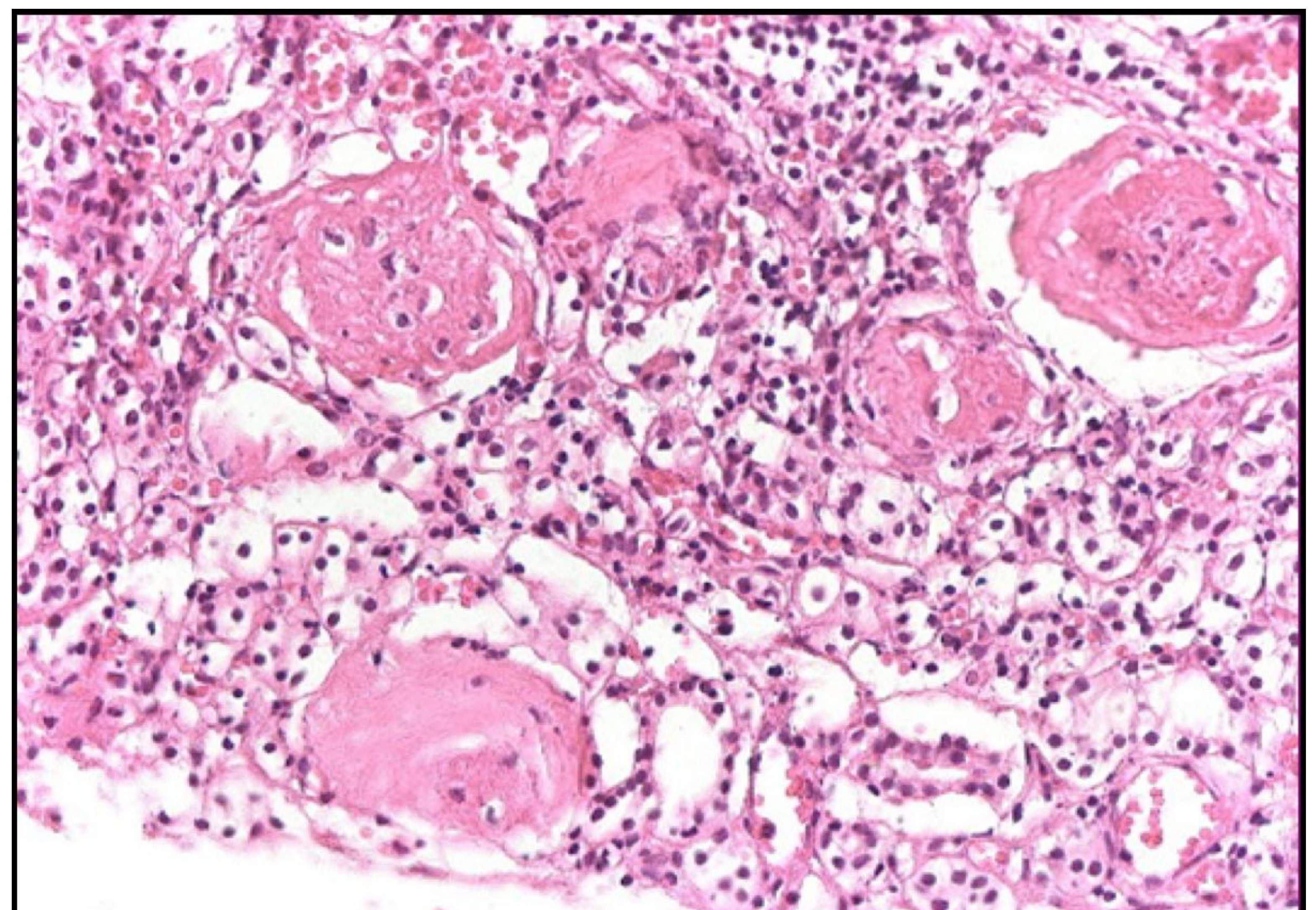
- 1-Markedly dilated renal pelvis and
- 2-calyces with atrophic and thin renal cortex

Chronic Pyelonephritis - Histopathology



- 1-markedly dilated renal pelvis and
- 2-calyces with atrophic and thin renal cortex /parenchyma

Chronic Pyelonephritis presenting as complication to Hydronephrosis



- Sclerosis of glomeruli with atrophic tubules and interstitial inflammation.

# #6 Nephrotic Syndrome

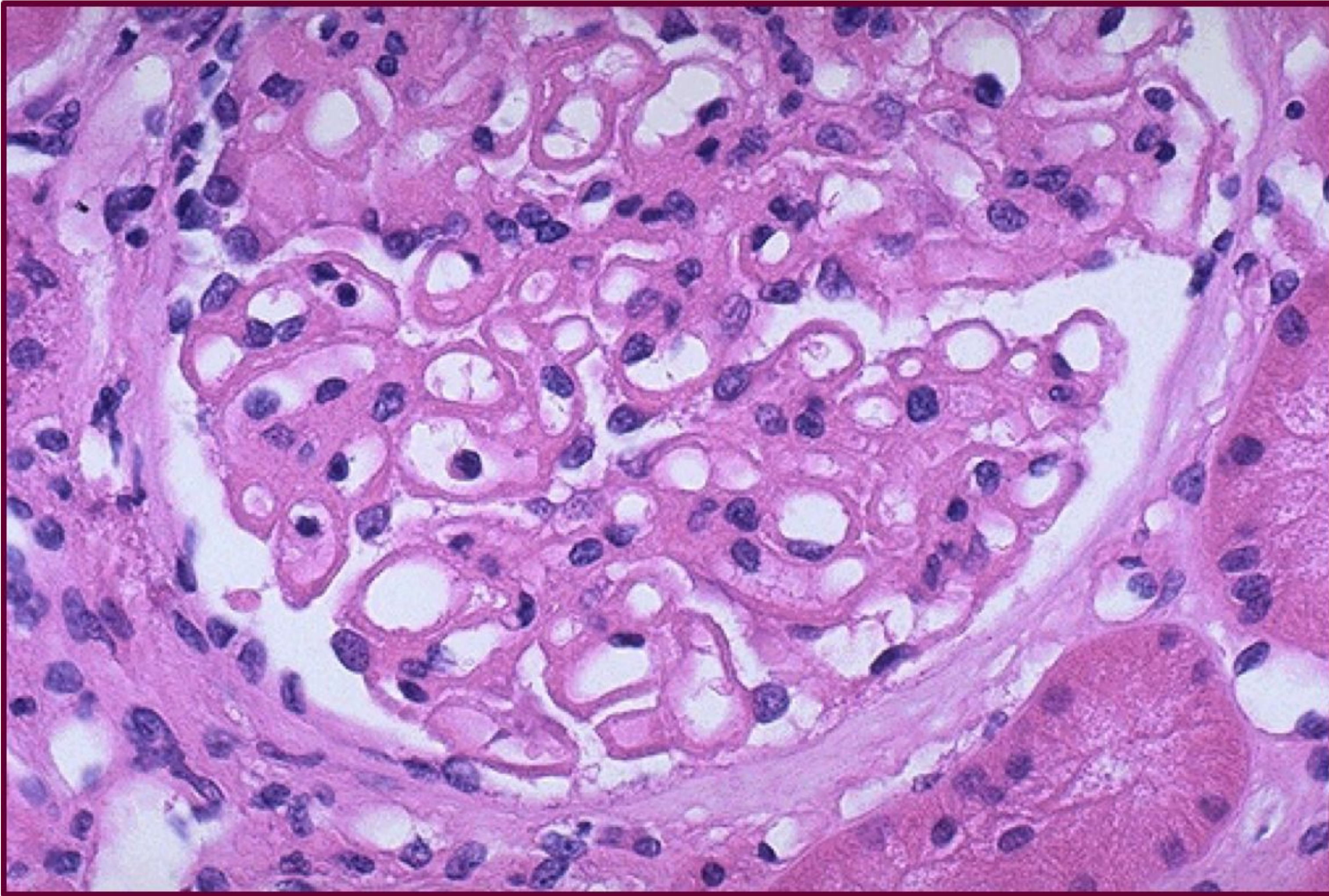
## Theoretical information

( The common cause of Nephrotic syndrome in adults): **Membranous Glomerulonephritis**

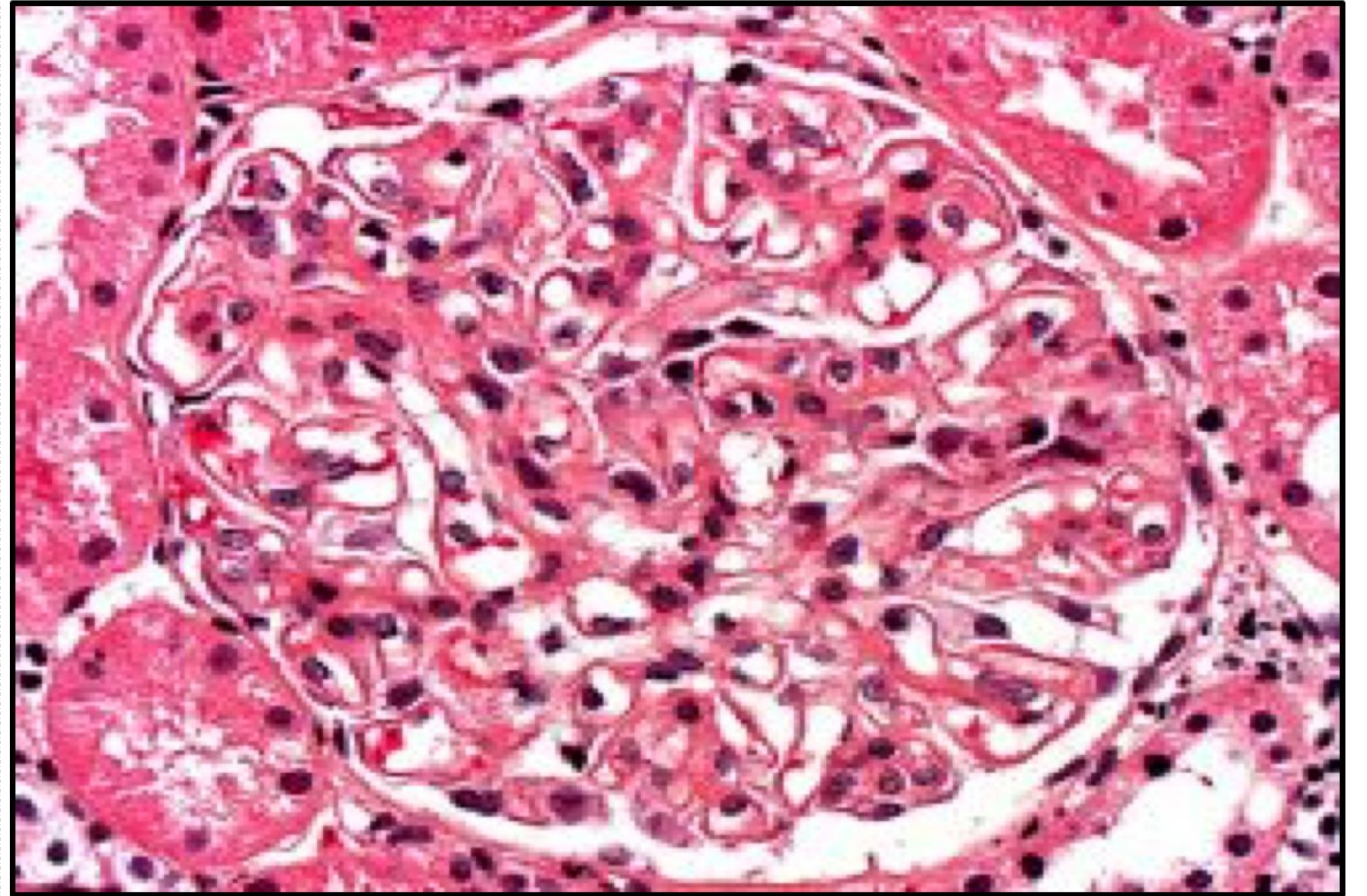
( The common cause of Nephrotic syndrome in children): **minimal change disease.**

Clinical sign: >3,5 g/day proteinuria

## Membranous Glomerulonephritis

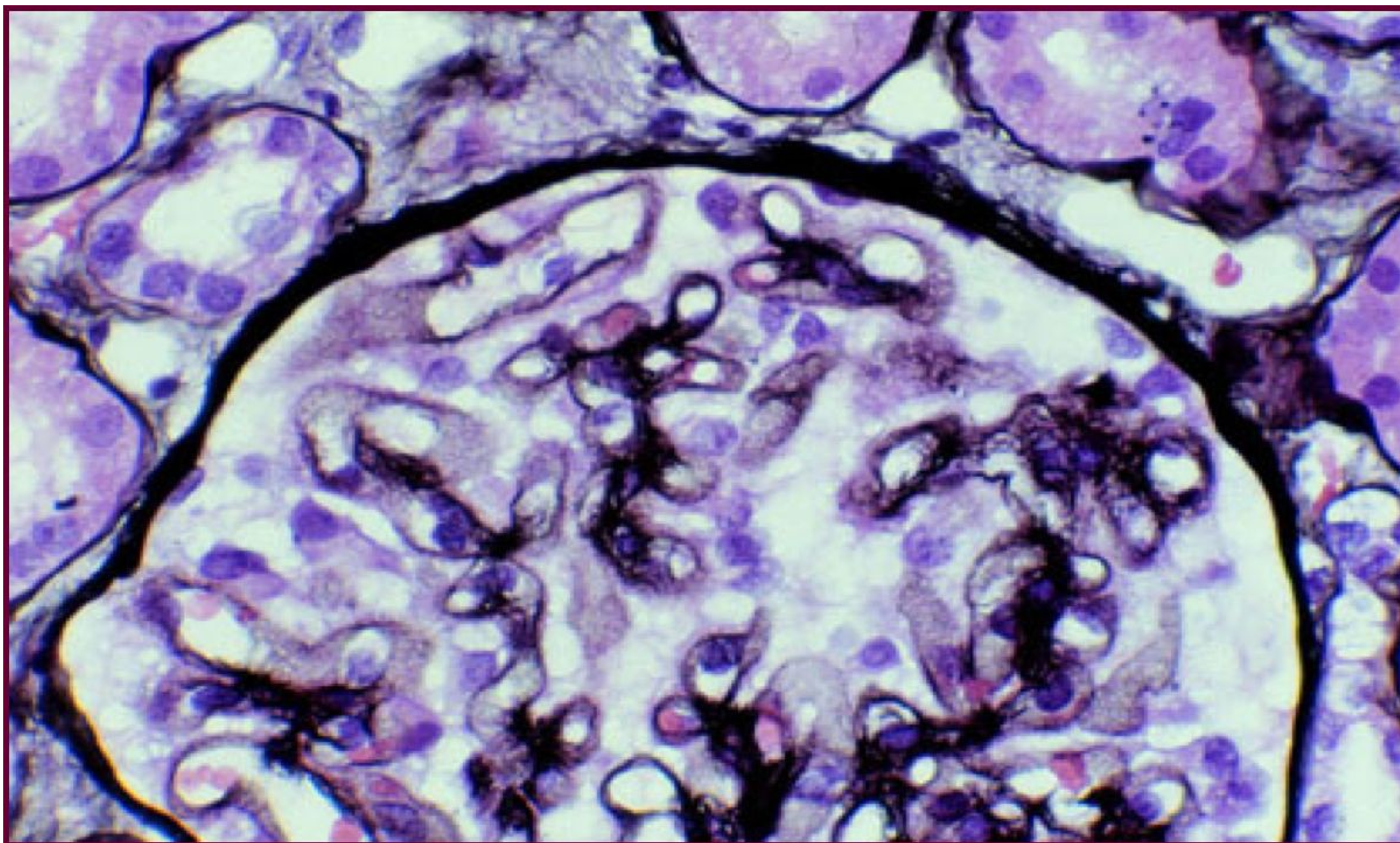


- **Capillary loops are thickened and prominent**
- **the cellularity is not increased.**



Close-up of glomerulus illustrating **rigid, uniformly-thickened capillary walls.**

## 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.



# #7 Nephritic Syndrome (RPGN)

Theoretical information:

**Crescentic glomerulonephritis** is known as **rapidly progressive glomerulonephritis (RPGN)** because this disease is very progressive.

