MED437
KING SAUD UNIVERSITY

PATHOLOGY
TEAM 437

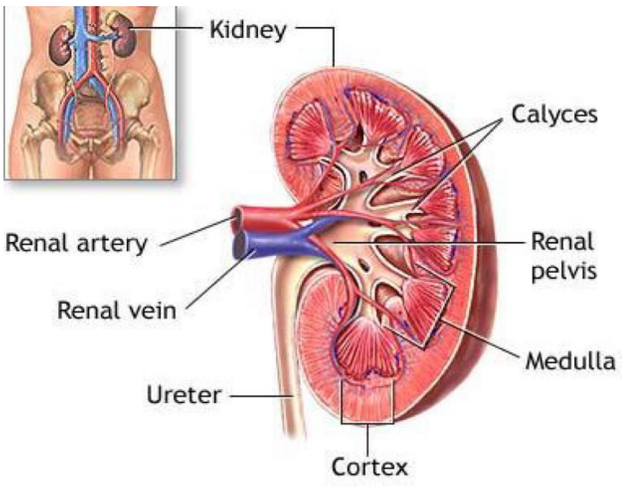
Pathology Practical

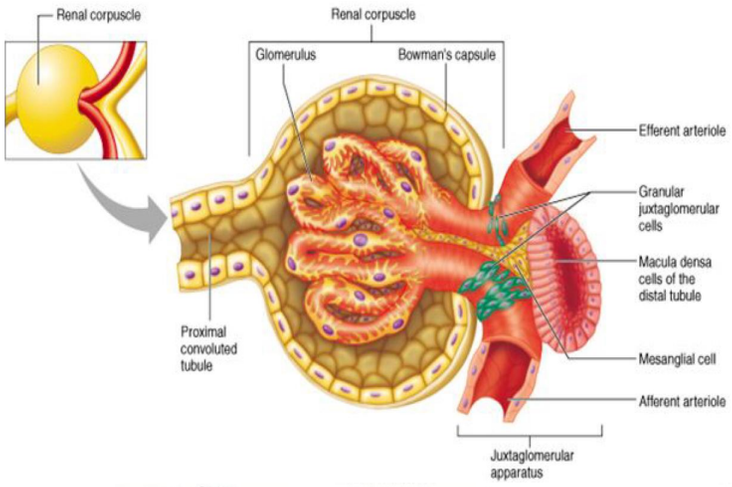
Renal Block

Pink: Only in female slides
Blue: Only in male slides
Grey: Notes

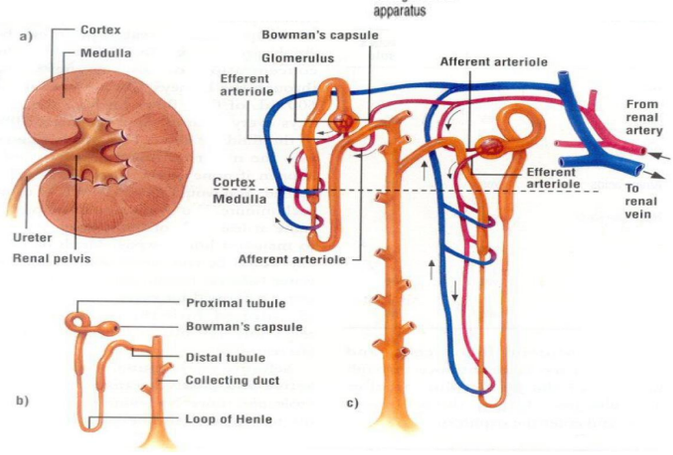
* لم تتم اضافة اضافات دكتورة البنات لانها غير مهمة + اضافات من الدكتورة فقط كـbackground informations لاننا ما اخذنا النظري قبل العملي

Normal anatomy & histology of the kidney

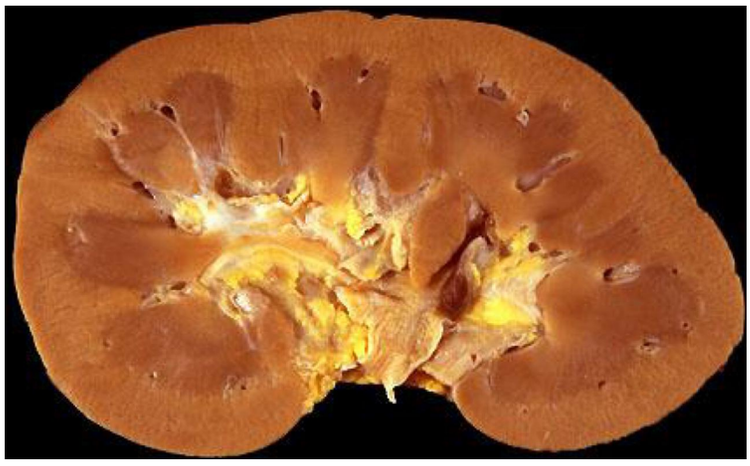




- Nephron is the functional unit of the kidney.
- Each kidney contains about 1,000,000 to 1,300,000 nephrons
- The nephron is composed of glomerulus and renal tubules
- The nephron performs its function by ultra filtration at glomerulus and secretion and reabsorption at renal tubules.

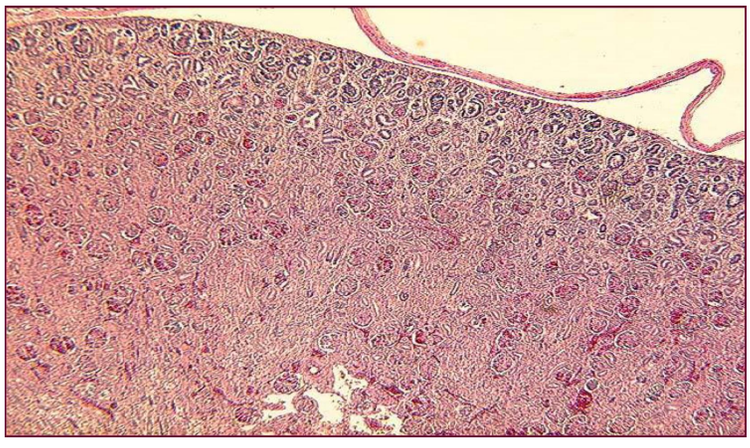


Gross:



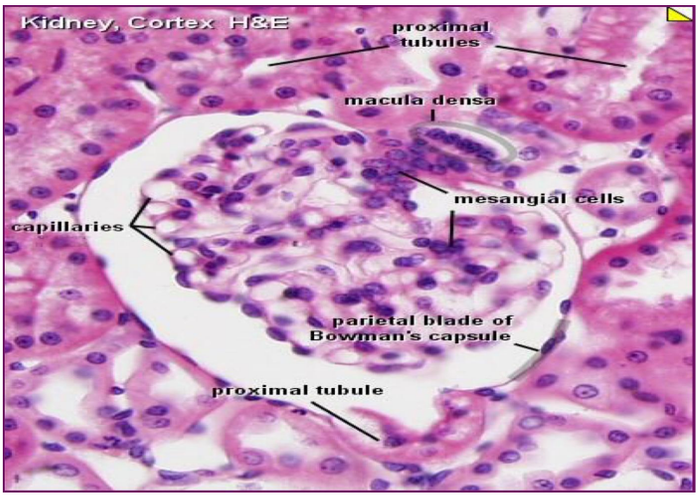
Normal adult kidney demonstrates:
- lighter outer cortex
- darker medulla
- renal pyramids into which the collecting ducts coalesce and drain into the calyces and central pelvis.

Histology:

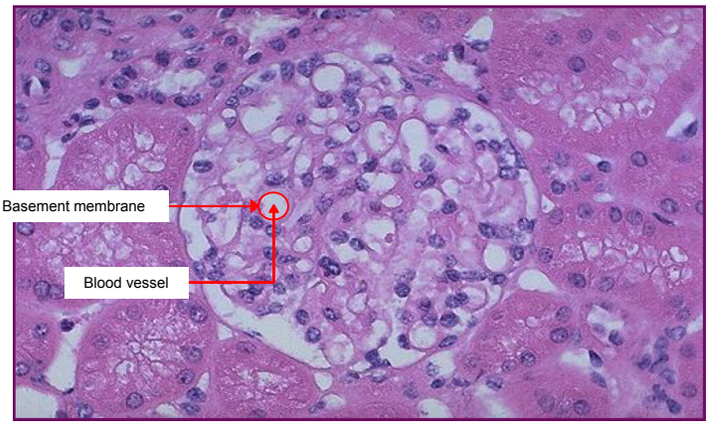


The kidneys show a well preserved lobular structure with indistinct corticomedullary demarcation

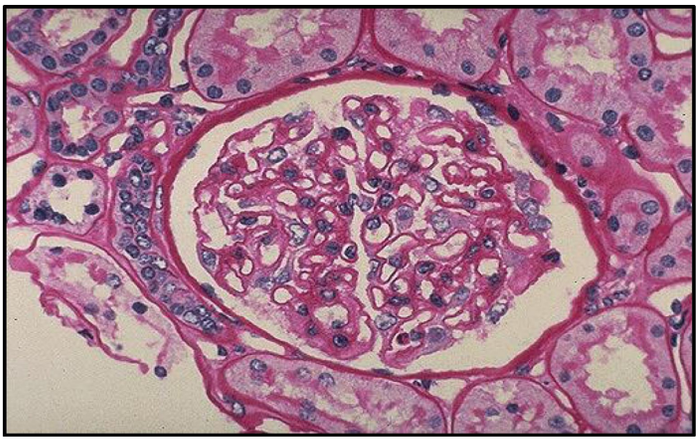
Renal Corpuscle:



No comment was written about this picture
(The wall of the cappilary should be thin, if it's thickened so there is disease)



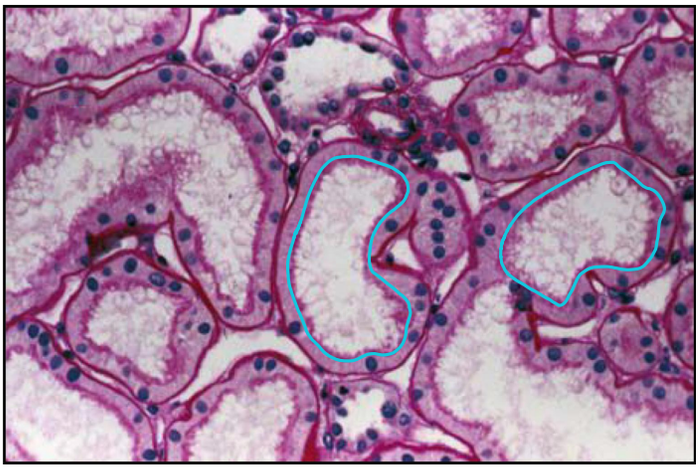
Normal glomerulus by light microscopy:
- The glomerular capillary loops are thin and delicate
- Endothelial and mesangial cells are normal in number
- The surrounding tubules are normal
- normally PCT has brush borders



- Normal glomerulus is stained with **PAS*** to highlight basement membranes of glomerular capillary loops and tubular epithelium.

*Periodic Acid Schiff

Normal Cortical Tubules:



- Normal cortical tubules
- Normal interstitium
- Normal peritubular capillaries
- most of the tubules are proximal
- well-defined brush borders **(PAS stain)**.

