The Urinary System Pathology

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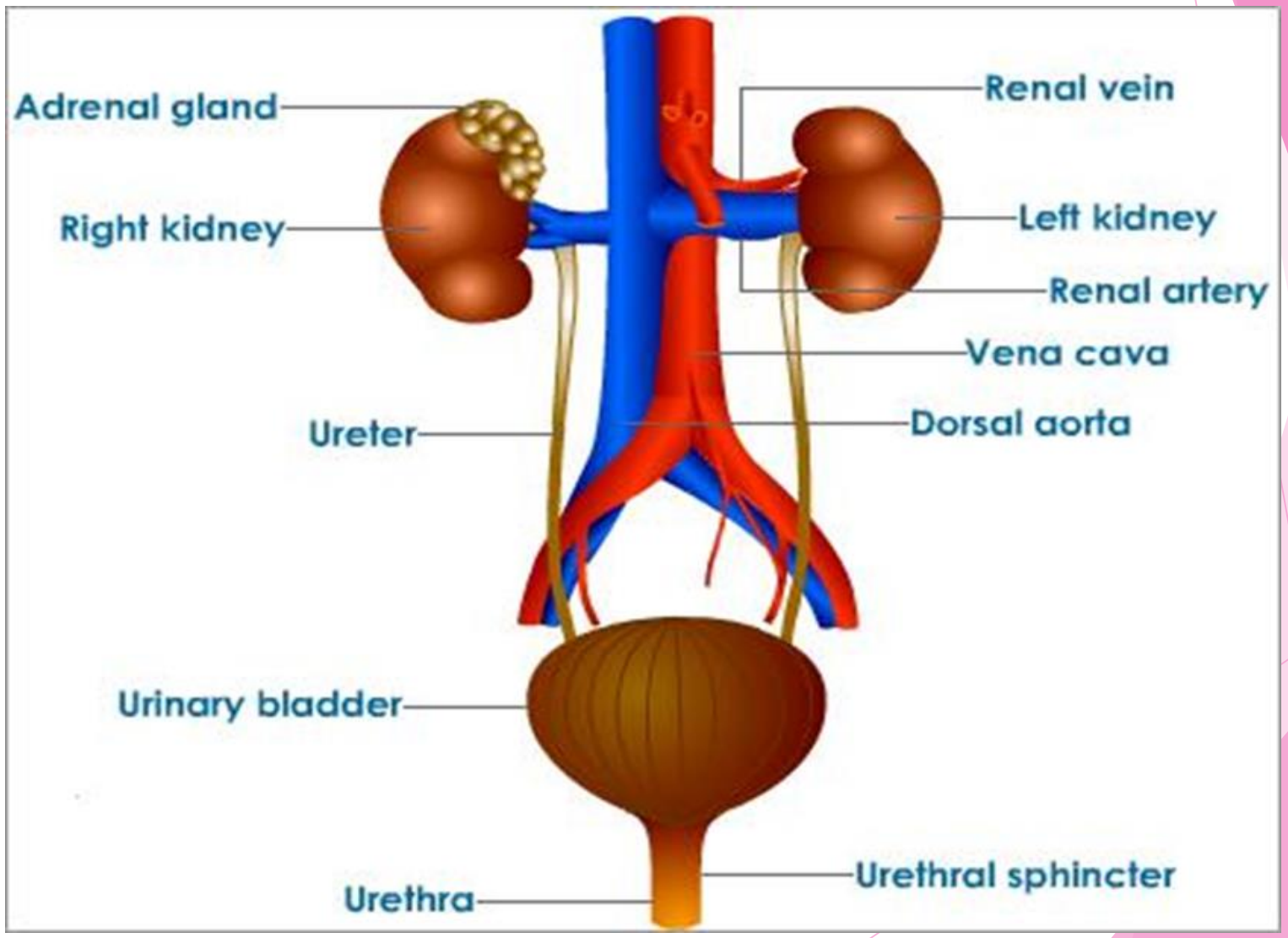
objectives

- To Know normal anatomy physiology and histology of renal system
- To discuss Kidney pathology: congenital, cystic disease , Glmerular disease
- To know tubluointerstitial disease, UTI
- To discuss urinary bladder pathology

Structures of the Urinary System

- Kidneys
- Nephrons
- Renal Pelvis
- Ureters
- Urinary Bladder
- The Urethra



Functions of the Urinary System

The urinary system performs many functions important to maintaining **homeostasis**

- Maintenance of water, salts, minerals and acids balance in the body
- Filters blood to remove **urea** and other waste products of metabolism from the bloodstream
  - **Urea** – the major waste product of protein metabolism
- Converts waste products and excess fluids into **urine** in the kidneys and excretes them from the body via the urinary bladder
- kidneys serve to convert more than 1700 L of blood per day into about 1 L of a highly concentrated fluid (urine).
- The kidney also serves as an endocrine organ, secreting such hormones as erythropoietin, renin, prostaglandins, and regulating vitamin D metabolism.

The Kidneys

Filter blood constantly to remove waste products and excess water which are excreted as urine (95% water and 5% other wastes)

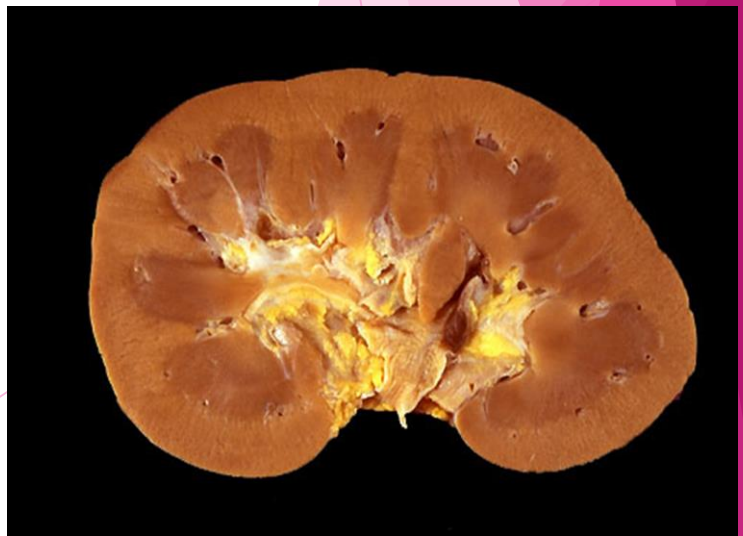
- 2 kidneys located retroperitoneally, one on each side of the vertebral column below the diaphragm consist of:

Renal cortex

- The outer region of the kidney
- Contains over 1 million microscopic units called nephrons

