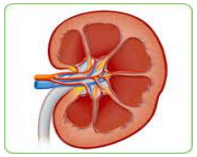
PATHOLOGY TEAM 436

MEDICINE
KING SAUD UNIVERSITY

RENAL BLOCK 

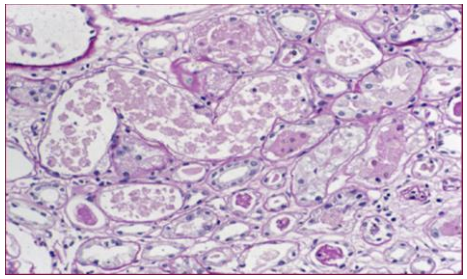
PATHOLOGY PRACTICAL

#case1
ACUTE KIDNEY injury

Causes:

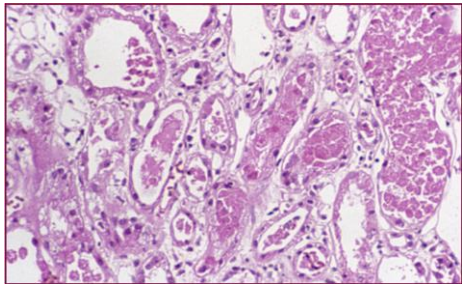
Pre-renal	Renal	Post-renal
(Decrease effective blood flow to the kidney) •Low blood volume, low blood pressure, and heart failure. •Renal artery stenosis, and renal vein thrombosis. •Renal ischemia.	•Glomerulonephritis (GN). • Acute tubular necrosis (ATN). •Acute interstitial nephritis (AIN). Four elements: 1. Glomeruli. 2. Tubules. 3. Blood Vessels. 4. Interstitium	(consequence of obstruction). •Benign prostatic hyperplasia. •Kidney stones. •Obstructed urinary catheter. • Bladder stone . •Bladder, ureteral or renal malignancy.

Acute **Tubular** Necrosis



Manifest by vacuolated cells and sloughed, necrotic cells in tubular lumina.
- *some tubules lined by flattened epithelium*
- *some showing frank necrosis*

Acute **Tubular** Necrosis



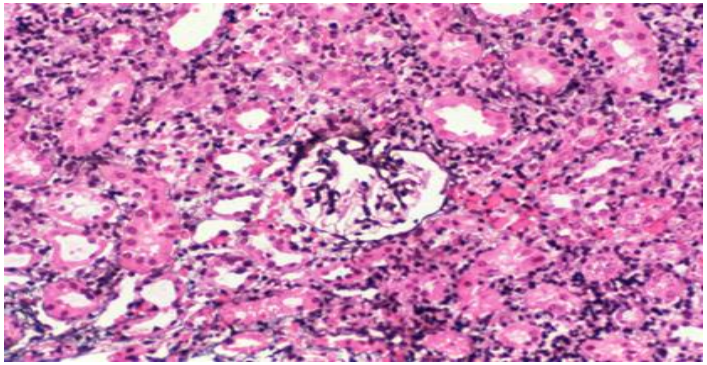
•degeneration and frank necrosis of individual cells or tubular segments or flattened, regenerating type epithelium with degenerated cells in the lumen (middle left)

Acute kidney injury

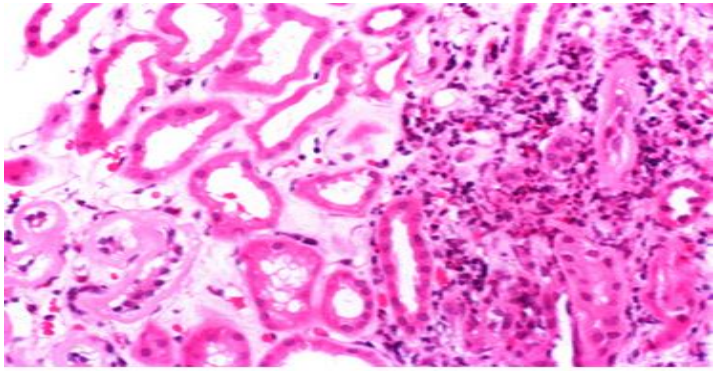
•Marked pallor of the renal cortex, contrasting to the darker areas of surviving medullary tissue .

Acute interstitial nephritis

Causes: *including toxins, viral infections and drug-induced hypersensitivity reactions*

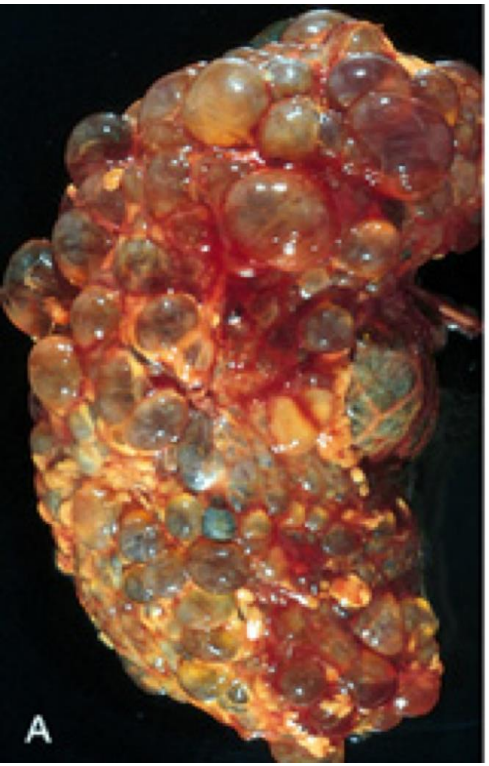


- There is edema associated with an interstitial lymphoplasmocytic infiltrate .
- Glomeruli are uninvolved , Unless there is Associated minimal change disease-type injury caused by NSAIDs.

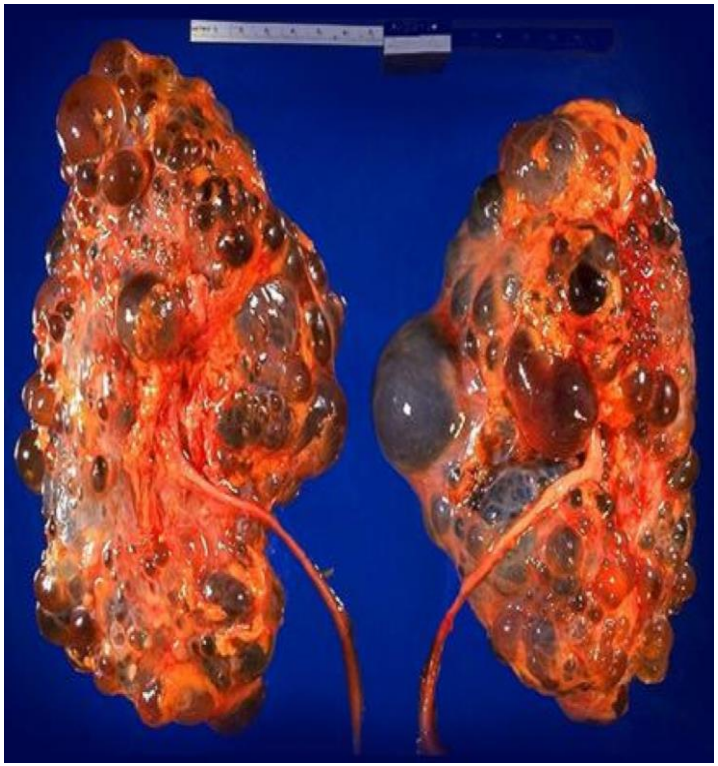


- There is Edema in addition to pre-existing mild tubuloinrestitial fibrosis .
- In case of Acute interstitial nephritis caused by drug induced hypersensitivity .
- There is prominent interstitial eosinophilic component , in addition to lymphocytes and plasma cells .

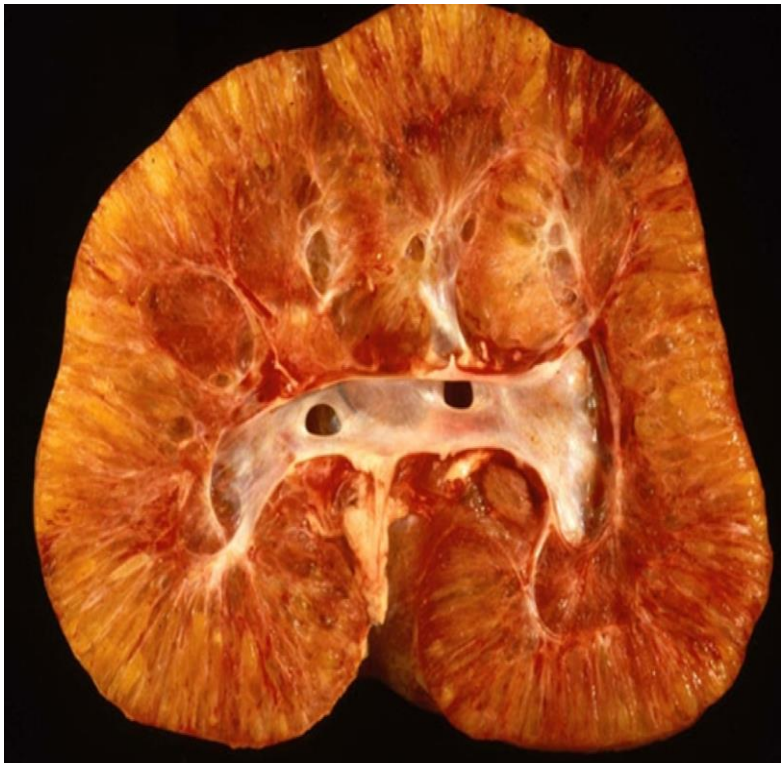
#Case2
Polysistic Kidney



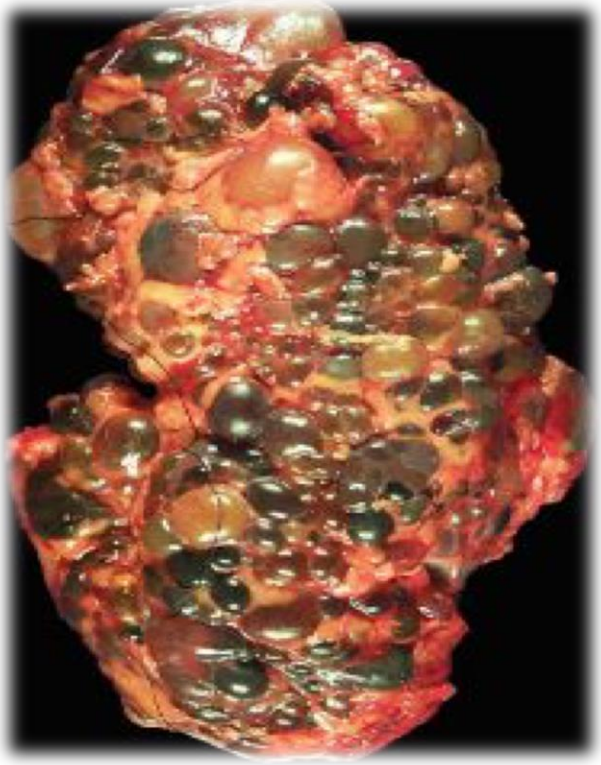
Markedly enlarged kidney
and replacement of the renal parenchyma by numerous cysts of variable sizes



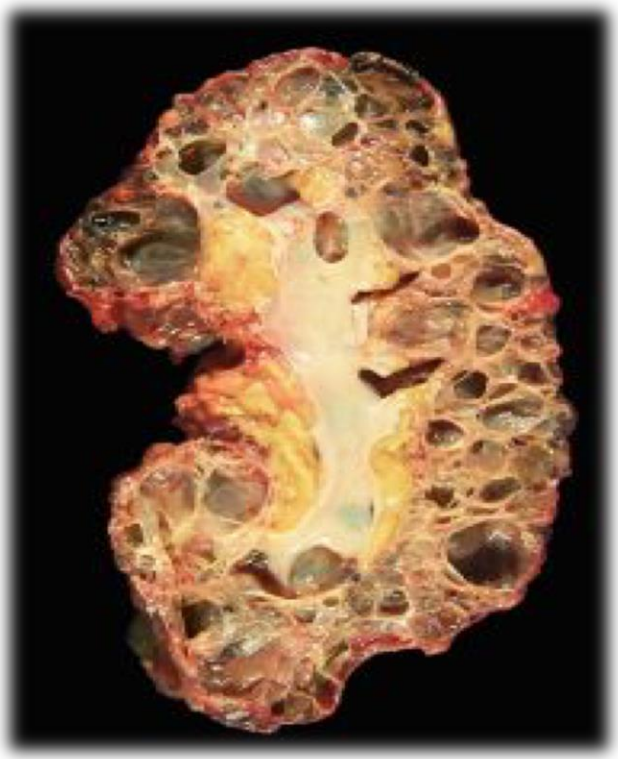
Bilateral autosomal dominant polycystic kidney disease