Case 1: Acute Kidney injury

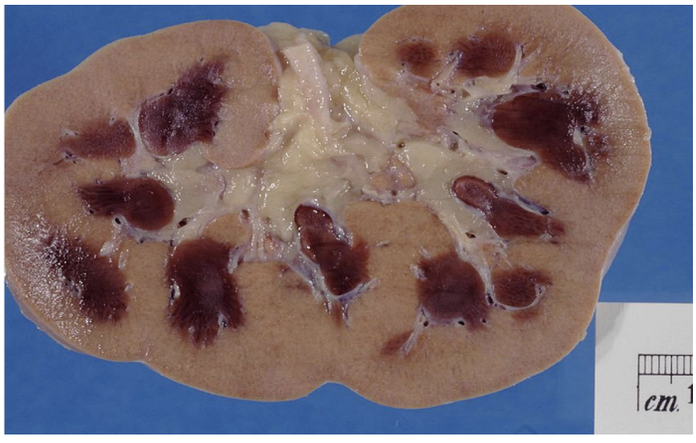
Causes

Pre-renal: (decreases effective blood flow to the kidney)
A- low blood volume, low blood pressure, heart failure
B- renal artery stenosis, renal vein thrombosis
C- renal ischemia

Renal:
A- Glomerulonephritis “GN”
B- Acute tubular necrosis “ATN”
C- Acute interstitial nephritis “AIN”

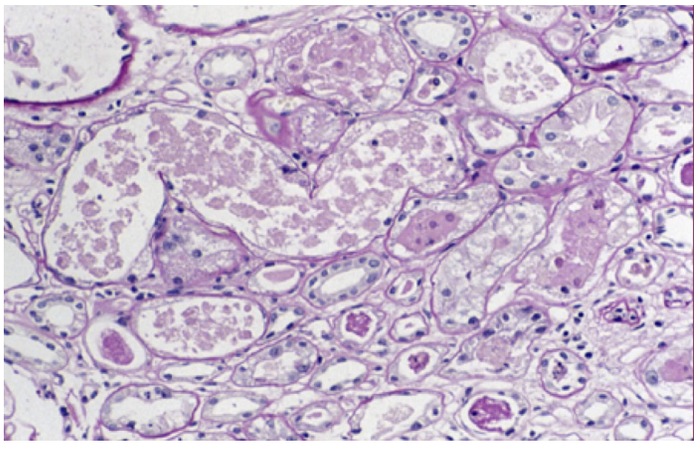
Post-renal: (consequence of urinary tract obstruction)
A- benign prostatic hyperplasia.
B- Bladder, ureteral or renal malignancy.
C- obstruction urinary catheter
D-bladder stones
E- Kidney stones.

Acute kidney injury :



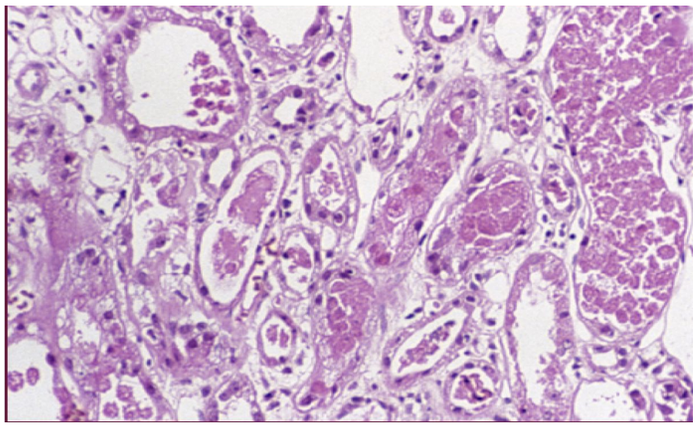
Kidney shows:
- Marked pallor of the cortex.
- Darker areas of surviving medullary tissue.

Acute Tubular Necrosis:



Acute tubular necrosis is manifest by:
- Vacuolated cells and sloughed.
- Necrotic cells in tubular lumina.
- Some tubules lined by flattened epithelium and some showing frank necrosis
- brush borders disappear due to necrosis

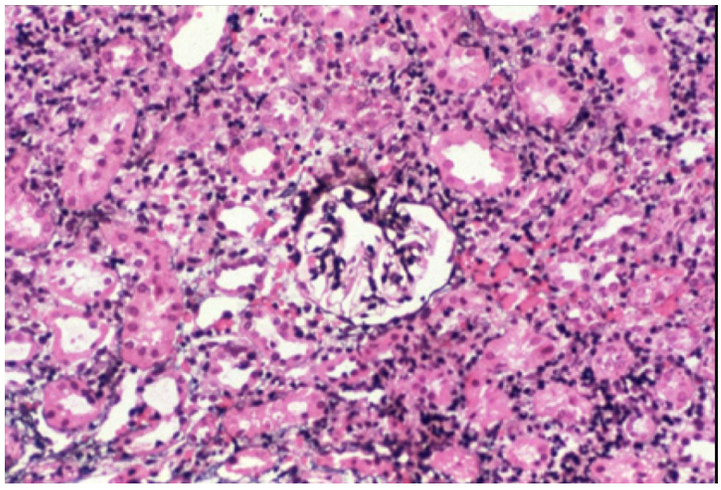
(PAS stain, x 400).



Acute tubular necrosis shows:
- Degeneration and frank necrosis of individual cells or tubular segments.
- Flattened, regenerating type epithelium with degenerated cells in the lumen **(middle left)**

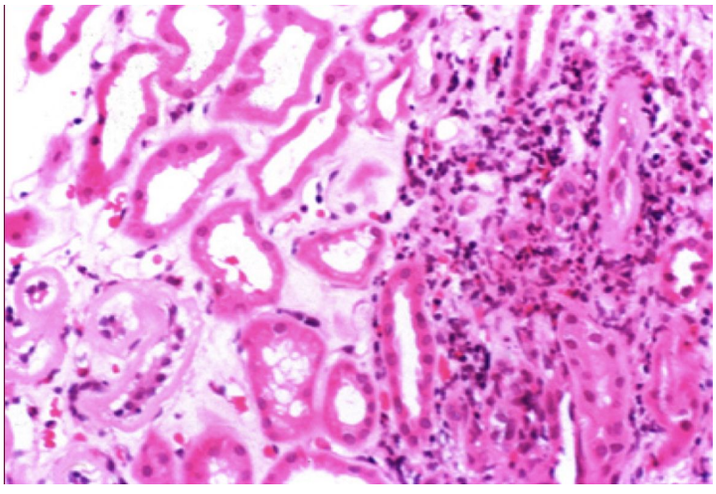
(H&E x 200)

Acute Interstitial Nephritis:



Acute interstitial nephritis shows:
- edema associated with an interstitial lymphoplasmocytic infiltrate.

There are numerous causes for acute interstitial nephritis:
A- toxins
B- viral infections
C- 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 (NSAID)



Acute interstitial nephritis shows:
- interstitial edema (left)
- preexisting mild tubulointerstitial fibrosis
- prominent interstitial eosinophilic component.
- lymphocytes and plasma cells

Cause: caused by drug-induced hypersensitivity.

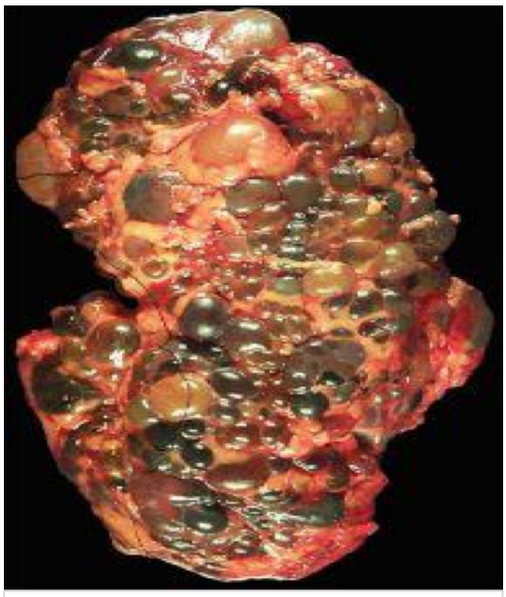
(H&E stain x 100)

Case 2: Polycystic Kidney

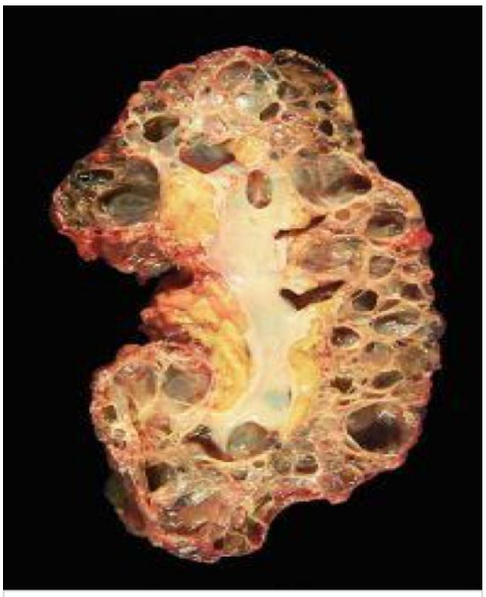
- The glomeruli is normal
- arise from the tubule and located in the cortex

Gross:

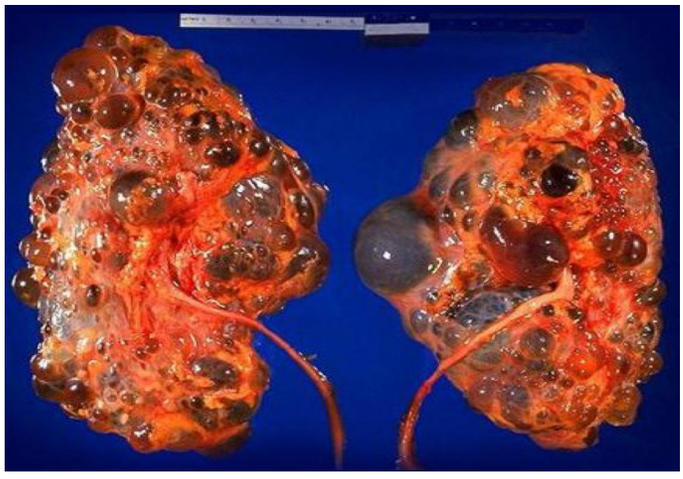
There is:
- Markedly enlarged kidney
- replacement of the renal parenchyma by numerous cysts of variable sizes



Massively enlarged kidney disrupted by numerous cysts

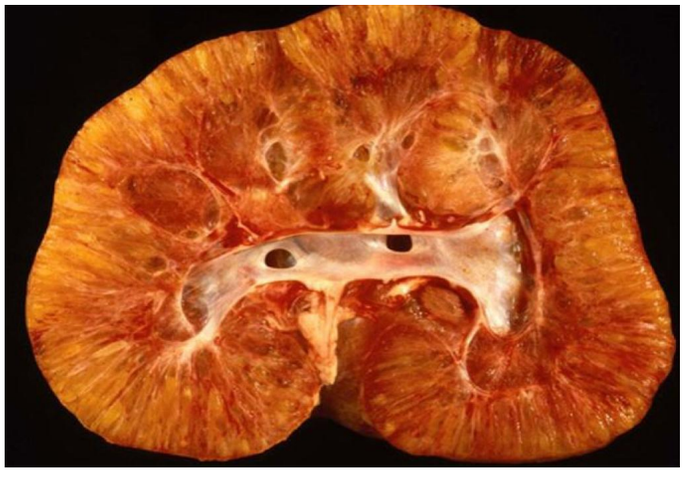


Cut surface of the kidney, showing:
- extensive cortical destruction by cysts

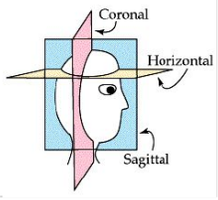


Bilateral autosomal dominant polycystic kidney disease

Infantile Polycystic kidney:

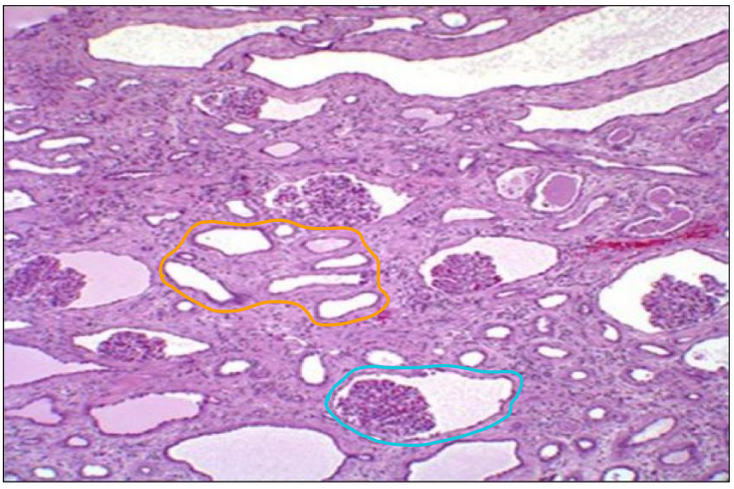


Coronal section of an infantile polycystic kidney

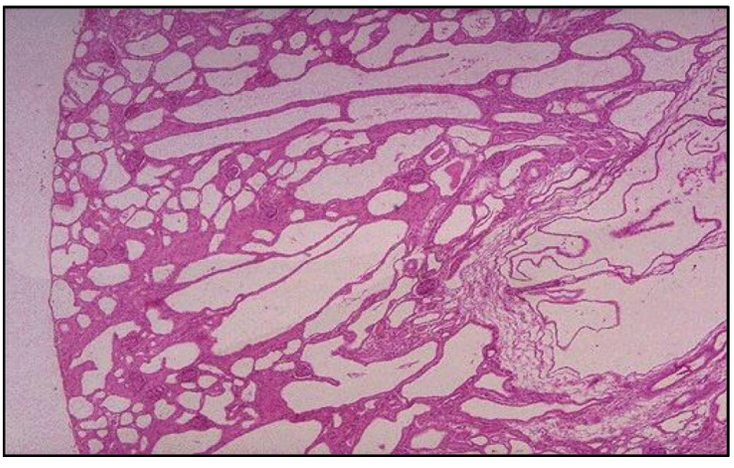


Histopathology:

*Polycystic kidney disease
PCKD or PKD



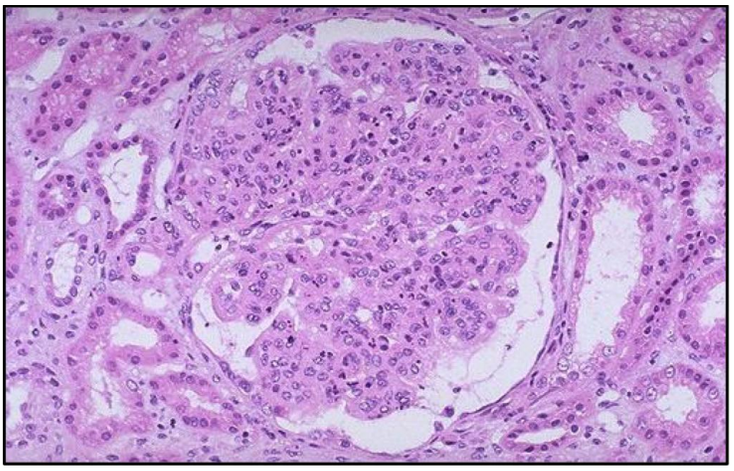
Kidney of child with autosomal dominant PCKD*.
- Coronal section of a polycystic kidney
- Histology demonstrating glomerular cysts
- normal-sized glomeruli
- enlarged Bowman's space
- tubular cystic changes
- (Occurs in adults)



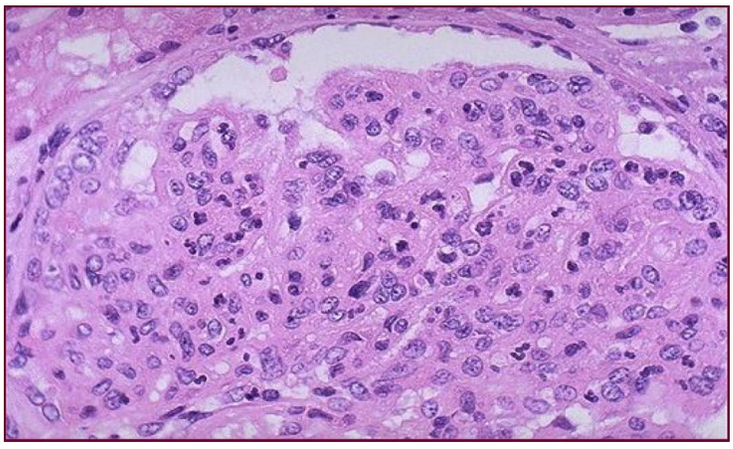
Autosomal Recessive Polycystic Kidney Disease (ARPKD)
- the cysts fill most of the parenchyma
- it is hard to find glomeruli
- (Occurs at age of birth)

Case 3: Acute (Post-streptococcal) Glomerulonephritis

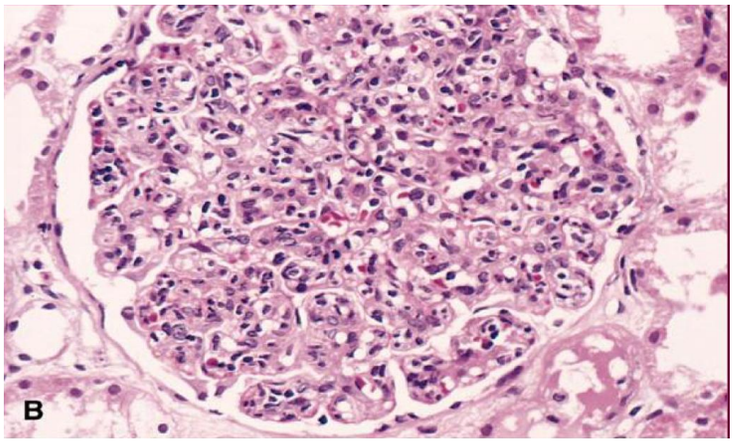
Section of the kidney will shows:
- The glomeruli are enlarged, lobulated and hypercellular with obliteration of capsular space.
- Cellularity is due to proliferation of endothelial and mesangial cells with some neutrophils.
- Many capillaries appear obliterated.
- Tubules show degenerative changes.



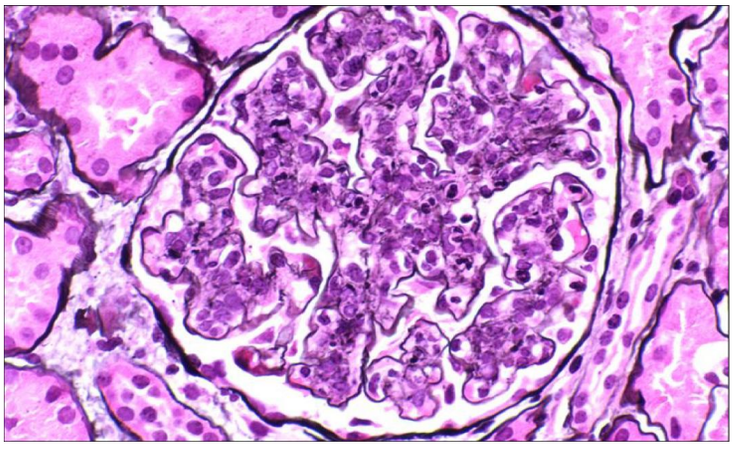
- This glomerulus is hypercellular and capillary loops are poorly defined.
- This is a type of proliferative (hypercellularity) glomerulonephritis known as **post-infectious glomerulonephritis**



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



High power LM of a hypercellular glomerulus shows:
- numerous capillaries contain inflammatory cells (mostly neutrophils)



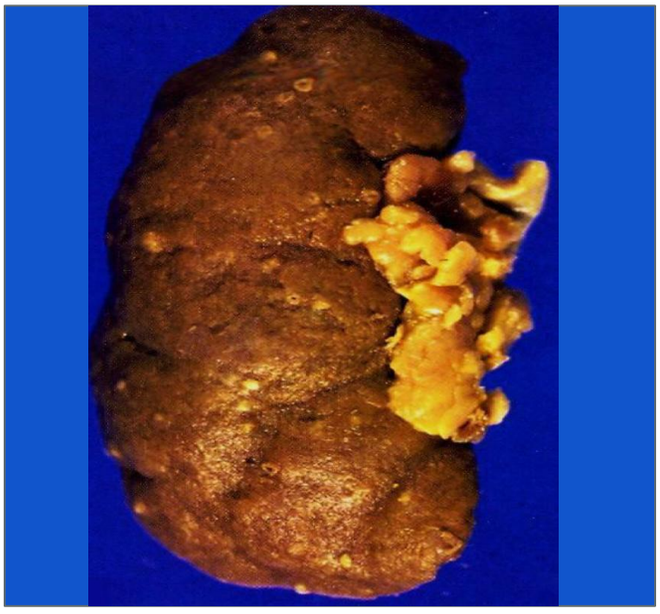
Acute Poststreptococcal Glomerulonephritis is evident in this high-power silver stain with large number of PMNs*.
- The glomerular basement membrane does not show **splitting** or **spikes**.
- There is **proliferation** of endothelial and mesangial cells and infiltrating cells and filling and distending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

*Polymorphonuclear neutrophils

Case 4: Pyelonephritis

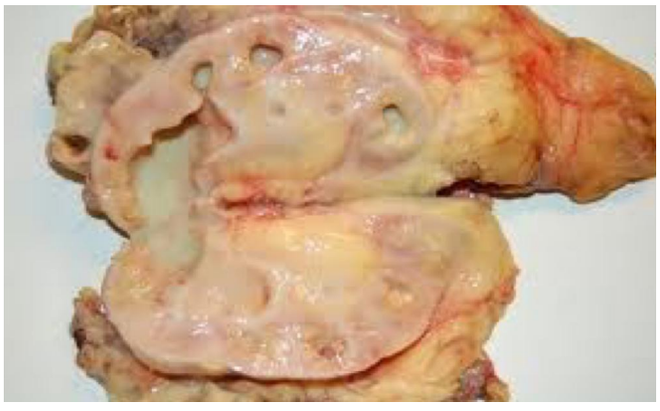
A- Acute Pyelonephritis

Gross:



Acute Pyelonephritis with small cortical abscesses

Note: pus = acute

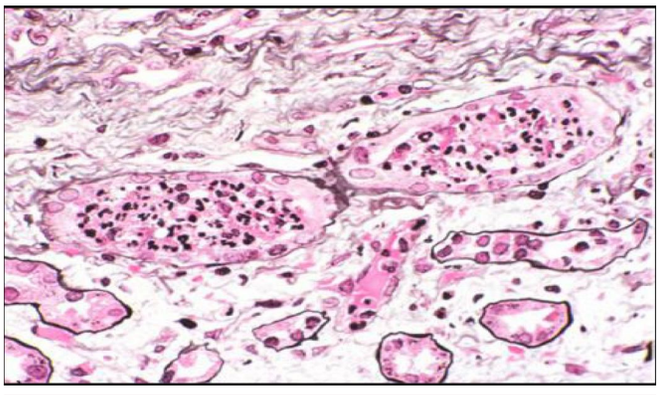


This kidney is bisected to reveal:
- a dilated pelvis
- calyxes filled with a yellow-green purulent pus which is consistent with a pyelonephritis.

The cortex and medulla are:
- pale
- the corticomedullary junction is ill-defined.

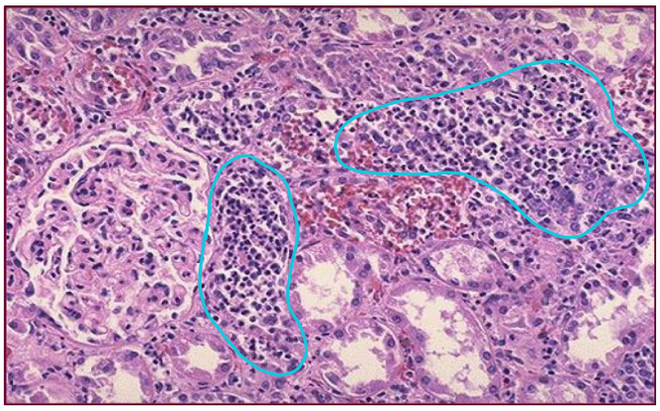
No tumors are seen

Histopathology:



Acute pyelonephritis is diagnosed by intratubular aggregations of polymorphonuclear neutrophils (PMNs).
There may be:
- surrounding interstitial inflammation
- mixture of PMNs, lymphocytes and plasma cells

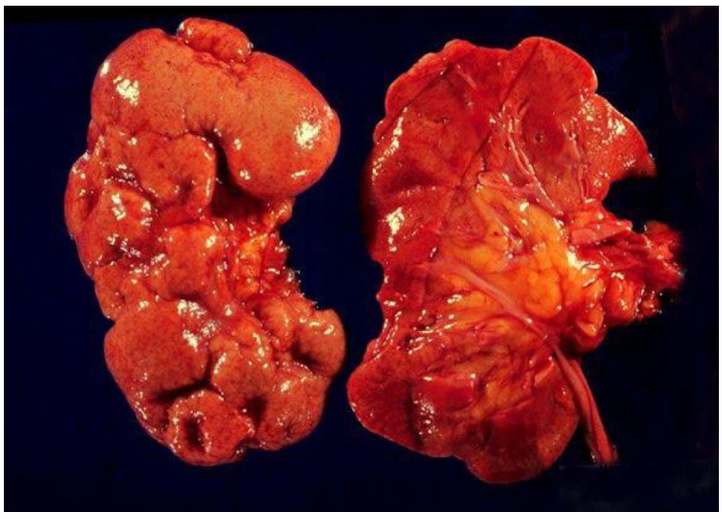
The predominant inflammation is within the tubule