Medulla

- The inner region of the kidney
- Contains the urine collecting tubules



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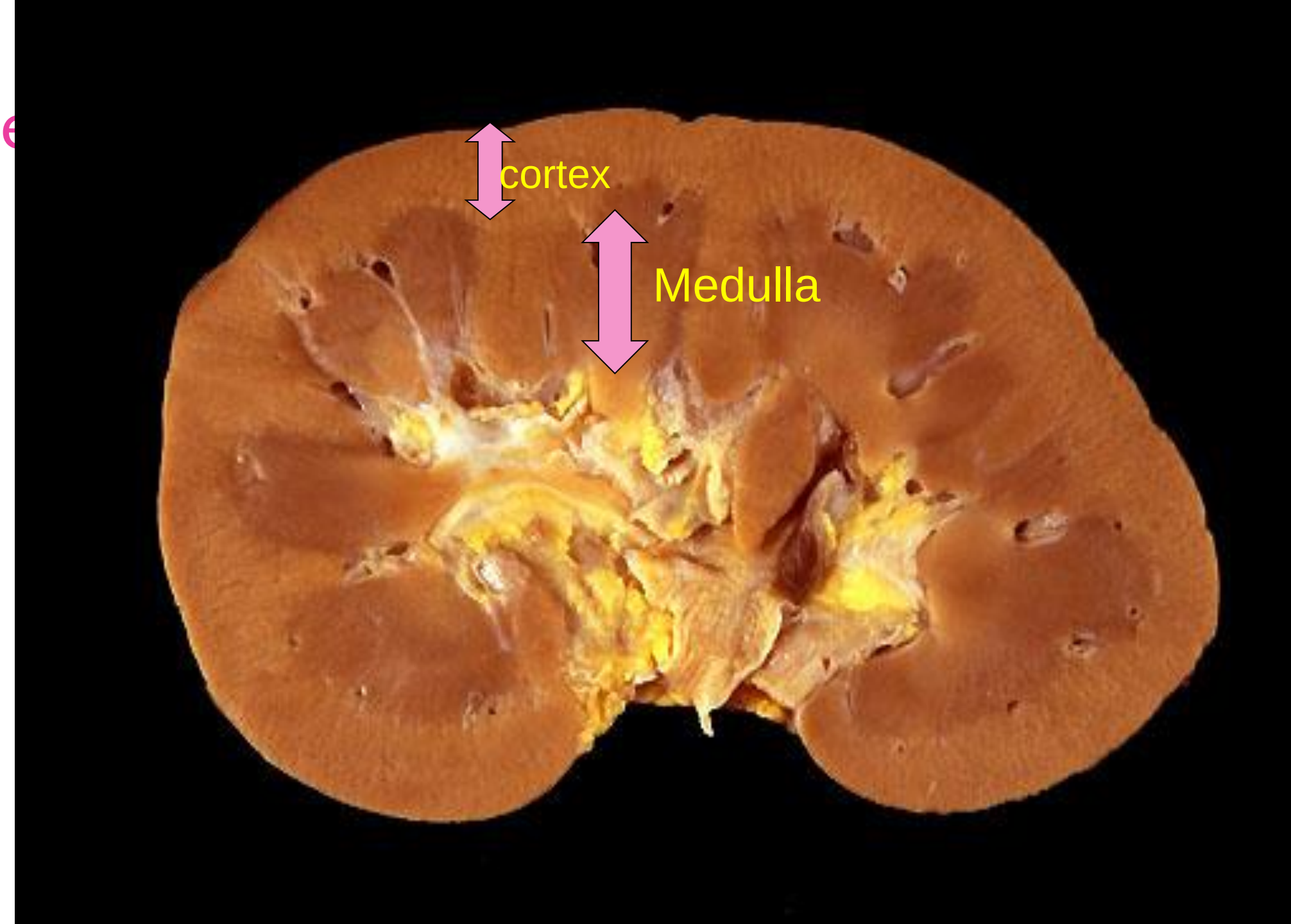
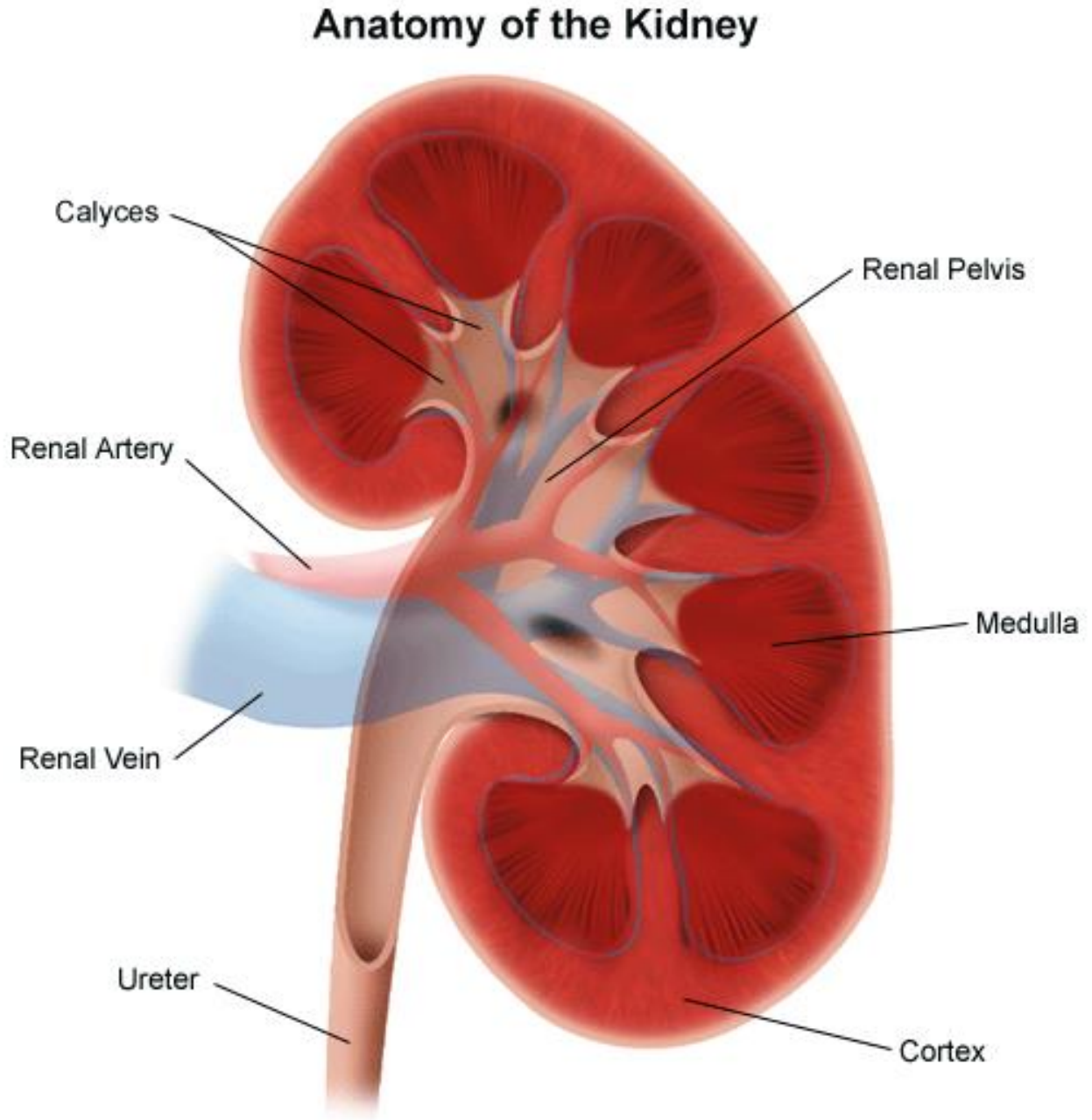
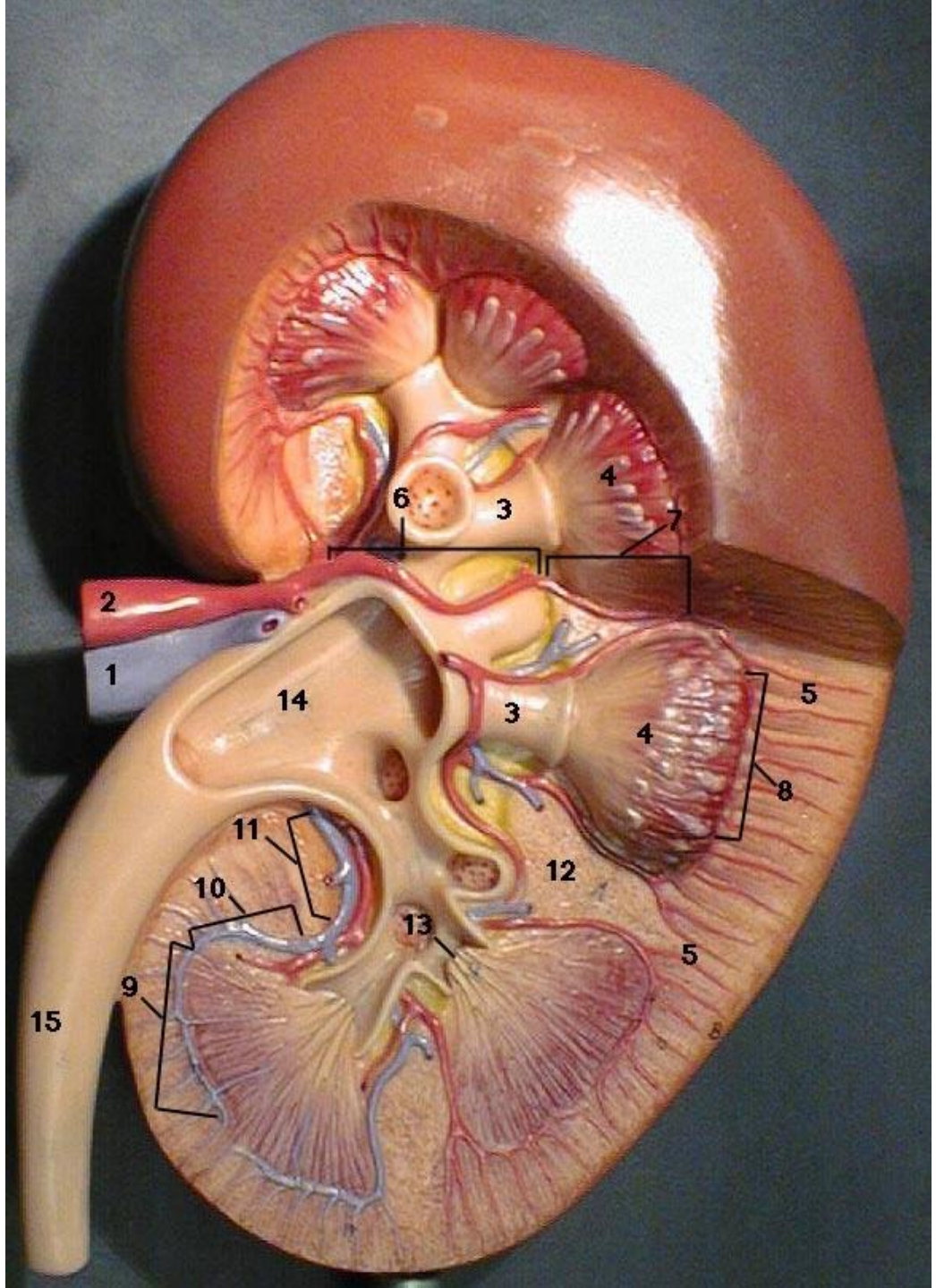