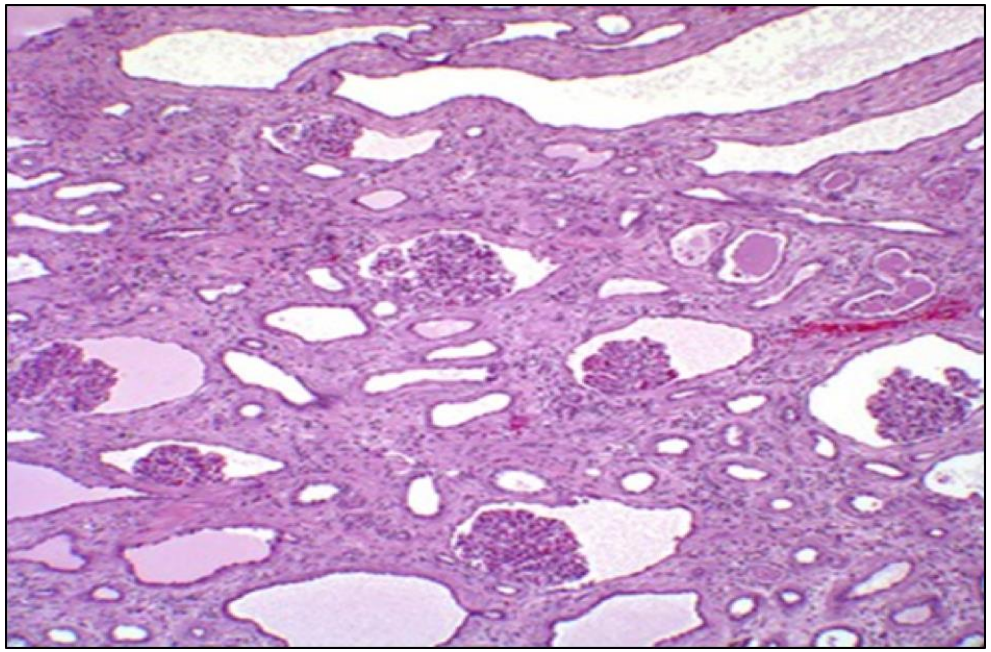
Coronal section of an infantile polycystic kidney



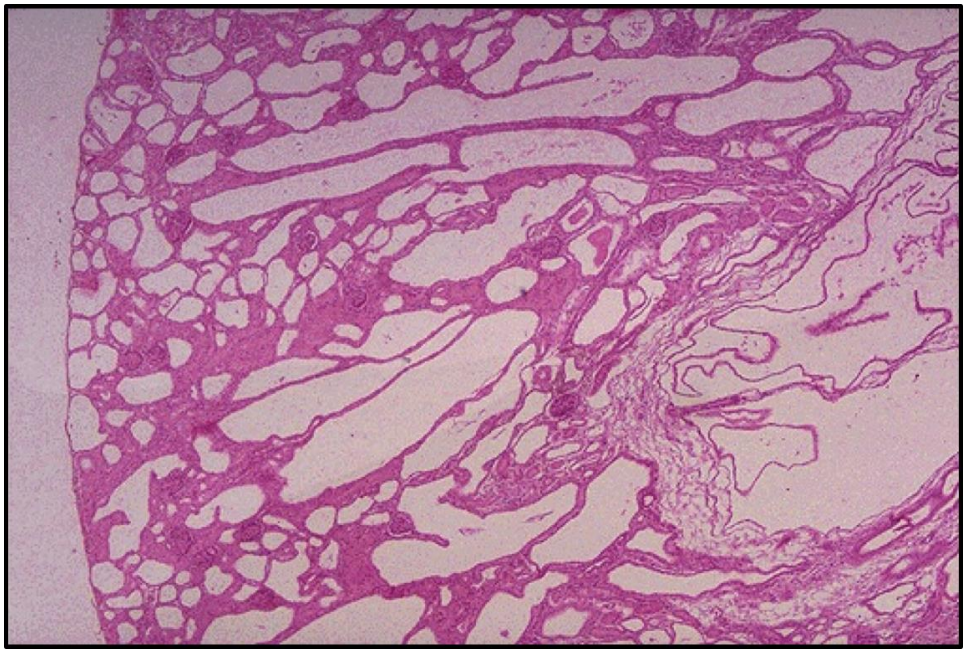
Massively enlarged kidney disrupted by numerous cysts



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



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



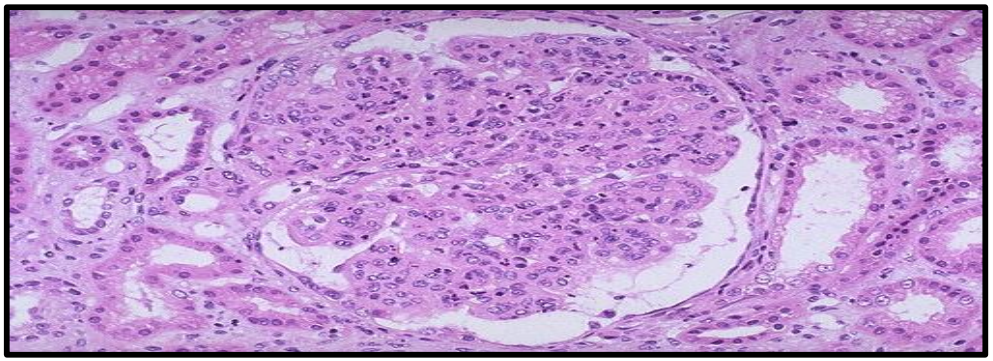
Autosomal Recessive Polycystic Kidney Disease (ARPKD). Note that the cysts fill most of the parenchyma, and it is hard to find glomeruli.

INFECTION OF THE URINARY TRACT

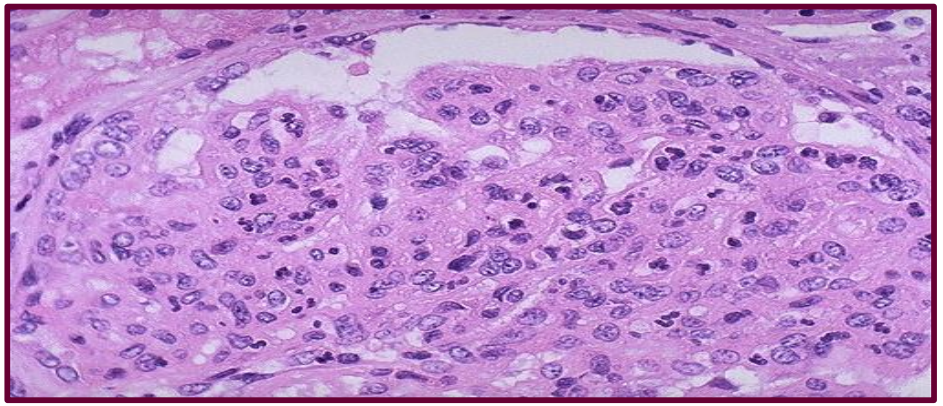
#Case3
Acute (Post-streptococcal) Glomerulonephritis

Section of the kidney shows: (Acute – post-streptococcal glomerulonephritis)

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

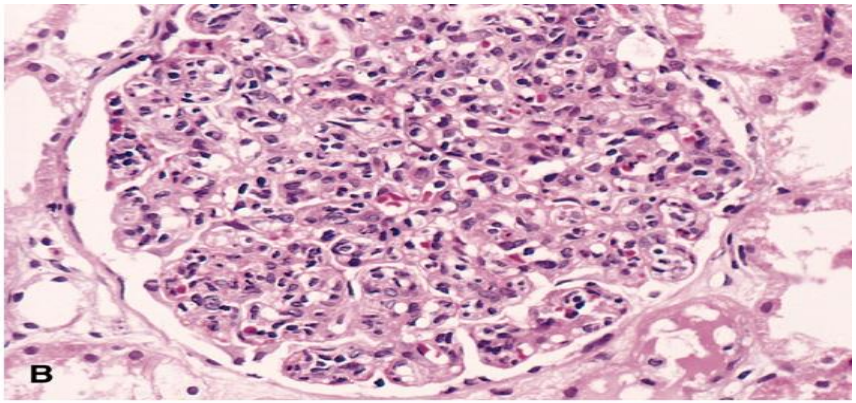


- Glomerulus is hypercellular.
- Capillary loops are poorly defined.
- This is a type of proliferative glomerulonephritis known as post-infectious glomerulonephritis.



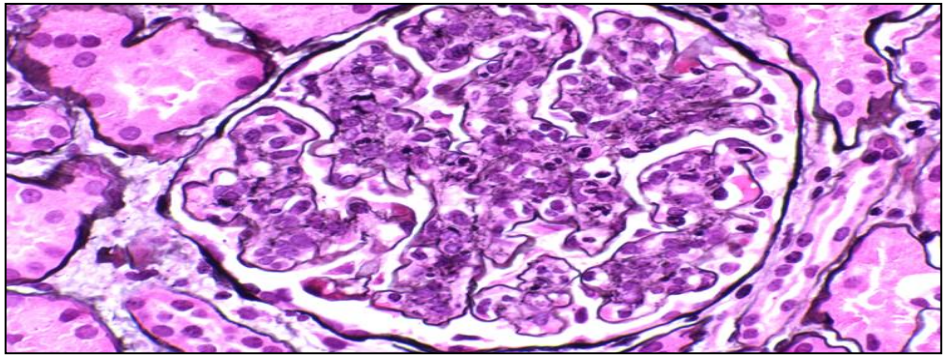
- Glomerulus of post infectious is hypercellular due to increased number of epithelial, endothelial, and mesangial cells as well as neutrophils in and around the glomerular capillary loops.

High power LM of a hypercellular glomerulus



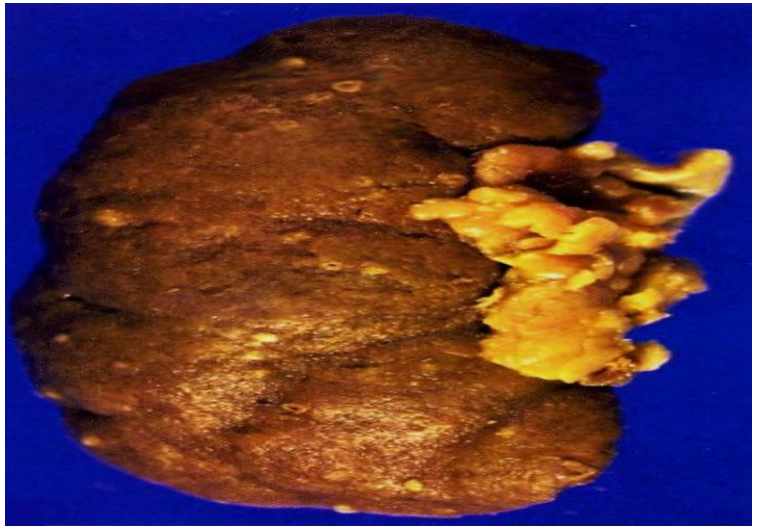
- Hypercellular glomerulus with numerous capillaries that contain inflammatory cells, mostly neutrophils.

High-power silver stain



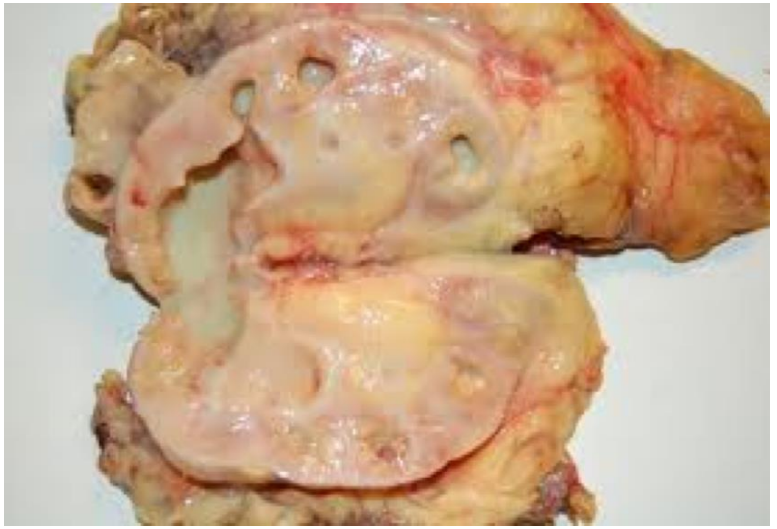
- Acute Post-streptococcal Glomerulonephritis is evident in this high-power silver stain.
- Large number of PMNs.
- The glomerular basement membrane does not show splitting or spikes.
- Proliferation of endothelial and mesangial cells and infiltrating cells and filling and distending capillary loops.