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

B- Chronic Pyelonephritis

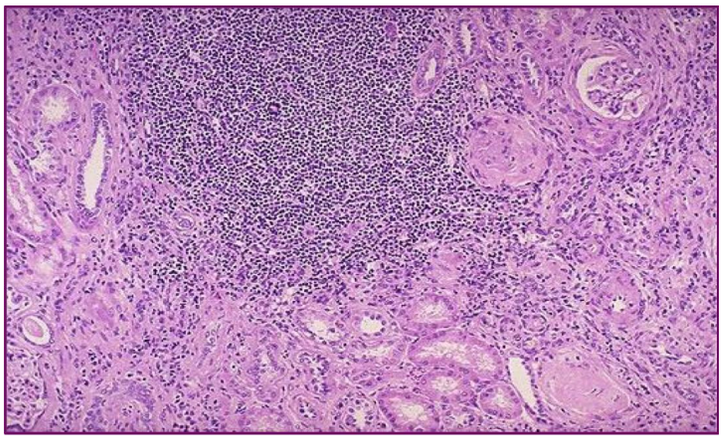
Gross:



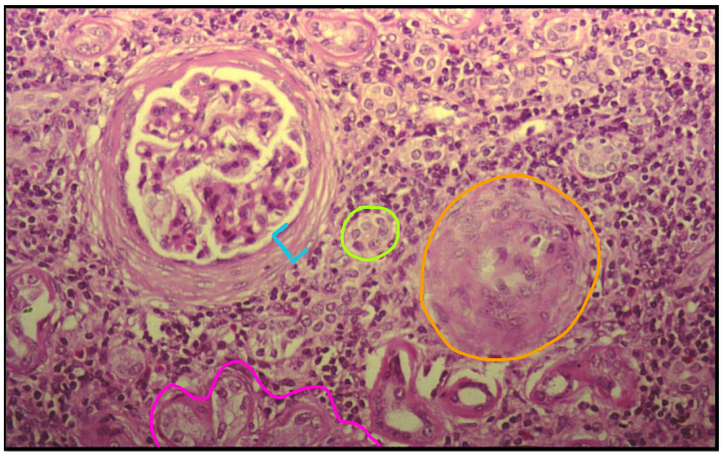
The picture shows:
- slightly atrophic kidney
- deformed kidneys
- cortical coarse scars
- shrinking of the kidney

The most common causes are:
- Obstruction by renal stones and others.
- Reflux uropathy – urinary reflux.
- Drugs like NSAID's, methicillin etc.
- Recurrent urinary tract infections.

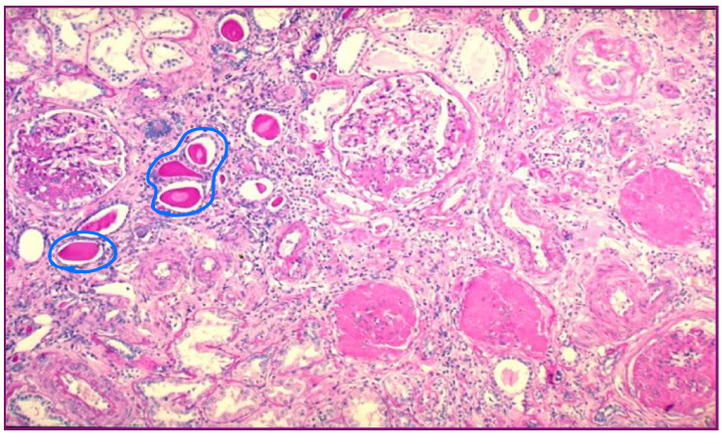
Histopathology:



- There is a large collection of chronic inflammatory cells (lymphocytes, plasma cells)
- The severity of disease depends upon the amount of remaining functional renal parenchyma (Renal failure – insufficiency or end – stage renal disease).



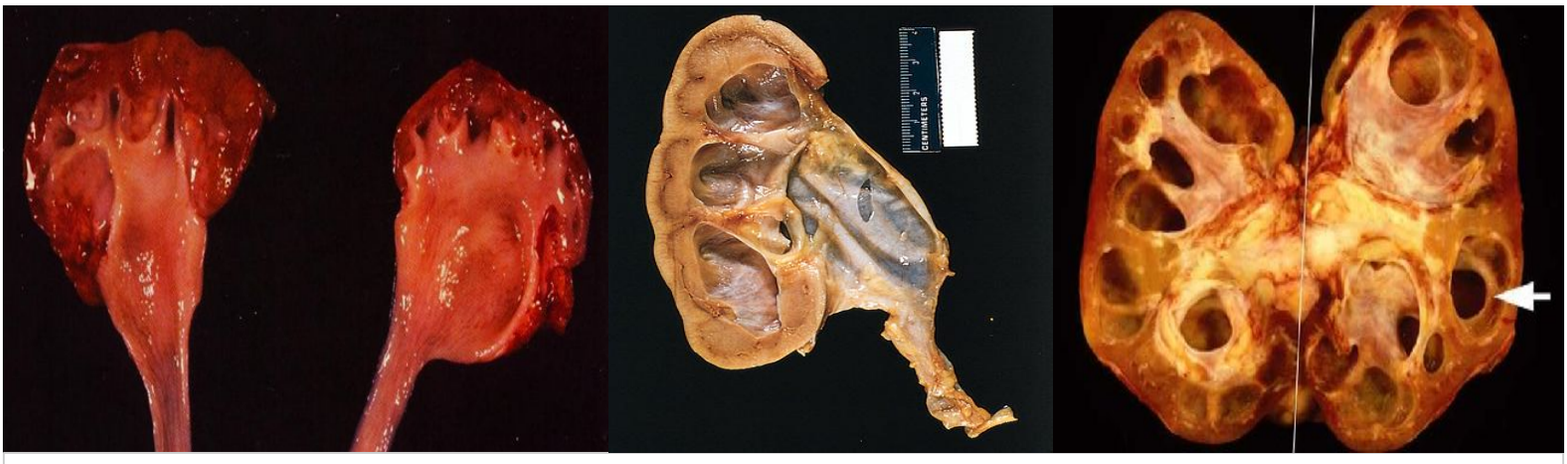
High power shows:
- periglomerular fibrosis
- glomerular sclerosis
- hyalinization and atrophy of renal tubules.
- marked chronic interstitial inflammation



- **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.

Case 5: Hydronephrosis

Gross:



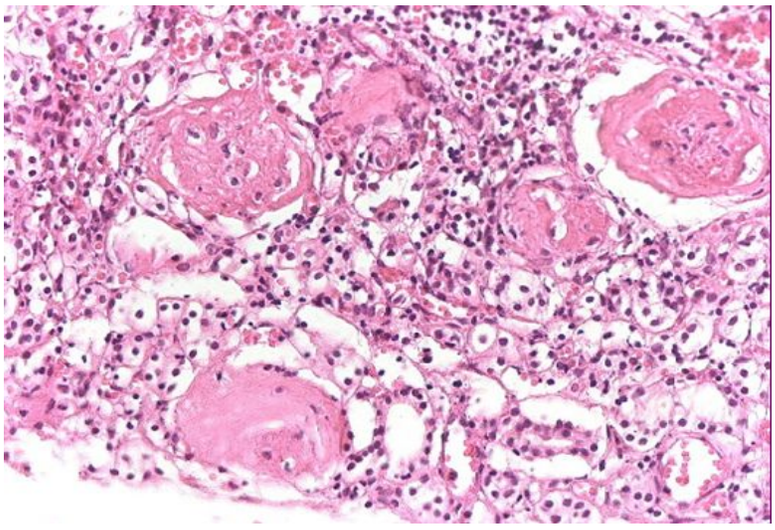
(Bisected kidney on the left) all 3 pictures showing:

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

The most common causes are:

1. Foreign bodies like calculi with obstruction,
2. Atresia (مغلقة) of the urethra,
3. Benign prostatic hyperplasia ,
4. Neoplasia of the prostate and bladder
5. Spinal cord damage with paralysis of the bladder .

Histopathology:



Chronic Pyelonephritis presenting as complication to Hydronephrosis, showing:
- Thinning renal parenchyma
- residual large renal vessels in the hilum.
- Sclerosis of glomeruli
- atrophic tubules

Case 6: Nephrotic syndrome

Causes of nephrotic syndrome: (only in girls slides)

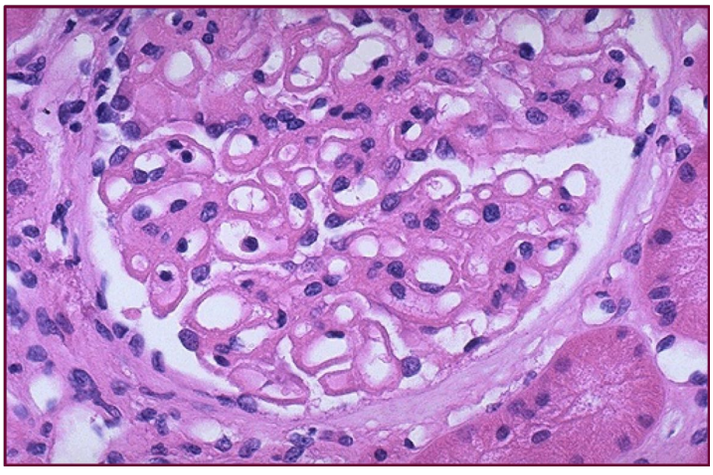
- **primary :** minimal-change nephropathy, membranous nephropathy, and focal glomerulosclerosis.
- **secondary**: systemic diseases such as diabetes mellitus, lupus erythematosus, myloidosis, infection and drugs.

The most common possible causes are: (only in males slides)

- Carcinoma (malignancy) - Drugs - Infections (Hepatitis B, Malaria) - Systemic lupus erythematosus. - Diabetic nephropathy.

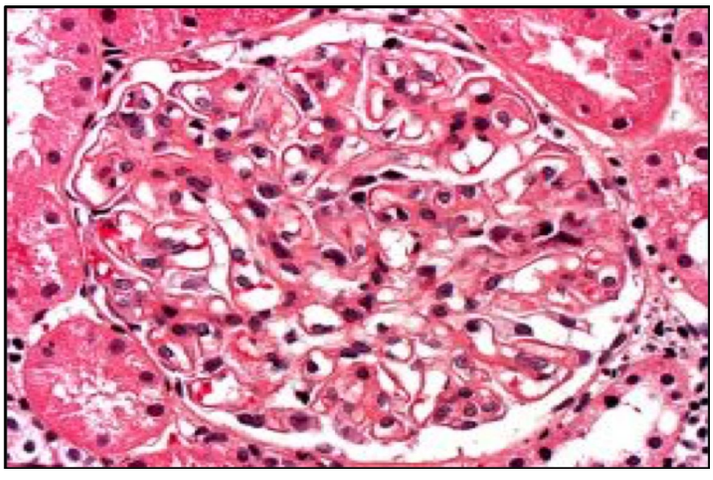
Membranous glomerulonephritis :

(The most common cause of Nephrotic syndrome in adults)

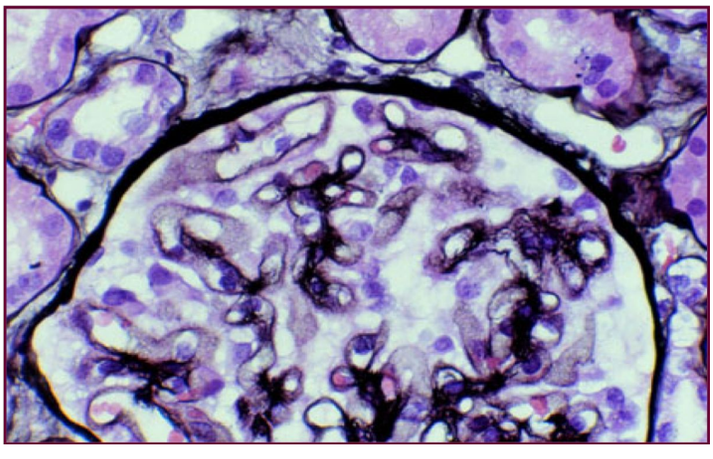


We can see:

- Thickened and primanet capillary wall
- The cellularity is not increased



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



stage II membranous glomerulonephritis:

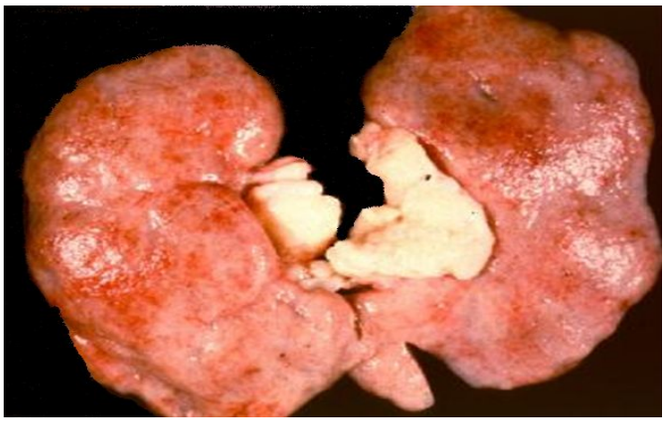
- The thickened capillary wall shows numerous "holes"indicating deposits (Deposits do not take up the silver stain.)
- Well-developed spikes around the deposits are not present here

ليش مو واضحة الspikes ؟ لانها خلاص بلعت الـdeposits والتقت الطرفين حقتها فصارت مو واضحة عالميمبرين

(Silver stain)

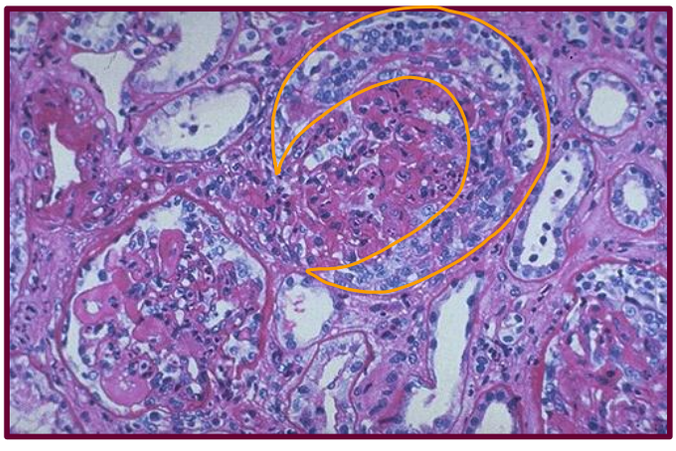
Case 7: NEPHRITIC SYNDROME (RPGN)

Gross: Rapid Progressive Glomerulonephritis (RPGN)



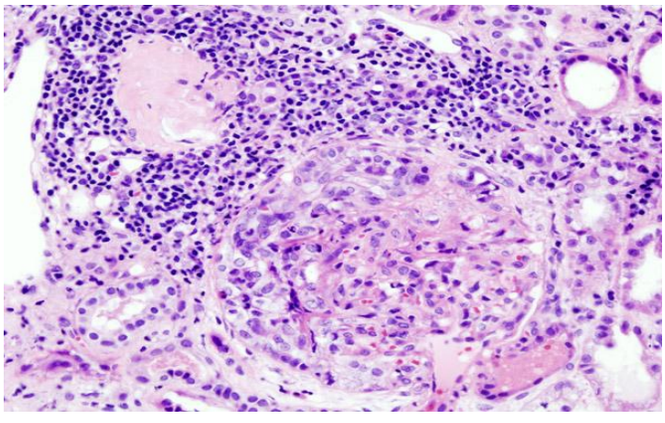
Gross appearance of RPGN:
- flea beaten appearance

Histopathology: Rapid Progressive Glomerulonephritis (RPGN)



Seen here:
- within the glomeruli are crescents composed of proliferating epithelial cells.

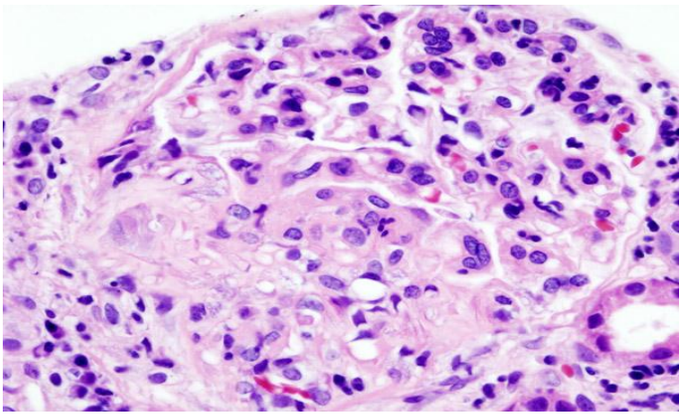
Crescentic glomerulonephritis is known as rapidly progressive glomerulonephritis (RPGN) because this disease is very progressive.



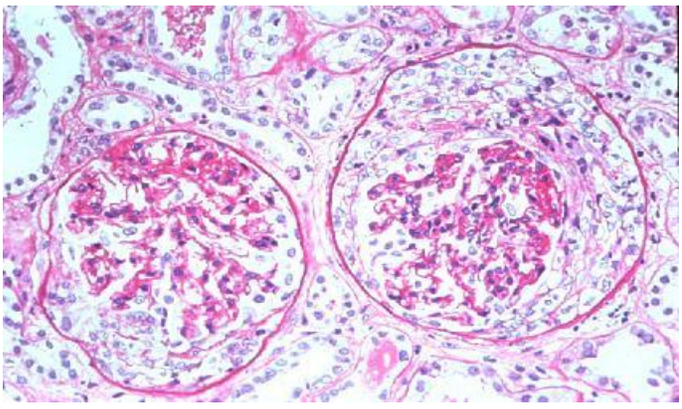
Crescentic glomerulonephritis in a patient with Rapid Progressive Glomerulonephritis (RPGN) . showing:

1. glomerular injury
2. formation of crescents
3. monocytes and macrophages proliferation compressing the glomerulus.

All types of RPGN are characterized by glomerular injury and formation of crescents with monocytes and macrophages proliferation compressing the glomerulus.

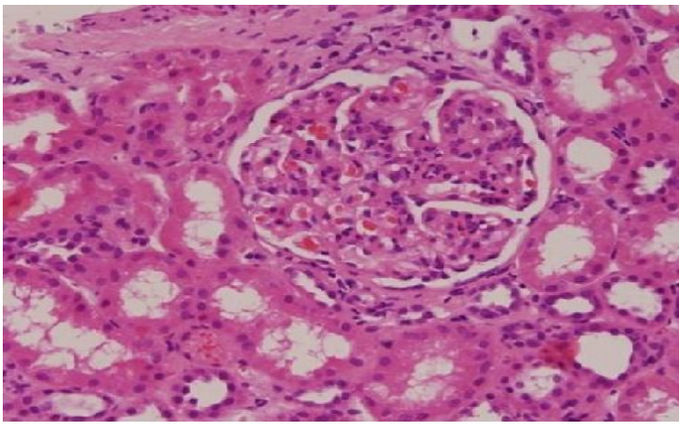


In severe injury:
- fibrin contribute most strongly to crescent formation.
- Epithelial cells of Bowman's capsule are proliferated.
- Infiltrating WBCs such as monocytes and macrophages also proliferate compressing the glomerulus, forming a **crescent-shaped scar.**



Rapid Progressive Glomerulonephritis (RPGN):
1-Epithelial cells of Bowman's capsule are proliferated
2-Infiltrating WBCs such as monocytes and macrophages also proliferate compressing the glomerulus, forming a crescent-shaped scar

Histopathology: Nephropathy : Nephritic / Nephrotic Syndrome

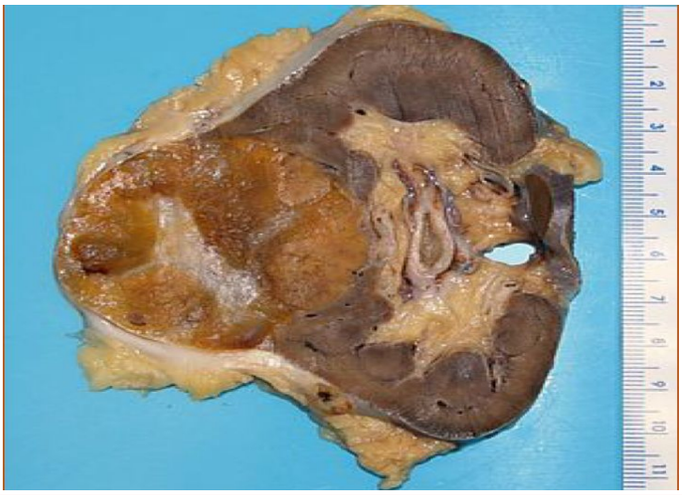


Nephritic / Nephrotic Syndrome showing:
- The glomeruli showed mesangial proliferation.
- The glomerular basement membrane was normal.
- The interstitium and blood vessels were unremarkable

Case 8: BENIGN RENAL TUMORS

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

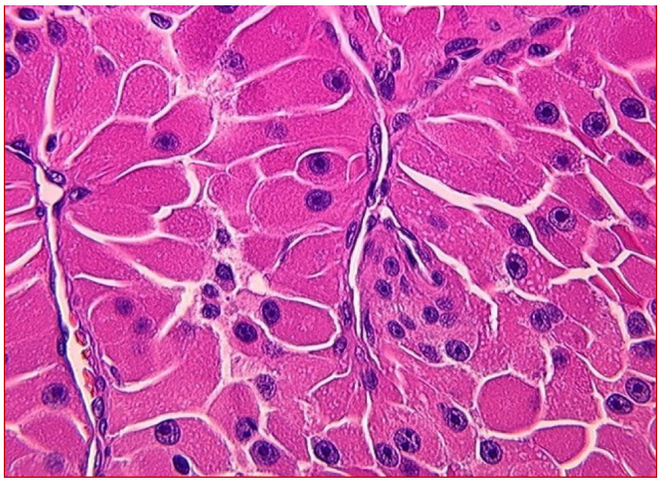
Gross:



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

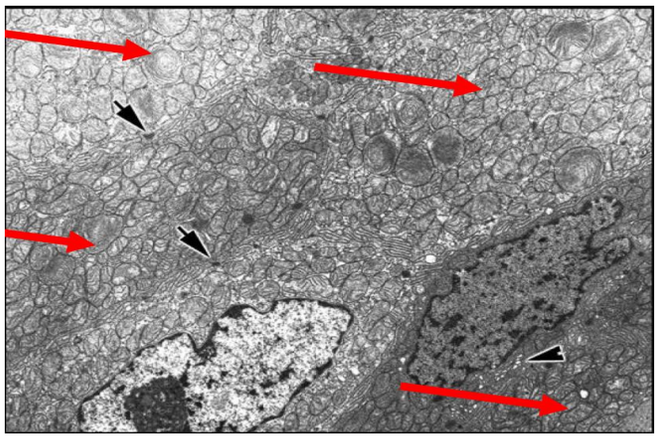
Note the:
- rounded contour (well circumscribed)
- Well-circumscribed mahogany colored renal mass
- central pale scar

Histopathology: (Oncocytoma)



Oncocytes are:
- very RED
- granular cytoplasm
- vesicular nuclei
- prominent nucleoli

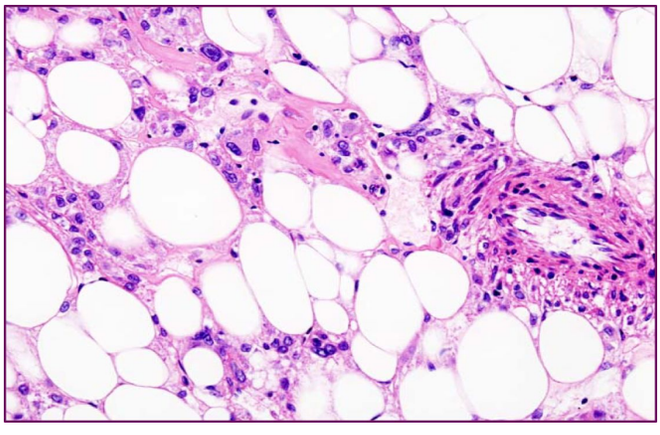
Nesting, alveolar or tubular patterns of uniform round / polygonal cells with abundant, intensely eosinophilic and granular cytoplasm, uniform small, round and central nuclei



Oncocytoma neoplastic cells filled with: (male slides only)
- mitochondria to the exclusion of almost all other organelles (red arrows).
- Note desmosomes (arrows)
- rare lysosomes, Golgi apparatus (arrowhead)
- ribosomes, and interdigitating plasmalemma.