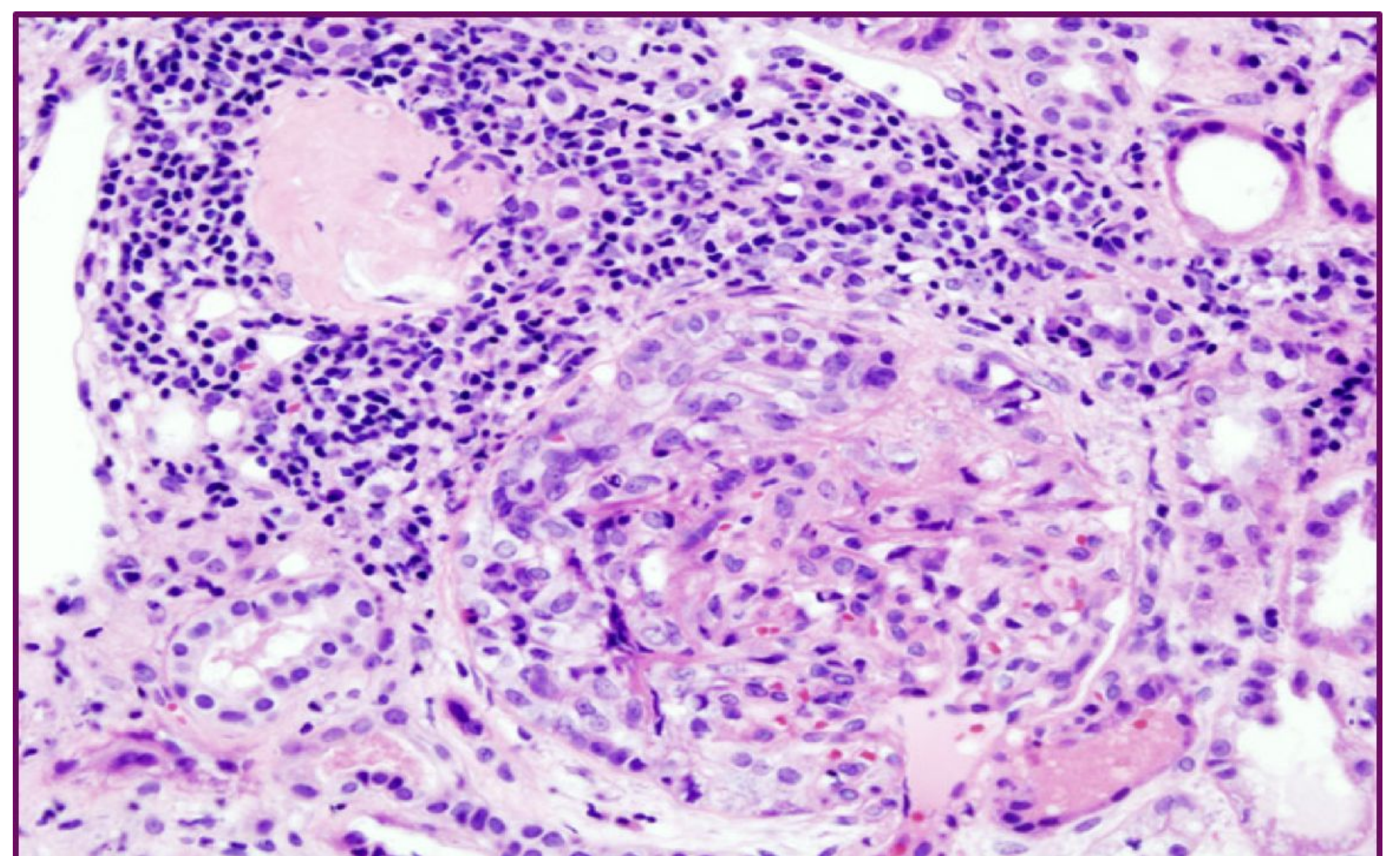
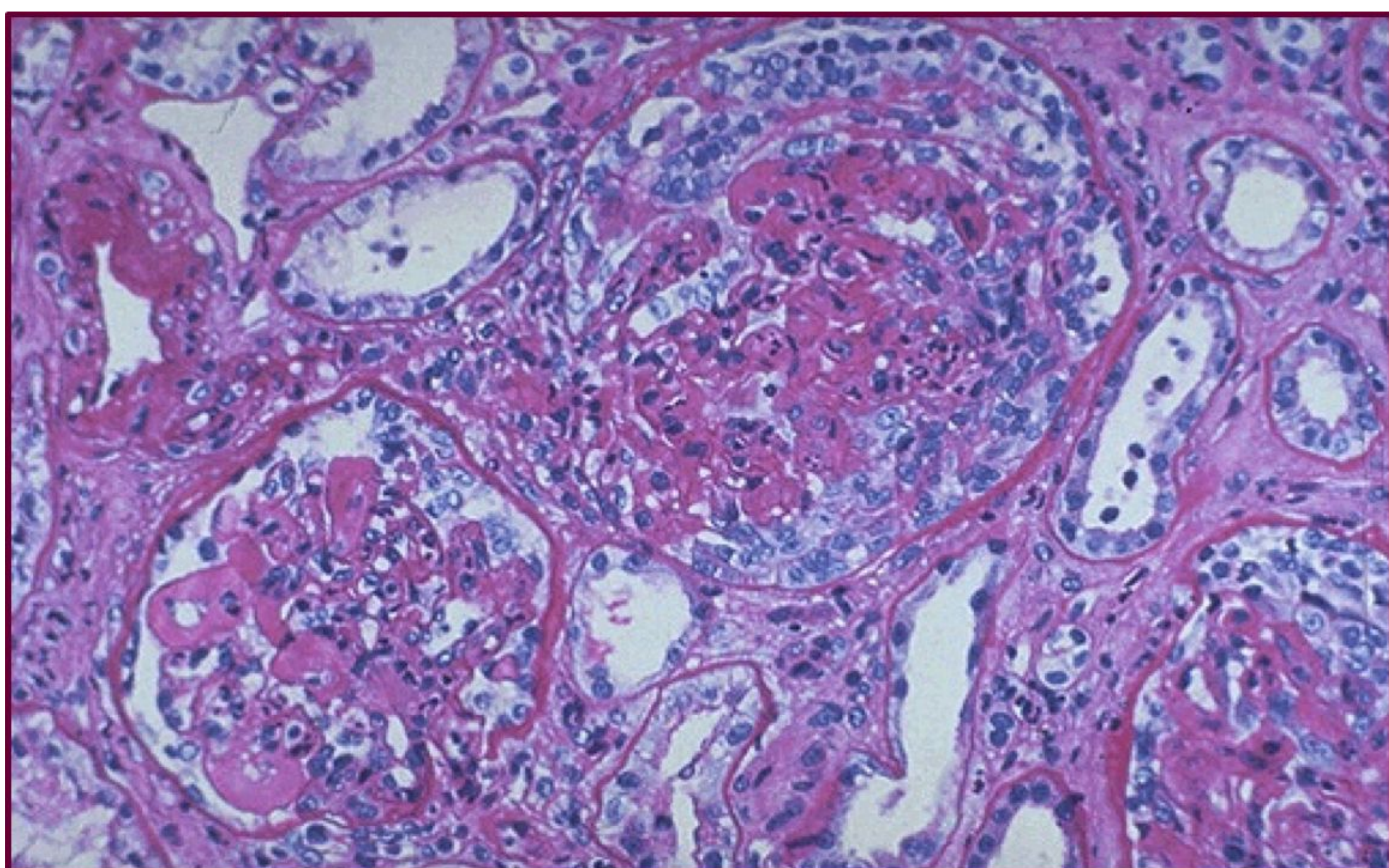
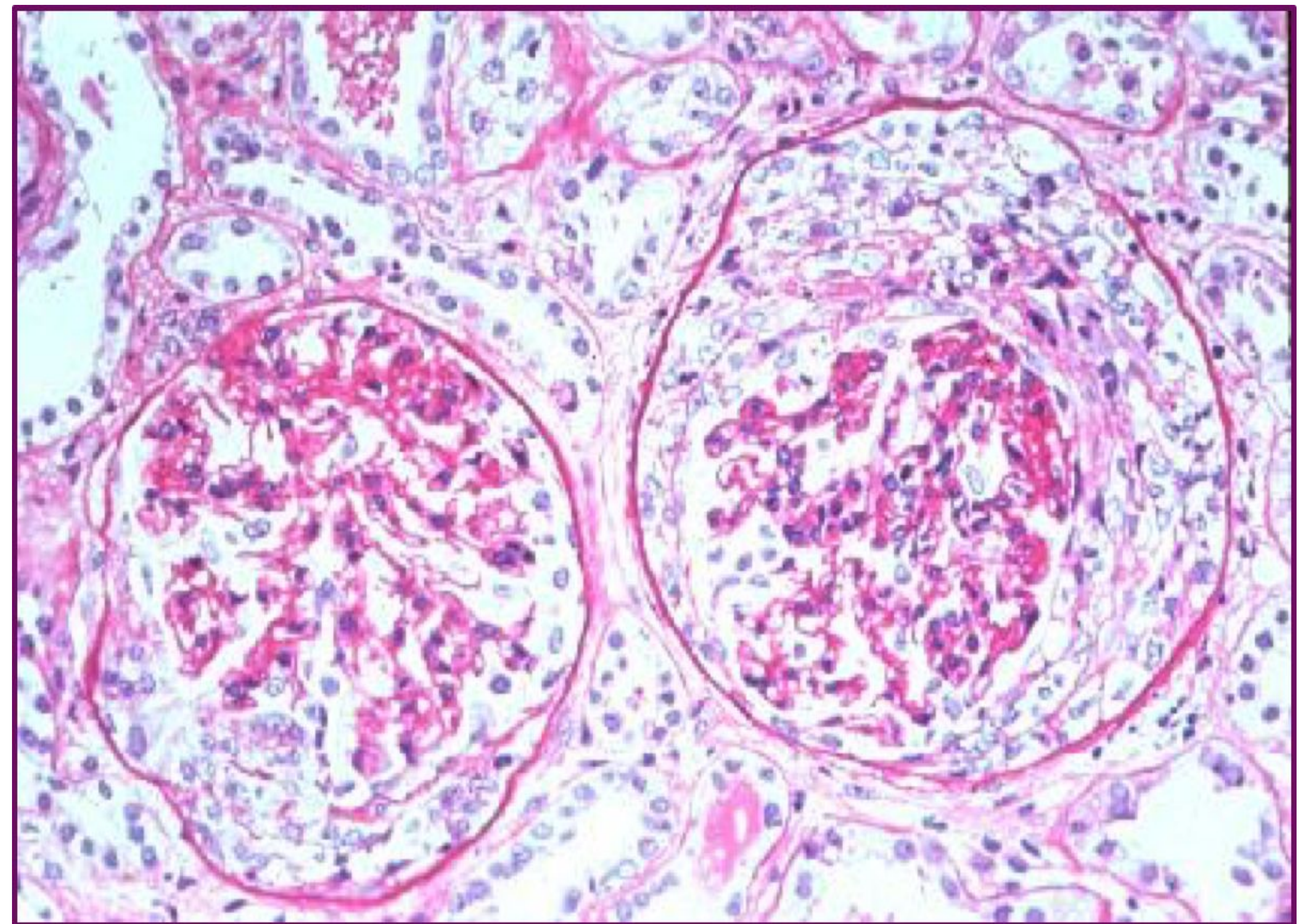
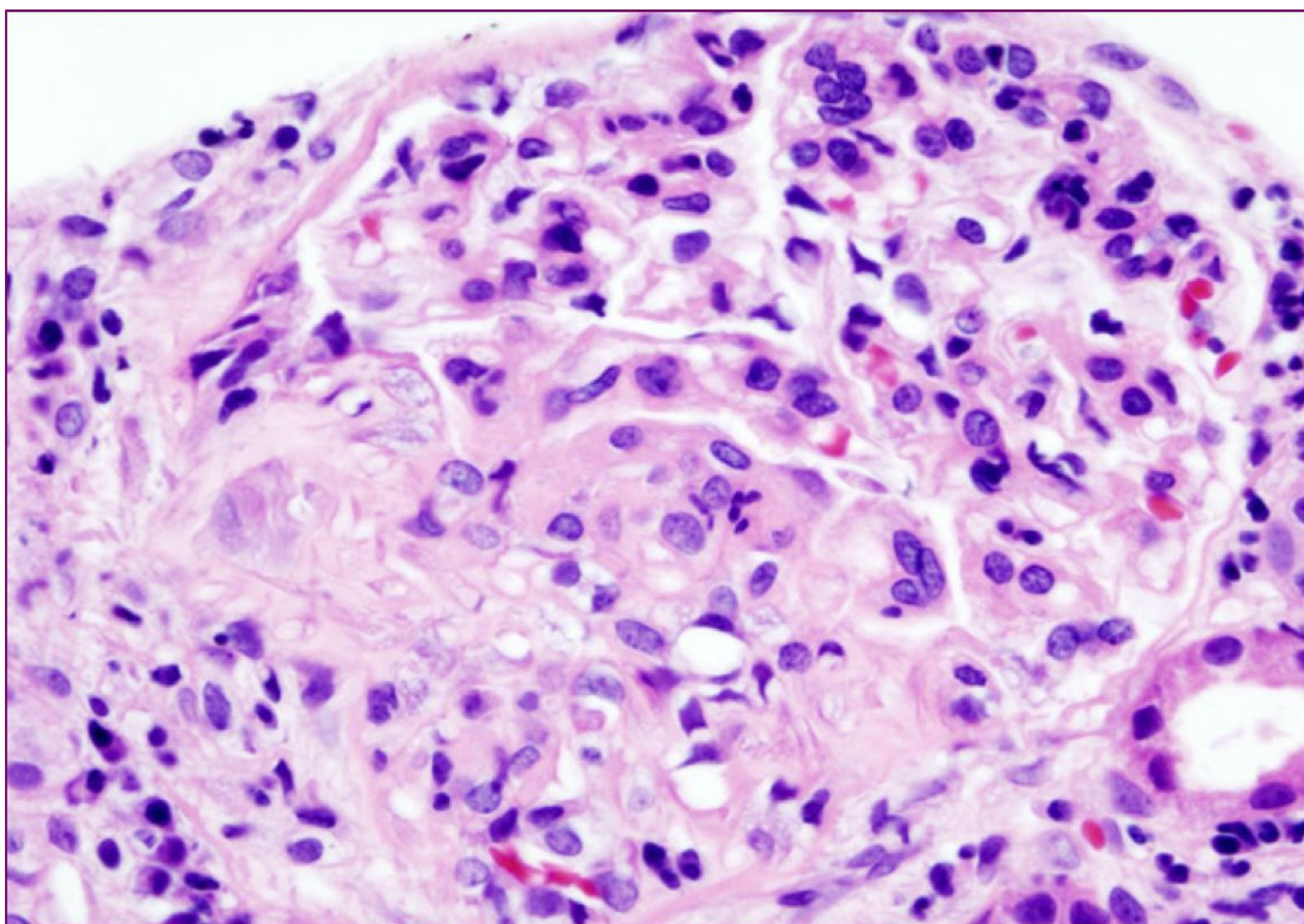
Clinical sign: **sever gross hematuria**.

## *Rapid Progressive Glomerulonephritis (RPGN)*



Gross appearance of RPGN note the flea beaten appearance

## Rapid Progressive Glomerulonephritis (RPGN)



**Crescents formation in BOWMAN'S CAPSULE** composed of:

- Proliferation of epithelial cells
- Monocytes and Macrophages

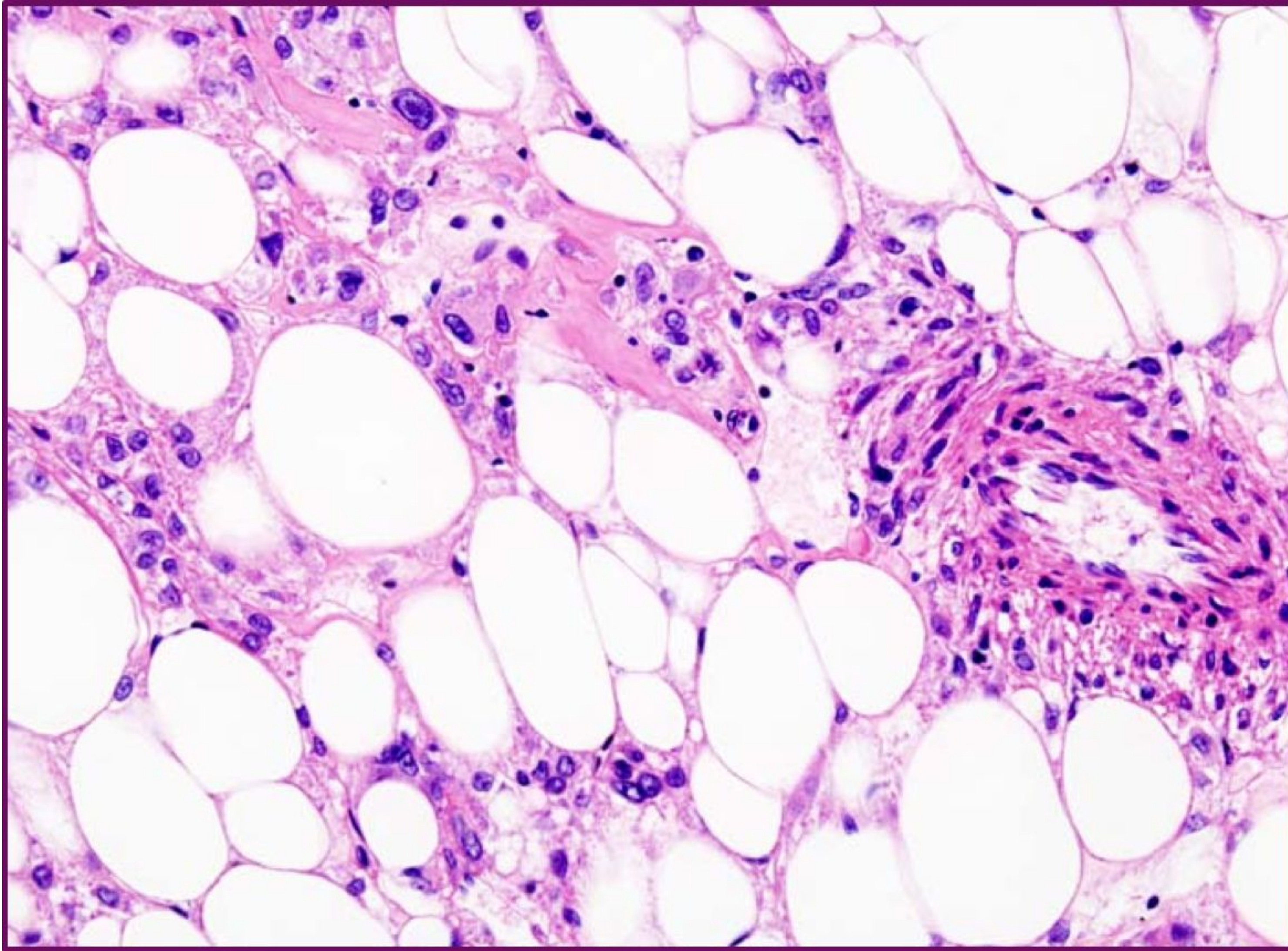
# #8 BENIGN RENAL TUMORS

Theoretical information:

RARE Tumors

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

## Angiomyolipoma



Benign tumor composed of

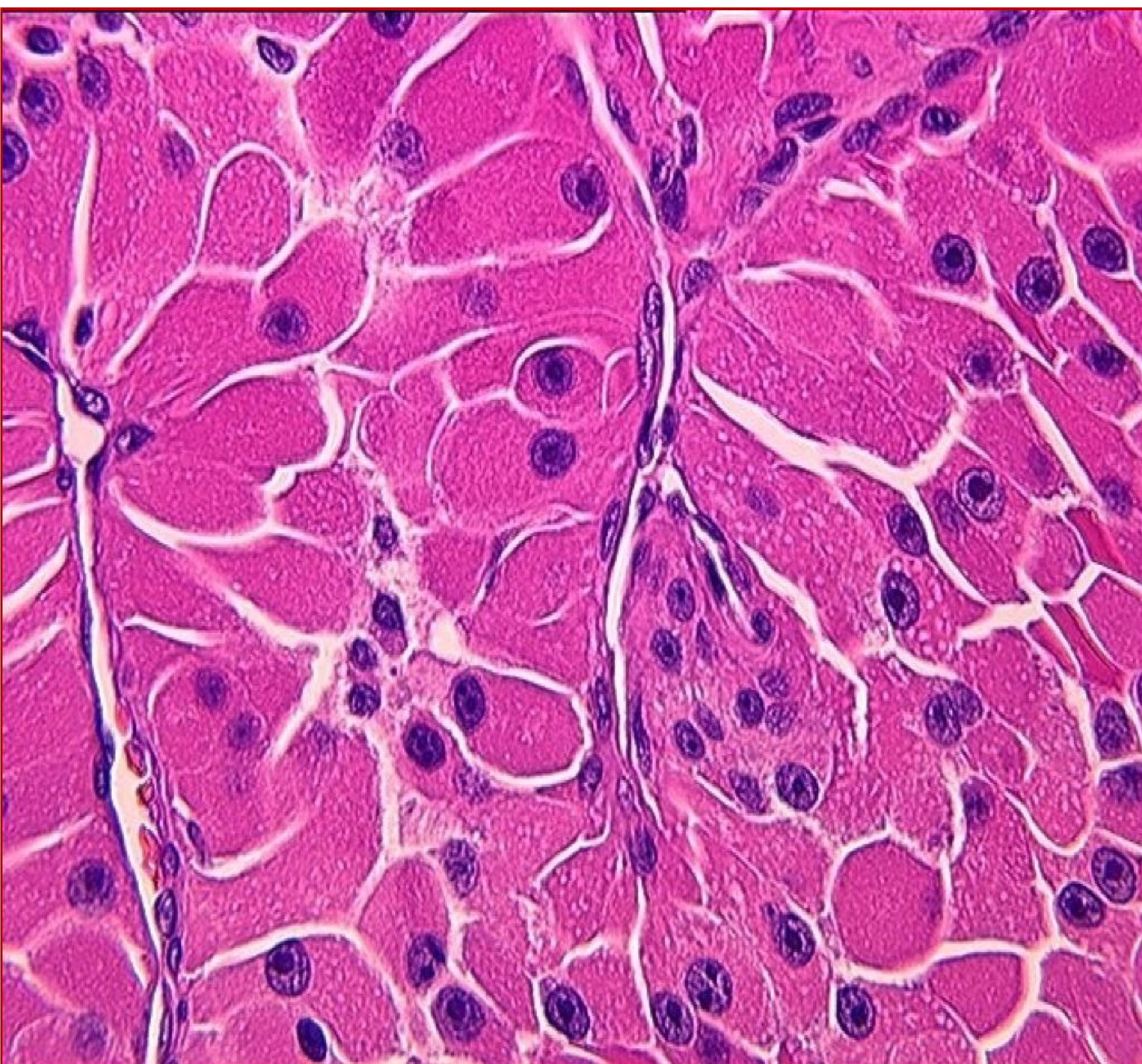
1- Vessels

2- Smooth muscle

3- Adipose tissue

angiomyolipoma associated with tuberous sclerosis

## Oncocytoma



- Oncocytes are very RED cytoplasm.
- Large eosinophilic cells.