#Case4
Acute Pyelonephritis



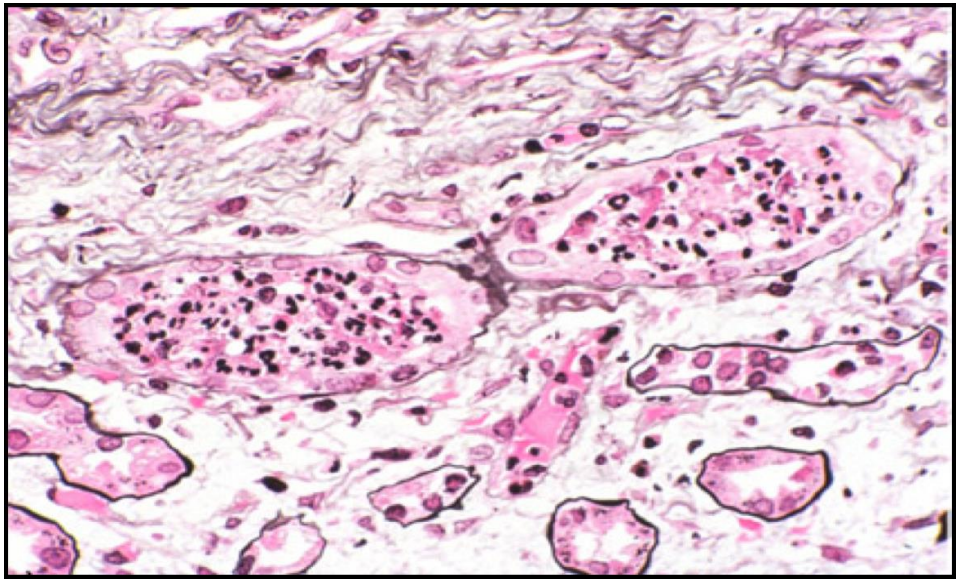
Pyelonephritis with small cortical abscess:

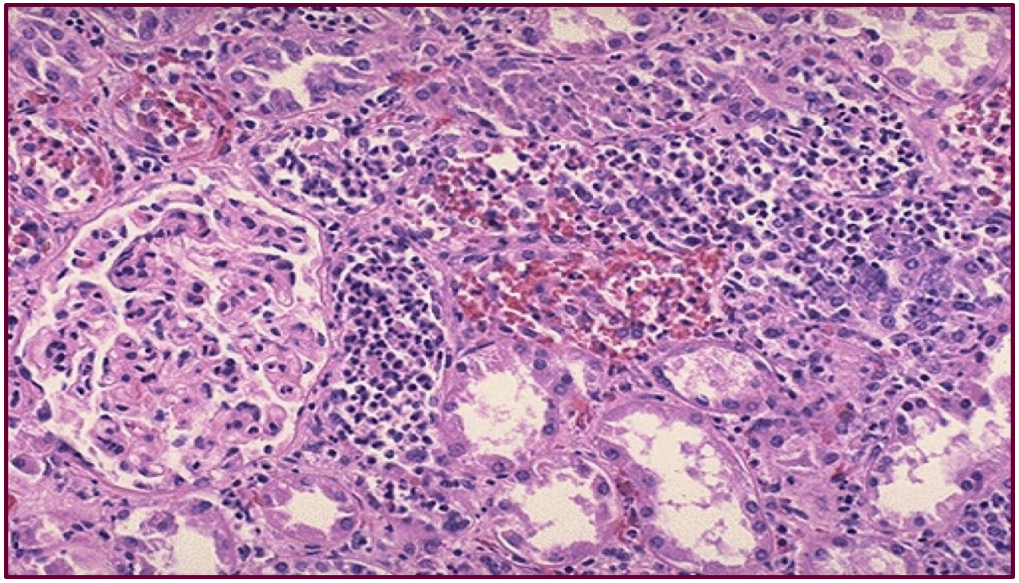
Pyelonephritis = inflammation within the kidney.



Classic picture of Pyelonephritis

- pelvis and calyxes filled with pus .
- The cortex and medulla are pale .
- The corticomedullary junction is ill-defined.





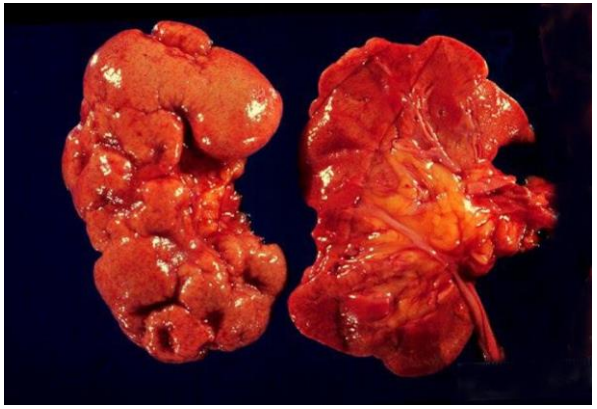
-Acute Pyelonephritis is diagnosed by intra-lobular aggregations od polumorphynuclear neutrophils (PMNs).
- Interstitial inflammation with mixure of PMN’s , netrophils, lymphocytes and plasma cells .
- The predominant inflammation is within the tubule.

- 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 urine originate in the distal renal tubules and collecting ducts .

#Case5
Chronic Pyelonephritis

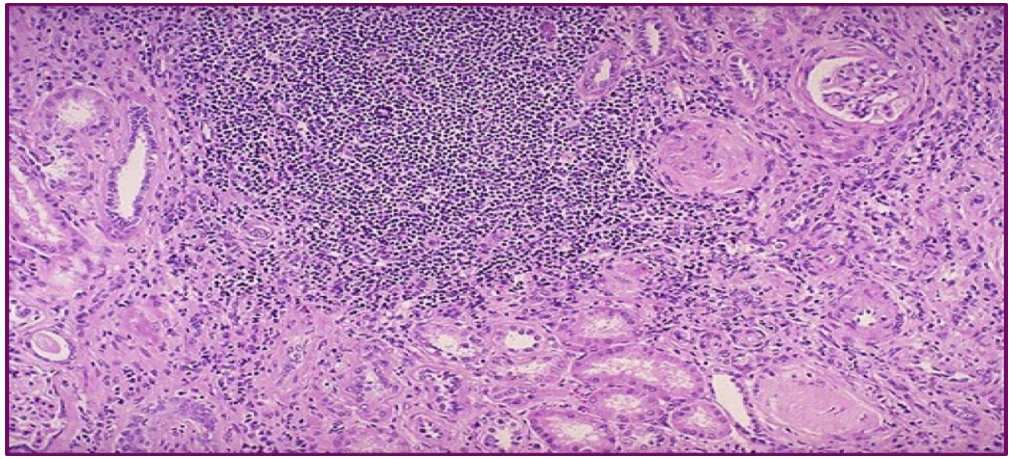
Theoratical information: Causes	
Acute Pyelonephritis	Chronic Pyelonephritis
• Hematogenous spread	• Recurrent attacks of acute pyelonephritis. • Drug-induced interstitial nephritis. • Urinary tract obstruction or reflux.

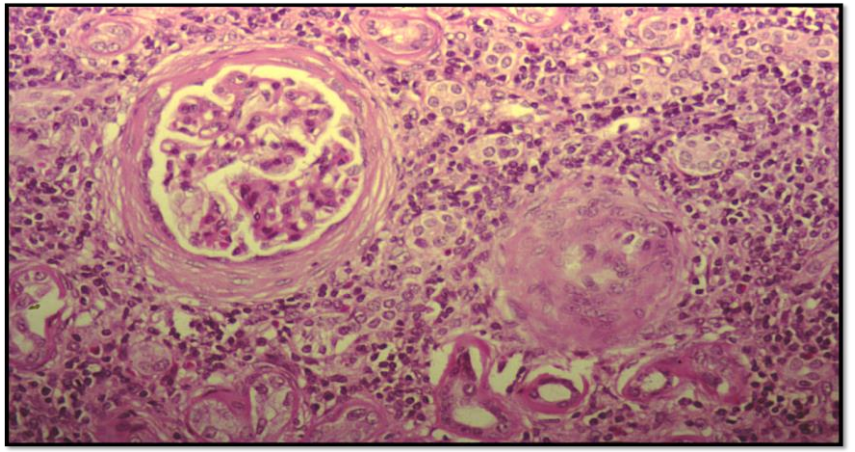
Gross:



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

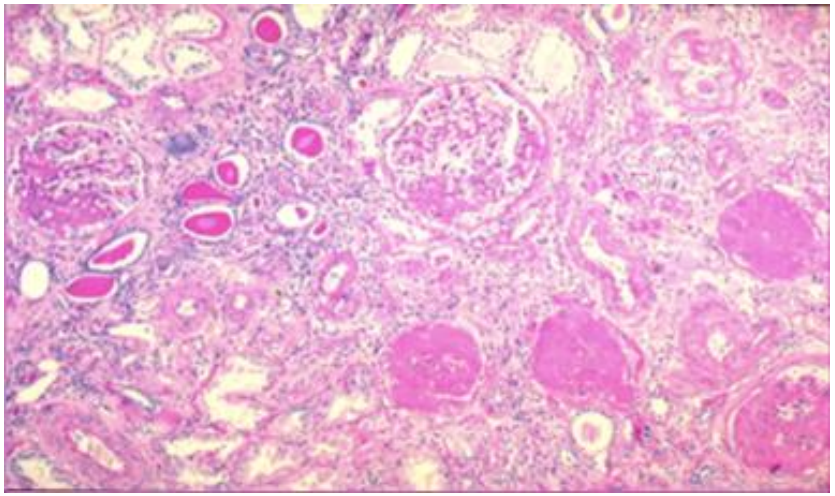
Most common cause of this condition in children – reflux nephropathy .





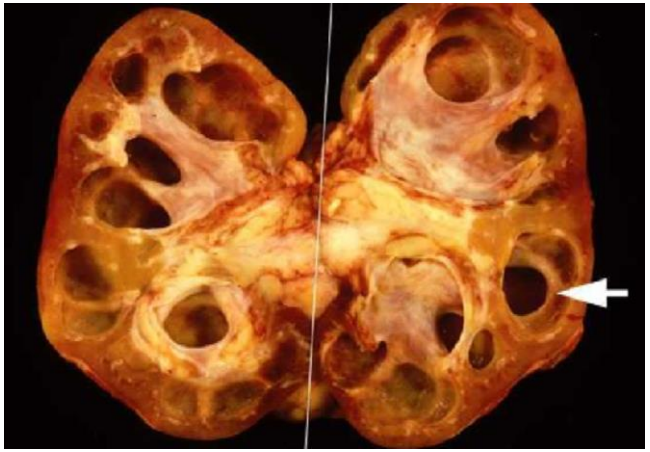
- This is chronic pyelonephritis where a large collection of chronic inflammatory cells .
- When eosinophilic infiltration is present in the interstitium , the most likely cause of
- nephritis in such cases - Drug induced interstitial nephritis.

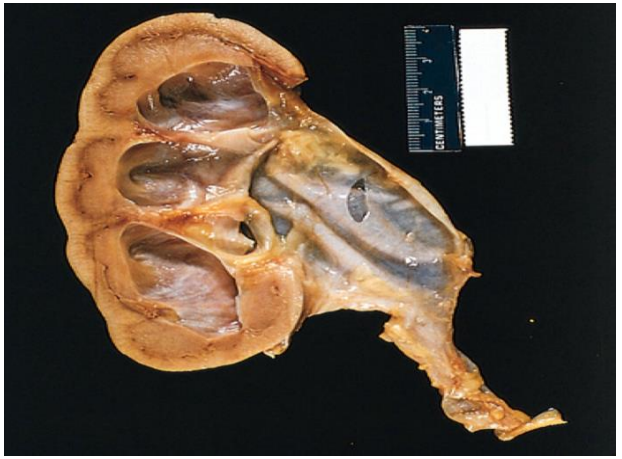
- Peri-glomerular fibrosis.
- Interstitial inflammation.
- Hyalinized /fibrotic glomeruli

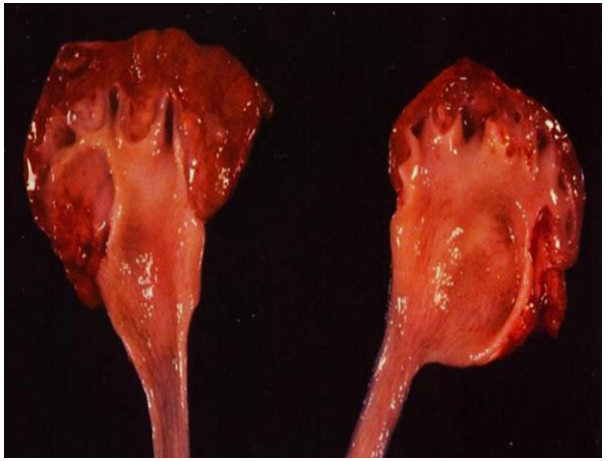


- **Glomeruli** show varying degrees of sclerosis & periglomerular fibrosis.
- **Tubules** show varying degrees of atrophy, Some tubules are dilated and filled with Eosinophilic hyaline casts resembling colloid (thyroidization).
- **Interstitial** tissue shows chronic inflammatory cells infiltrate and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

#Case6
HYDRONEPHROSIS





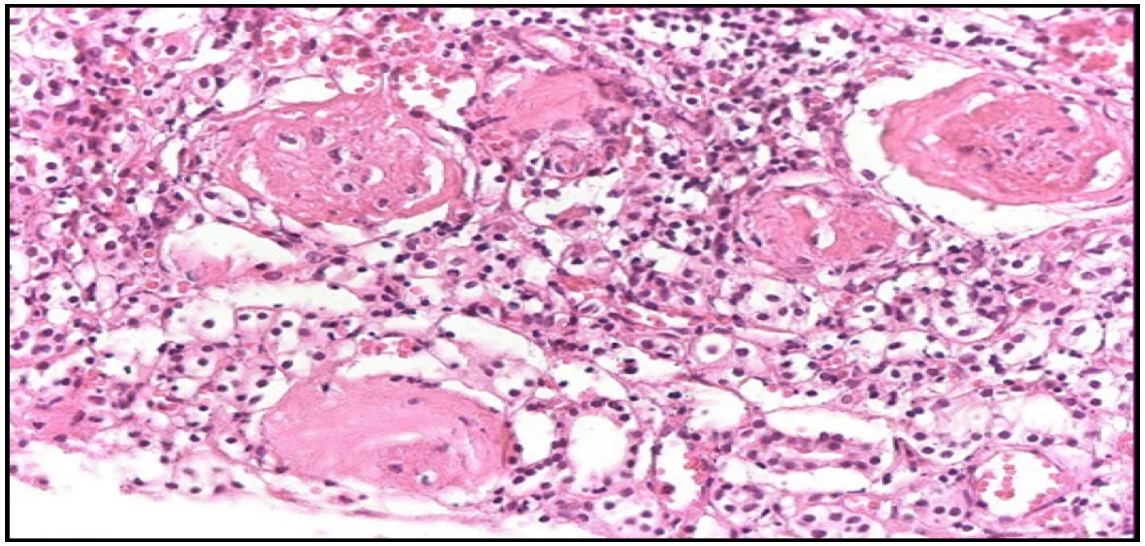


-All pictures show : markedly dilated renal pelvis and calyces with atrophic and thin renal cortex .