These correspond to the oncocytoma cells with abundant granular, eosinophilic cytoplasm seen by light microscopy.

Histopathology: (Angiomyolipoma)



Benign tumor composed of:
- vessels (thick walled hyalinized vessels)
- smooth muscle (originate from vessel walls)
- fat (mature adipose tissue)

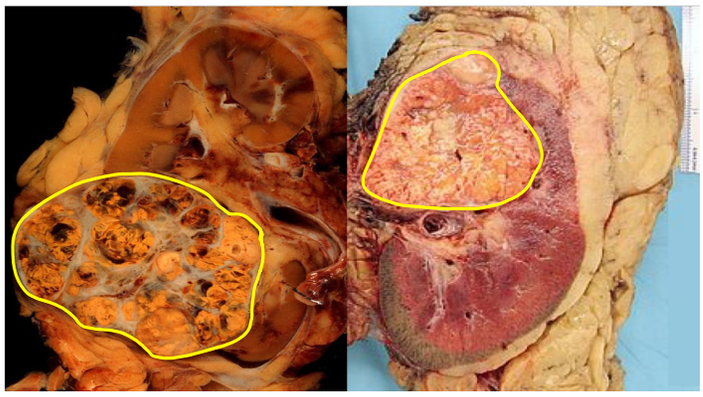
- Angiomyolipomas are present in 25% to 50% of patients with tuberous sclerosis.
- Tuberous sclerosis is characterized by lesions of the **cerebral cortex** that produce **epilepsy** and **mental retardation**, a variety of skin abnormalities, and unusual benign tumors at other sites, such as the heart.
- The clinical importance of angiomyolipoma is due largely to their susceptibility to spontaneous hemorrhage.

Case 9:Malignant tumor -Renal Clear Cell Carcinoma-.

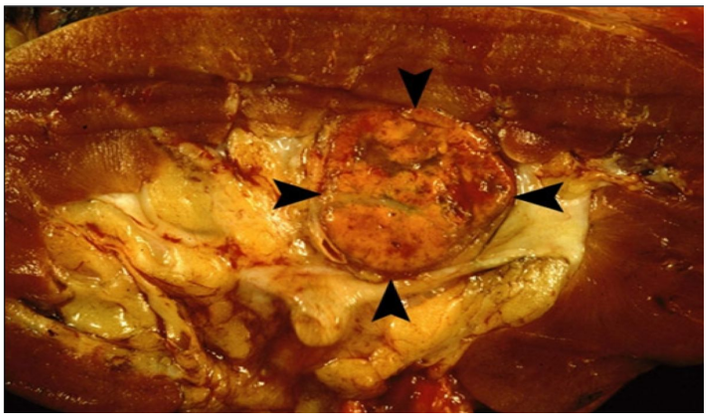
Renal Cell Carcinoma:
- Clear Cell Carcinoma
- Adenocarcinoma
- Hypernephroma

Urothelial (Transitional)

Gross:



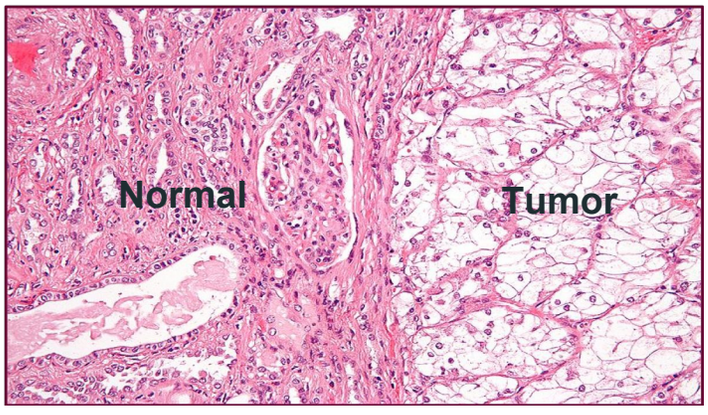
A well circumscribed renal cortical mass which is:
- partly yellow due to presence of fat
- partly hemorrhagic
- lobulated cut surface



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

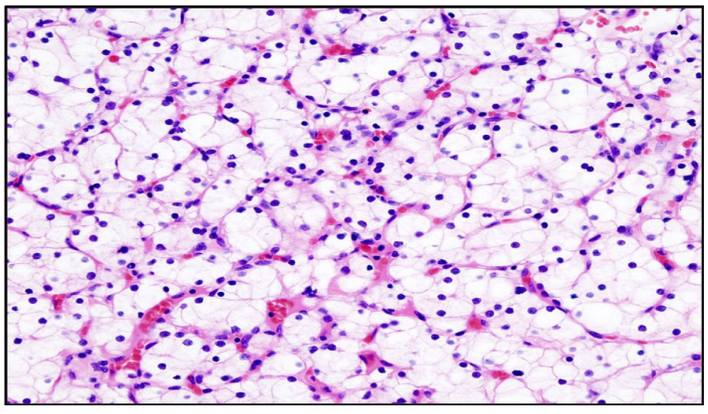
Histopathology:

- 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.
- Commonly associated with Von Hippel-Lindau disease that shows alterations in a gene localized on chromosome 3



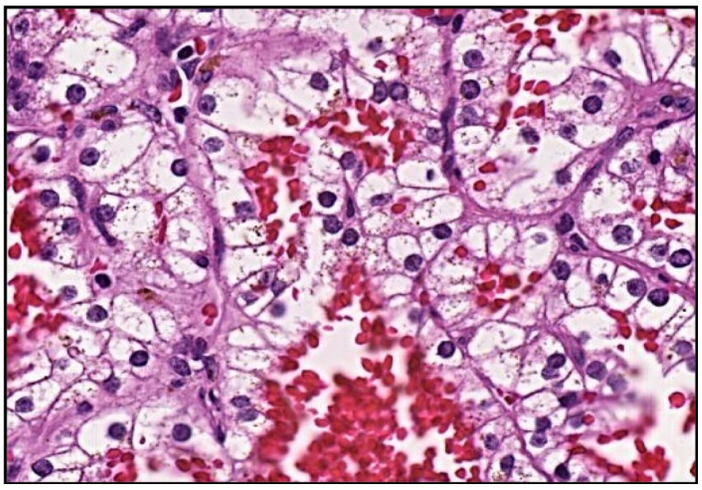
The most common type of renal cell carcinoma (clear cell) -on right of the image:
- Cells with clear cytoplasm, typically arranged in nests
- Nuclear atypia is common

Non-tumour kidney is on the left.



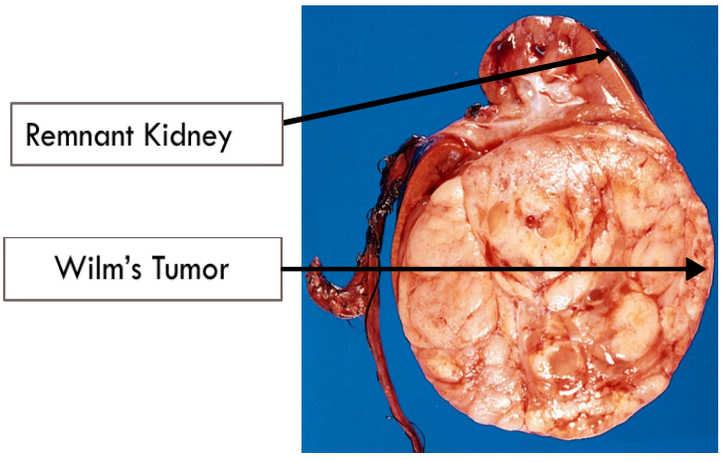
Tumor cells are:
- large polygonal
- clear cytoplasm (dissolved glycogen and lipid)
- piknotic nuclei

Cells show pleomorphism and mitosis.

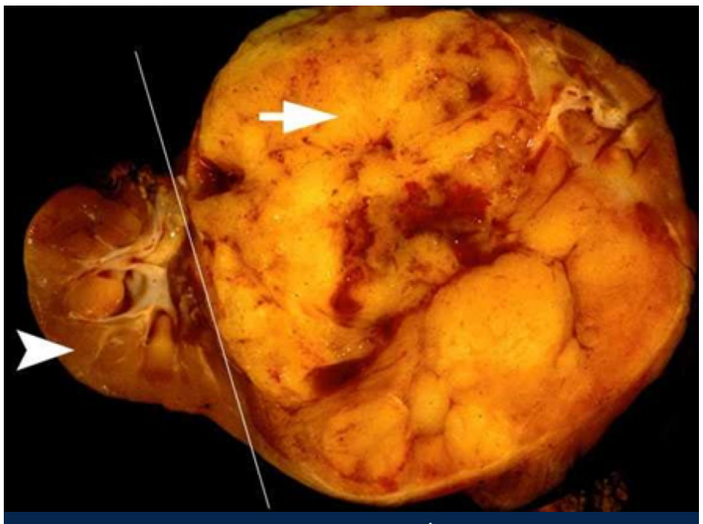


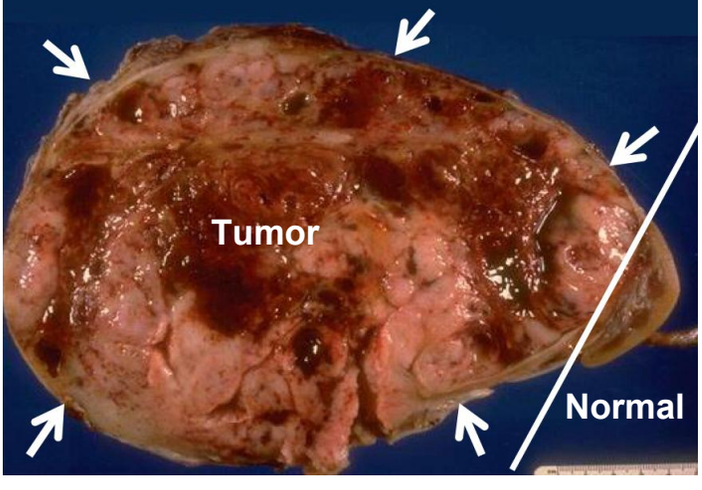
Section shows:
- clear tumor cells
- pleomorphic nuclei
- areas of hemorrhage

Case 10: WILM'S TUMOR



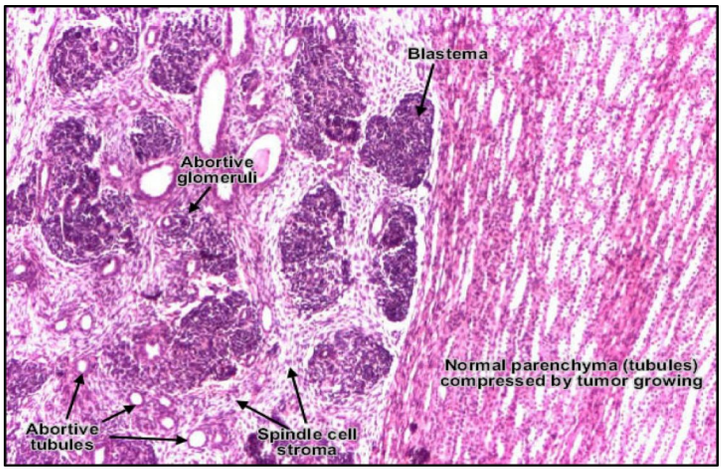
Gross:



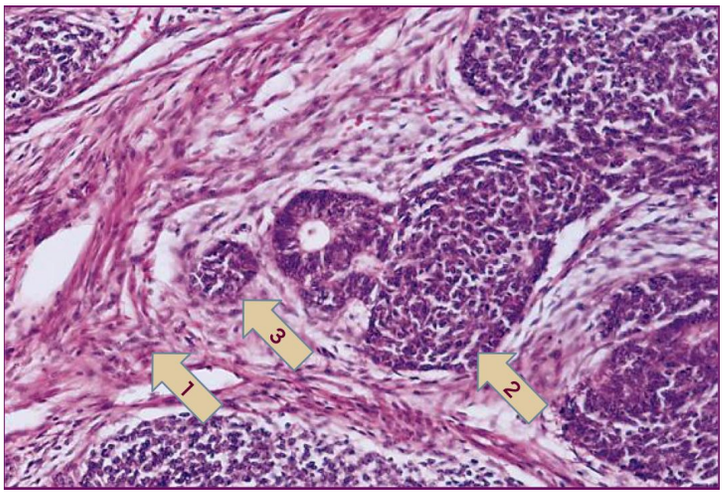


Gross picture shows:
- Partly pale
- Partly hemorrhagic solid tumor replacing almost the entire renal parenchyma
- Areas of necrosis

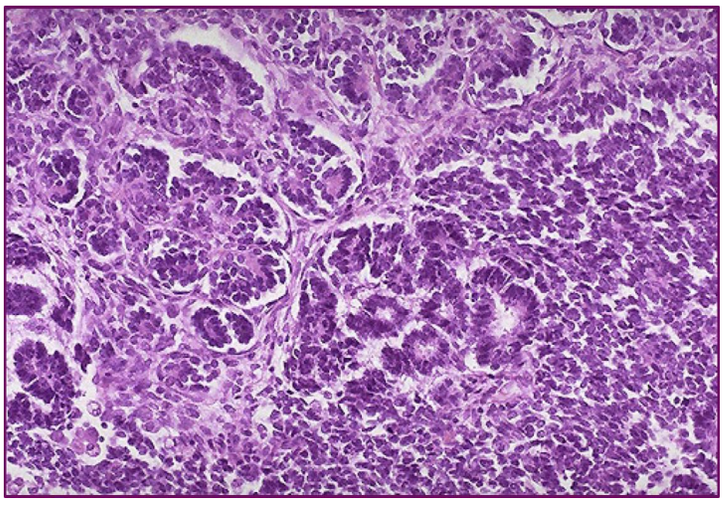
Histopathology:



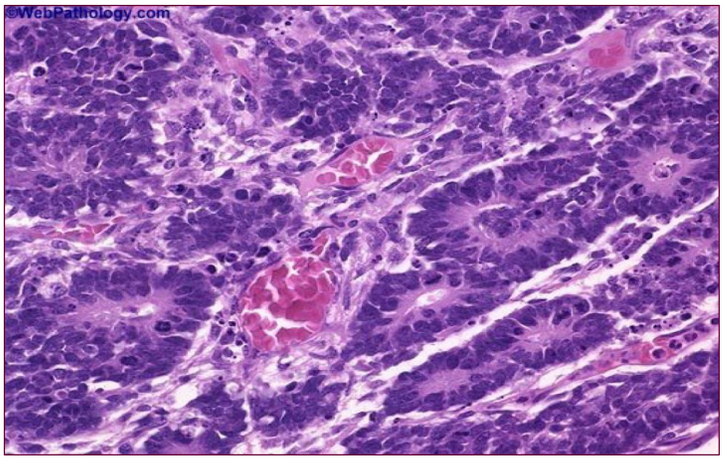
Blastema in WT consists of:
- sheets of densely packed small blue cells
- hyperchromatic nuclei
- little cytoplasm
- conspicuous mitotic activity.



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



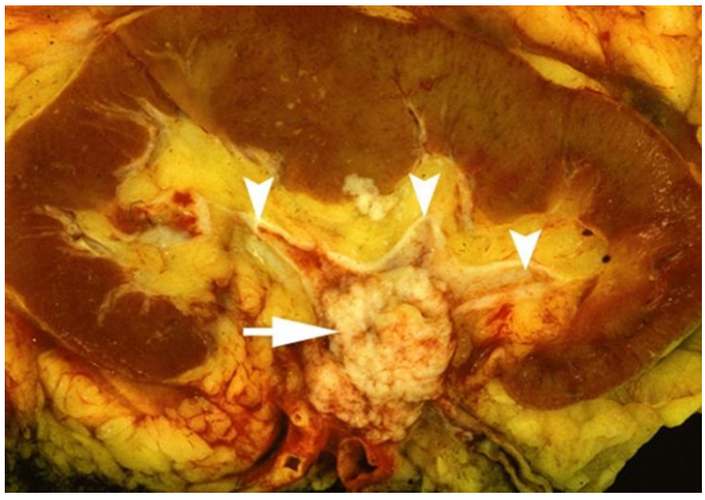
Wilm's tumor resembles the fetal nephrogenic zone of the kidney.
Three major components:
- Undifferentiated blastema cells
- epithelial tissue which shows attempts to form primitive glomerular & tubular structures
- mesenchymal (stromal) tissue



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

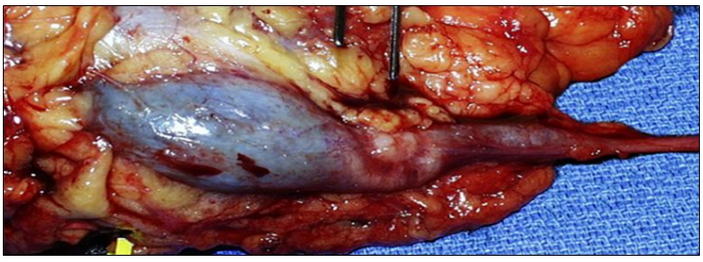
Case 11: Carcinoma of Renal Pelvis and Ureter

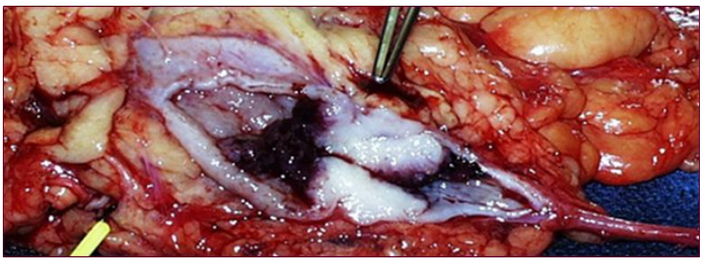
Gross: Urothelial (Transitional) Carcinoma of Renal Pelvis



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

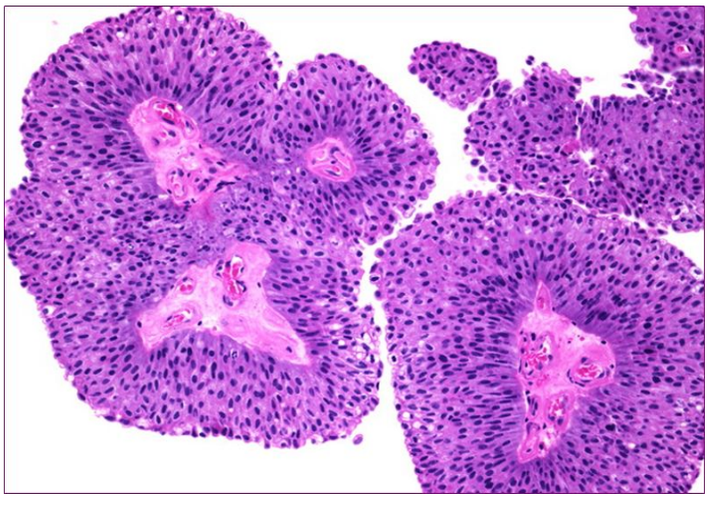
Gross: Urothelial Carcinoma involving Ureter





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

Histopathology:



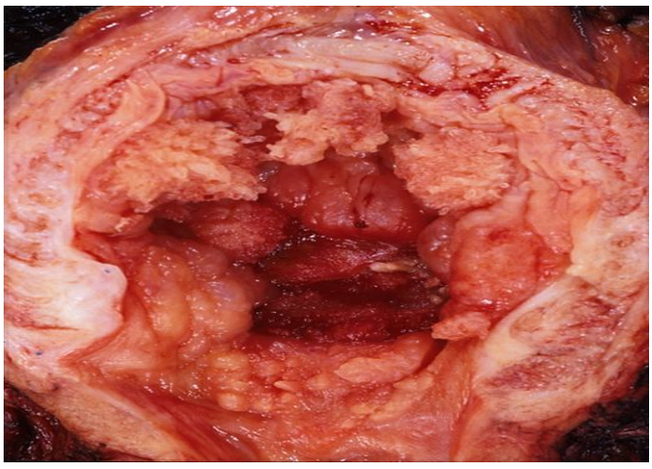
Papillary Urothelial carcinoma of the renal pelvis – Low Grade

Low-grade papillary urothelial carcinoma shows:
- minimal cytologic
- architectural atypia.

Adjacent papillary fronds may fuse, as seen in this image.

Case 12: CARCINOMA OF THE URINARY BLADDER

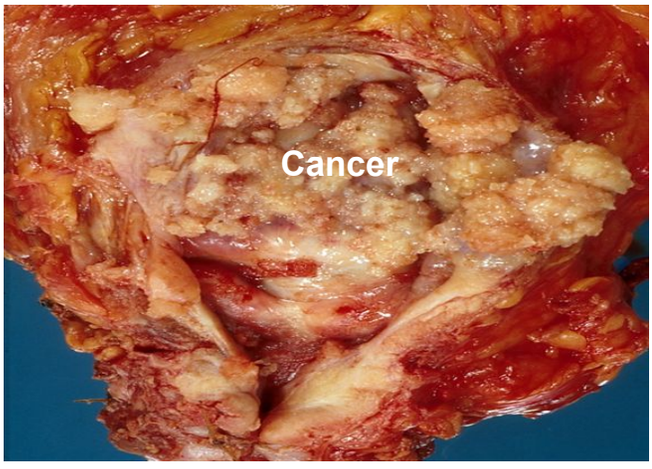
Gross: Urinary Bladder Carcinoma - Urothelial (Transitional cell) papillary Carcinoma



90% of bladder cancers are **transitional cell carcinoma.**

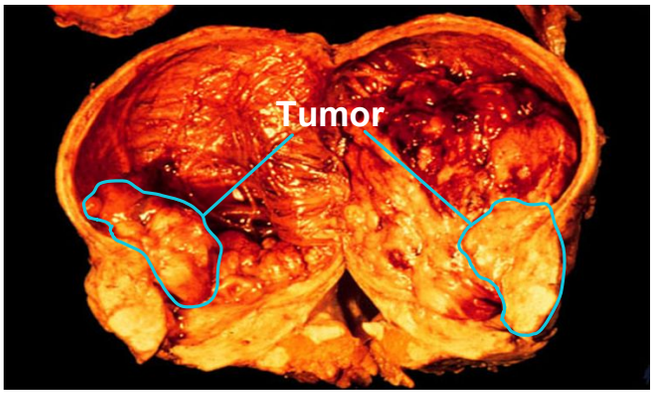
The other 10% are:
- squamous cell carcinoma
- adenocarcinoma
- sarcoma
- small cell carcinoma
- secondary metastases.

Gross: Papillary Urothelial Carcinoma of Bladder.



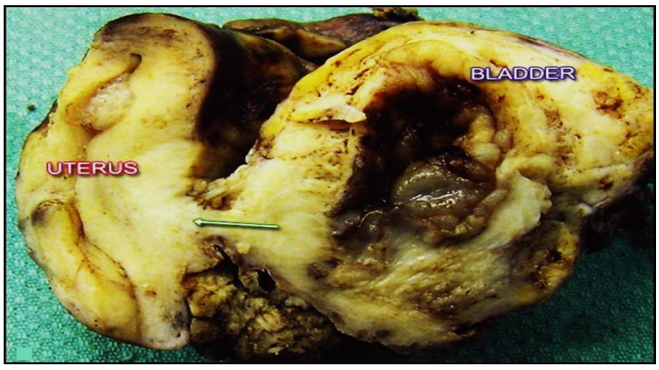
Radical cystectomy specimen showing multifocal papillary urothelial carcinoma.

Gross: Transitional Carcinoma of Bladder.