- Rounded contour the mahogany colour.
- the central scar.

# #9 MALIGNANT RENAL TUMORS

Theoretical information:

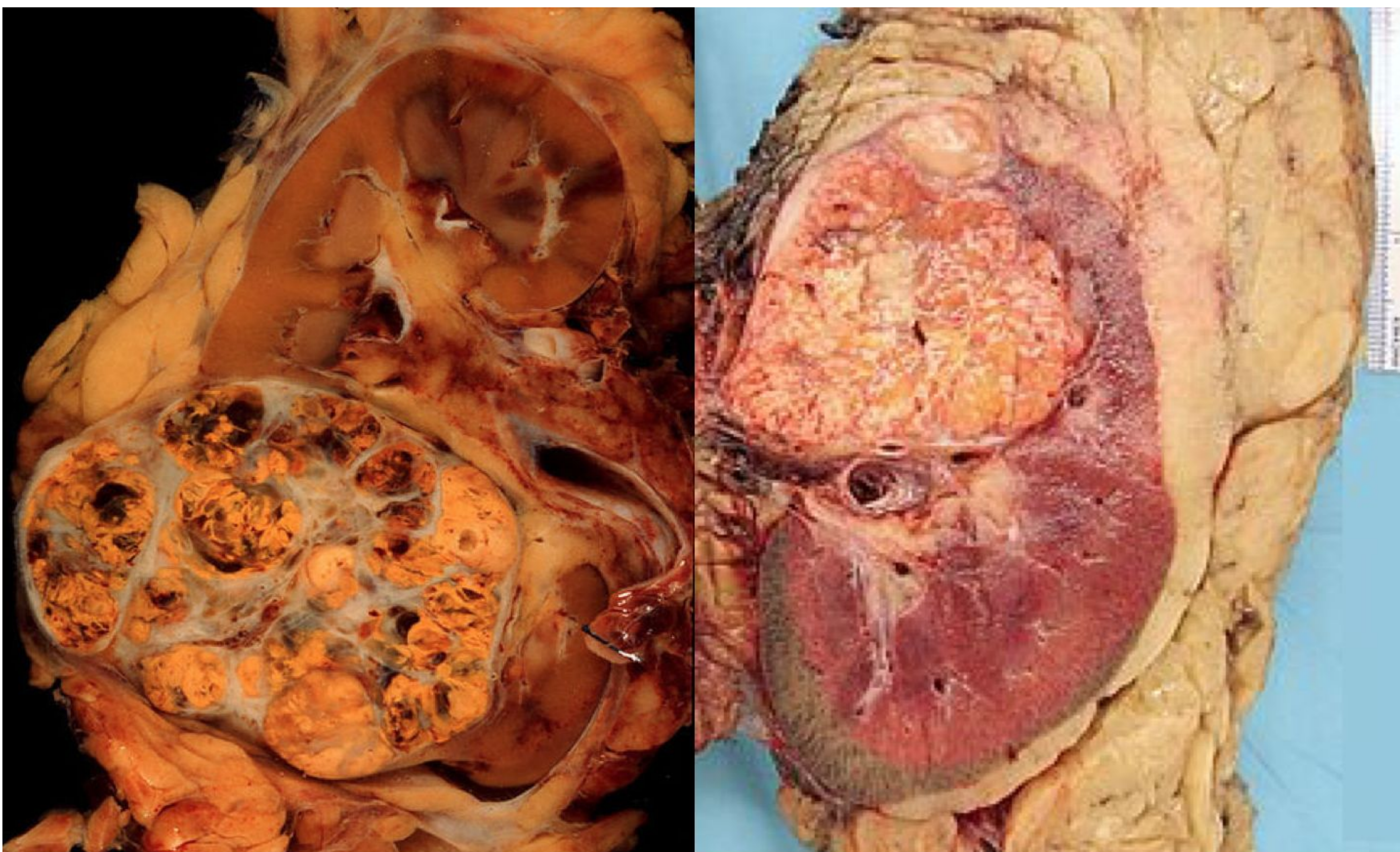
**Renal Cell Carcinoma:**

**Clear Cell Carcinoma, Adenocarcinoma, Hypernephroma Urothelial (Transitional).**

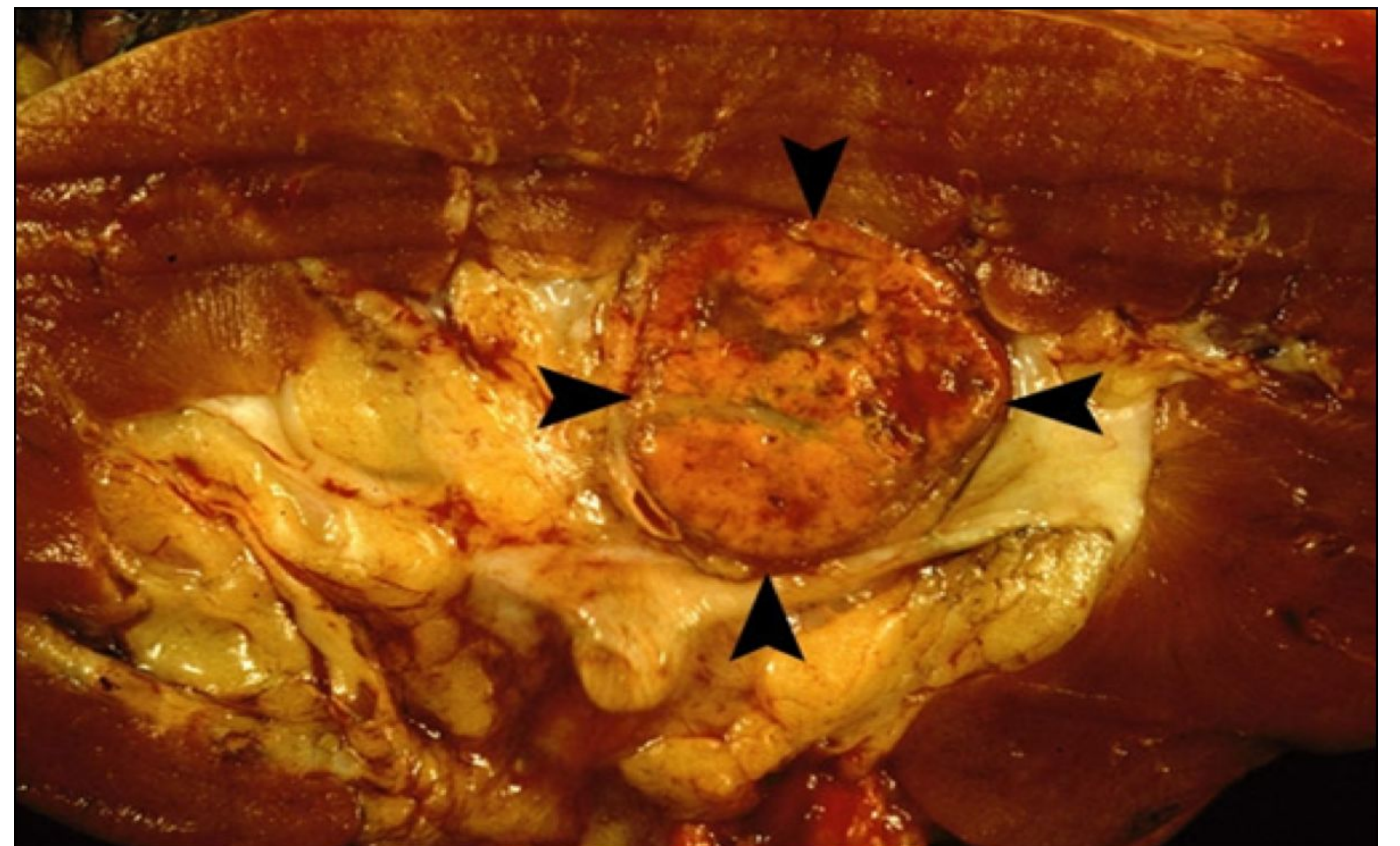
Risk Factors: **aniline dye, immunosuppression, smoking..**

- Gene which may be responsible for this condition: **VHL gene on chromosome 3.**
- The most common type of renal cell carcinoma: **(clear cell carcinoma)**
- Patient presents with: **hematuria, flank pain and palpable mass (abdomen).**

## Renal Clear Cell Carcinoma – Gross pathology

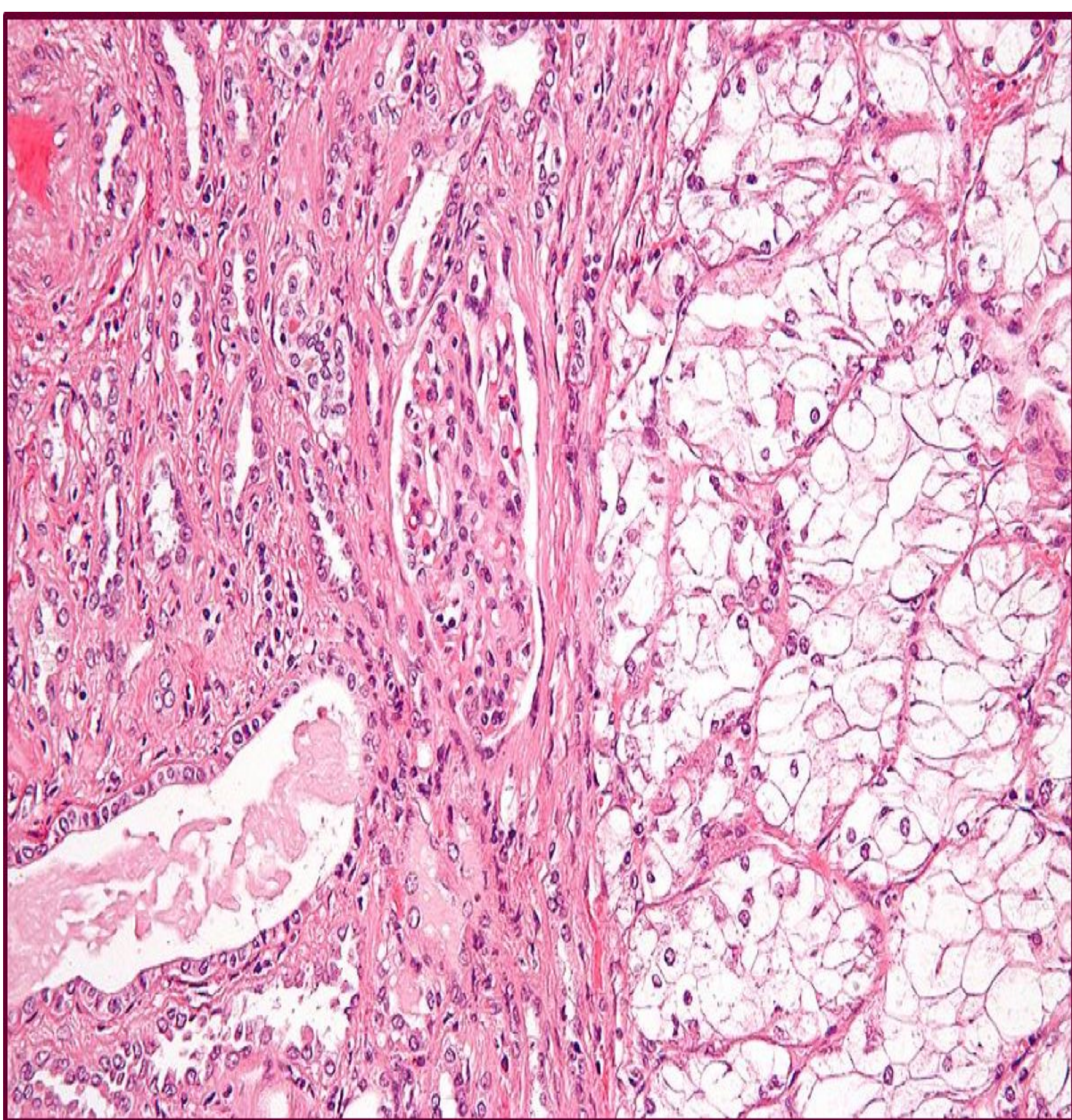


**A well circumscribed renal cortical mass which is partly yellow fat and necrosis and hemorrhage with lobulated cut surface .**

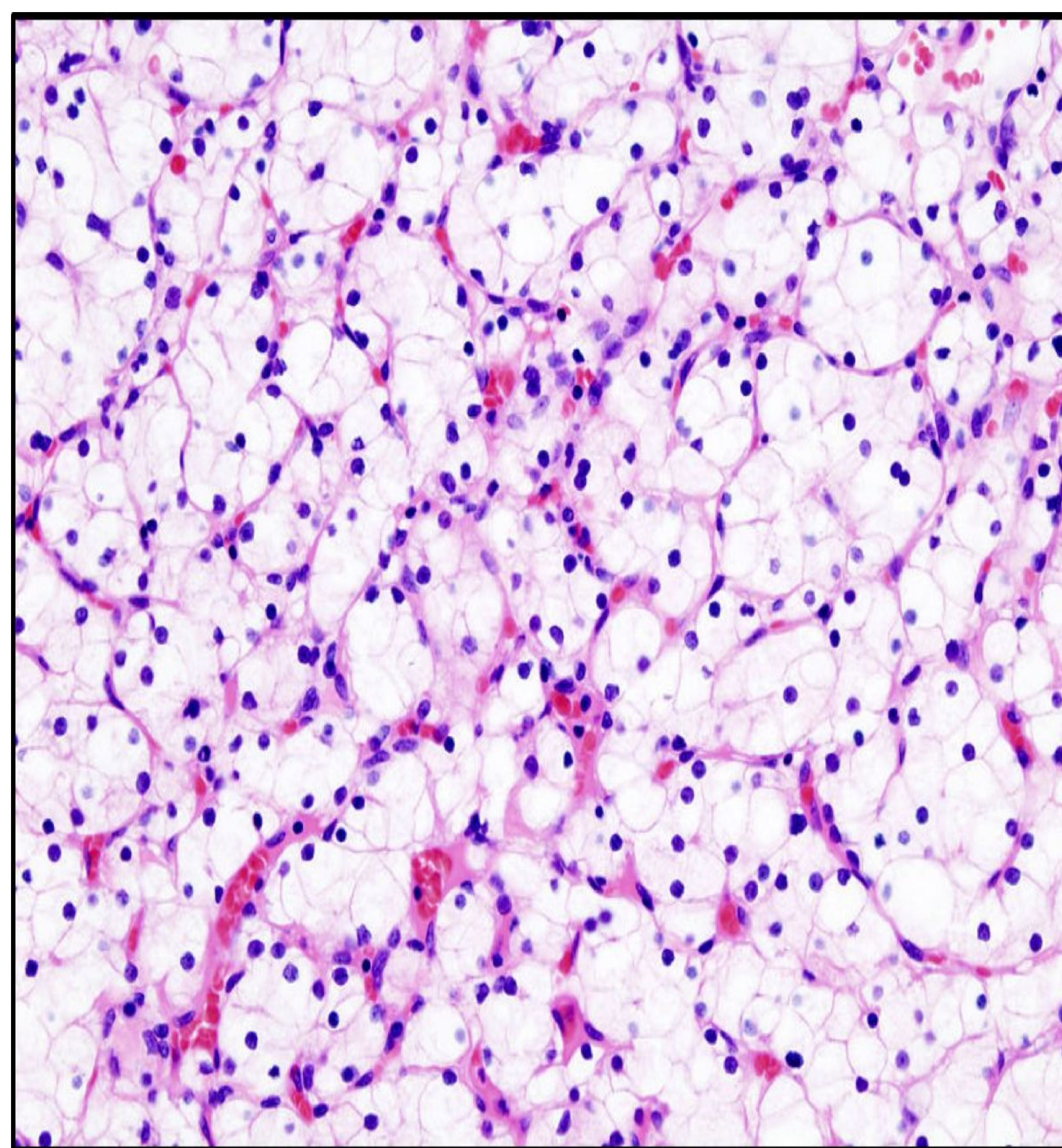


The tumor is well demarcated from the surrounding non-neoplastic renal parenchyma by a pseudocapsule