The most common causes of Hydronephrosis are:

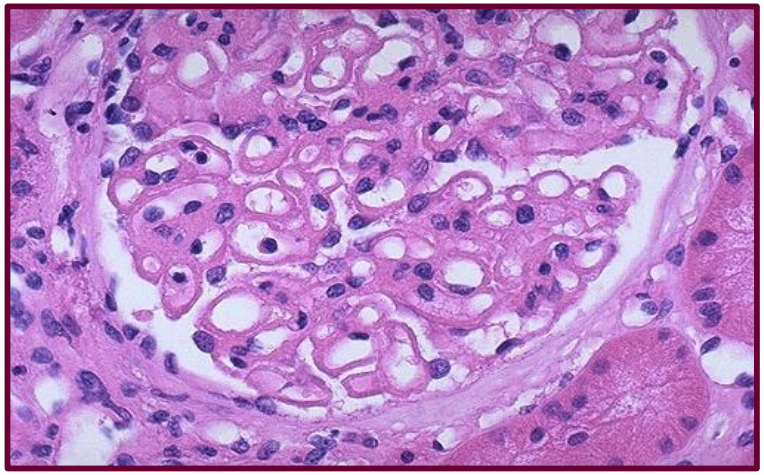
- Foreign bodies like calculi with obstruction,
- Atresia of the urethra
- Benign prostatic hyperplasia and neoplasia of the prostate and bladder .
- Spinal cord damage with paralysis of the bladder

Complication: Chronic Pyelonephritis



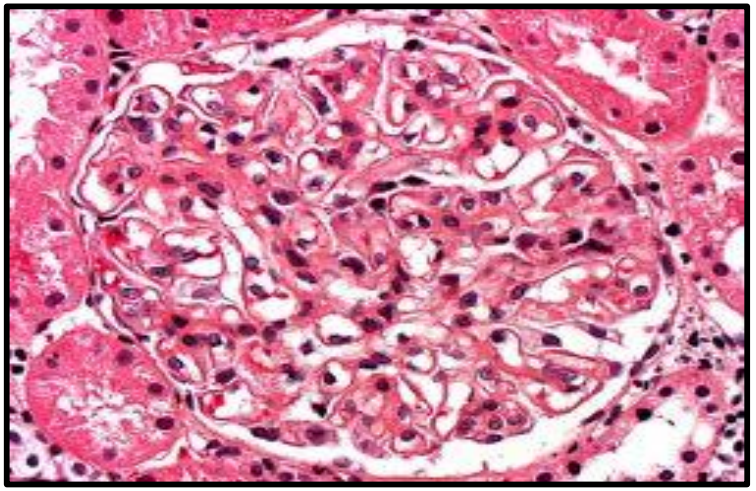
Chronic Pyelonephritis presenting as complication to Hydronephrosis .
This picture shows :
- Sclerosis of glomeruli with atrophic tubules .

#Case7
NEPHROTIC SYNDROME



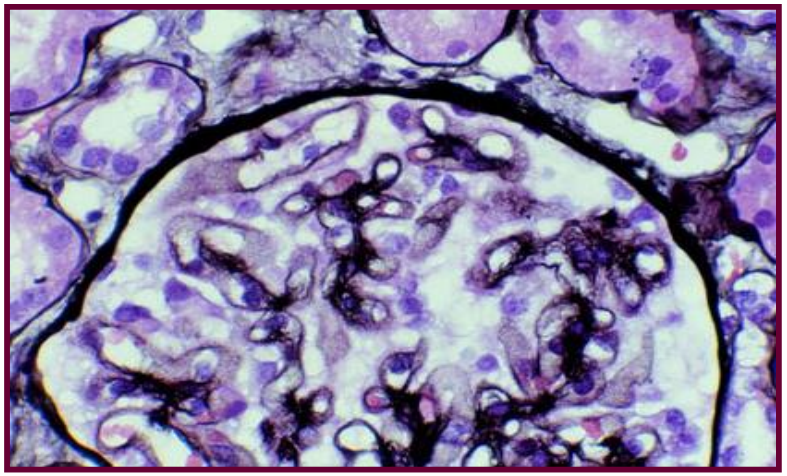
Membranous glomerulonephritis (The common cause of Nephrotic syndrome in adults):

- The capillary loops are thickened
- The capillary loops are prominent
- The cellularity is not increased.

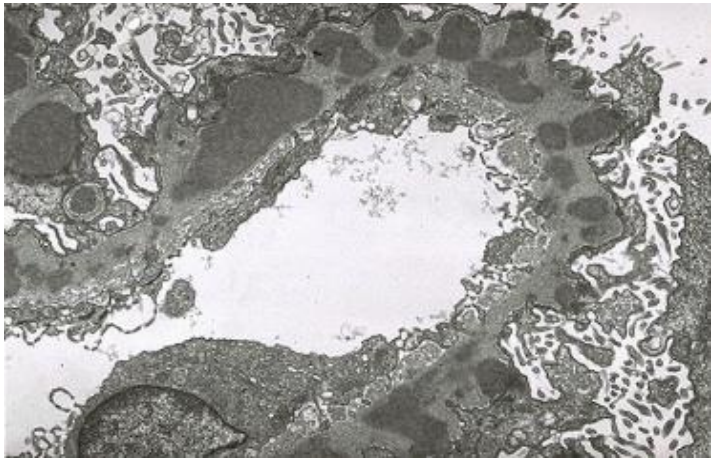


Close-up of glomerulus illustrating rigid, uniformly -thickened capillary walls (H&E stain, 400x original magnification).

Nephrotic Syndrome : 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.



Electron microscopy features:

a- Effacement of epithelial foot processes.

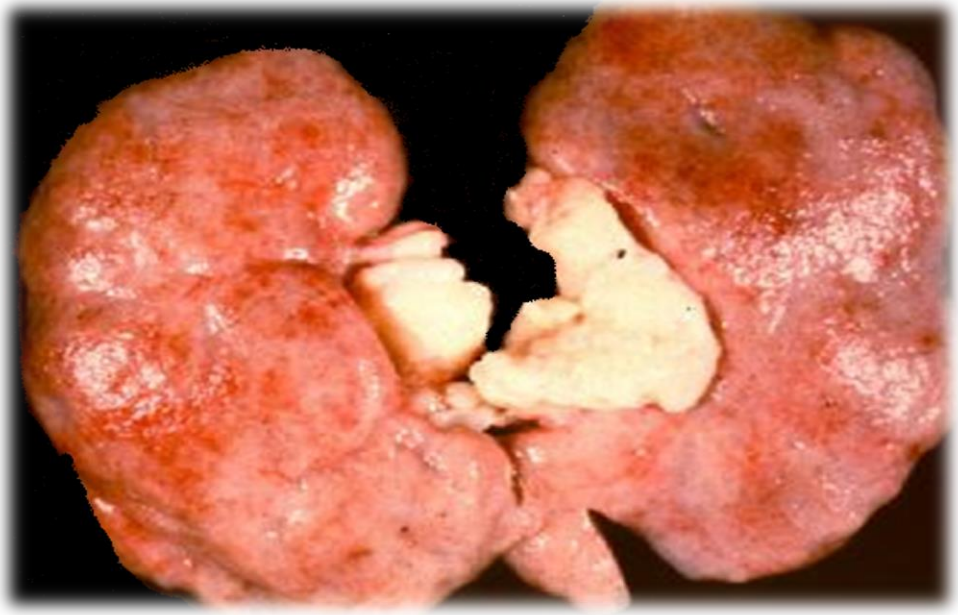
b- Immune complexes deposition in basement membrane leading to domes" appearance "spikes and

.

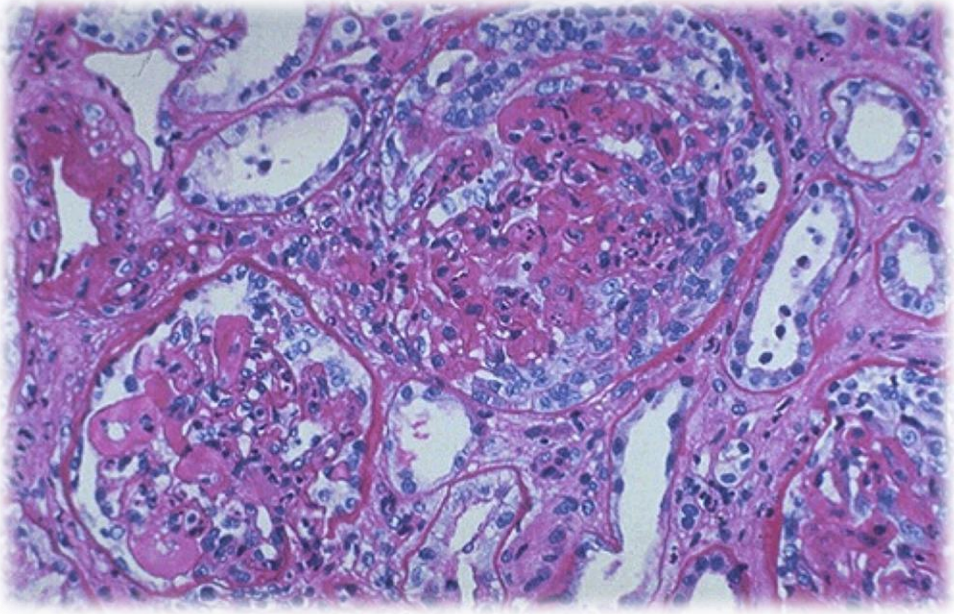
Disorders that can predispose to membranous glomerulonephritis:

a- Infections: Hepatitis B, Syphilis and Malaria.
b- Malignant tumours (Lung and colon).
c- SLE and autoimmune disorders.
d- Exposure to inorganic salts and certain drugs

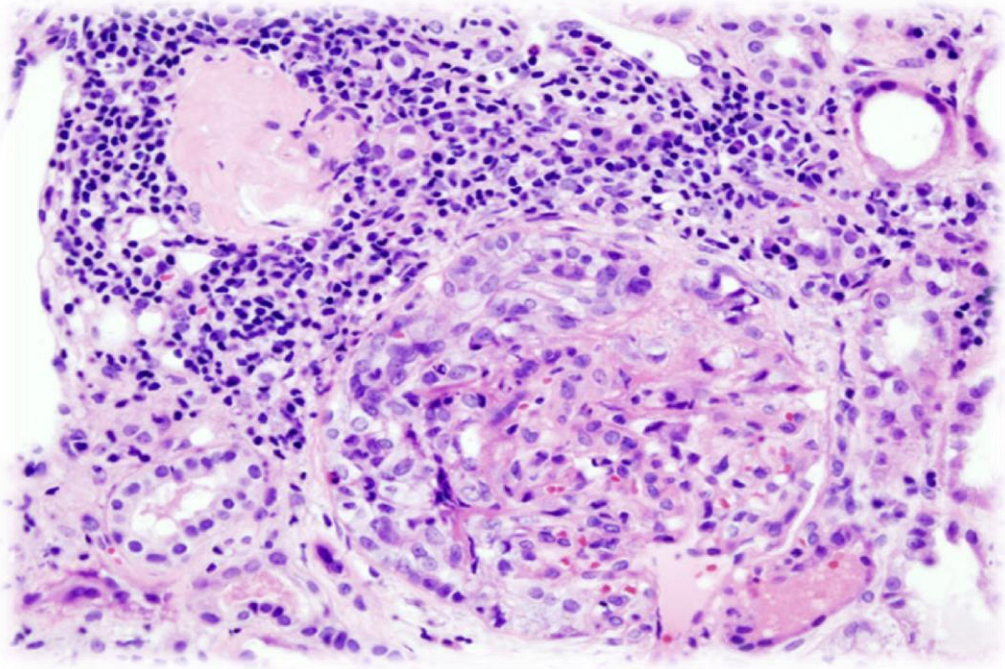
#Case8
NEPHRITIC SYNDROME
(RPGN)



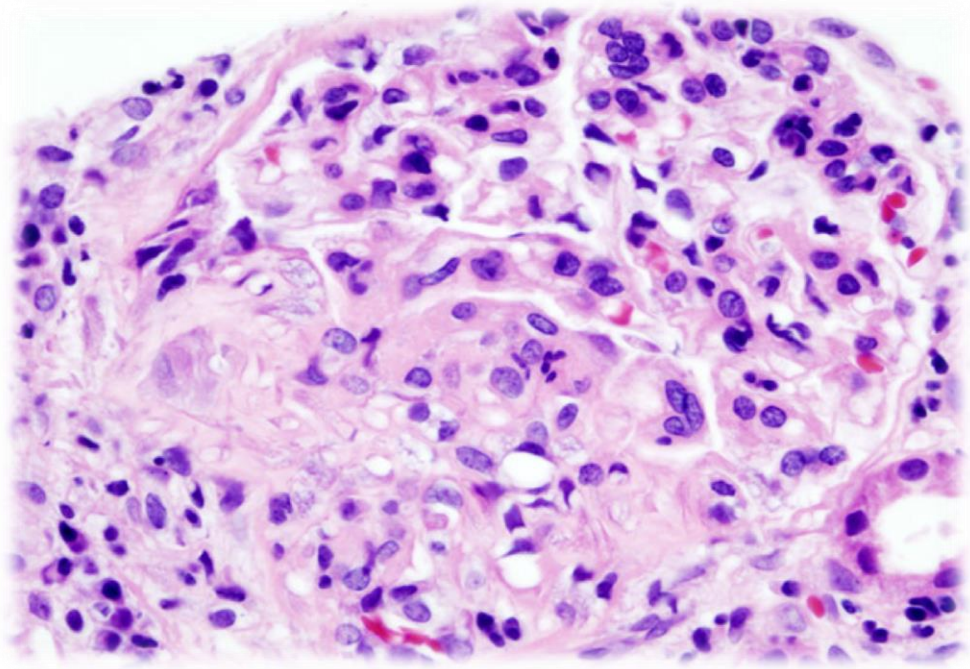
Gross appearance of RPGN - note the flea beaten appearance.