- Each human adult kidney weighs about **150 gm.**
- Anatomically the ureter forms the pelvis (the dilated upper ureter) which is divided into 2 or 3 major calyces, each one giving 3 or 4 minor calyces.
- The kidney is divided into the cortex 1.2-1.5 cm and medulla.
- The medulla consists of renal pyramids, the apices of which are called papillae, each related to a calyx.


Anatomy of the Kidney
Calyces
Renal Pelvis
Renal Artery
Medulla
Renal Vein
Ureter
Cortex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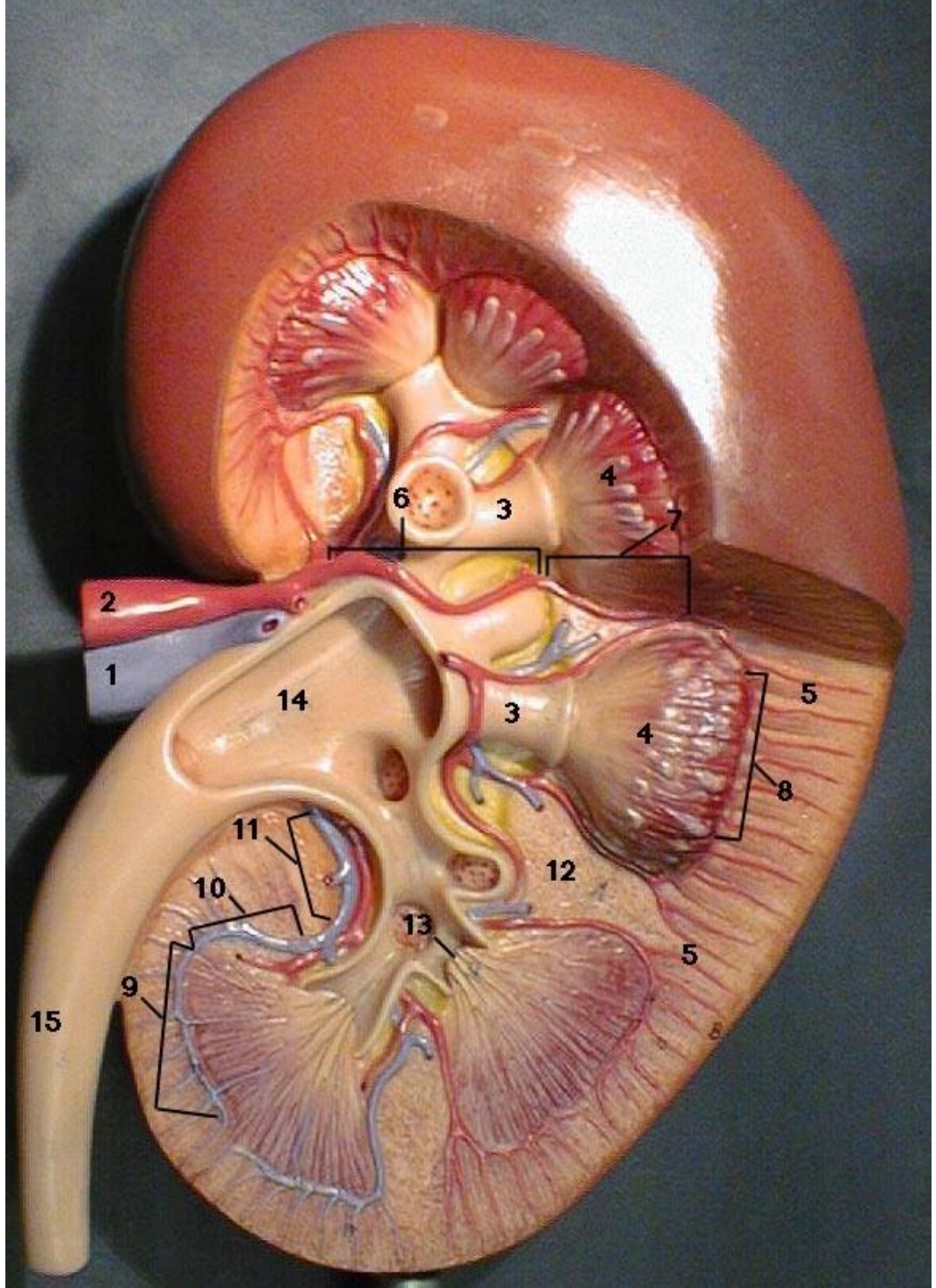
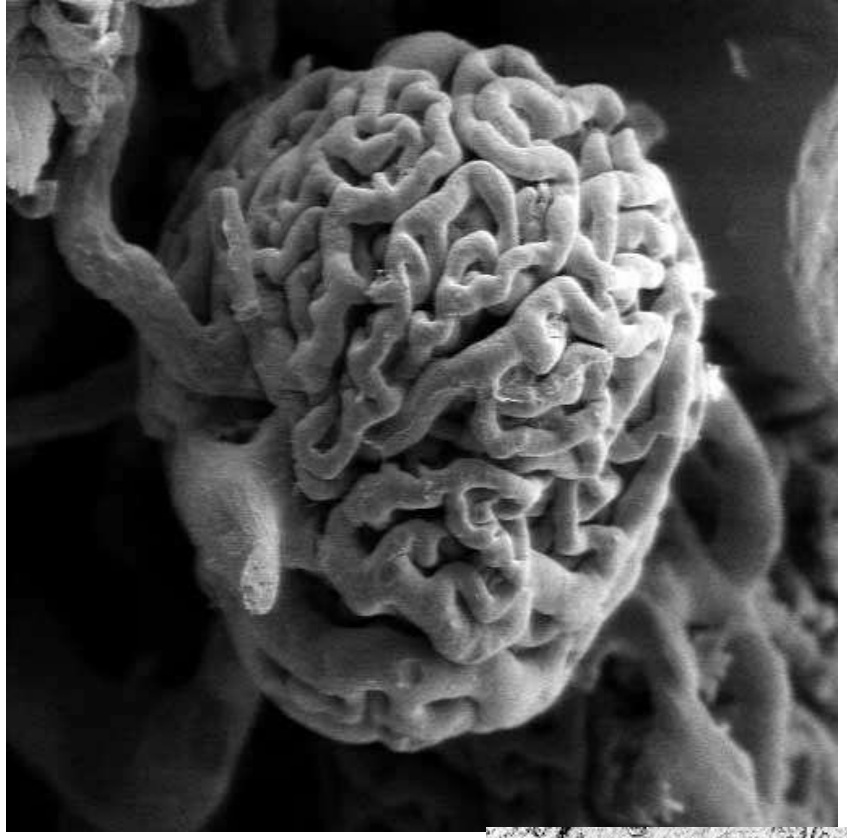
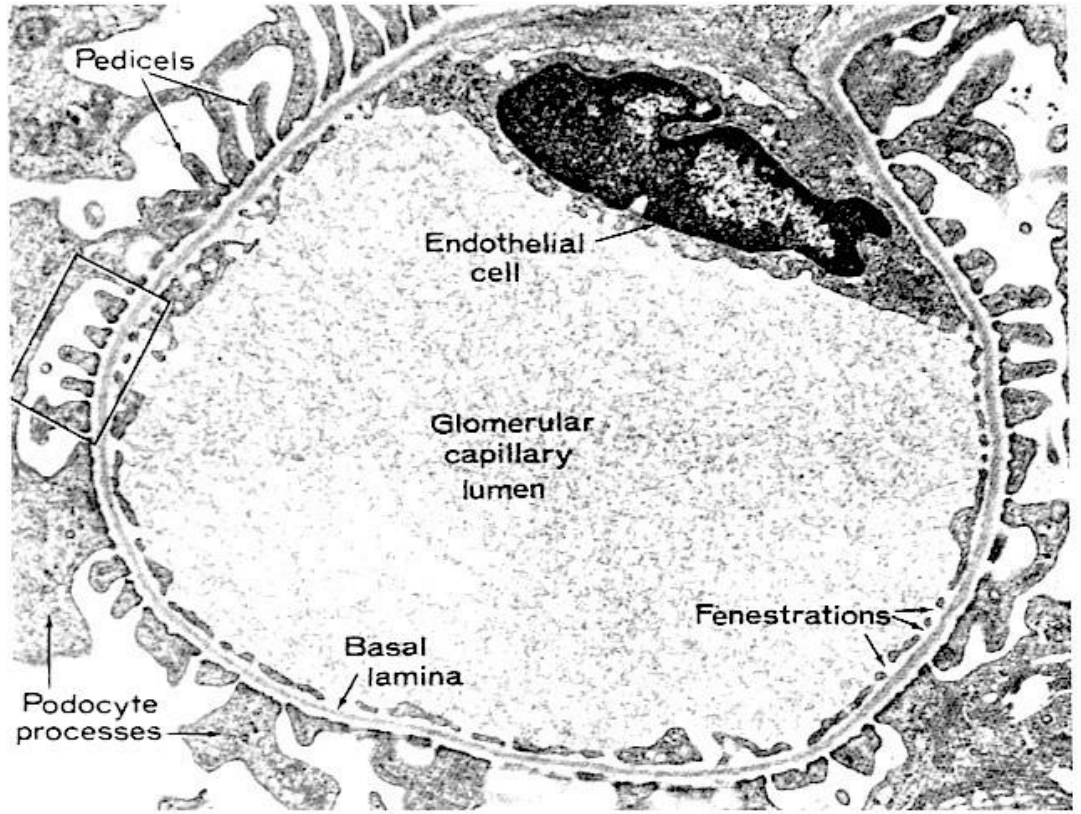