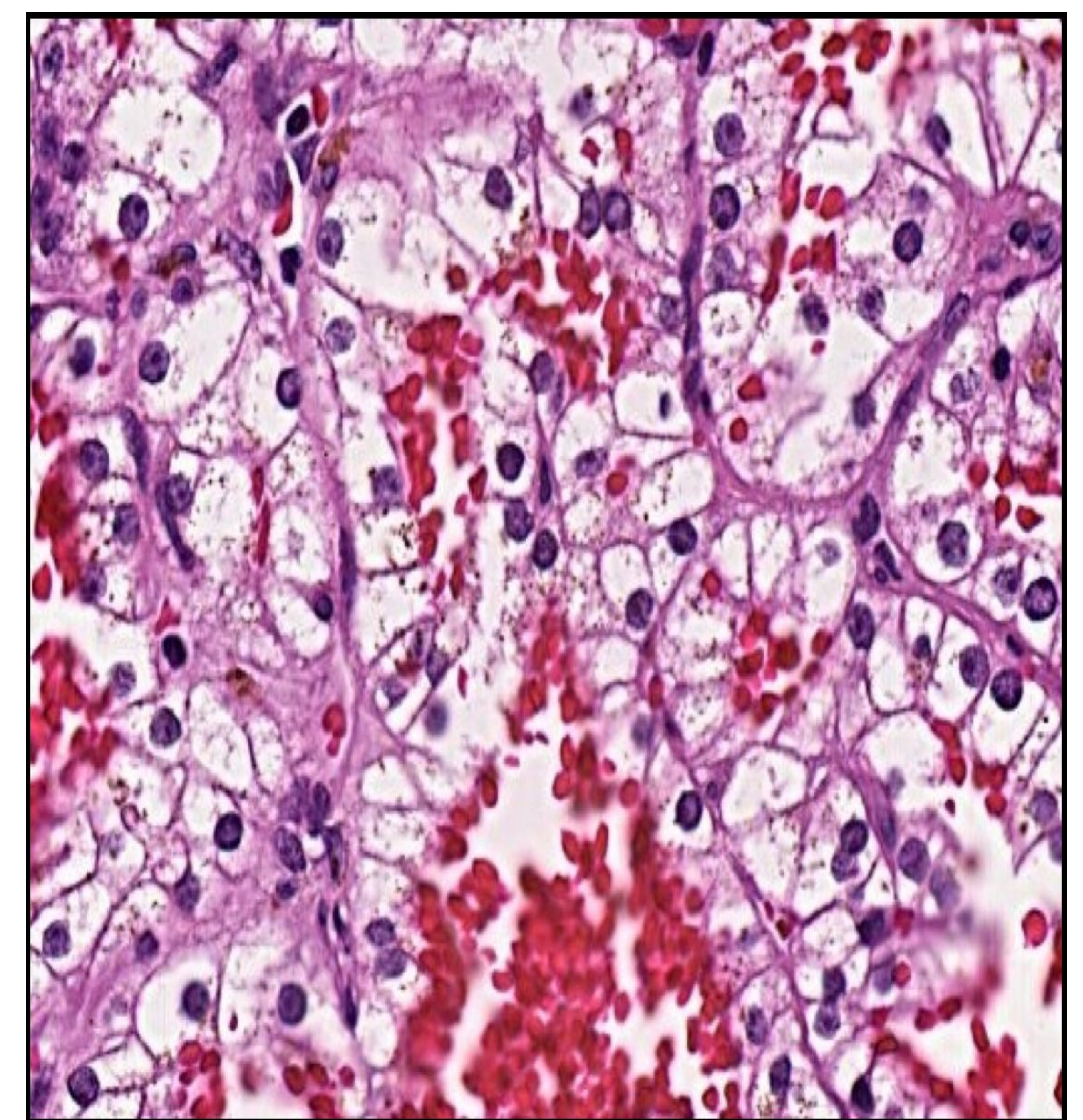
## Renal Clear Cell Carcinoma - Histopathology



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



Tumor cells are large polygonal with clear cytoplasm (dissolved glycogen and lipid) and piknotic nuclei.  
- Cells show pleomorphism and mitosis.



- **Chicken wire appearance**
- **Hemorrhage**
- **Large nuclei with prominent nucleoli**

# #10 WILM'S TUMORS

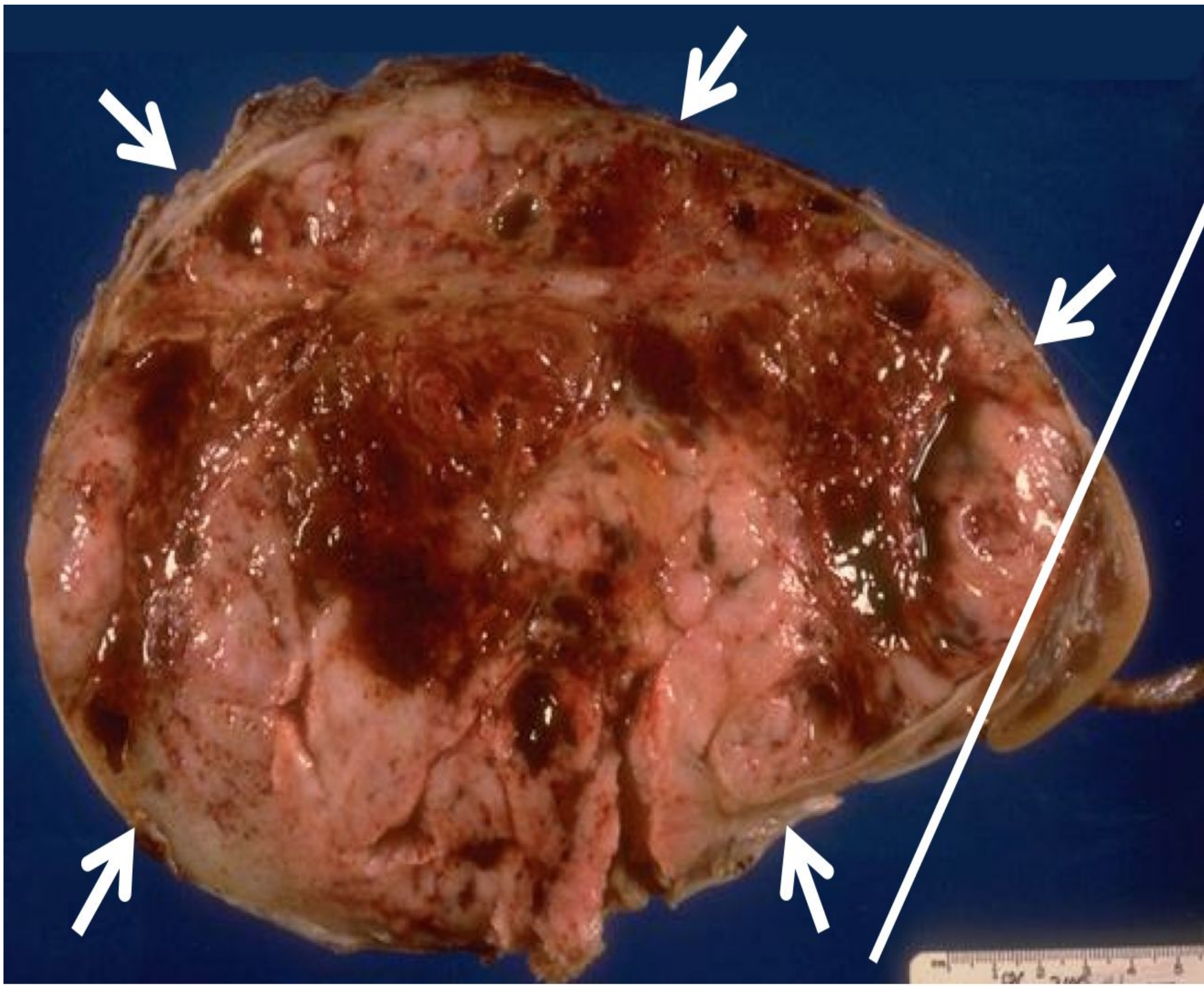
Theoretical information:

Wilm's tumor usually affect small children.

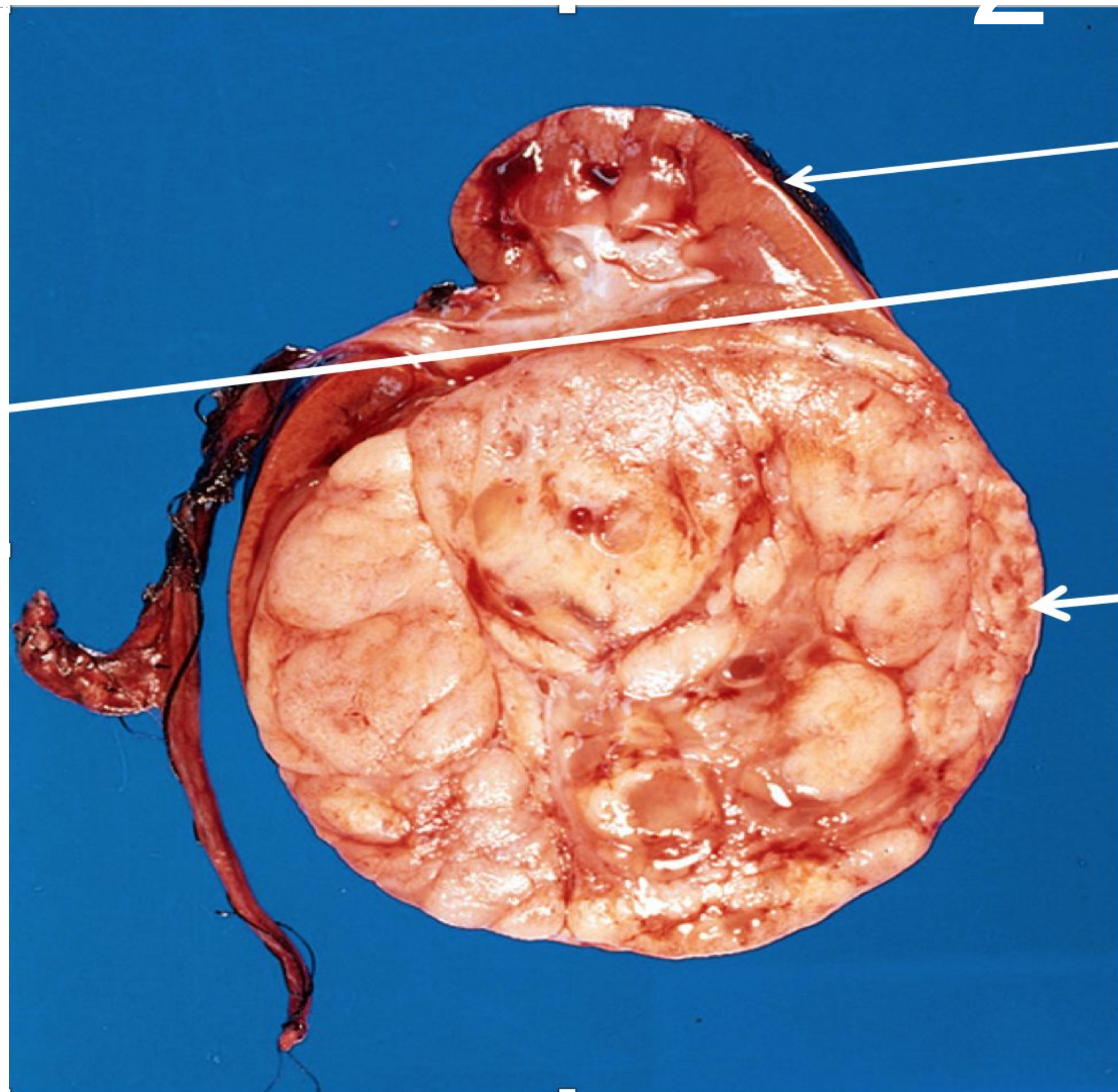
Good prognosis when diagnosed in he's 1st year.

common presentation: flank pain, abdominal mass

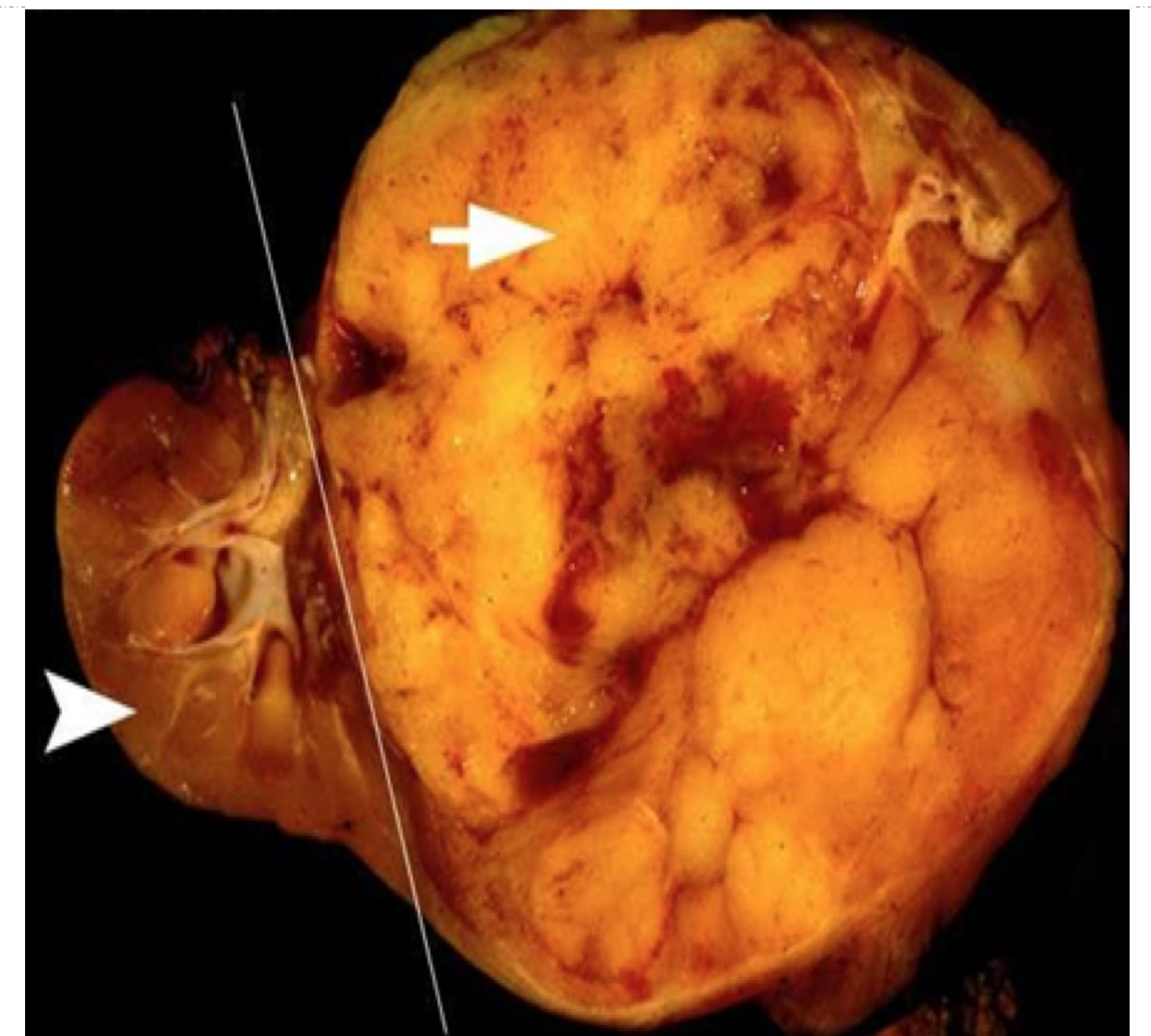
## Wilm's Tumor – Gross Pathology



- Huge hemorrhagic solid mass in the center
- Normal renal tissue in the periphery