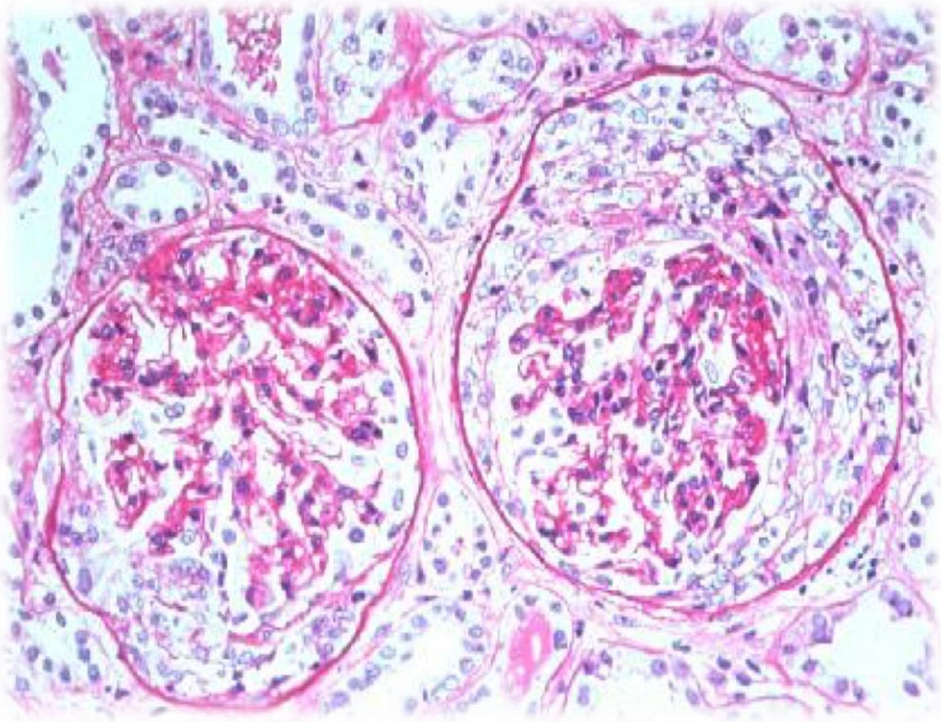
Seen here within the glomeruli are crescents composed of proliferating epithelial cells. Crescentic glomerulonephritis is known as rapidly progressive glomerulonephritis (RPGN) because this disease is very progressive.



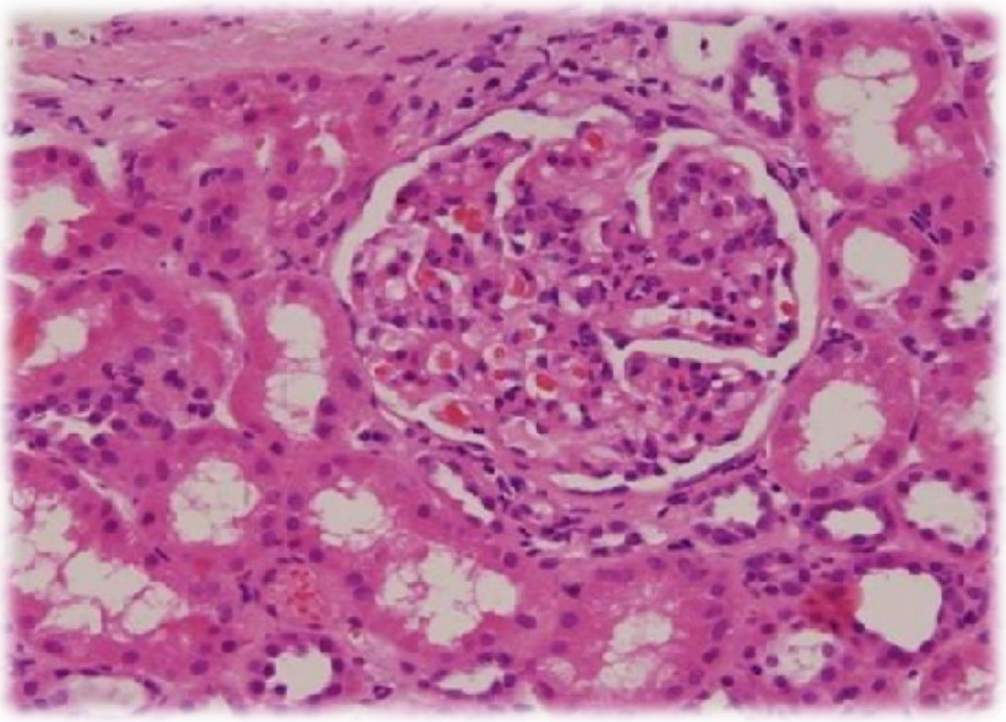
Crescentic glomerulonephritis in a patient with Rapid Progressive Glomerulonephritis (RPGN) . All types of RPGN are characterized by glomerular injury and formation of crescents with monocytes and macrophages proliferation compressing the glomerulus.



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 .



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

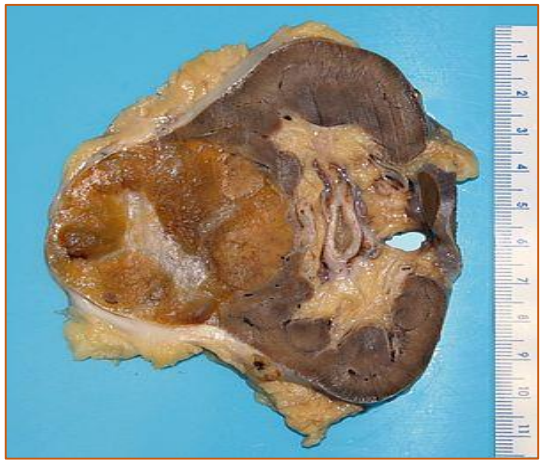


The glomeruli showed mesangial proliferation. The glomerular basement membrane was normal. The interstitium and blood vessels were unremarkable .

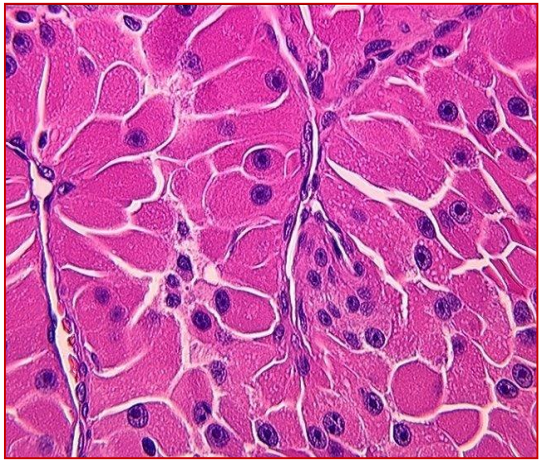
#Case9
BENIGN RENAL TUMORS

RARE Tumors:

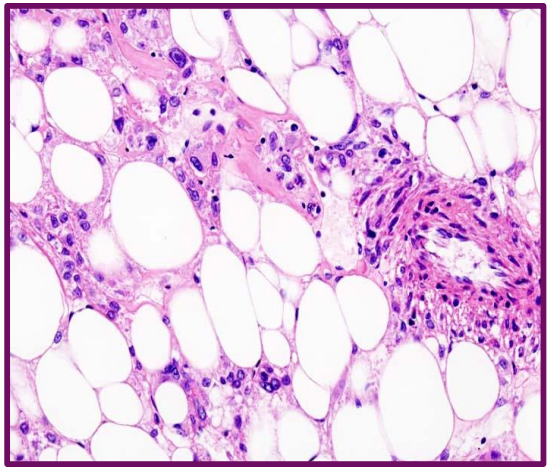
- ***Papillary Adenoma (SIZE very important)***
- ***Fibroma/ Hamartoma***
- ***Angiomyolipoma***
- ***Oncocytoma (very** red, granular, mitochondria**)***



Gross appearance of a renal oncocytoma (left of image) and a slice of a normal kidney (right of image). Note the rounded contour, the mahogany colour and the central scar

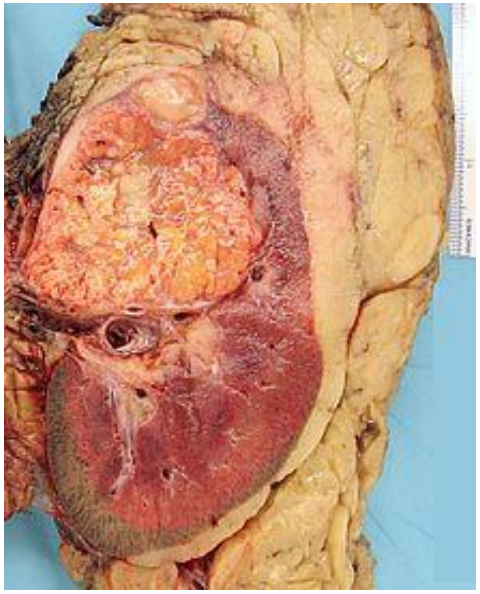


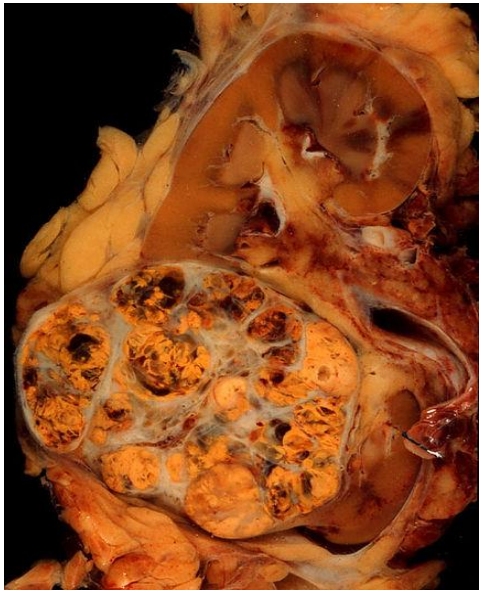
Oncocytes are RED and granular .

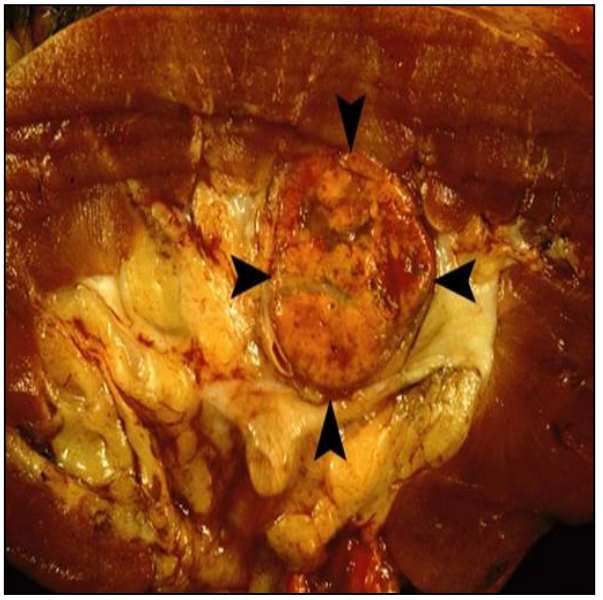


Benign tumor composed of vessels, smooth muscle and fat

#Case10
MALIGNANT RENAL TUMORS





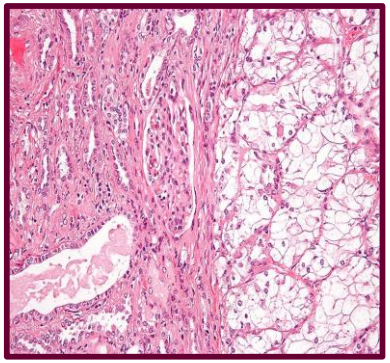


A well circumscribed renal cortical mass which is partly yellow due to presence of fat and partly hemorrhagic with lobulated cut surface .

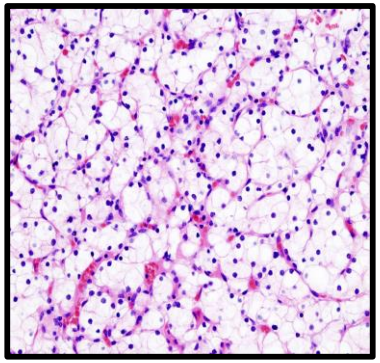
Renal clear cell carcinoma. The tumor is well demarcated from the surrounding non-neoplastic renal parenchyma by a pseudocapsule.

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.