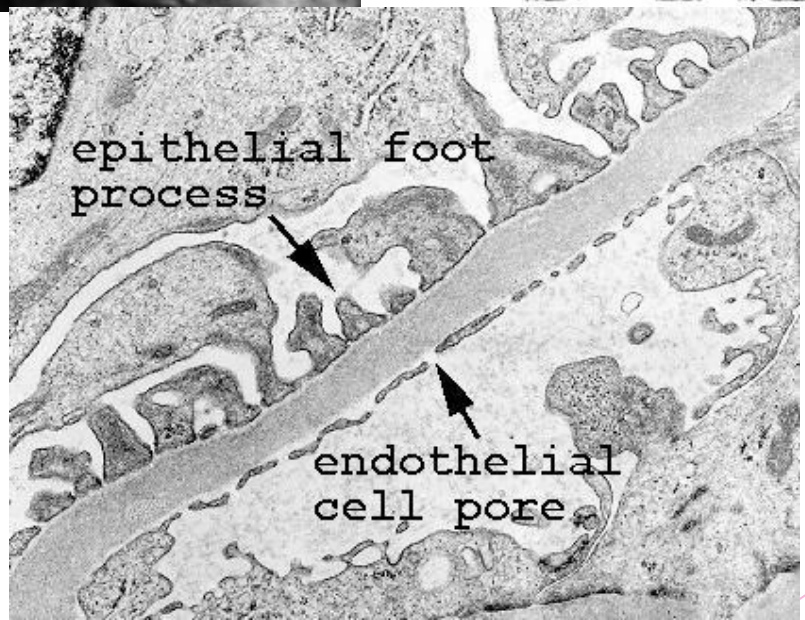
1. Renal Vein
2. Renal Artery
3. Renal Calyx
4. Medullary Pyramid
5. Renal Cortex
6. Segmental Artery
7. InterlobAR Artery
8. Arcuate Artery→ interlobULAR
9. Arcuate Vein
10. Interlobar Vein
11. Segmental Vein
12. Renal Column
13. Renal Papillae
14. Renal Pelvis
15. Ureter

Nephrons

- The functional units of the kidneys
- Form urine by the process of filtration, reabsorption, and secretion
  - Reabsorption: is the return of substances that were removed from filtration back to the bloodstream
- Each nephron contains a **glomerulus**
  - Cluster of capillaries surrounded by a cup-shaped membrane called the Bowman's capsule

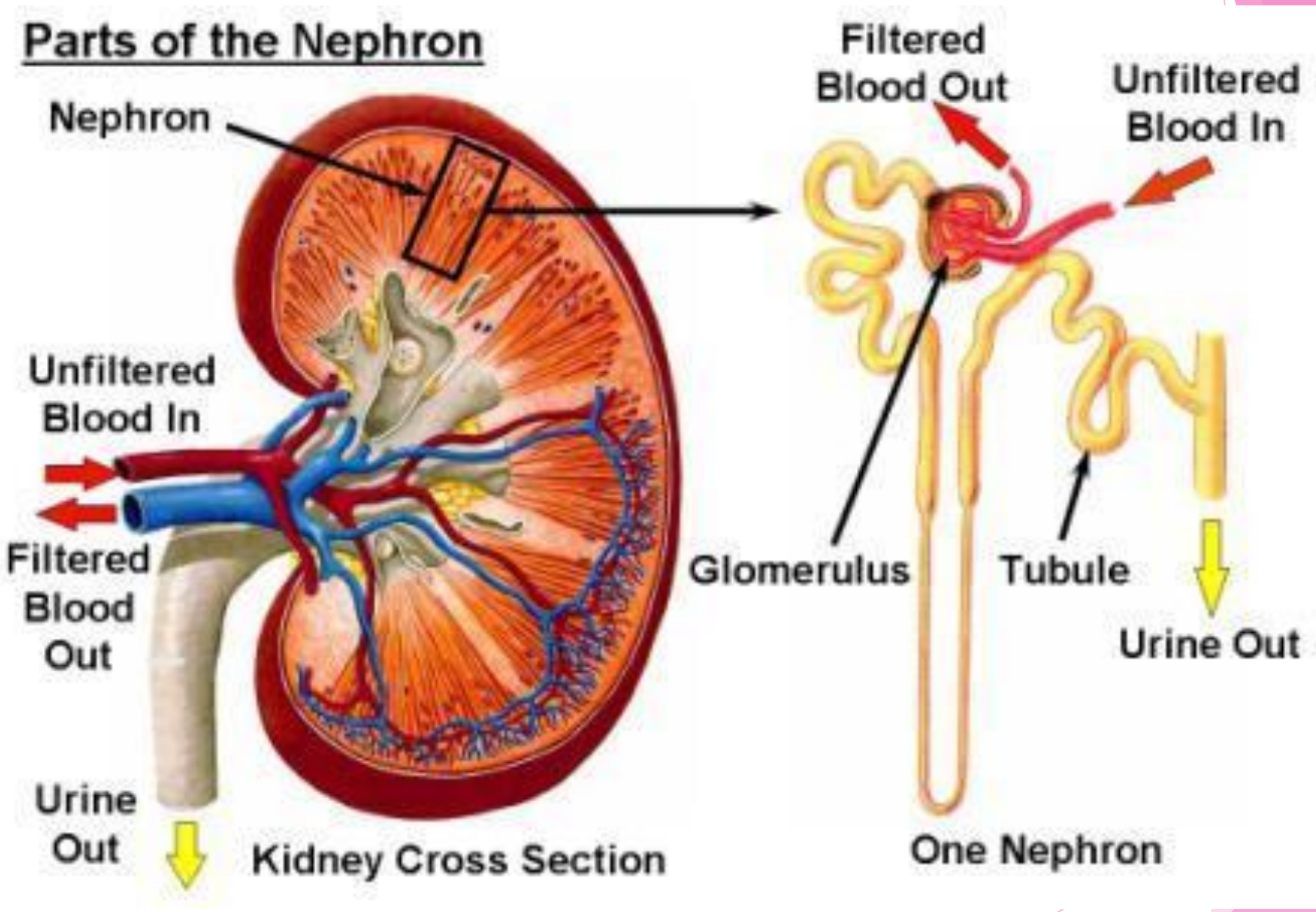



Pedicels
Endothelial cell
Glomerular capillary lumen
Fenestrations
Basal lamina
Podocyte processes


epithelial foot process
endothelial cell pore

S.E.M.