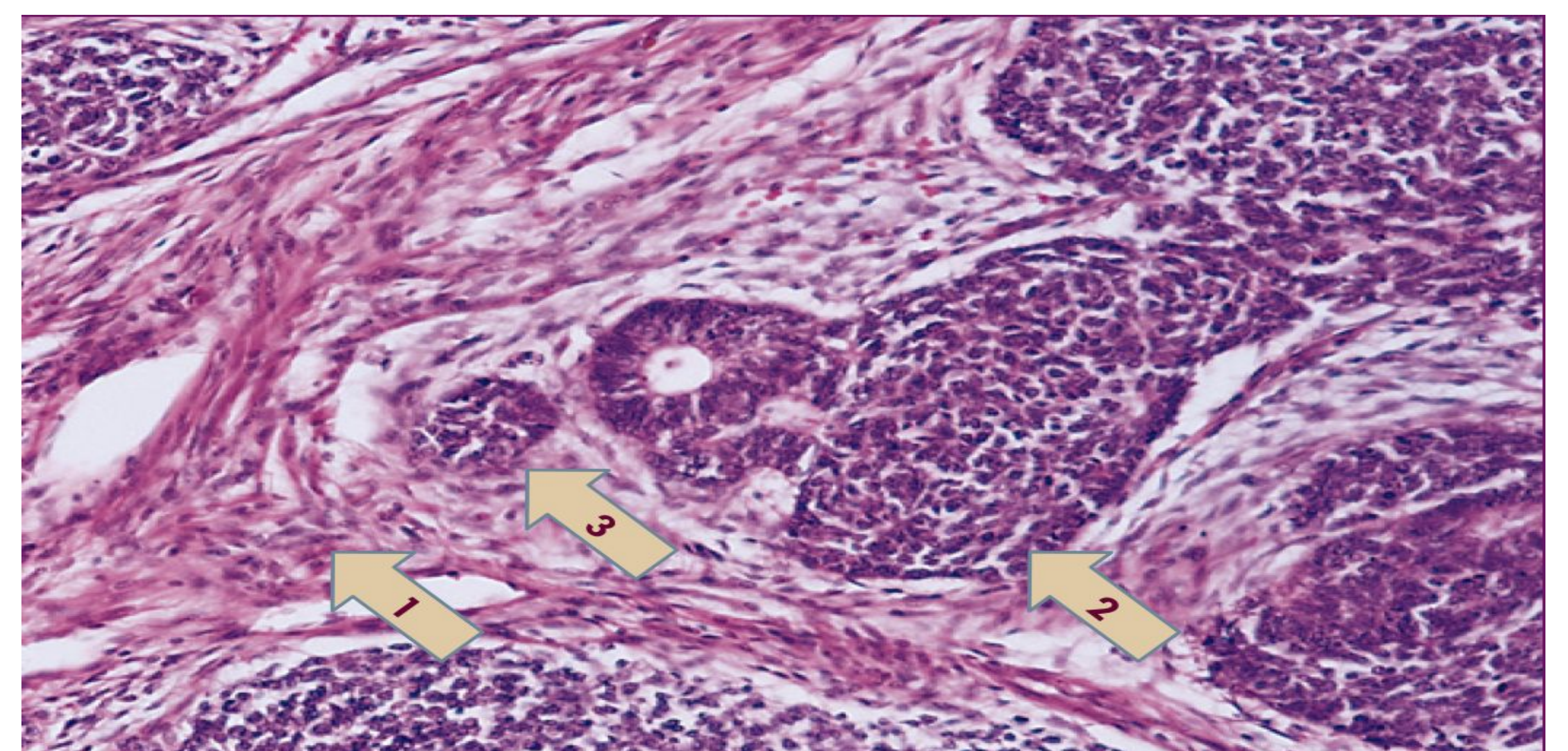
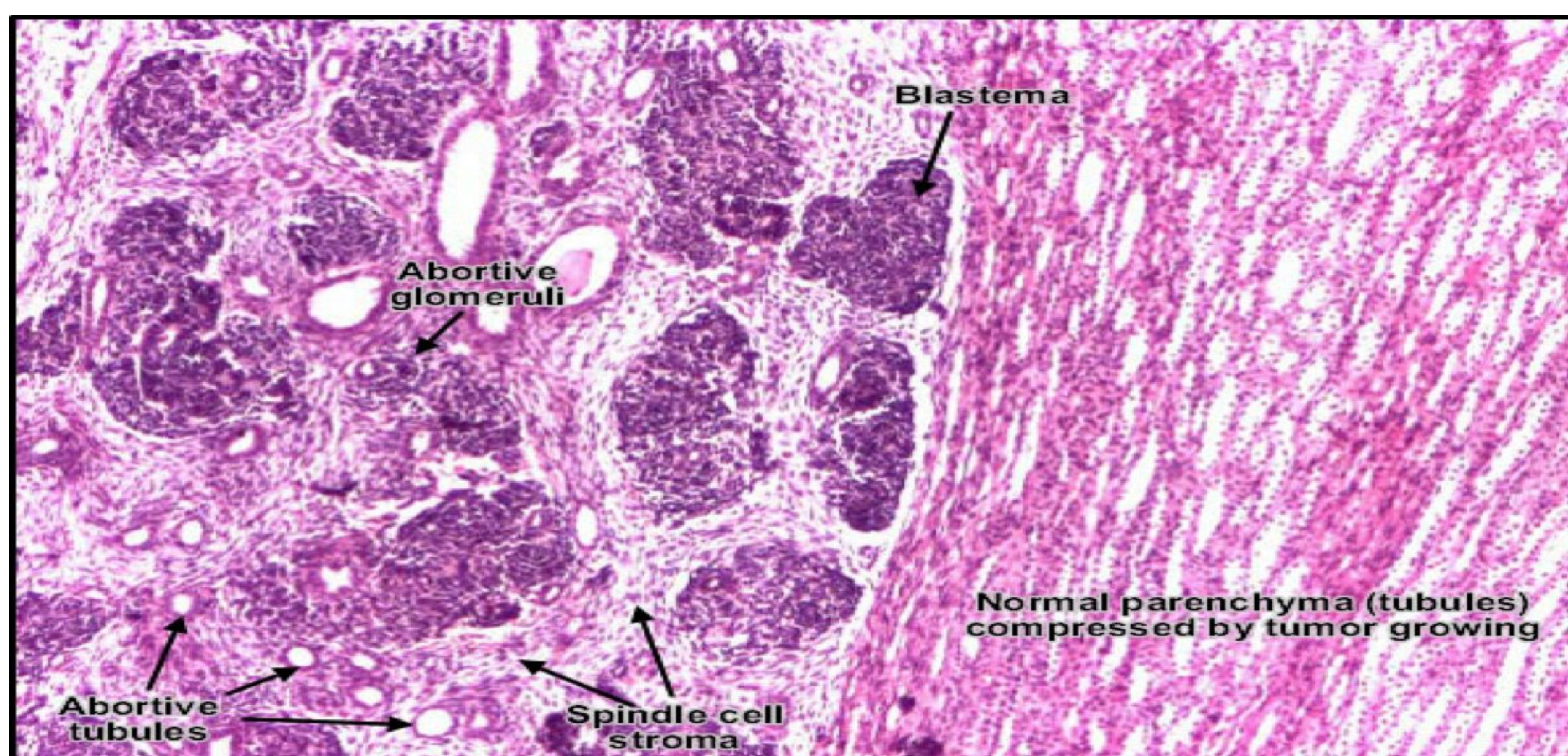
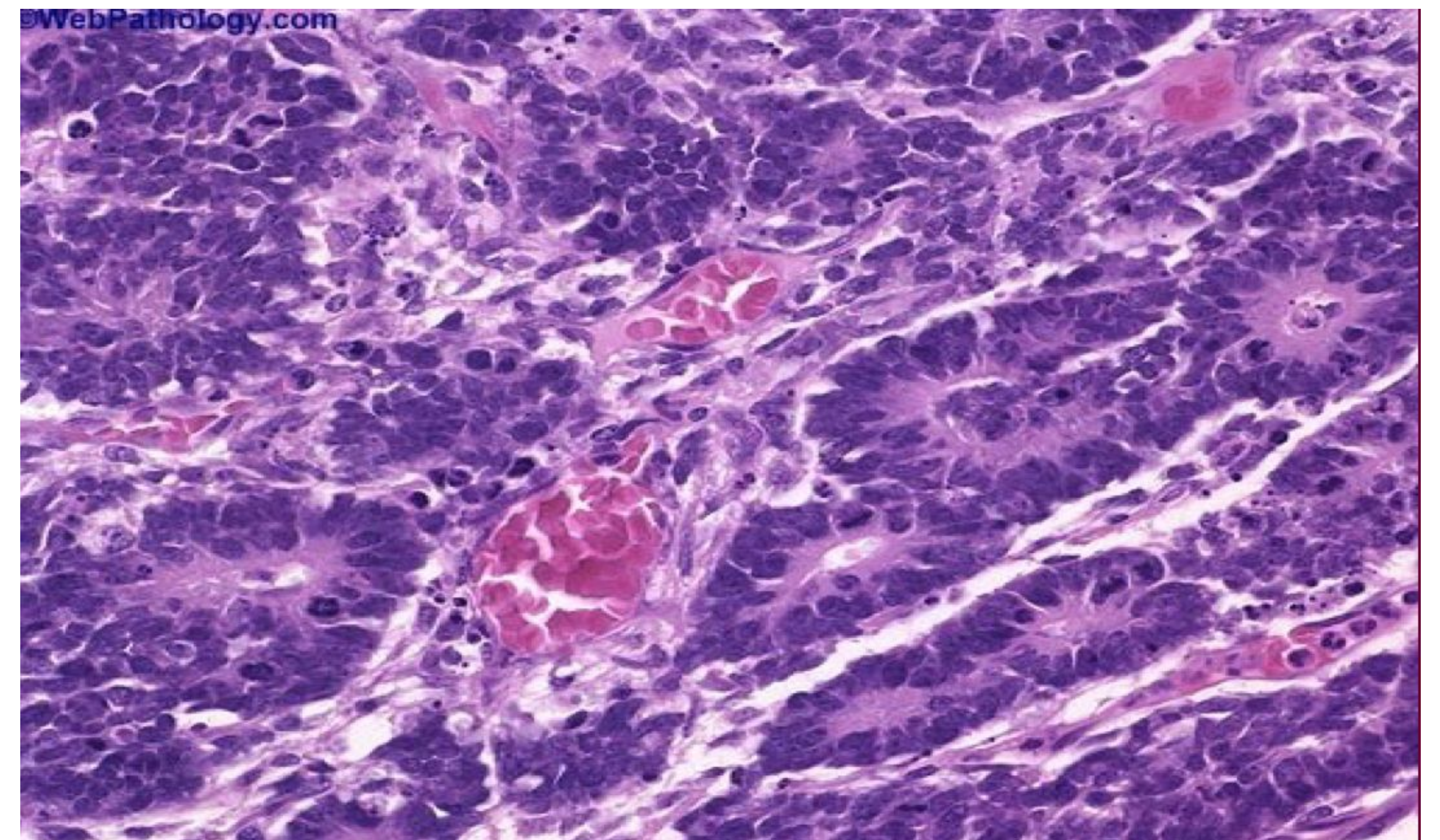
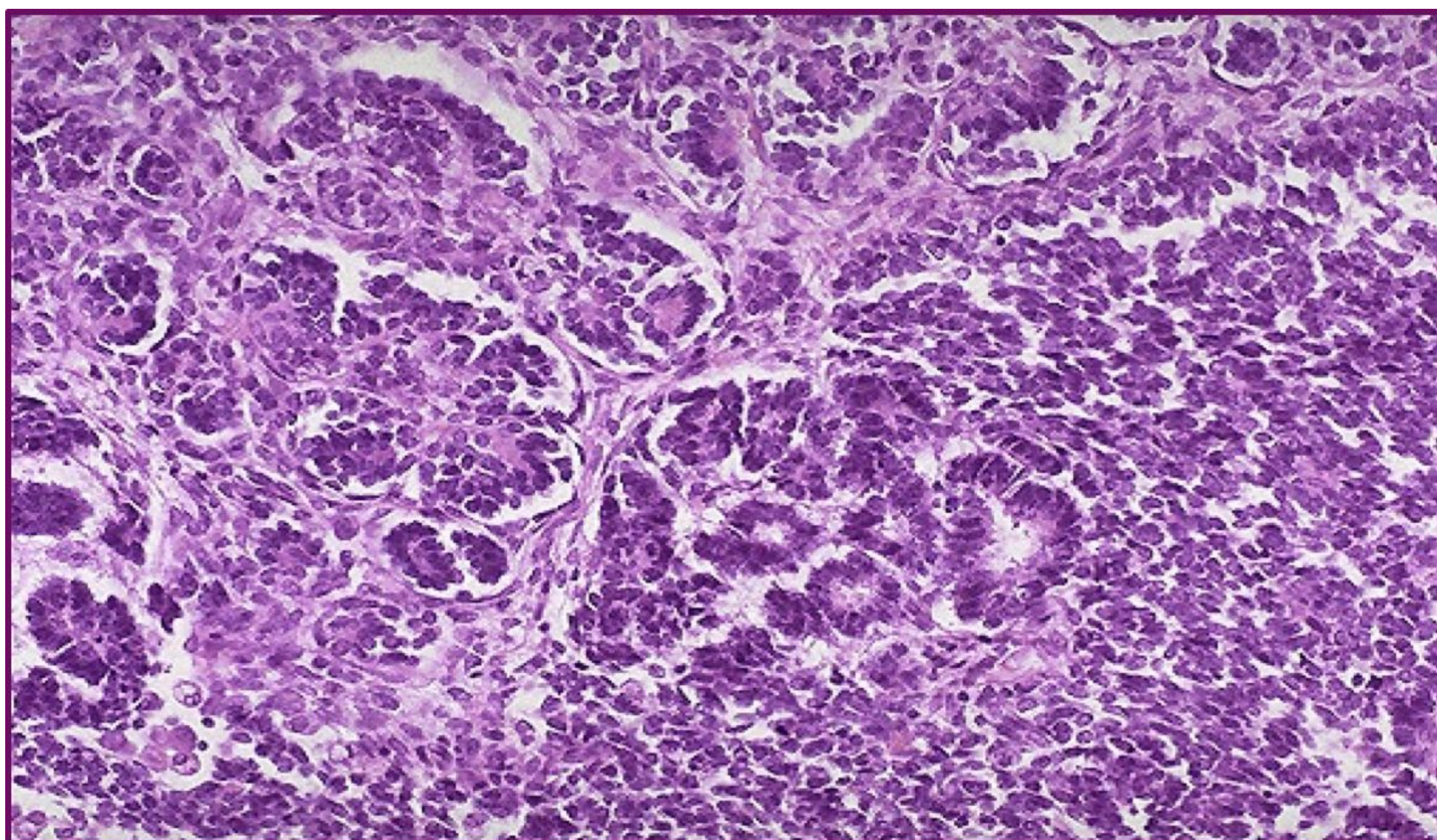


- 1-Remnant Kidney
- 2-Wilm's Tumor



- Hemorrhagic whitish solid mass
- necrosis

## Wilm's Tumor – Histopathology



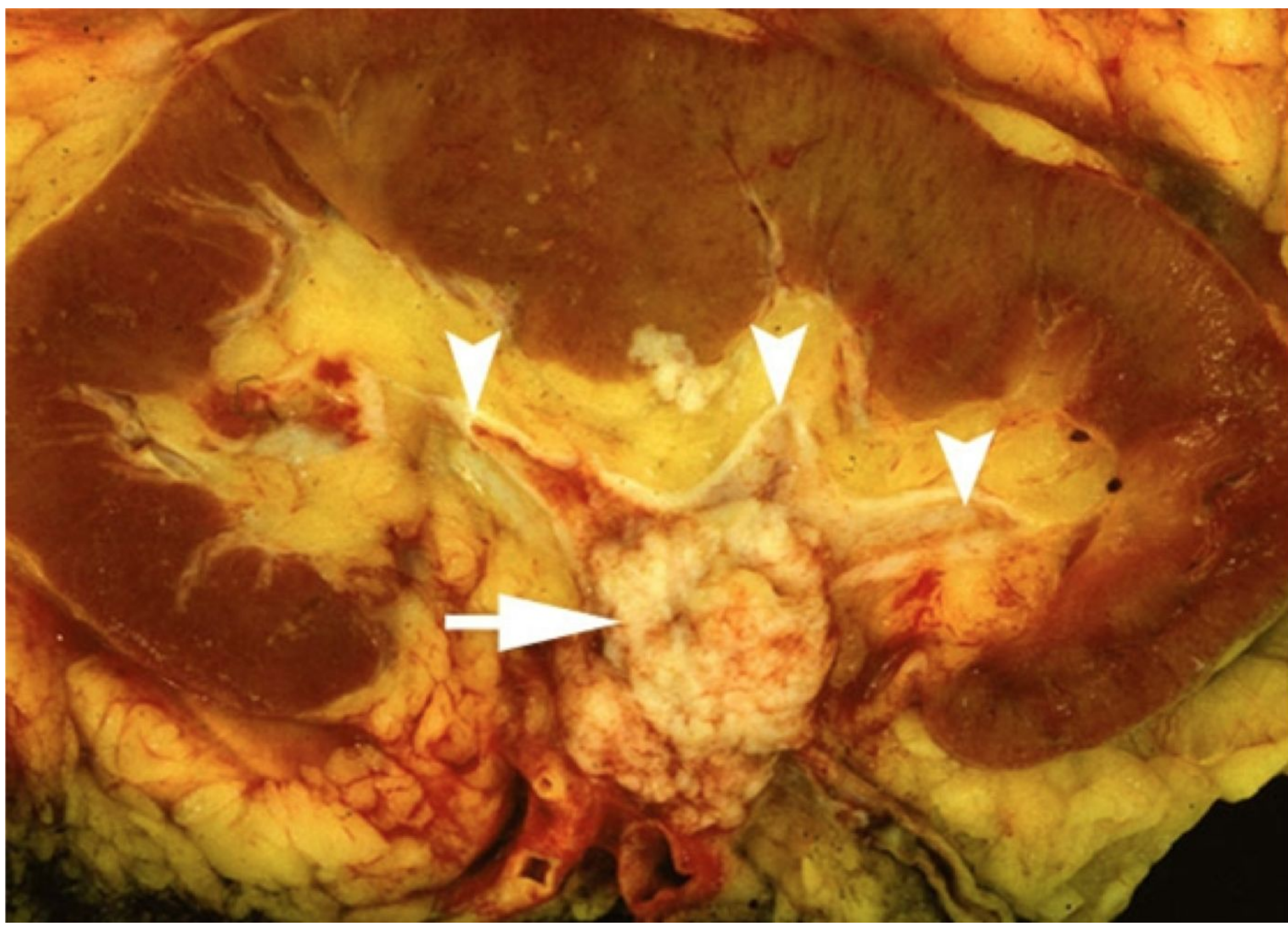
- 1.Spindle cell stroma. 2.Blastema. 3.Abortive glomeruli.

# #11 Carcinoma of Renal Pelvis and Ureter

Theoretical information:

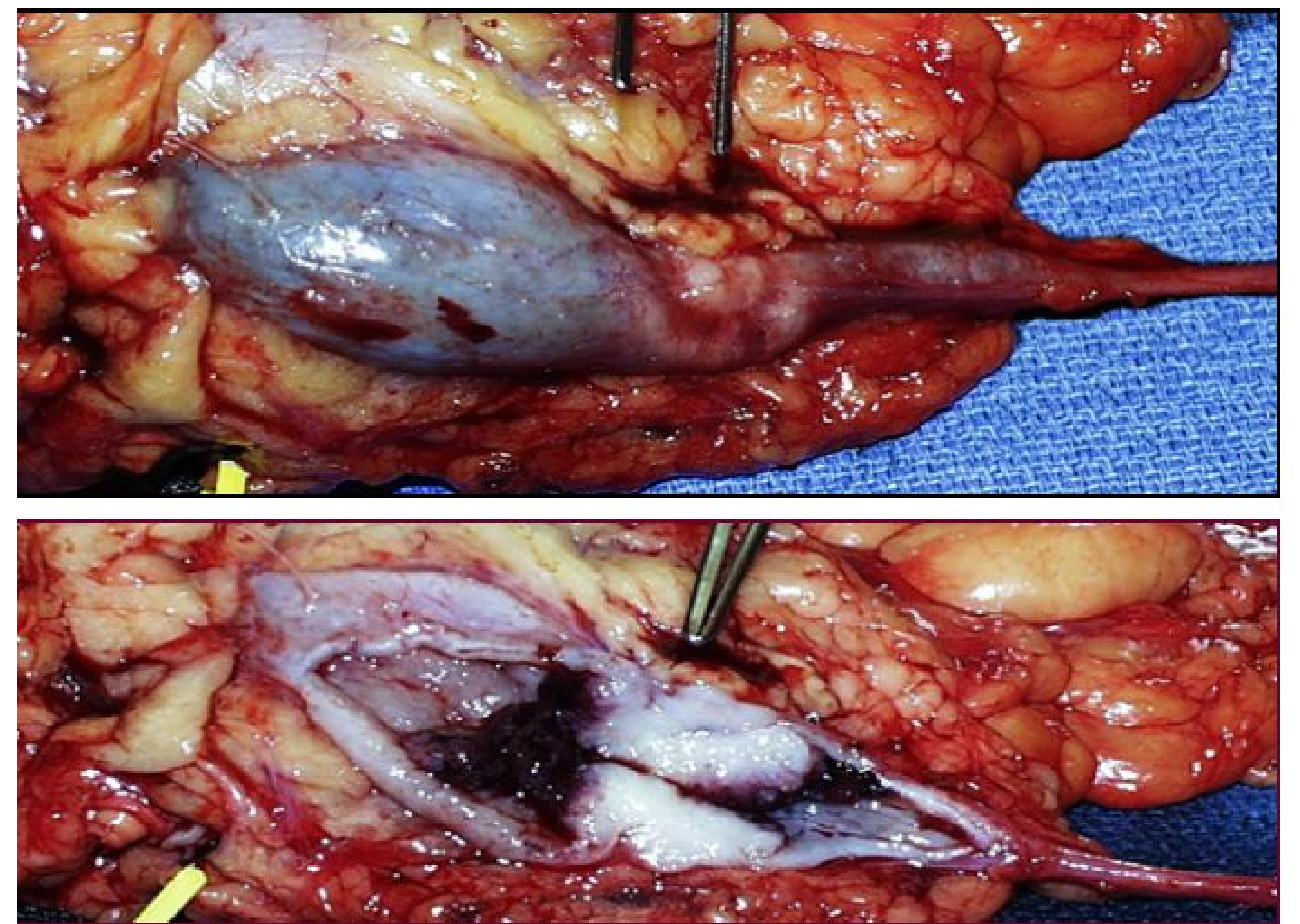
- Renal pelvis carcinoma prognosis is more worse than urothelial carcinoma of the bladder.
- 