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



The most common type of renal cell carcinoma (clear cell) .
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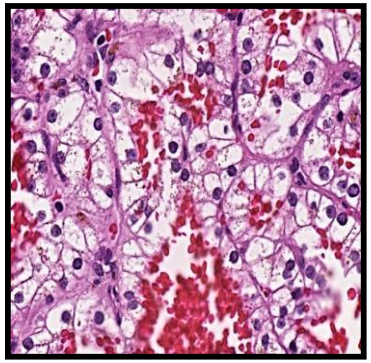
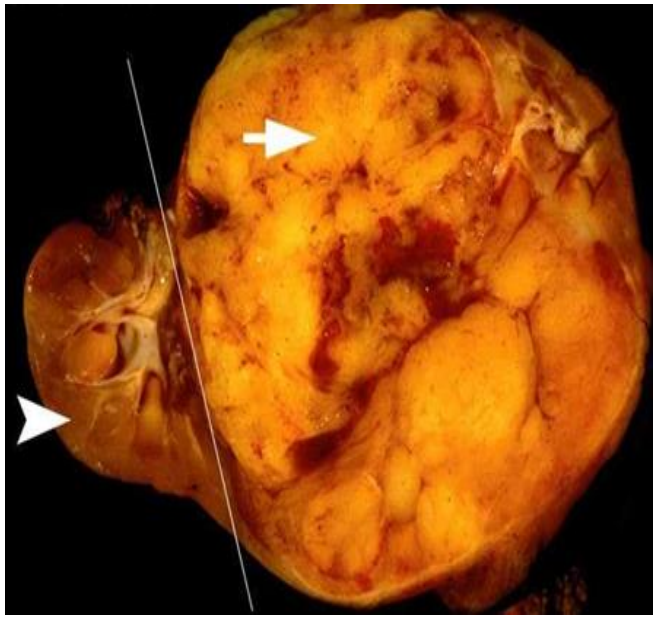
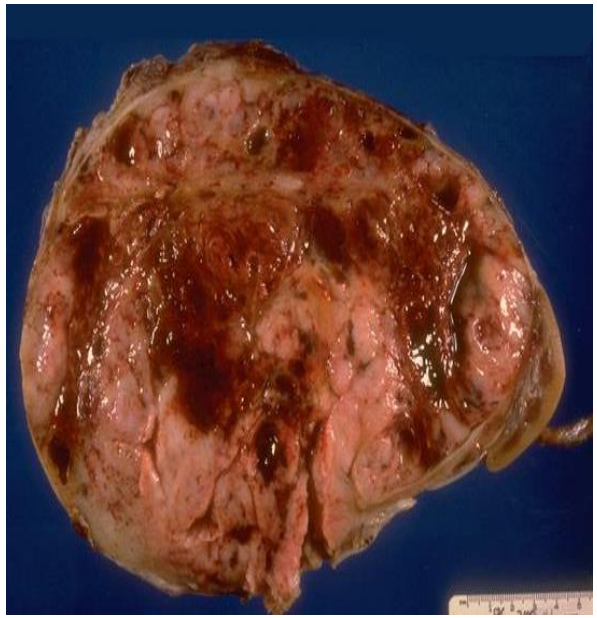


Section shows clear tumor cells with pleomorphic nuclei and areas of hemorrhage .

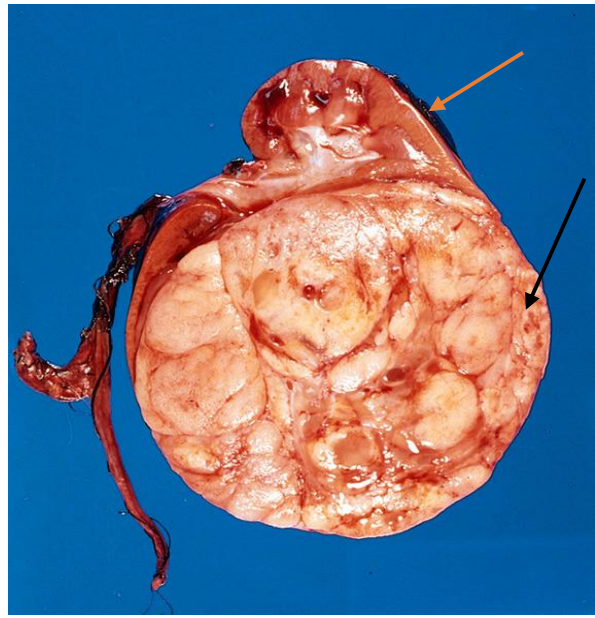
#Case11
WILM'S TUMOR



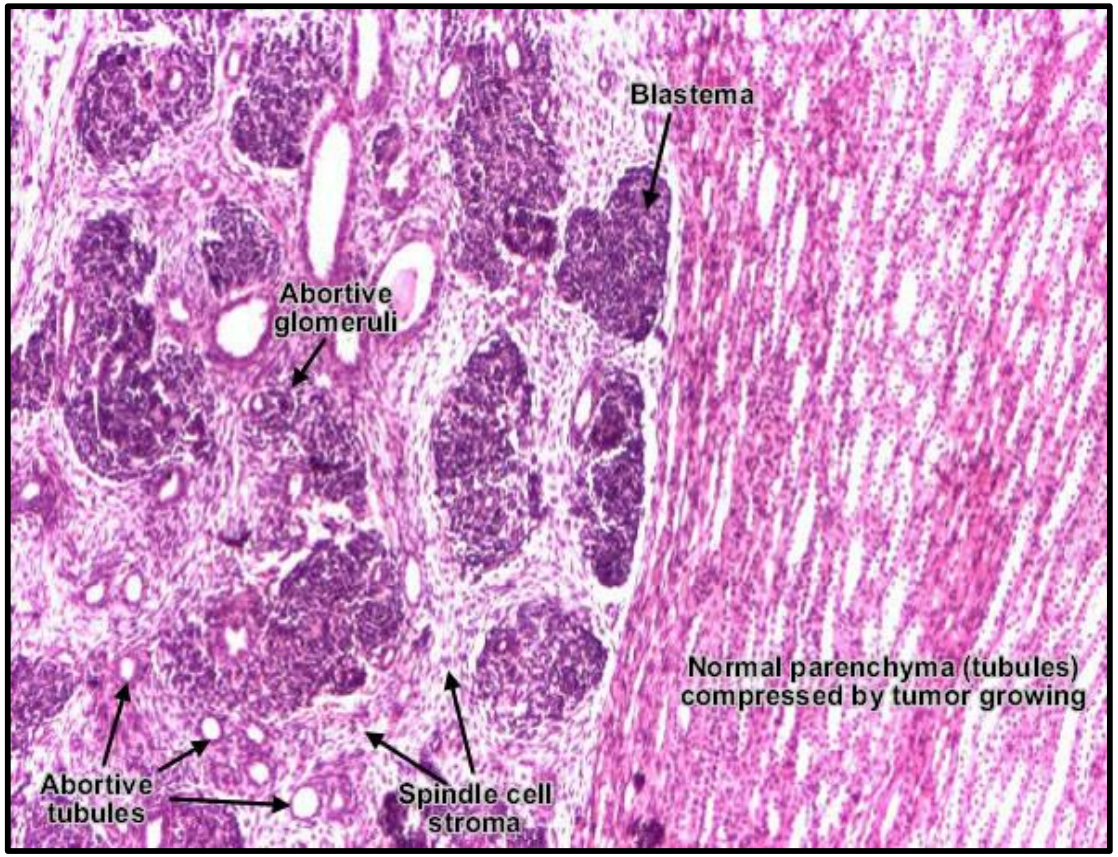
- Large well-circumscribed renal mass .
- Pale, gray and haemorrhagic cut surface.

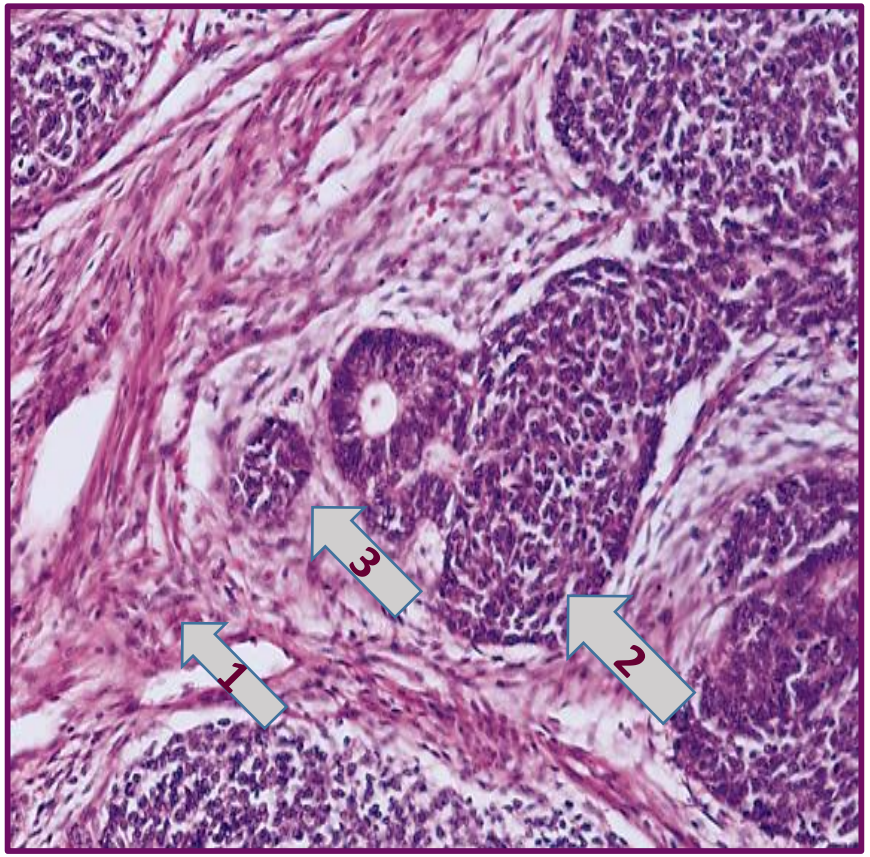


- Picture shows partly pale and partly hemorrhagic solid tumor replacing almost the entire renal parenchyma .
- *areas of necrosis also seen.*



Remenant kidney.
Wilm's tumor .





Blastema in WT consists of:

- sheets of densely packed small blue cells
- with hyperchromatic nuclei
- little cytoplasm
- conspicuous mitotic activity

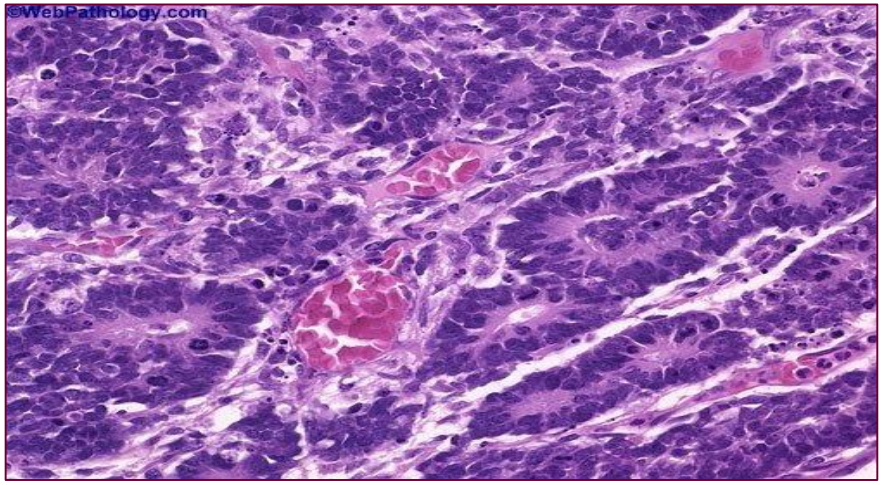
- Spindle cell stroma.
- Blastema.
- Abortive glomeruli.

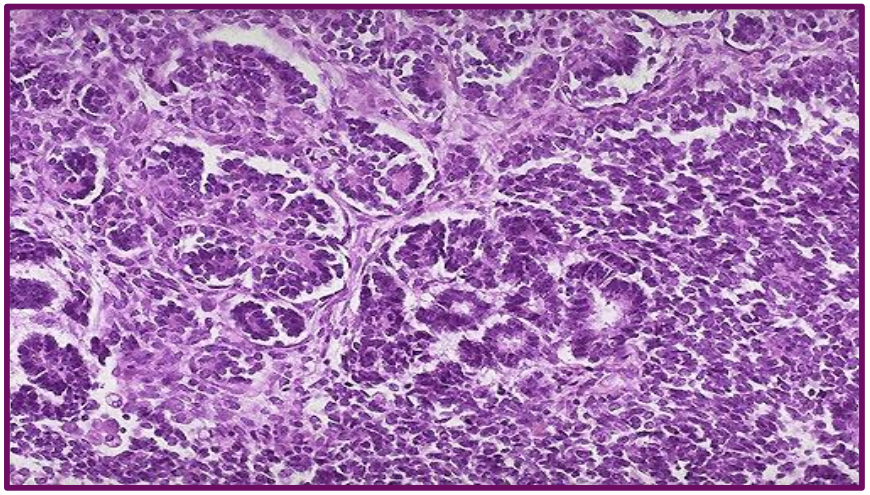
Syndromes that are associated with Wilms Tumor are:

a. *WAGR syndrome.*
b. *Denys-Drash syndrome.*

*G*ene is mutated in this condition:

WTI gene located on chromosome 11p13



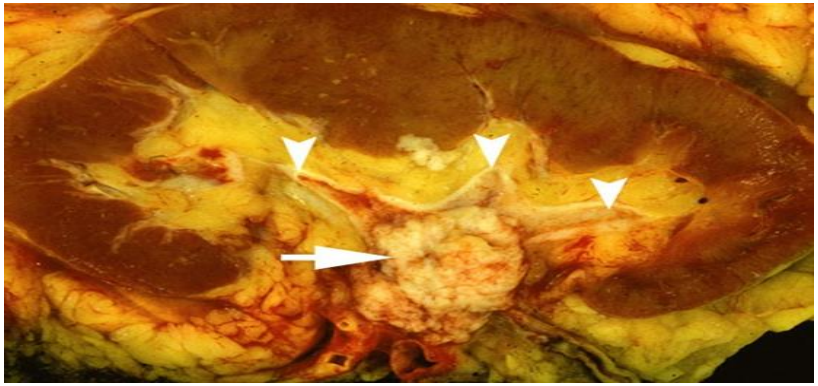


The epithelial component consists of primitive cuboidal cells forming tubular structures and rosettes.