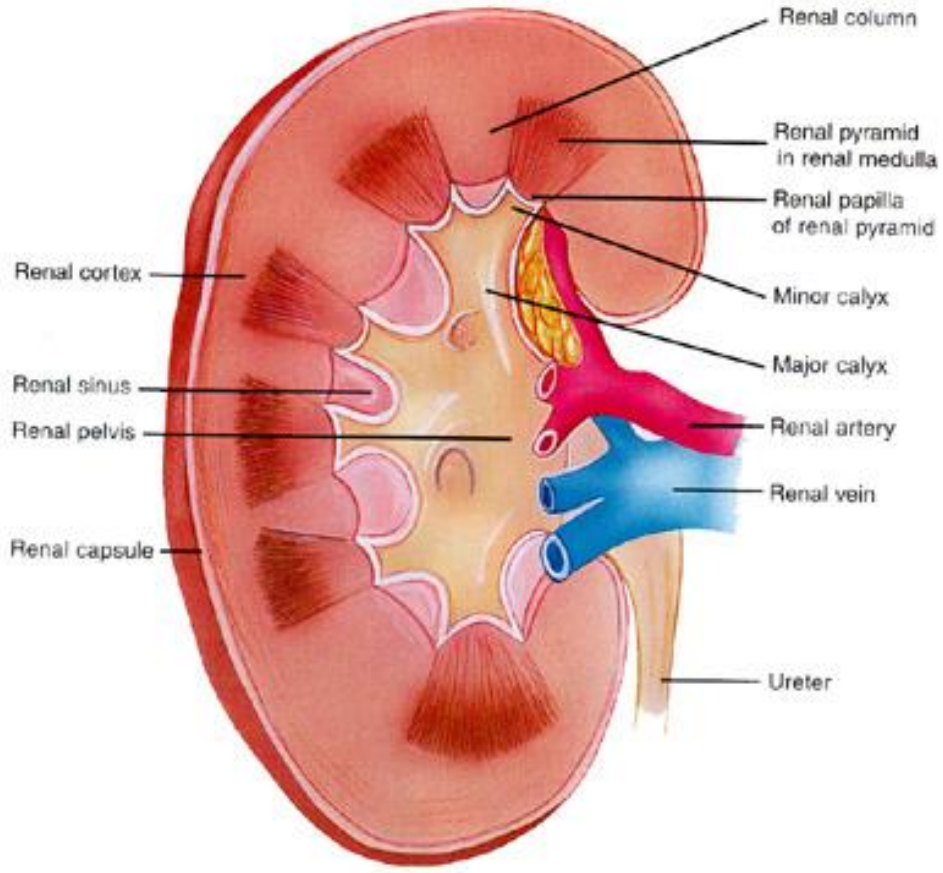
T.E.M.


Parts of the Nephron
Nephron
Unfiltered Blood In
Filtered Blood Out
Urine Out
Kidney Cross Section
Filtered Blood Out
Unfiltered Blood In
Glomerulus
Tubule
Urine Out
One Nephron

How is Urine Made?

- Blood enters the kidneys through the renal artery and flows into the nephrons
- After being filtered by the capillaries of the glomerulus, the blood leaves the kidney through the renal vein
- Waste products that were filtered out of the blood remain behind in the kidney where they pass through urine-collecting tubules
- Urine is then transported to the renal pelvis and collected in preparation for entry into the ureters

The Renal Pelvis

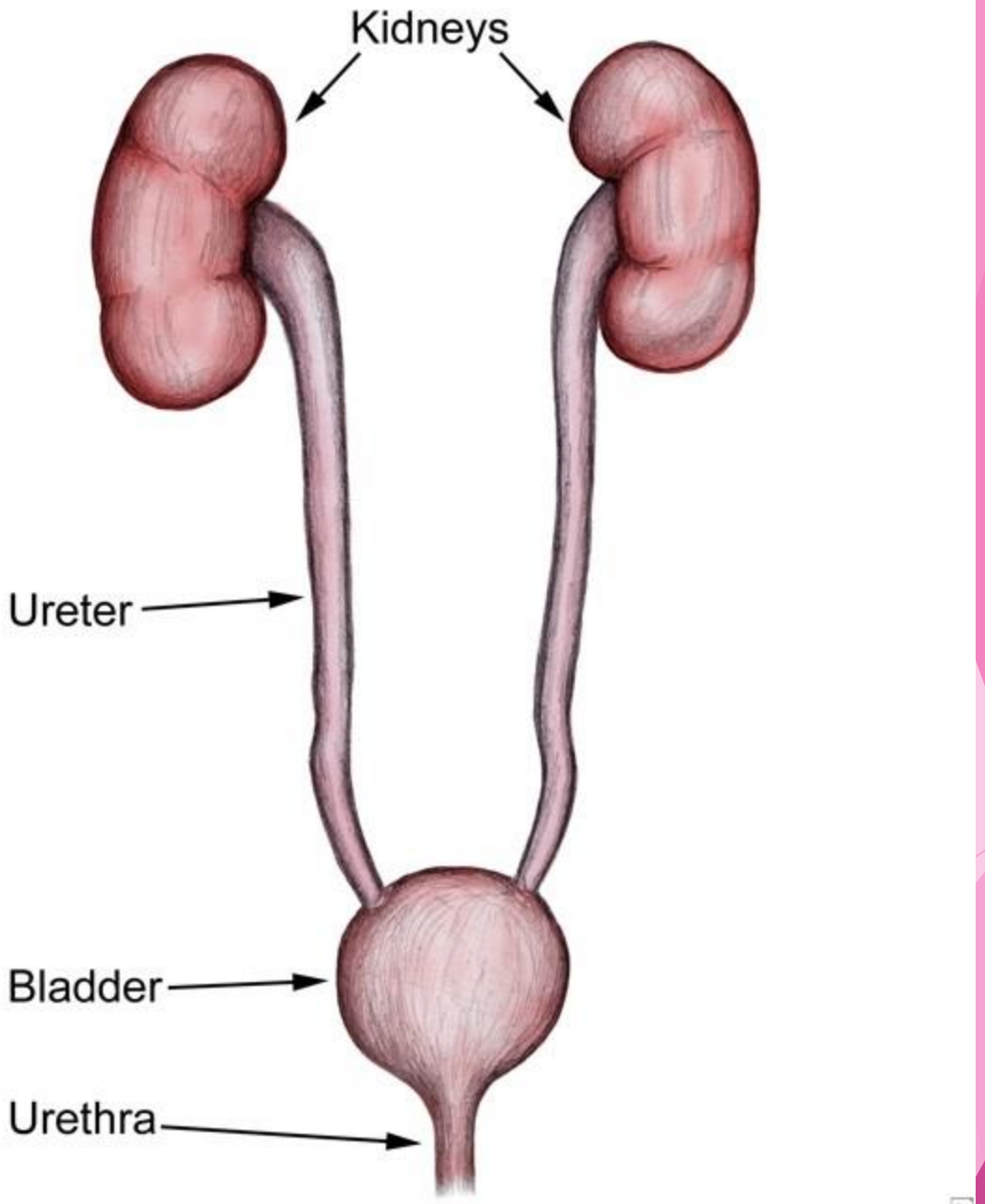


- Funnel-shaped area in each kidney that is surrounded by the renal cortex and medulla
- Newly formed urine collects here before flowing to the ureters