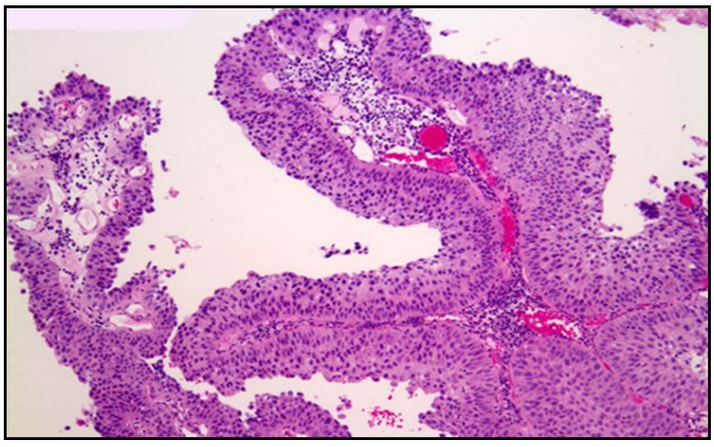
- The mucosa of the open urinary bladder appears edematous.
- There are several whitish or red nodules and patches indicative of a multi-focal nature of this tumor.

Gross: Bladder Tumor invading the Uterus.



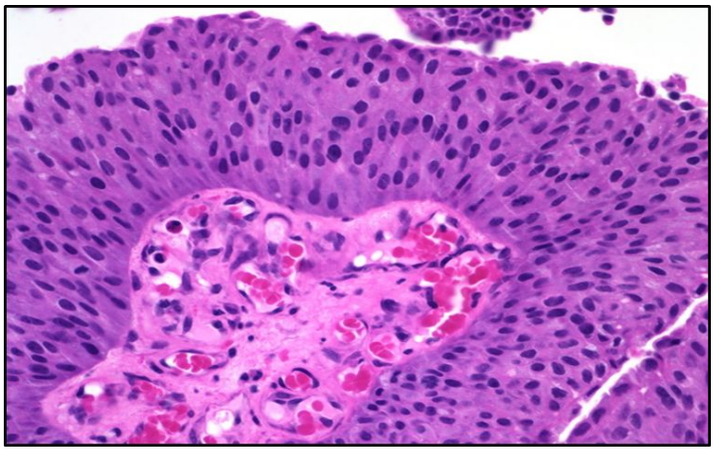
Urinary bladder carcinoma infiltrating the urinary bladder wall with extension to the uterus .

Papillary Urothelial carcinoma – Low Grade



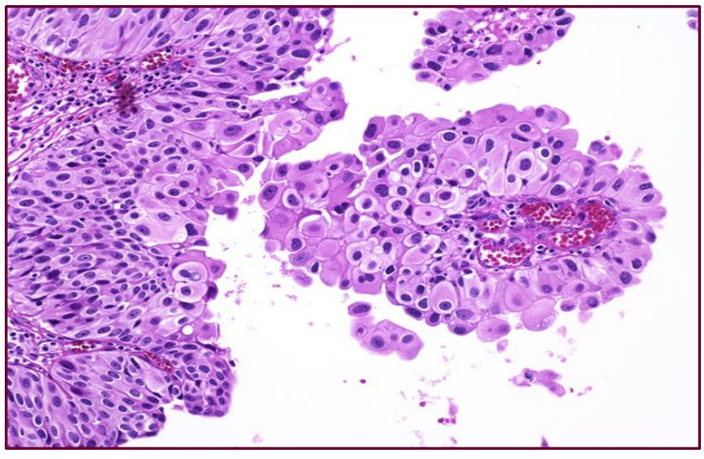
The low grade tumors show:
- overall preservation of cell polarity
- few mitosis (mitoses)
- lack of significant morphologic atypia.

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



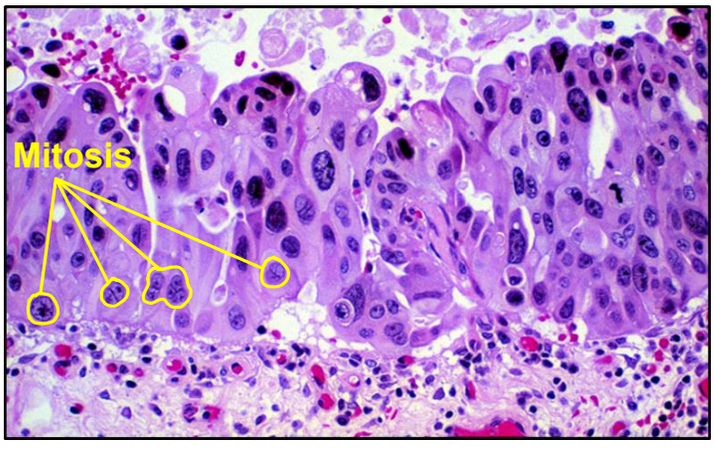
High power view of a low-grade papillary urothelial carcinoma shows:
- scattered hyperchromatic nuclei
- typical mitotic figures.

Papillary Urothelial carcinoma – High Grade



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

Urothelial (Transitional) carcinoma – HPF



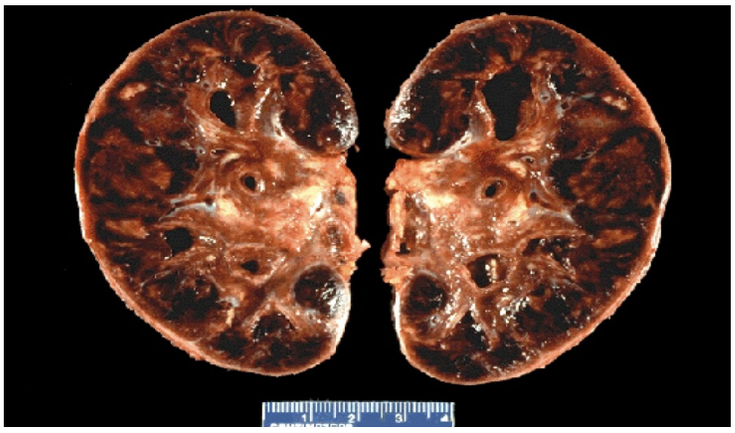
- Almost all cases of Bladder carcinomas are originating from the transitional epithelium.
- Bladder carcinoma might be squamous cell in nature
- Chronic inflammation of the bladder mucosa, caused by stones or schistosomiasis may lead to it .
- Rarely, it presents as adenocarcinoma.

Predisposing conditions and exposures that can lead to Urothelial (Transitional) carcinoma: (male slides only)
• Exposure to aniline and Azo dyes. • Cigarette smoking. • Cyclophosphamide.

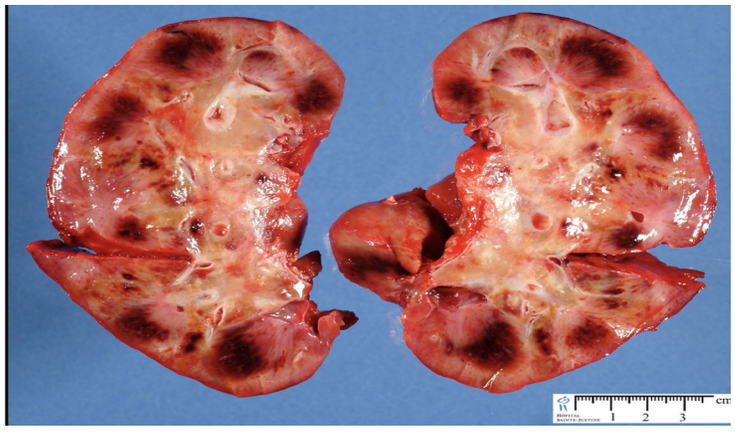
Case 13: PATHOLOGY OF RENAL ALLOGRAFT

Acute Cellular Allograft Rejection:

Gross:



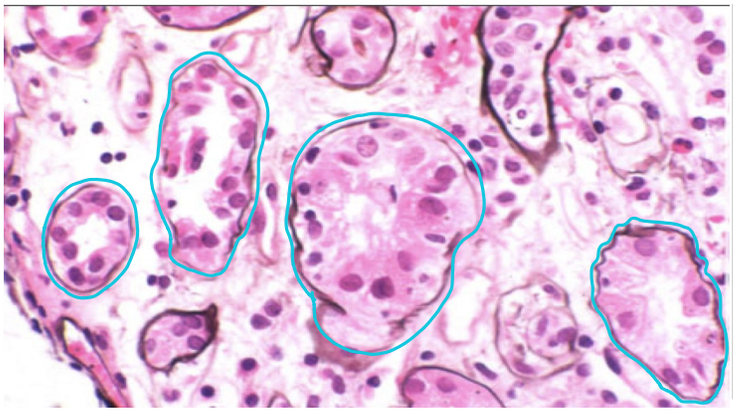
- This kidney was removed because of acute transplant rejection.
- **swollen** and **hemorrhagic** appearance of this entire kidney.



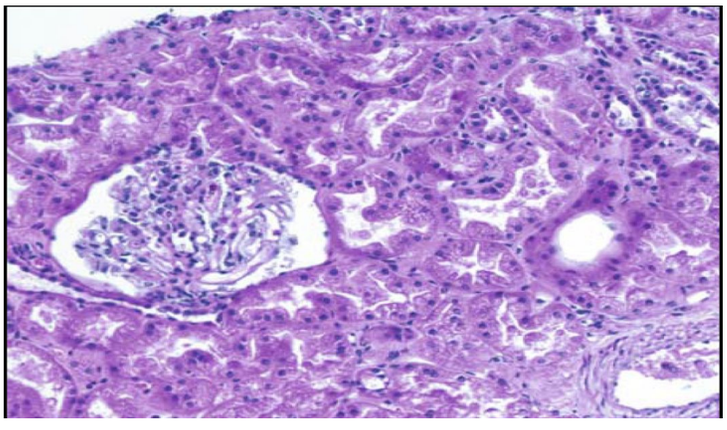
- **Swollen** and **hemorrhagic** appearance of acutely rejected renal allograft

Acute Cellular Allograft Rejection - type 1:

Histopathology

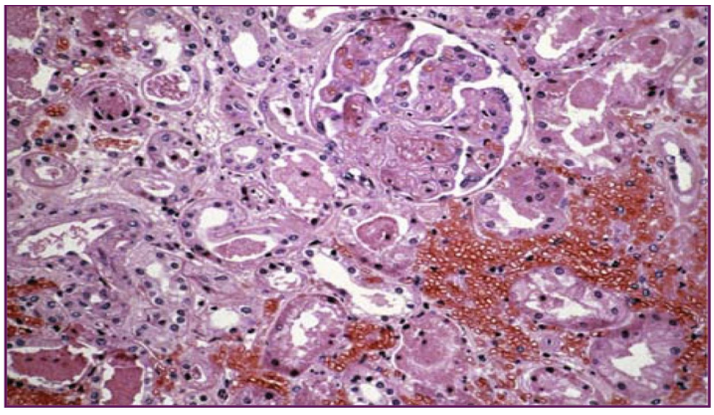


the hallmark of type I interstitial acute rejection are:
- Tubulitis
- infiltration of tubular epithelium by lymphocytes.



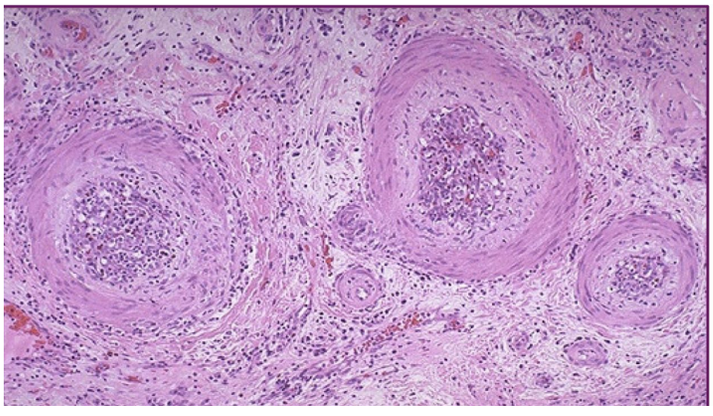
- Humoral (Antibody-mediated) rejection
- type I. Acute tubular injury is evident, **without neutrophils** in capillaries.
- Peritubular and glomerular capillary inflammation **with neutrophils.**
- necrosis of arteries

Hyperacute Allograft Rejection:



cortex shows:
- diffuse hemorrhage and neutrophils in peritubular capillaries
- prominent glomerular thrombi 1 day after transplantation

Chronic Allograft Rejection:



Chronic vascular rejection of a renal transplant which has a poor prognosis

There is:
- thickened arteries with intimal fibrosis
- chronic inflammation.

MED437
KING SAUD UNIVERSITY

PATHOLOGY
TEAM 437

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