- Immature tubules
- Immature stromal elements and blastema

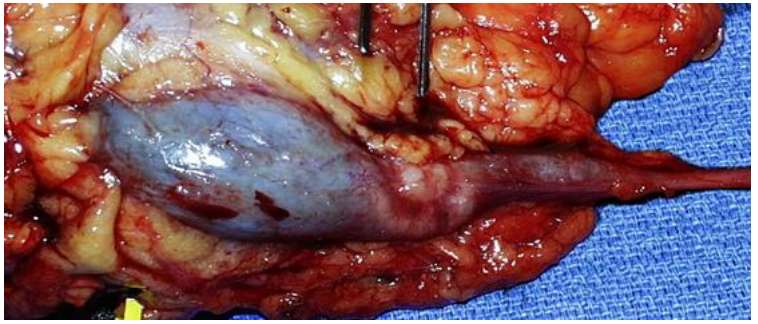
#Case12
CARCINOMA OF RENAL PELVIS AND URETET

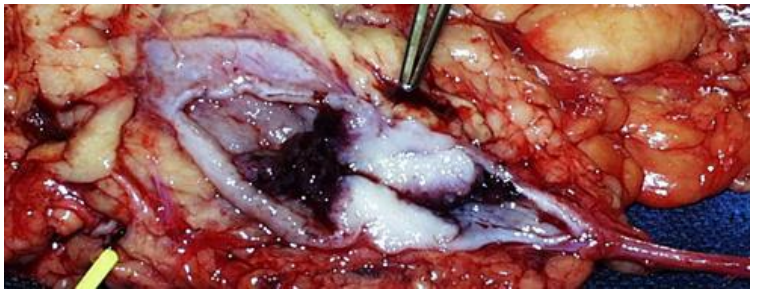
- Urothelial (Transitional) Carcinoma of Renal Pelvis



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

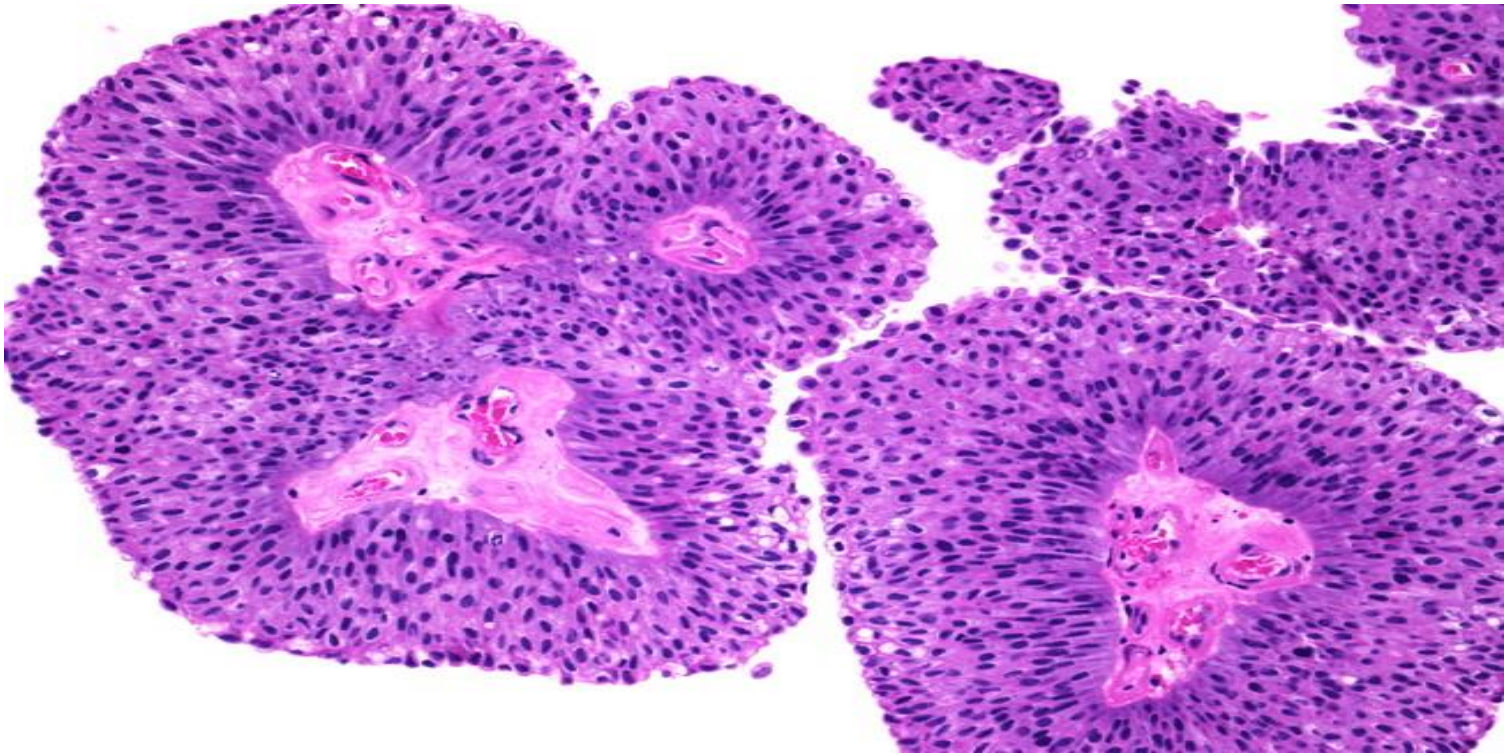
- Urothelial Carcinoma involving Ureter – Gross





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

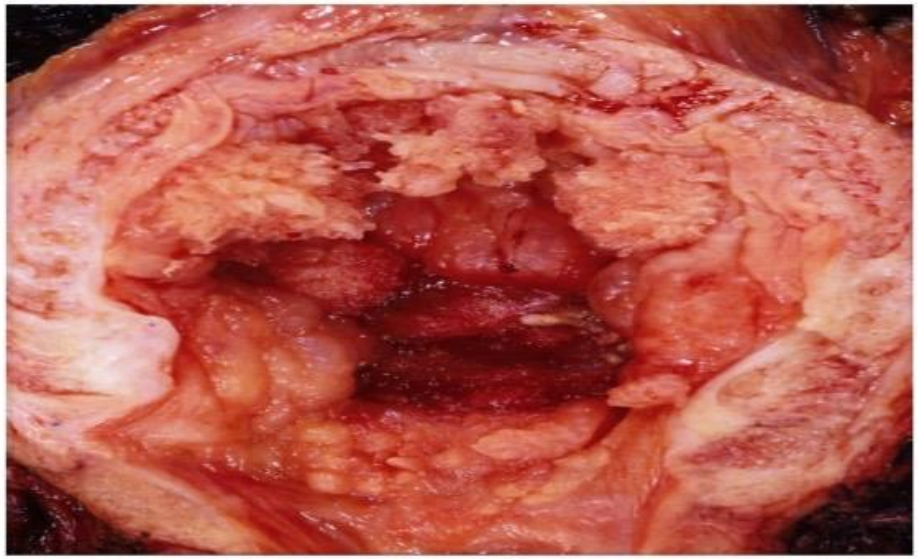
- Papillary Urothelial carcinoma of renal pelvis - lowe grade



Low-grade papillary urothelial carcinoma shows:
minimal cytologic and architectural atypia.
Adjacent papillary fronds may fuse

#Case13
CARCINOMA OF THE URINARY BLADDER

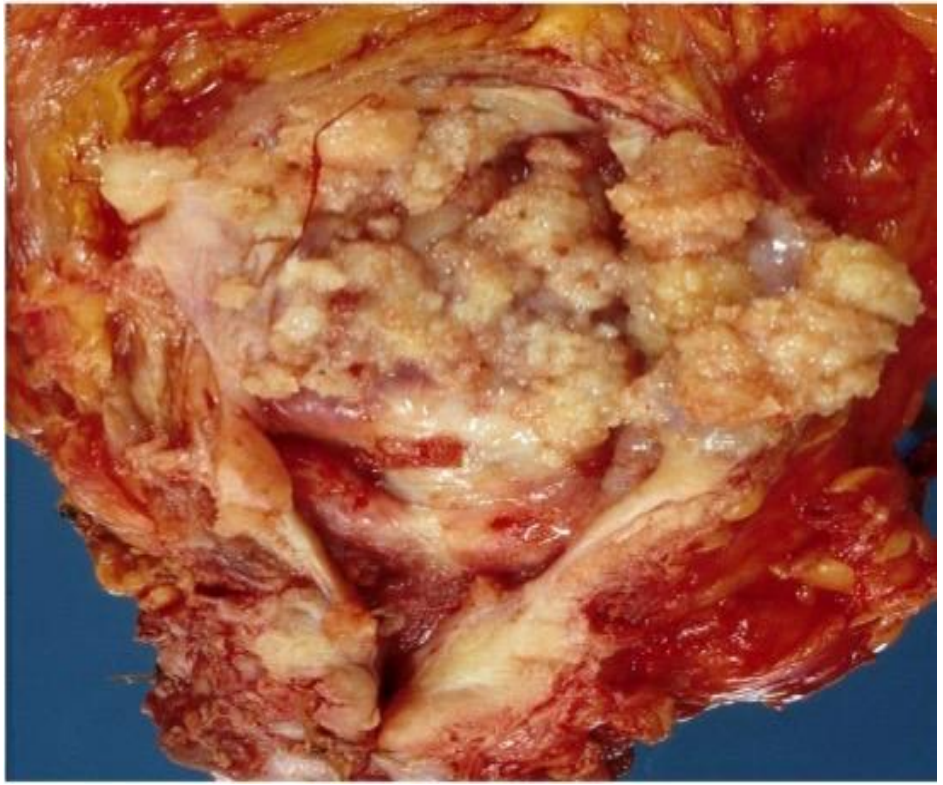
- Urothelial (Transitional cell) papillary carcinoma



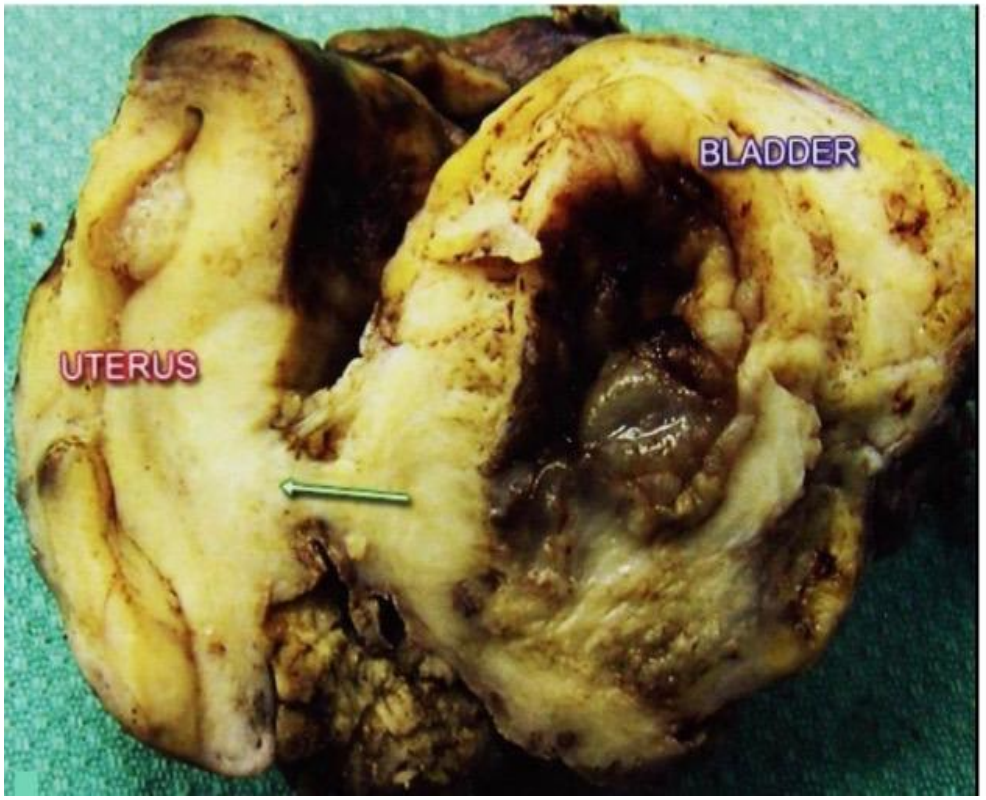
- 90% of bladder cancers are (Transitional) .
- Other 10% are squamous cell carcinoma , adenocaricnoma , sarcoma , small cell carcinoma and secondary metasteses .