The Ureters

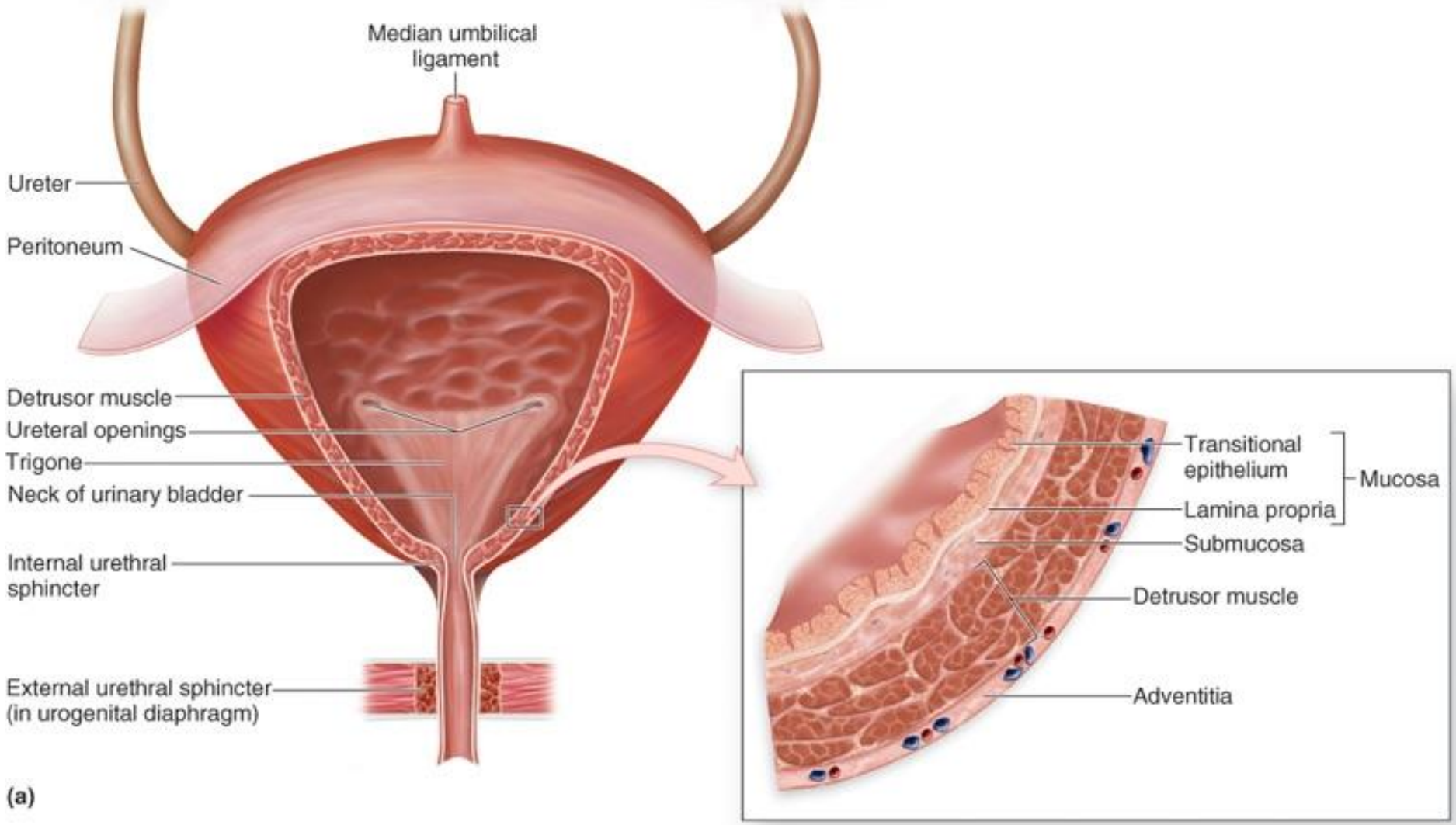
- 2 narrow tubes (10-12 inches each) that transport urine from each kidney to the bladder

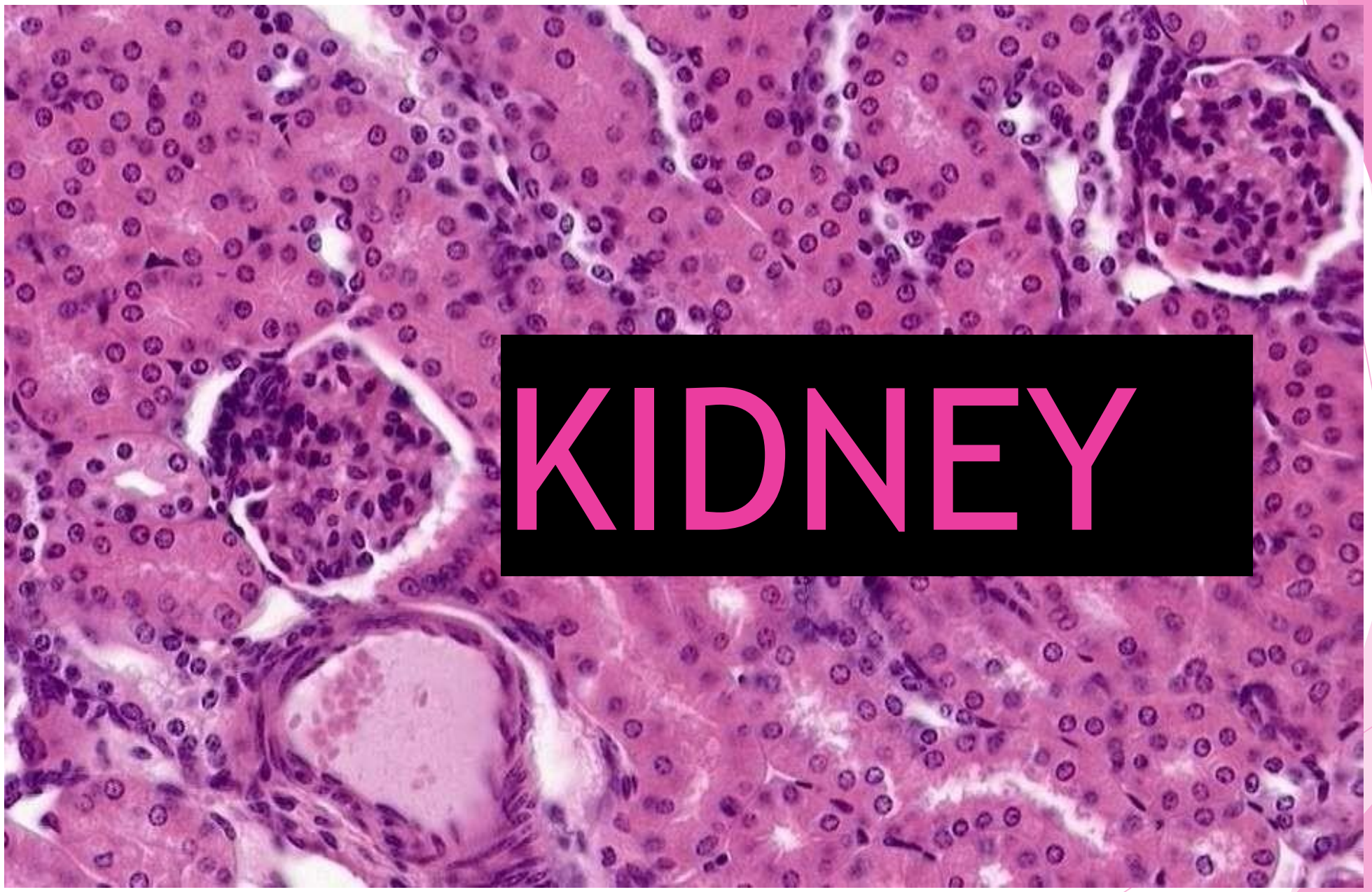
- Peristalsis moves urine down each ureter into the bladder



The Urinary Bladder

- Hollow muscular organ that is a reservoir or holding tank for urine before it is excreted from the body
- Located in the anterior portion of the pelvic cavity behind the pubic symphysis
- Lined with *rugae* that allow it to expand and contract
- Trigone- smooth triangular area on the inner surface of the bladder located between the openings of the ureters and the urethra




KIDNEY

Kidney PATHOLOGY

- **CONGENITAL MALFORMATION**
- **“CYSTS”**
- **GLOMERULAR DISEASES**
- **TUBULAR/INTERSTITIAL**
- **BLOOD VESSELS**
- **OBSTRUCTION**
- **TUMORS**

CONGENITAL

- **AGENESIS**
- **HYPOPLASIA**
- **ECTOPIC**
- **HORSESHOE**

Congenital anomalies

Agenesis of the kidney(absence):

- Bilateral agenesis is incompatible with life, seen in stillborn.
- Unilateral type is uncommon.
- The opposite kidney is usually enlarged as a result of compensatory hypertrophy.