## Urothelial (Transitional) Carcinoma of **Renal Pelvis**



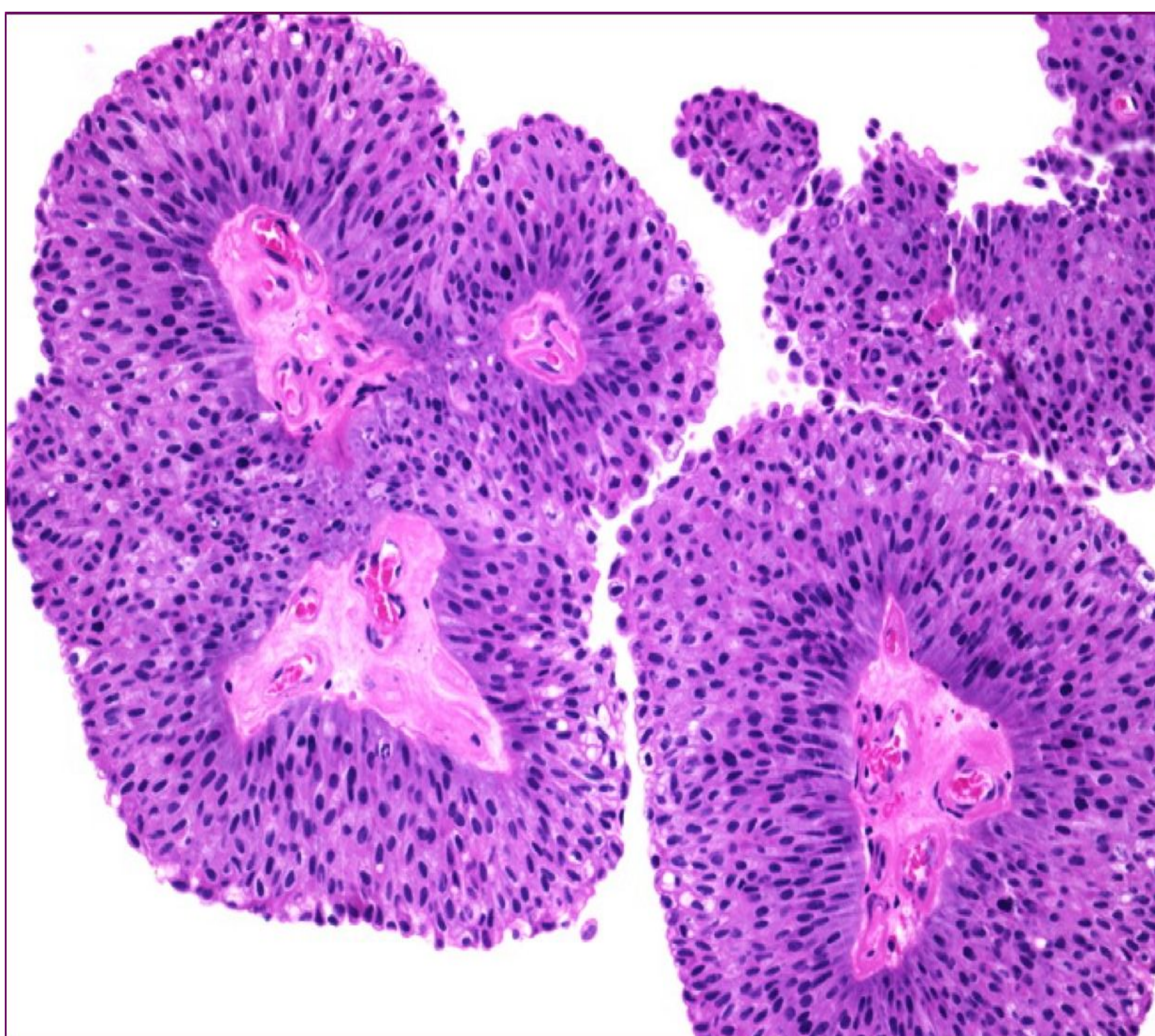
More commonly infiltrative

## 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 ( finger like projection )
- Adjacent papillary fronds surrounded by transitional epithelium

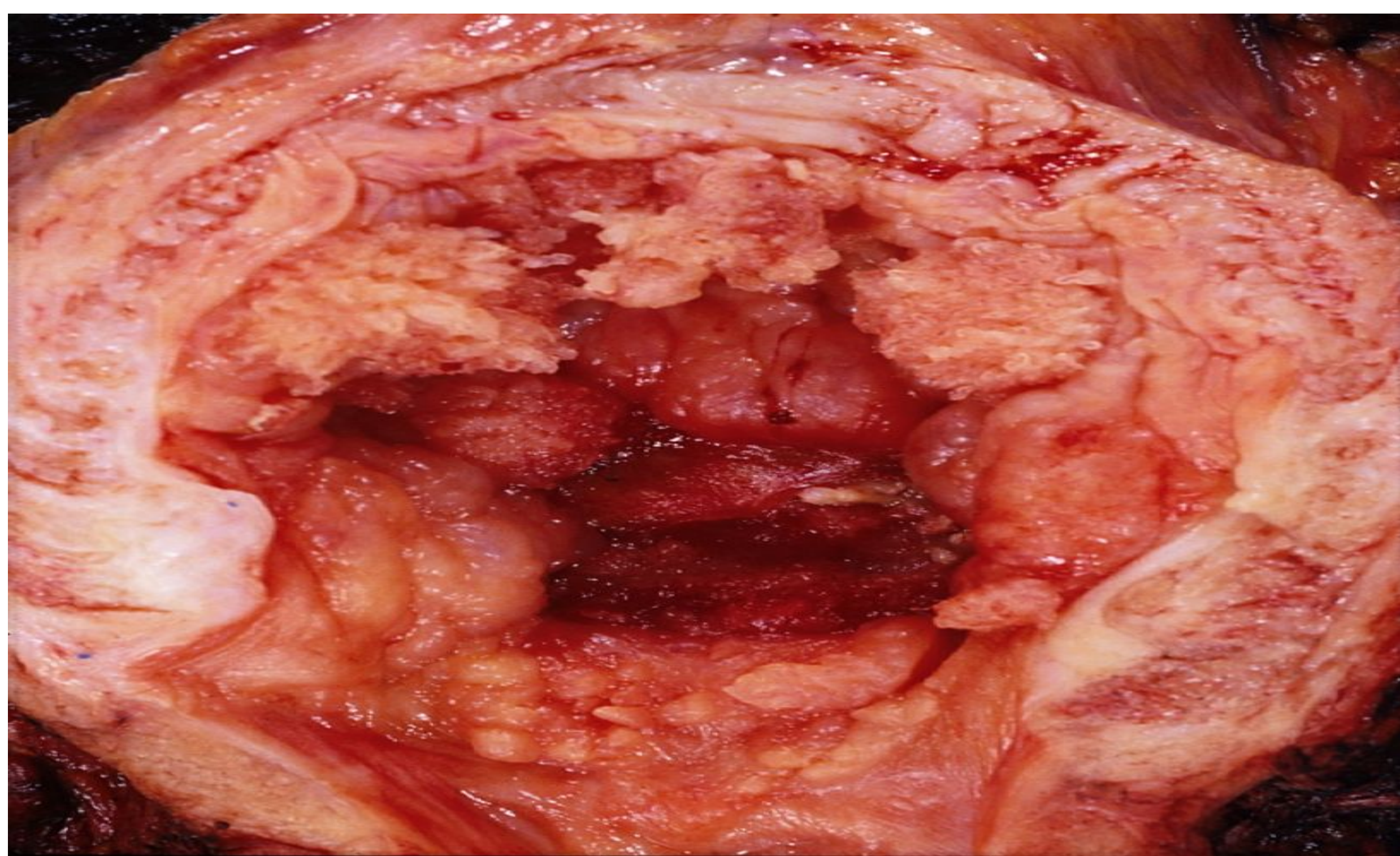
# #12 CARCINOMA OF THE URINARY BLADDER (1)

Theoretical information:

Risk factors for the development of papillary urothelial carcinoma of bladder:

- a- Exposure to aniline dyes. (before people were aware of it's dangerousness)
- b- Cigarette smoking. (common nowadays causing urothelial)
- c- Treatment with cyclophosphamide.
- d- Schistosoma haematobium infestation (Squamous cell carcinoma is the most common with patient presented with schistosoma. ).
- e- Persistent urachus.

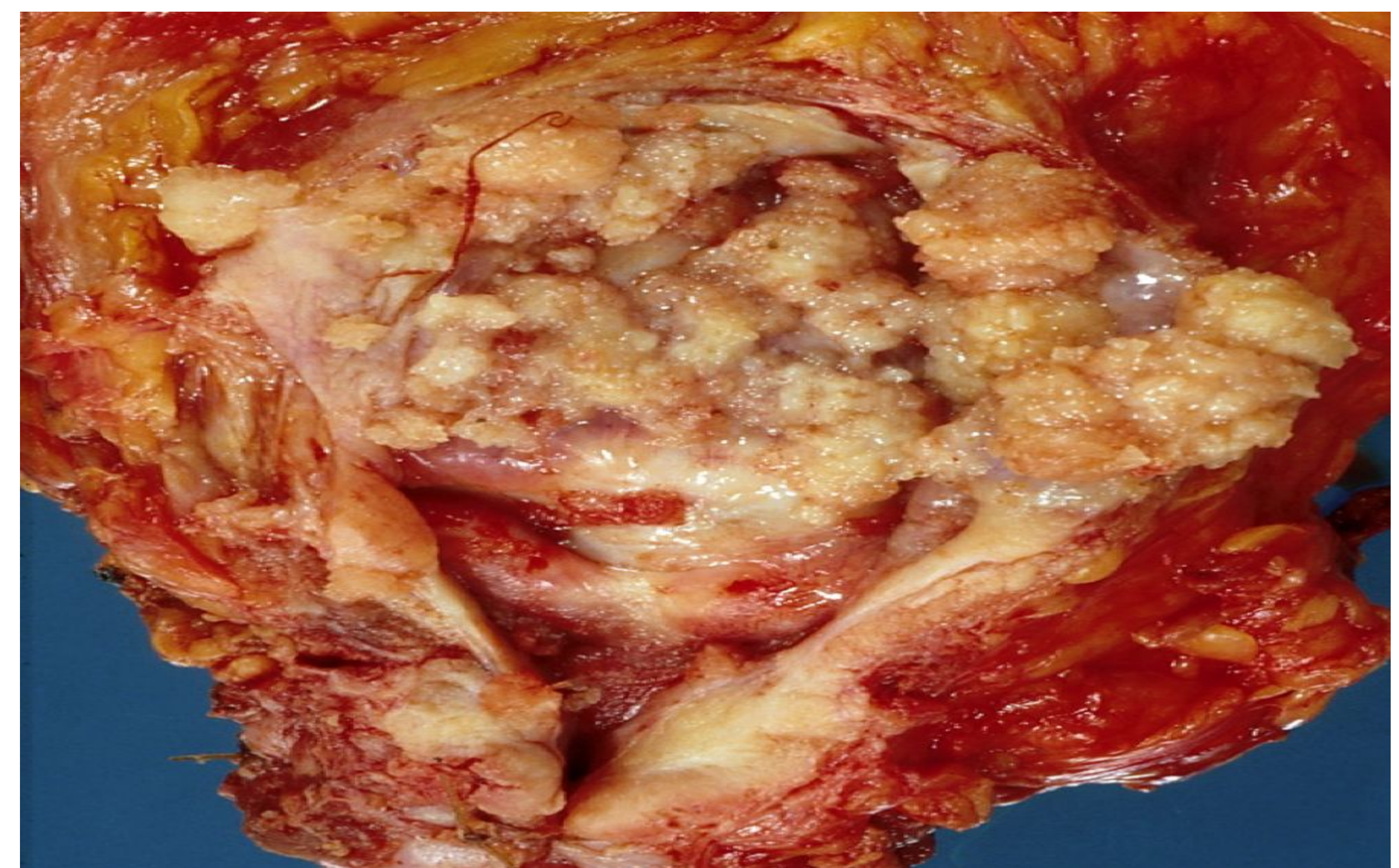
## Urinary Bladder Carcinoma Urothelial (Transitional cell) papillary Carcinoma - Gross



90% of bladder cancers are transitional cell carcinoma.

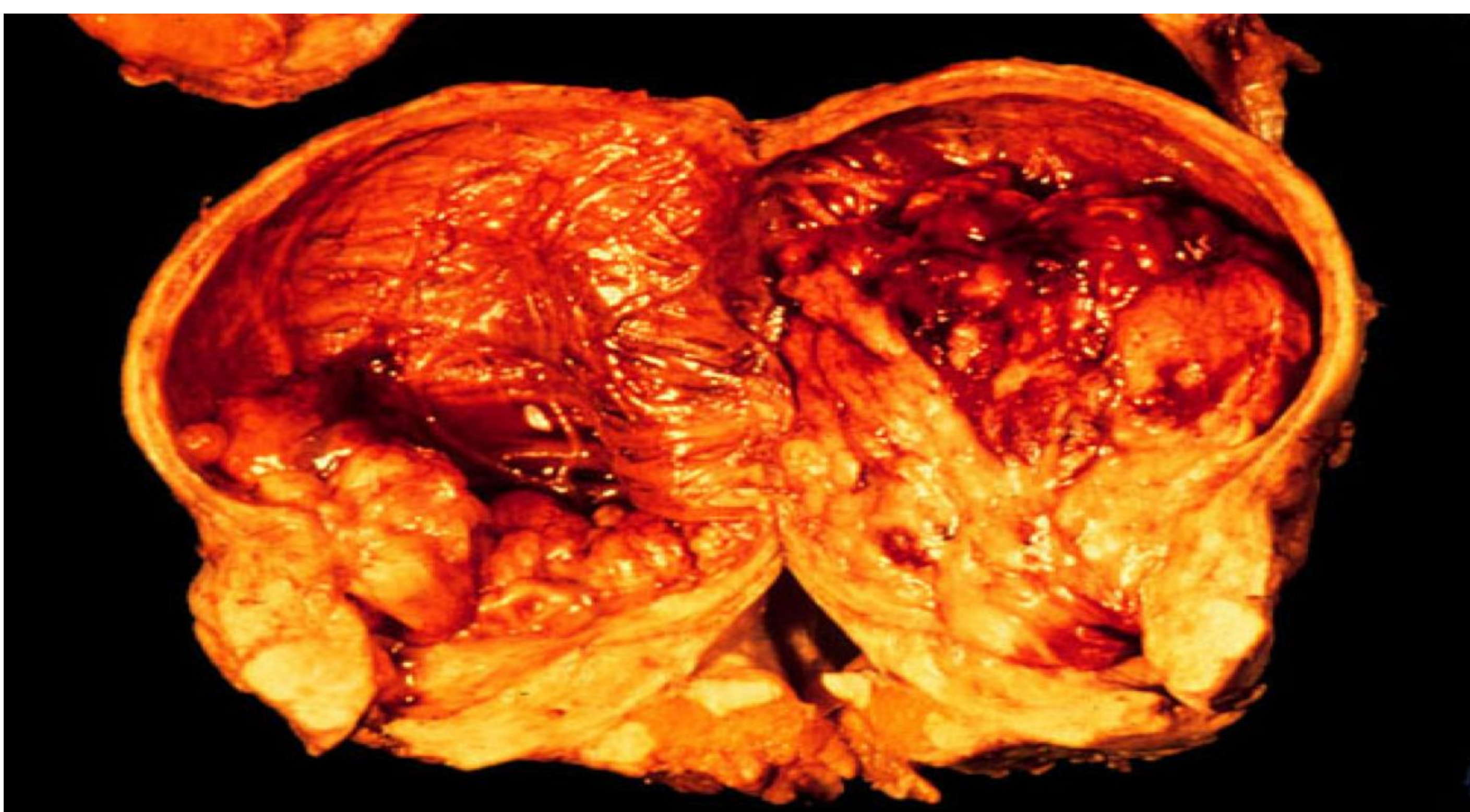
The other 10% are: squamous cell carcinoma, adenocarcinoma, sarcoma, small cell carcinoma, and secondary metastases.

## Papillary Urothelial Carcinoma of Bladder - Gross



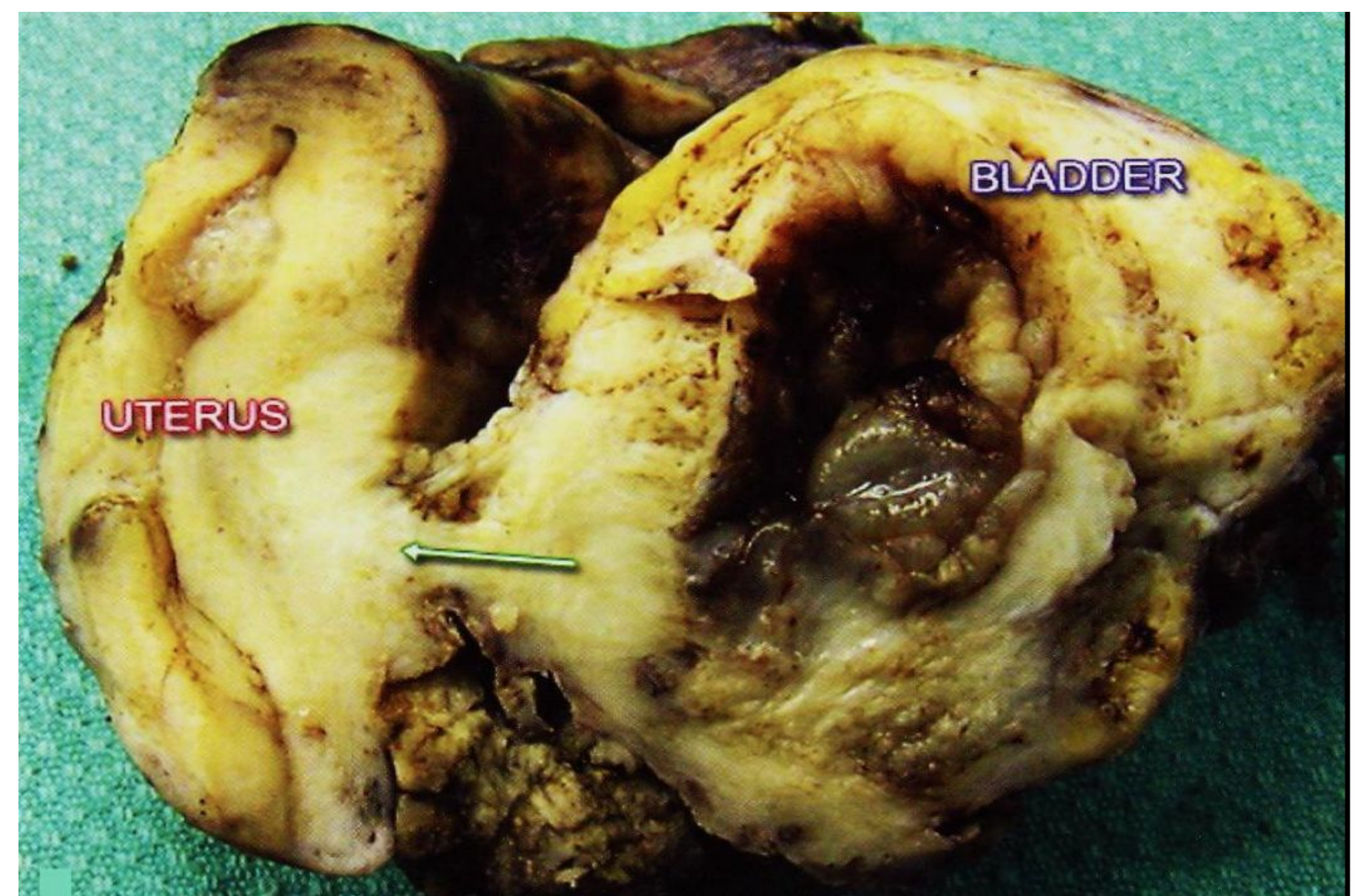
Bladder showing **multifocal** papillary mucosal neoplasm.