Predisposing risk factors :

- Exposure to Beta Naphythlamine
- Schistosoma haemtobium infestation
- Cigarrete smoking
- Exposure to industrial solvents and dyes
- Exposure to cyclophosphamide
- Chronic cystitis

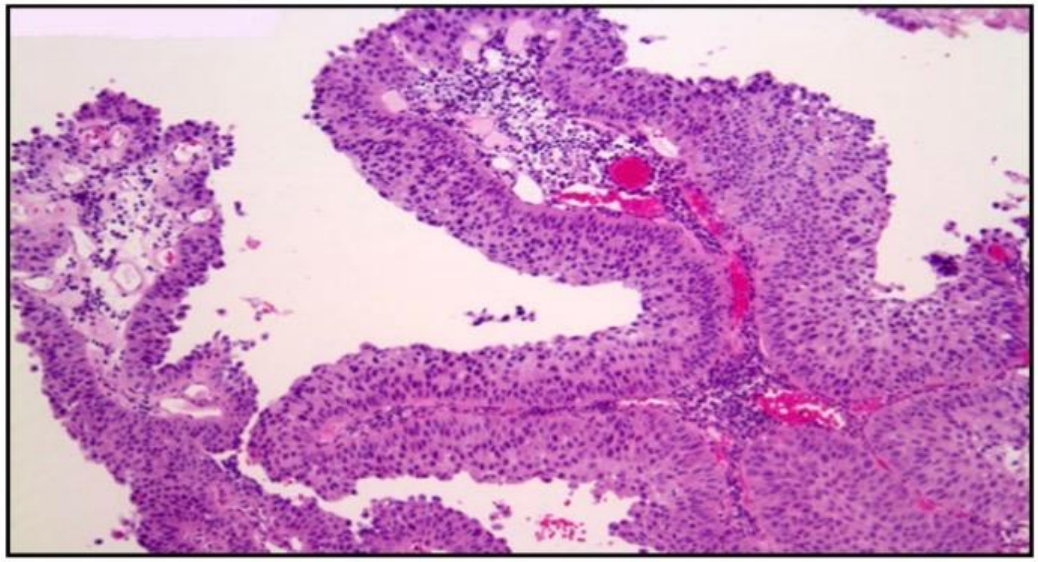


High grade transitional cell carcinoma of the UB showing solid and papillary pale neoplasm infiltrating the bladder wall and filling bladder lumen .



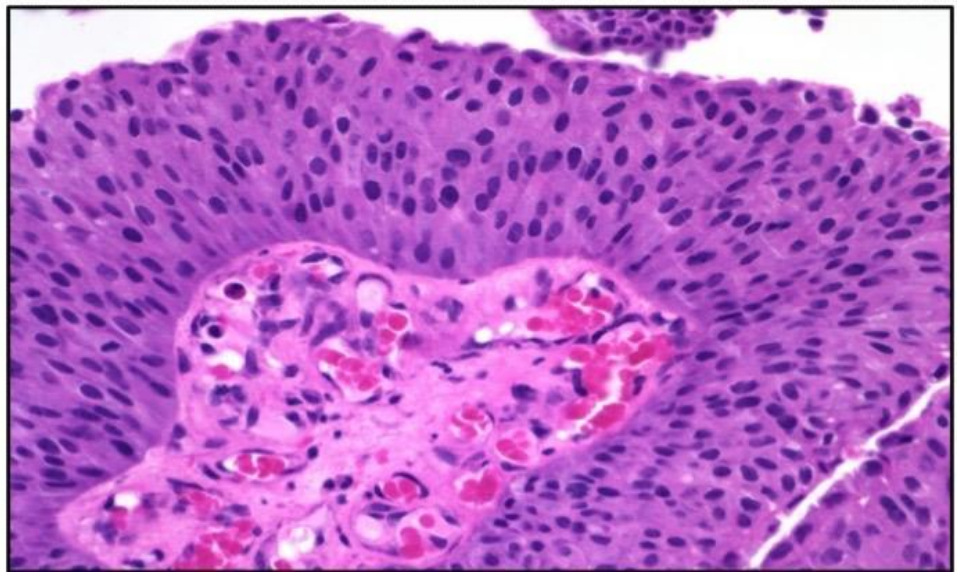
Urinary bladder carcinoma infiltrating the UB wall with extension of the uterus

- Papillary Urothelial carcinoma – low grade



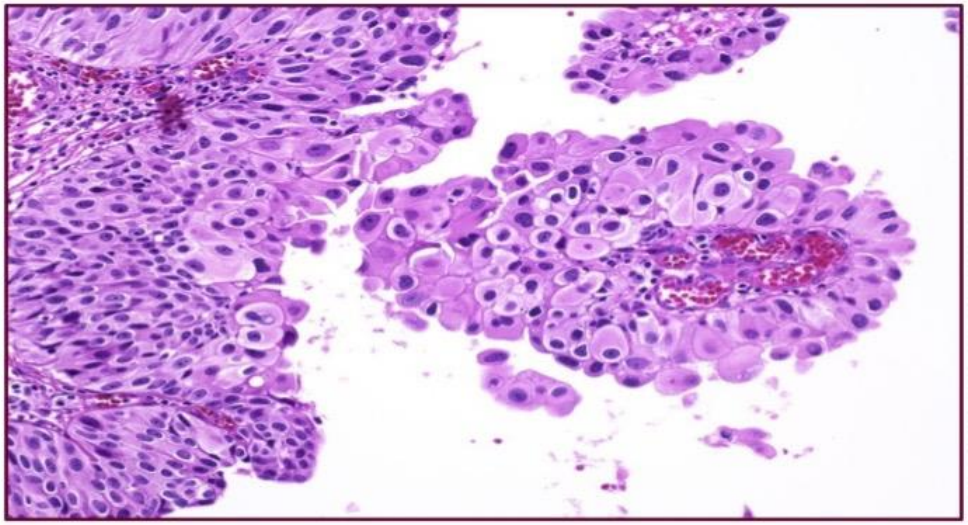
Low grade tumor show over all preservation of call polarity , few mitoses and lack of significant morphologic atypia . This exophytic papillary tumor shows multiple finger-like projections lined by multiple layers of urothelium (Transitional epithelium).

- Papillary Urothelial carcinoma – low grade

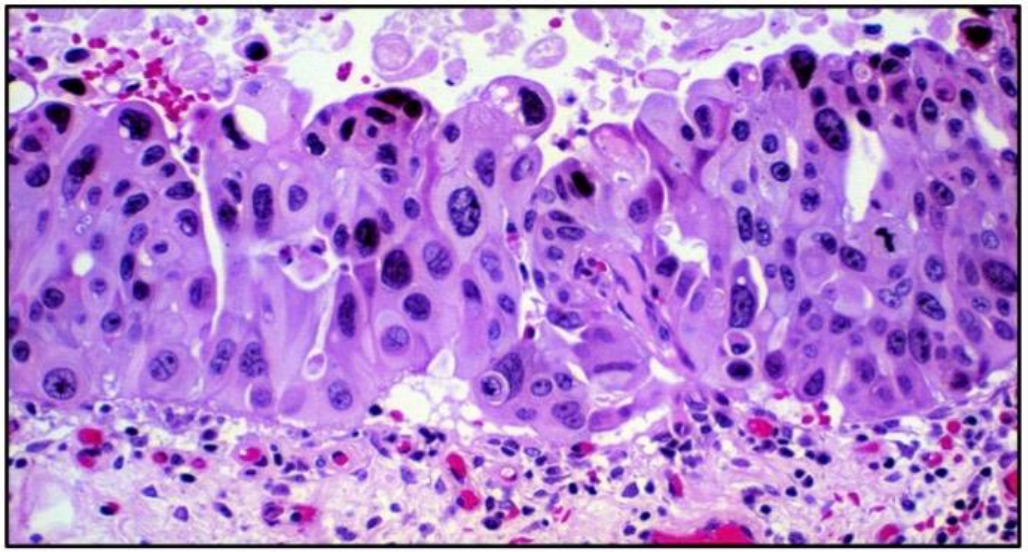


High power view of a low-grade papillary urothelial carcinoma . There are scattered hyperchromaatic nuclei and typical mitotic figures .

- Papillary Urothelial carcinoma – high grade



- Papillary Urothelial carcinoma – high grade



- Hyperchromasia
- Pleomorphisim
- Papillary transtional cells

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

#Case14
RENAL ALLOGRAFT

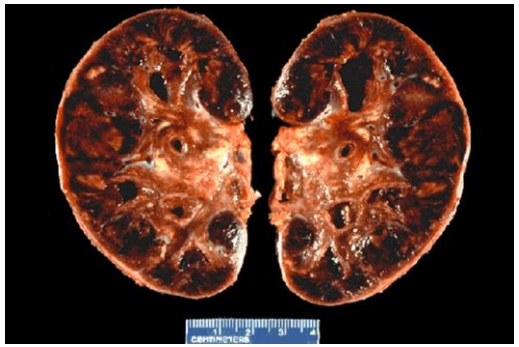
It is the transplantation of kidney from human to another, carrying different genotype.

Complications :

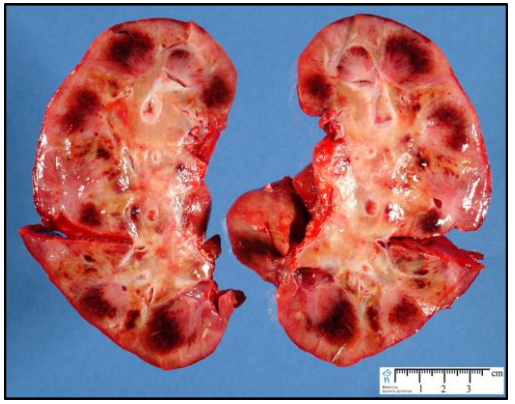
Rejection due to an immunological reaction and it has different types; acute, hyperacute and chronic.

1-Acute Cellular allograft rejection:

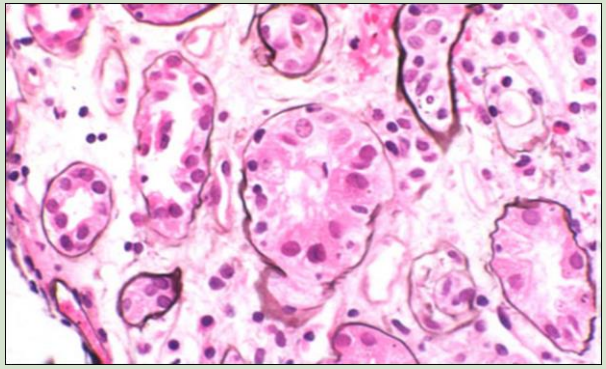
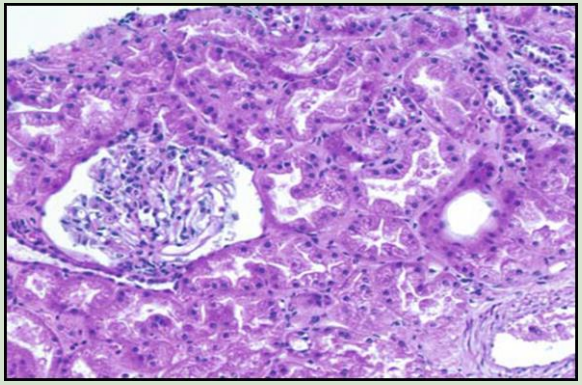
It could be interstitial or humoral.



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



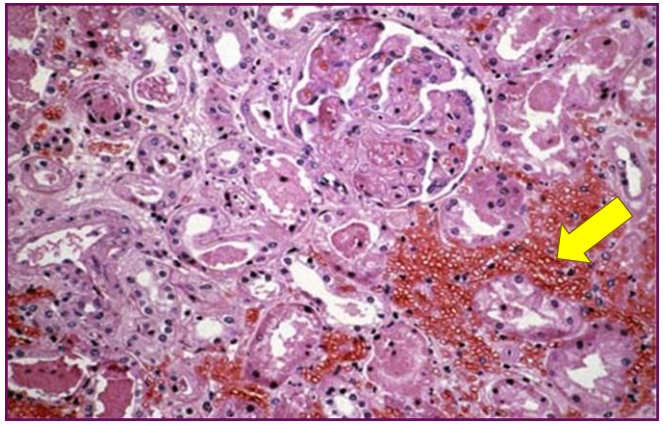
Swollen and hemorrhagic appearance of acutely rejected renal allograft

Acute Interstitial Allograft rejection-Type I	Acute Humoral Allograft rejection-Type I
	
Tubulitis, ie, infiltration of tubular epithelium by lymphocytes, is the hallmark of type I interstitial acute rejection.	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.

*We can differentiate between the Interstitial rejection and the humoral by the type of WBCs; Interstitial will have Lymphocytes while Humoral may have neutrophils. Also, the presence of necrosis. It presents in the Humoral.

Hyperacute Allograft Rejection:

Its hall mark is the presence of Glomeruli thrombi.

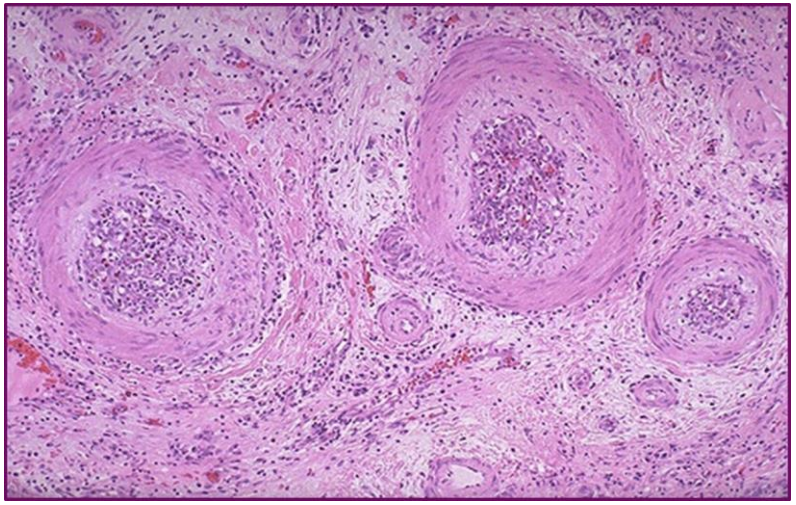


Hyperacute rejection. The cortex shows diffuse hemorrhage and neutrophils in peritubular capillaries with prominent glomerular thrombi 1 day after transplantation.

*My advice to you is to study the practical of Pathology with the lectures because Dr.Hala focused a lot on the histopathology.

Chronic Allograft Rejection:

The hall mark is Fibrosis.



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

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