Hypoplasia(small size kidney) :

- Is failure of the kidney to develop to a normal size. This anomaly may occur bilaterally, resulting in renal failure and early childhood death.
- Unilateral cases are more common. The opposite kidney is also enlarged due to compensatory hypertrophy.
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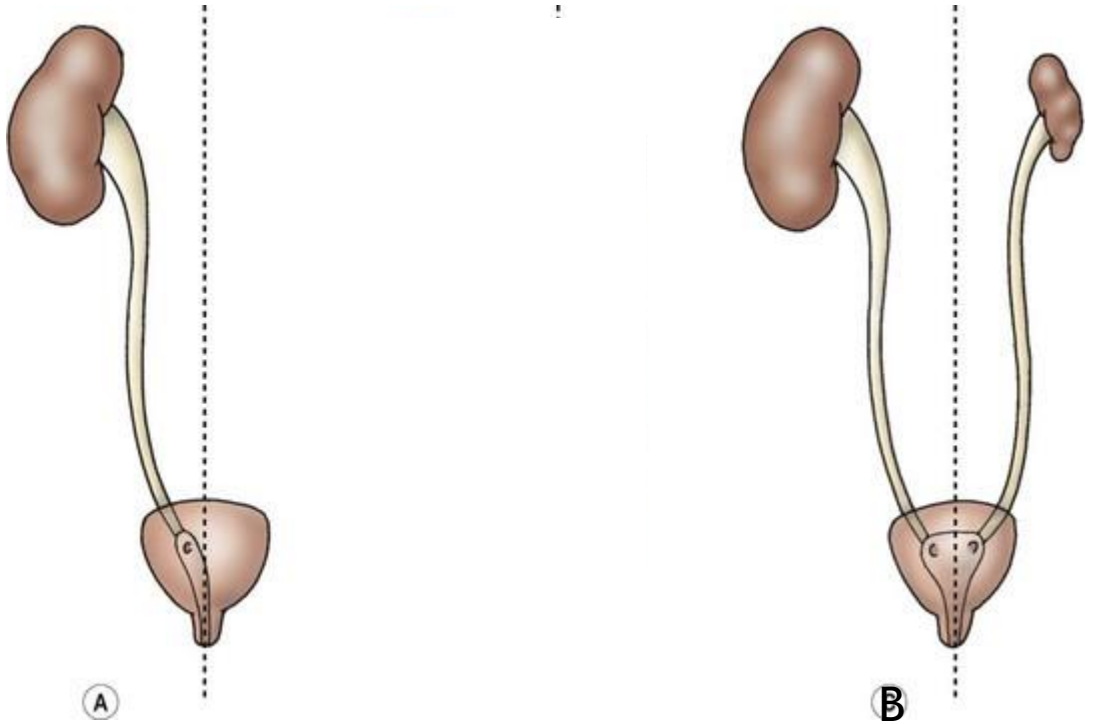
Renal (A) agenesis, and (B) hypoplasia.



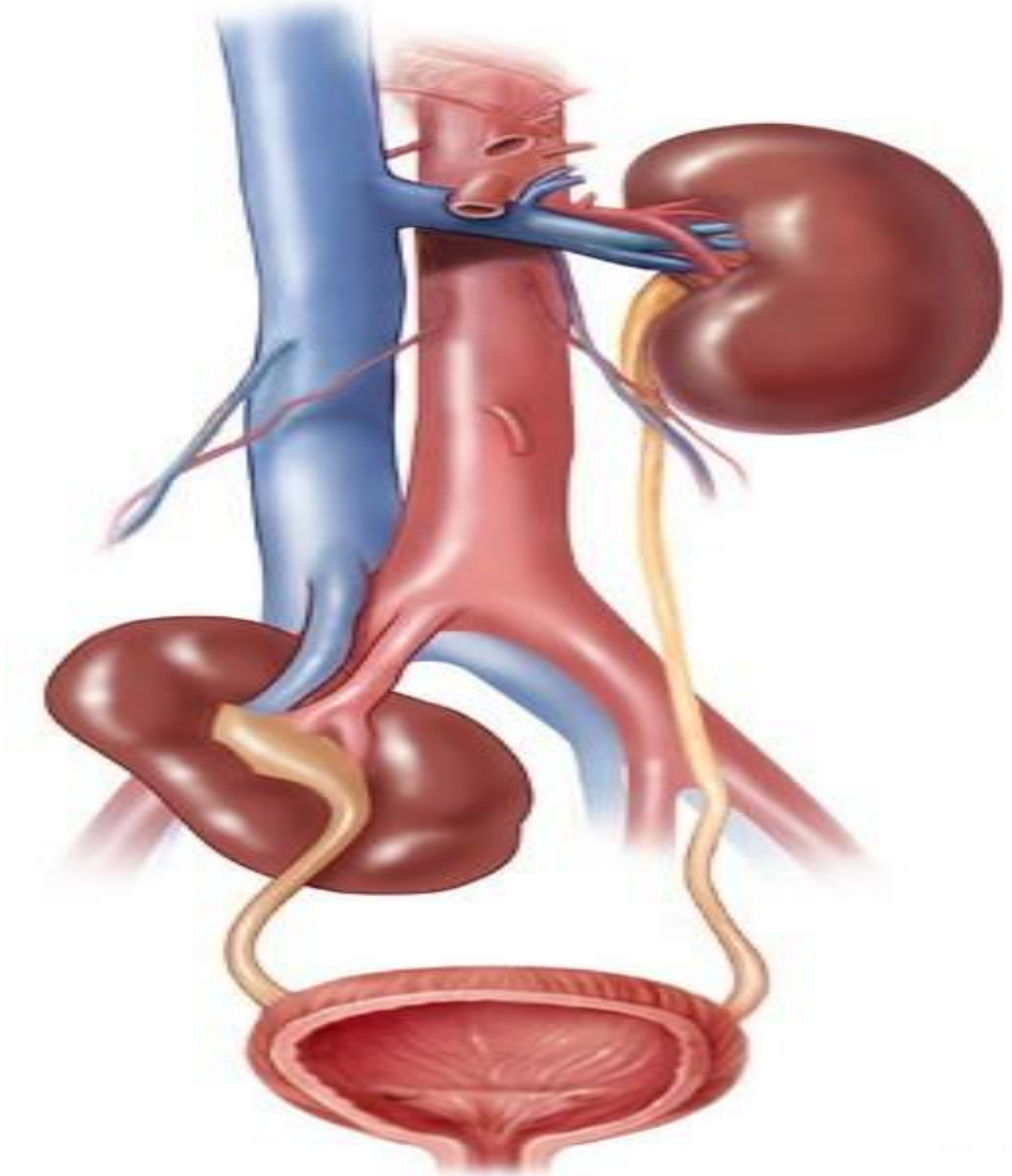
HYPOPLASIA

Congenital anomalies-cont

Ectopic Kidneys:

- These kidneys lie within the pelvis.
- They are usually normal or slightly small in size.
- Because of their abnormal location can cause :
- kinking or tortuoisity of the ureters may cause some obstruction to urinary outflow.
- difficulty in labor in females.
- Misdiagnosis as pelvic tumors & abscesses.
- Have long renal artery that may have many complications like damage during surgery.

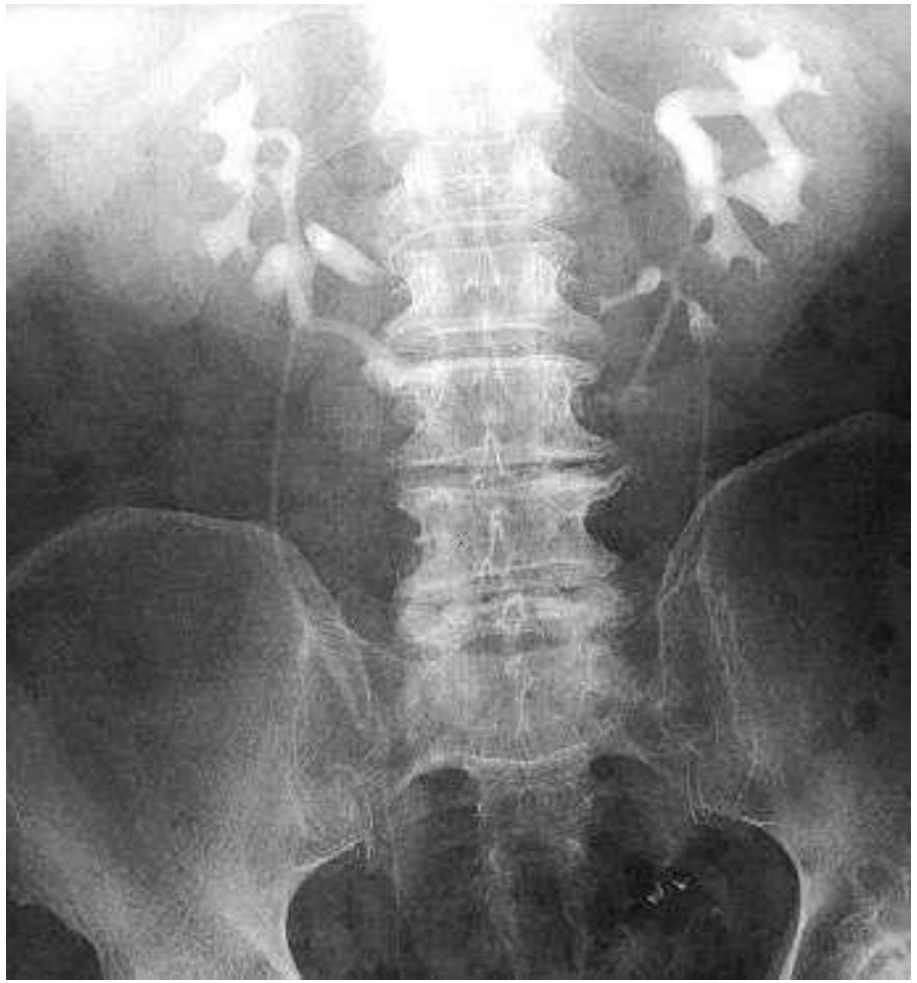
ECTOPIC (usually PELVIC)