## Transitional Carcinoma of Bladder - Gross



The mucosa of the open urinary bladder wall 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



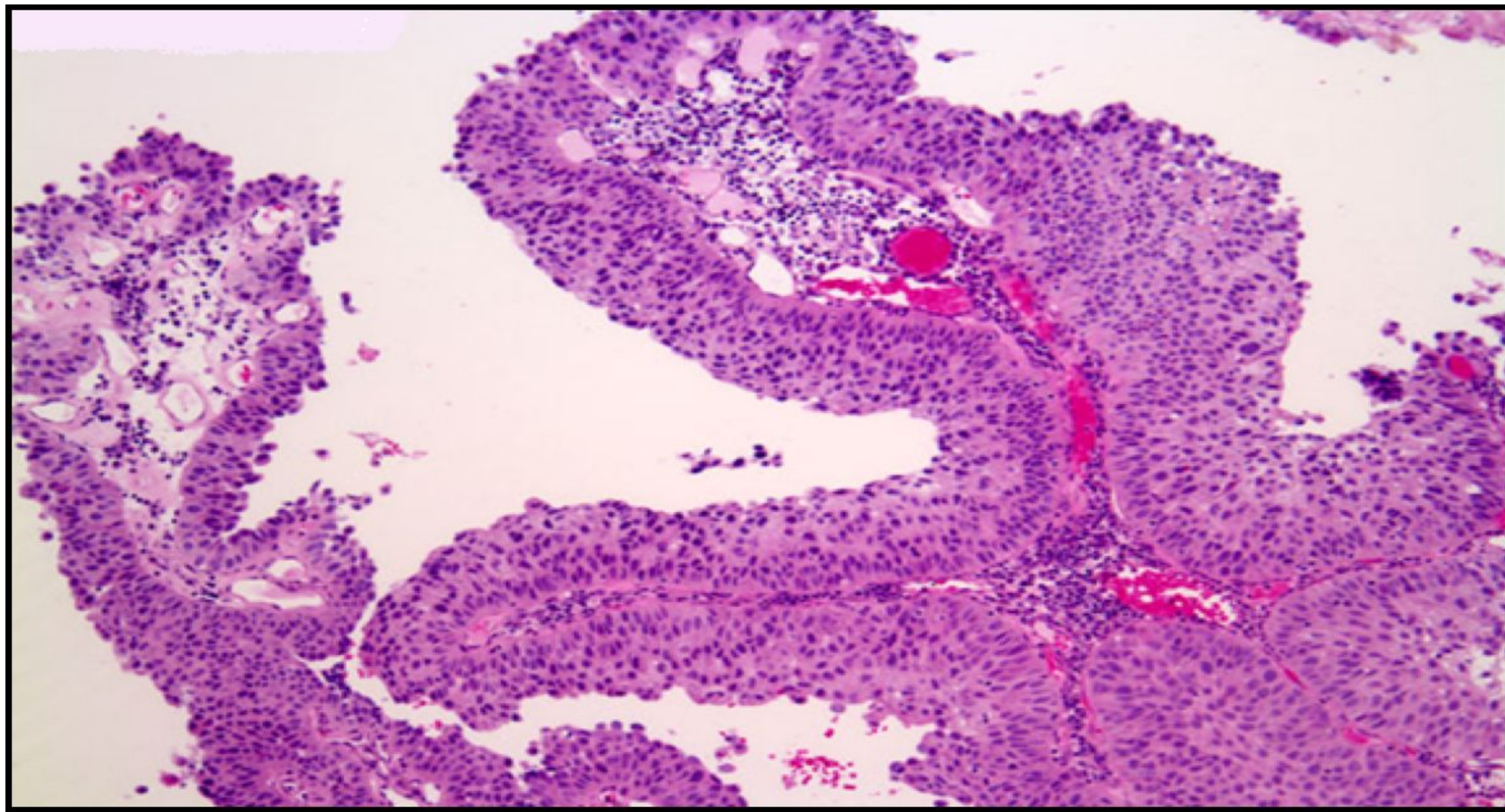
Picture shows **fistula between the bladder and the uterus**. Urinary bladder carcinoma infiltrating the urinary bladder wall with extension to the uterus .

# #12 CARCINOMA OF THE URINARY BLADDER

## (2)

Always know weather its low or high grade in histopathology

Papillary Urothelial carcinoma – Low Grade

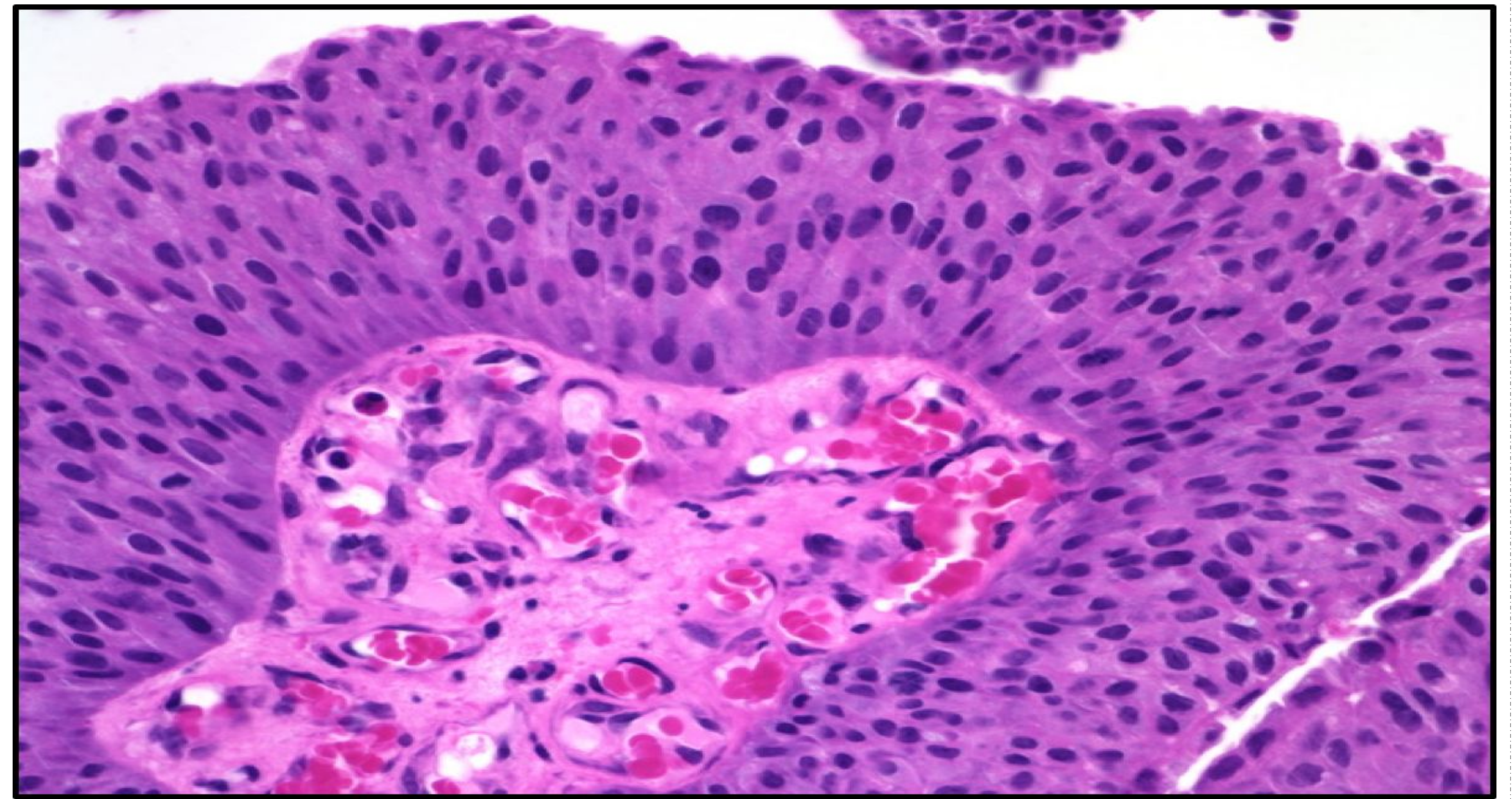


The **low grade tumors** show:

1. Overall preservation of cell polarity.
2. Few mitoses.
3. Lack of significant morphologic atypia.

This exophytic papillary tumor shows multiple finger-like projections lined by multiple layers of urothelium (transitional epithelium).

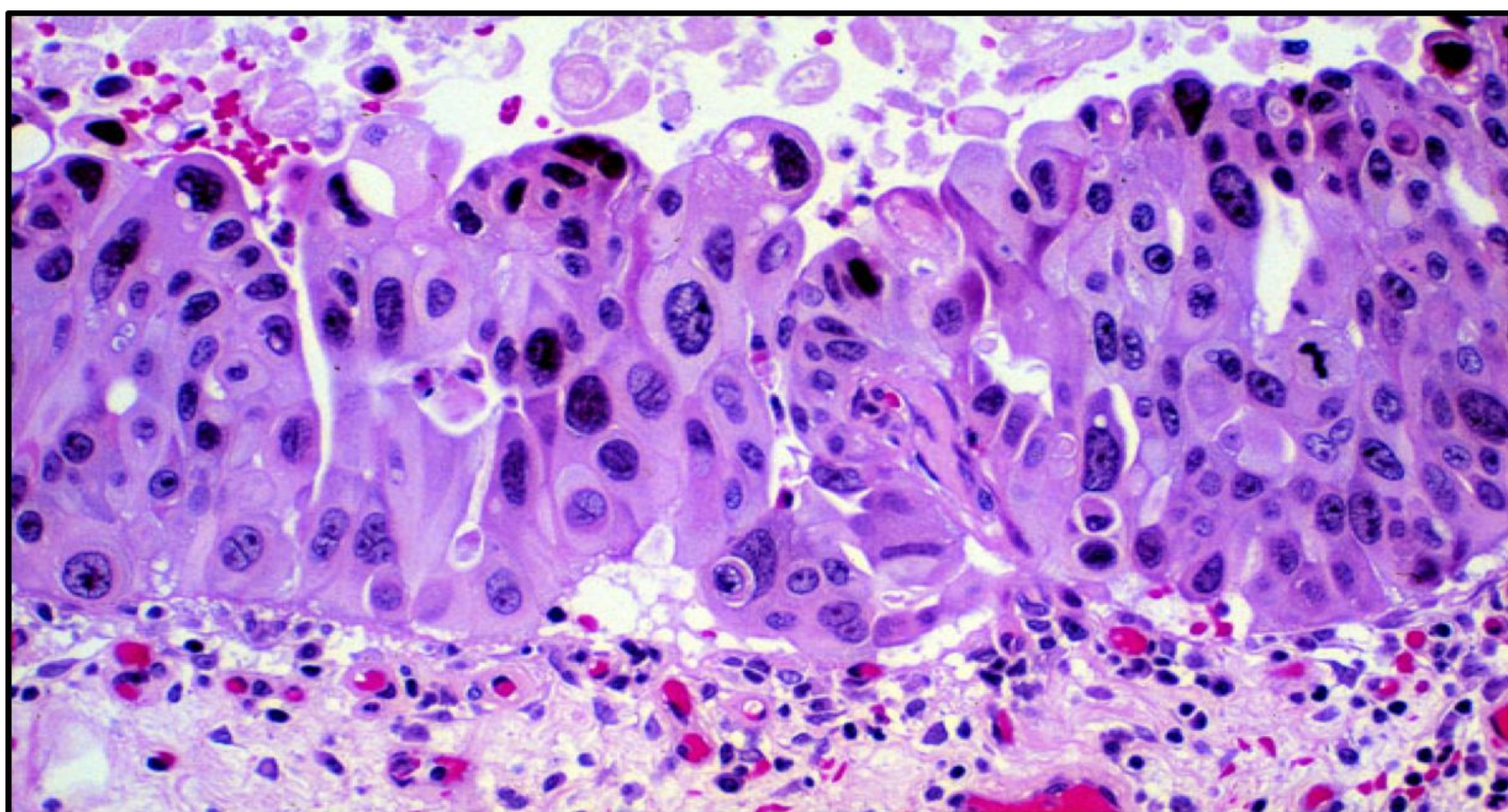
Papillary Urothelial Carcinoma – Low Grade



**low-grade papillary urothelial carcinoma.**

There are scattered hyperchromatic nuclei and typical mitotic figures.

Urothelial (Transitional) carcinoma – HPF



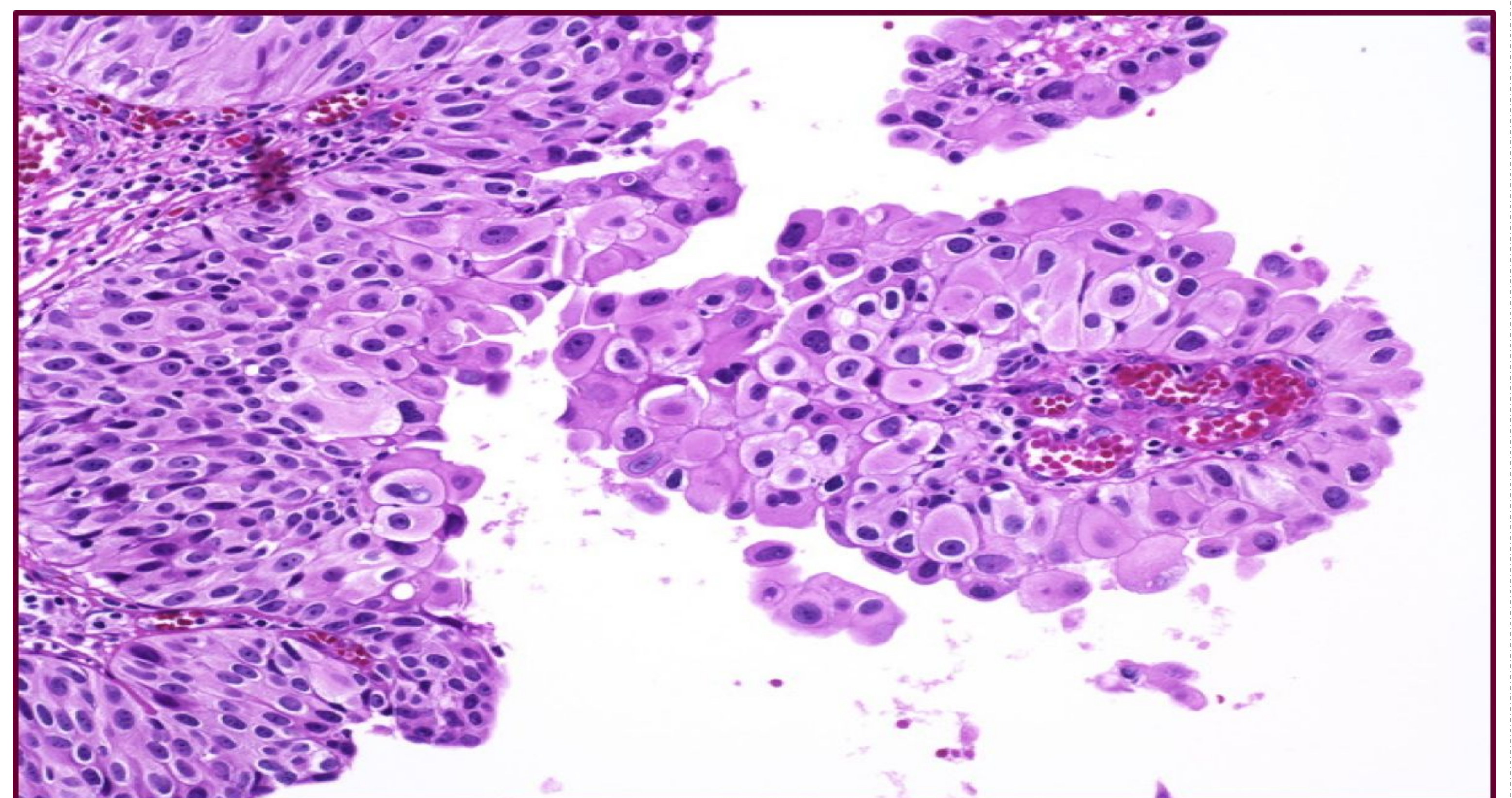
Almost all cases of Bladder carcinomas are **originating from the transitional epithelium.**

Bladder carcinoma might be squamous cell in nature.

Chronic inflammation of the bladder mucosa, **caused by stones or schistosomiasis.**

Rarely, it presents as adenocarcinoma (aggressive and high grade)

Papillary Urothelial carcinoma – High Grade

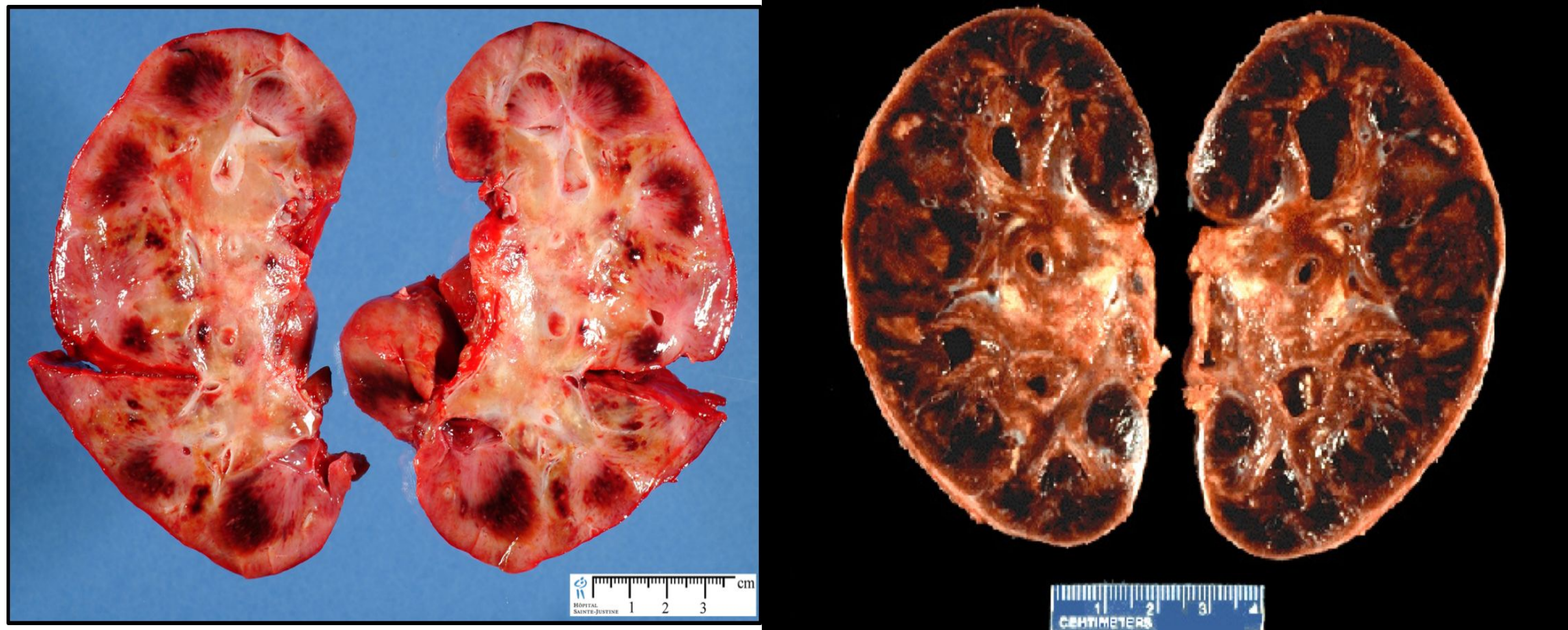


This high-grade papillary urothelial carcinoma shows:

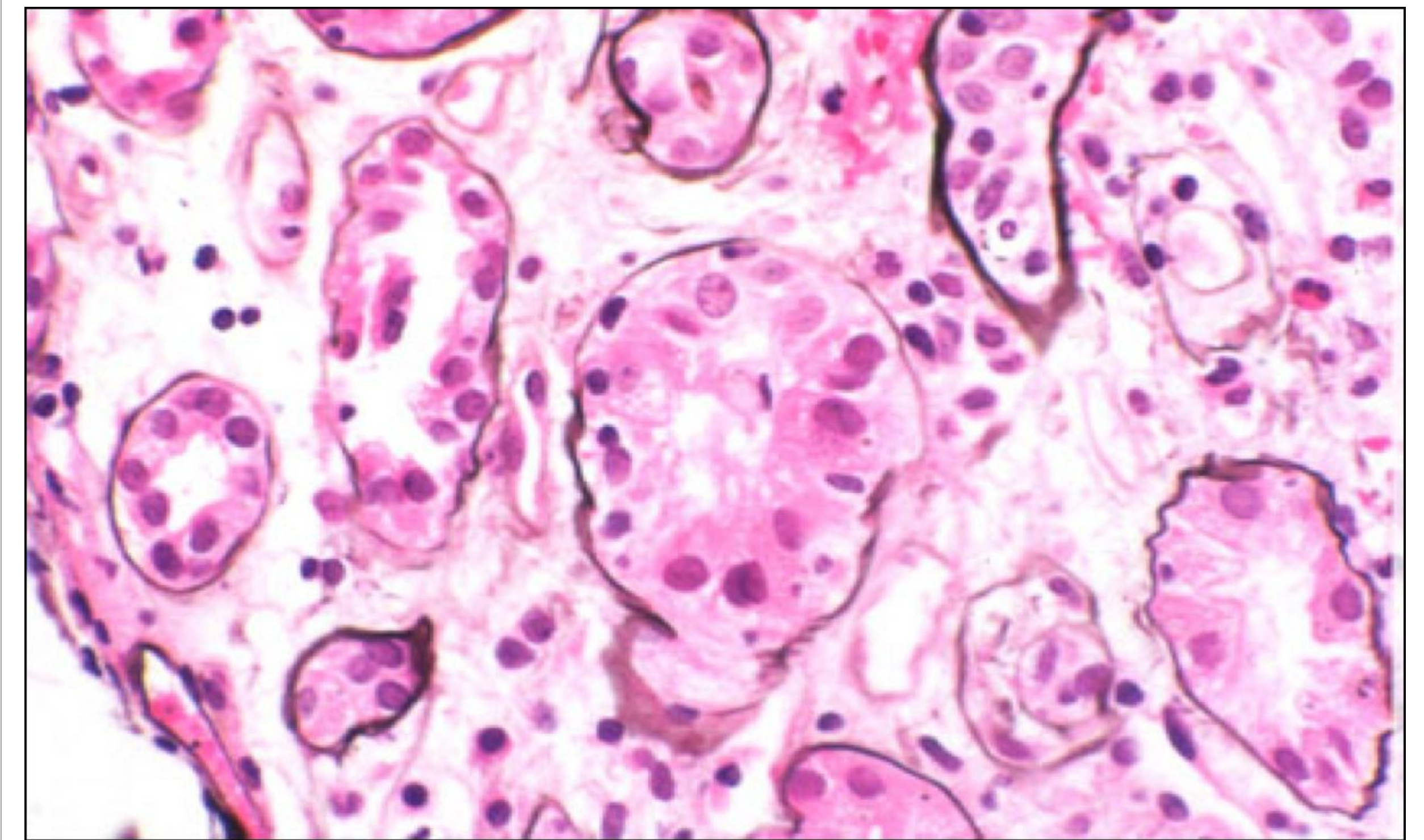
1. Highly pleomorphic.
2. Hyperchromatic nuclei with voluminous cytoplasm.

# #13 Pathology of Renal Allograft

## Acute Cellular Allograft Rejection

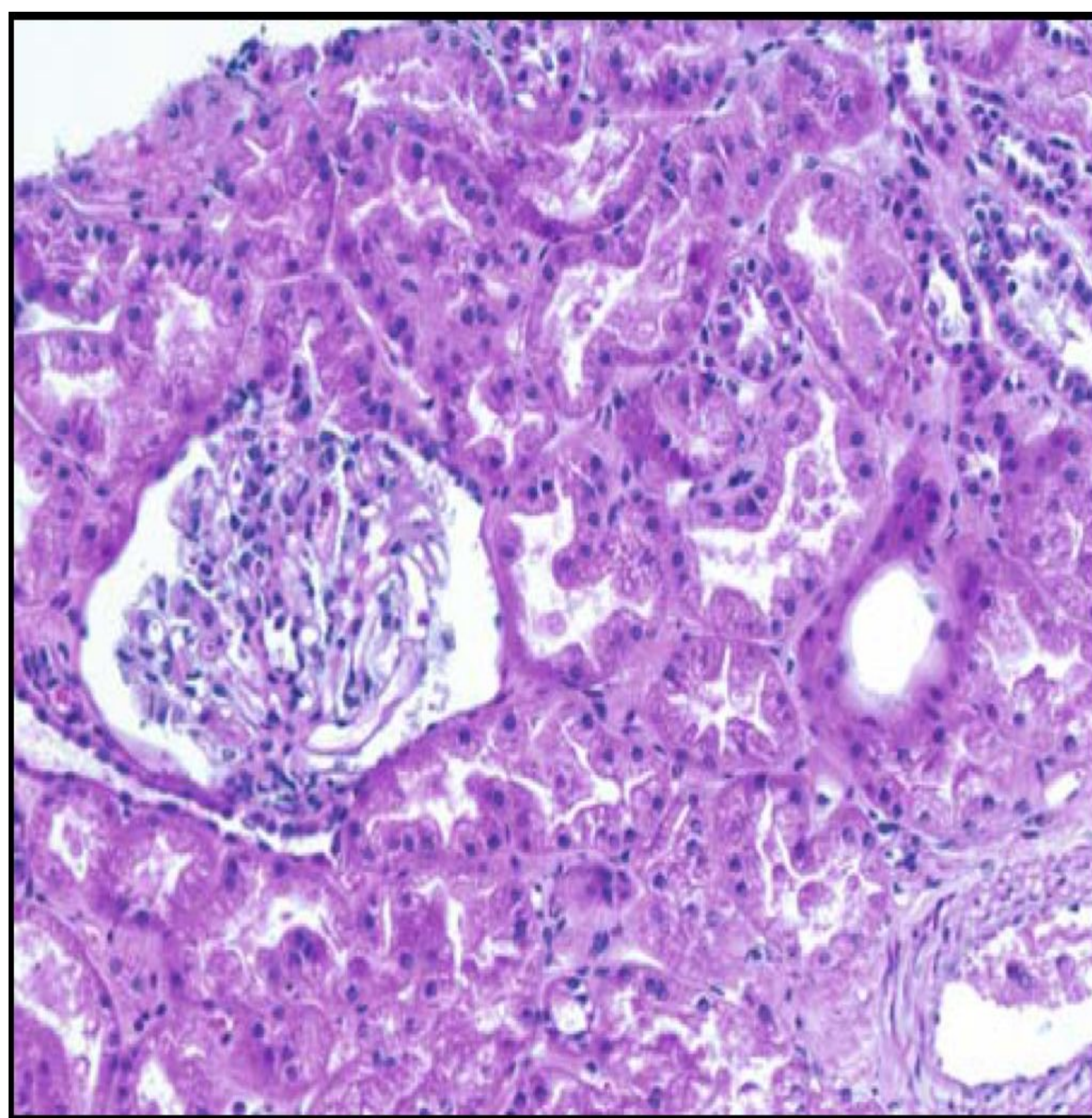


Swollen (Edema) and hemorrhage .



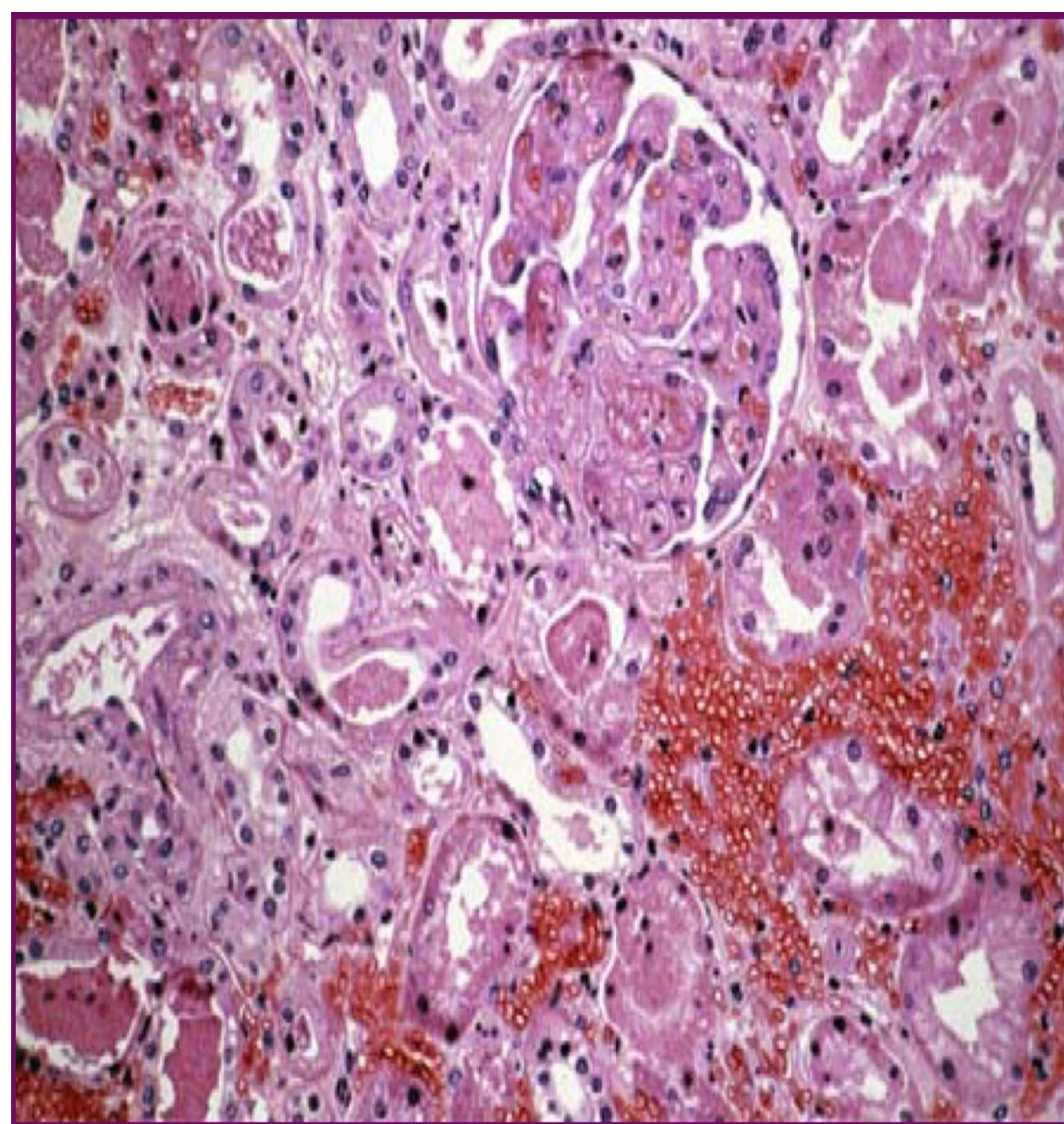
**Tubulitis**, infiltration of tubular epithelium by lymphocytes, is the hallmark of type I interstitial acute rejection

## Acute Humoral Rejection (AHR) – Type I



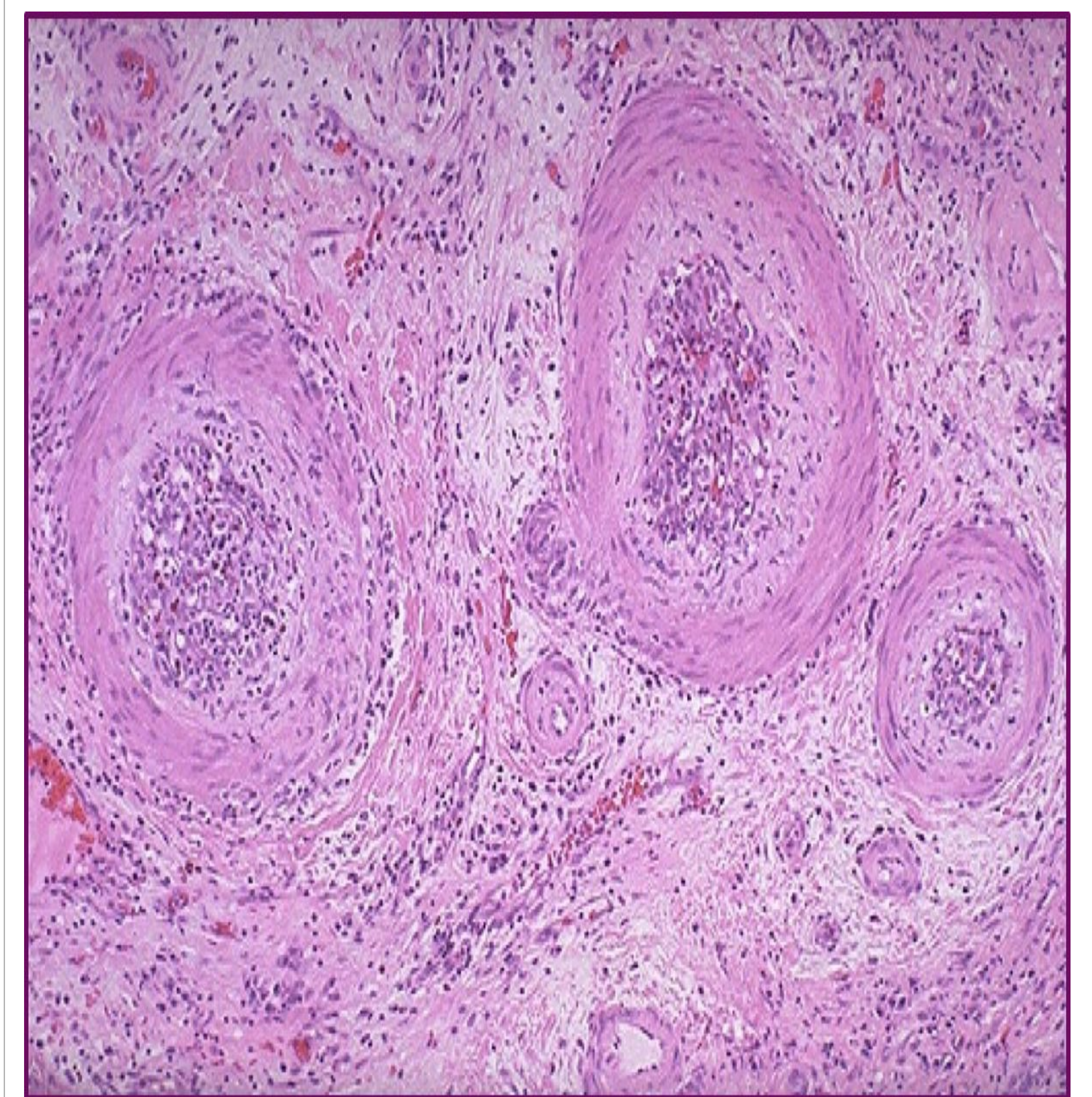
- Vasculitis
- Acute tubular injury

## Hyperacute Allograft Rejection



- diffuse hemorrhage and neutrophils in peritubular capillaries with prominent glomerular thrombi

## Chronic Allograft Rejection



- thickened arteries with intimal fibrosis and also chronic inflammation
- Bad prognoses



Notes:

4 cases with 4 questions, one of them is a theoretical based question ( you can find them in the orange boxes).

Thank you for checking our  
work.

Done by:

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