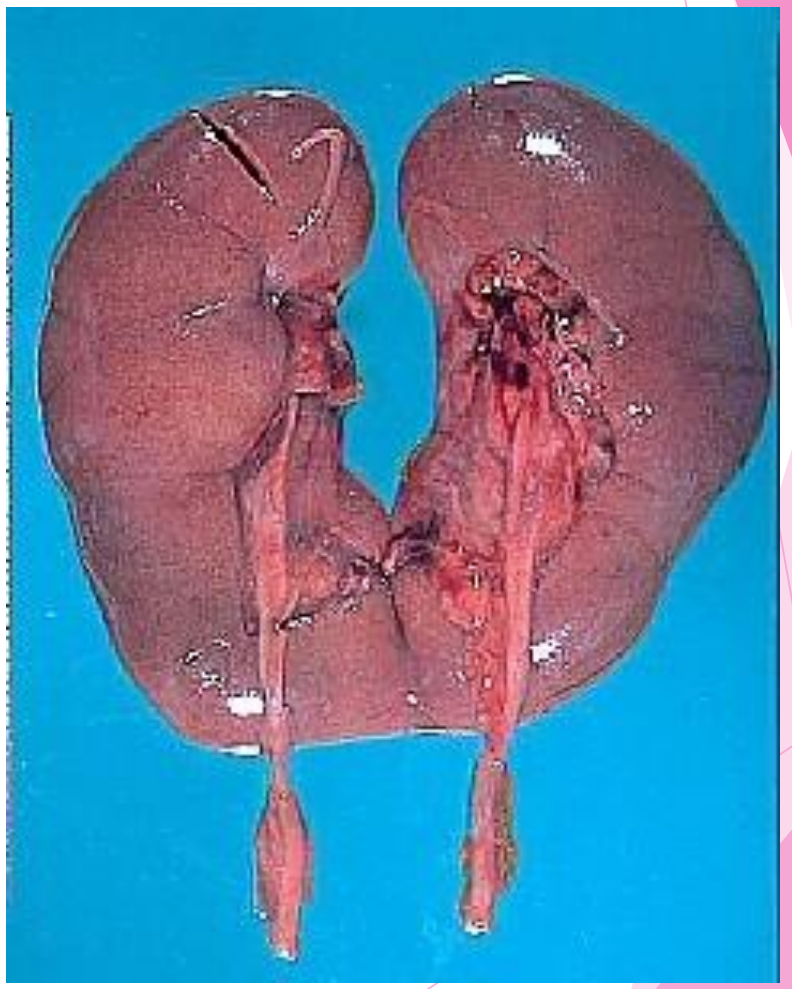


Horseshoe kidneys:

- Fusion of lower poles of the kidneys that is continuous across the midline anterior to the great vessels.
- may cause many complications:
- 1. Partially obstructed the ureters, which result in hydronephrosis.
- 2. Recurrent UTI.
- 3. Stone formation.

HORSESHOE





Cystic Diseases Of The Kidney

- They are heterogeneous group comprising **hereditary, developmental and acquired disorders**.
- They are important for several reasons;
  - 1. They are reasonably **common** and often represent diagnostic problems for clinicians, radiologists and pathologists.
  - 2. Some forms are major **causes of chronic renal failure.**
  - 3. They can occasionally be **confused with malignant** tumors.

Classification of CYSTIC DISEASES

- **Polycystic kidney :**

  Autosomal DOMINANT (AD-ULTS)

- **Autosomal RECESSIVE (CHILDREN)**
- **ACQUIRED**
- **SIMPLE**
- **(dialysis-associated) cystic disease.**
- **Parasitic cysts (e.g. hydatid cyst).**

Autosomal Dominant Polycystic Kidney Disease

- Multiple expanding cysts of both kidneys that ultimately destroy the intervening parenchyma.
- It affects roughly 1 of every 400 to 1000 live births and accounting for about 5-10% 0f cases of chronic renal failure.
- It can be caused by inheritance of at least two Autosomal dominant genes of high penetrance:
- 1. PKD 1, present on chromosome 16, mutant in 90% of cases.
- 2. PKD 2, present on chromosome 4, mutant

Gross:

- Large kidney (4 kg) of each kidney, & present as abdominal mass.
- Mass of cysts of varying sizes up to 3 to 4 cm in diameter without intervening parenchyma.
- Cysts are filled with fluid (clear, hemorrhagic).

Mic:

- Cysts arise from tubules or collecting ducts.
- Often have atrophic lining.
- Superimposed infection is common.
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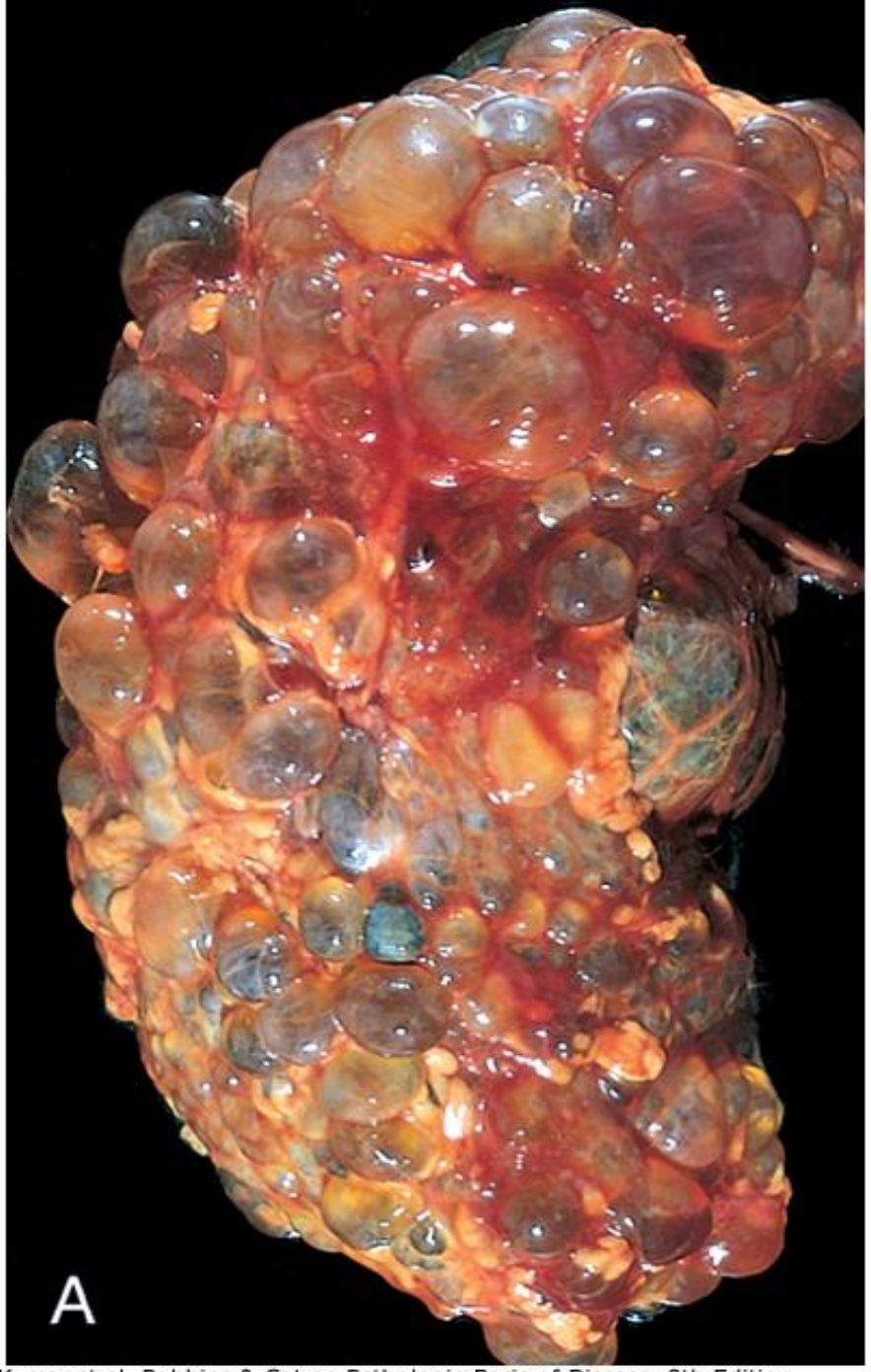
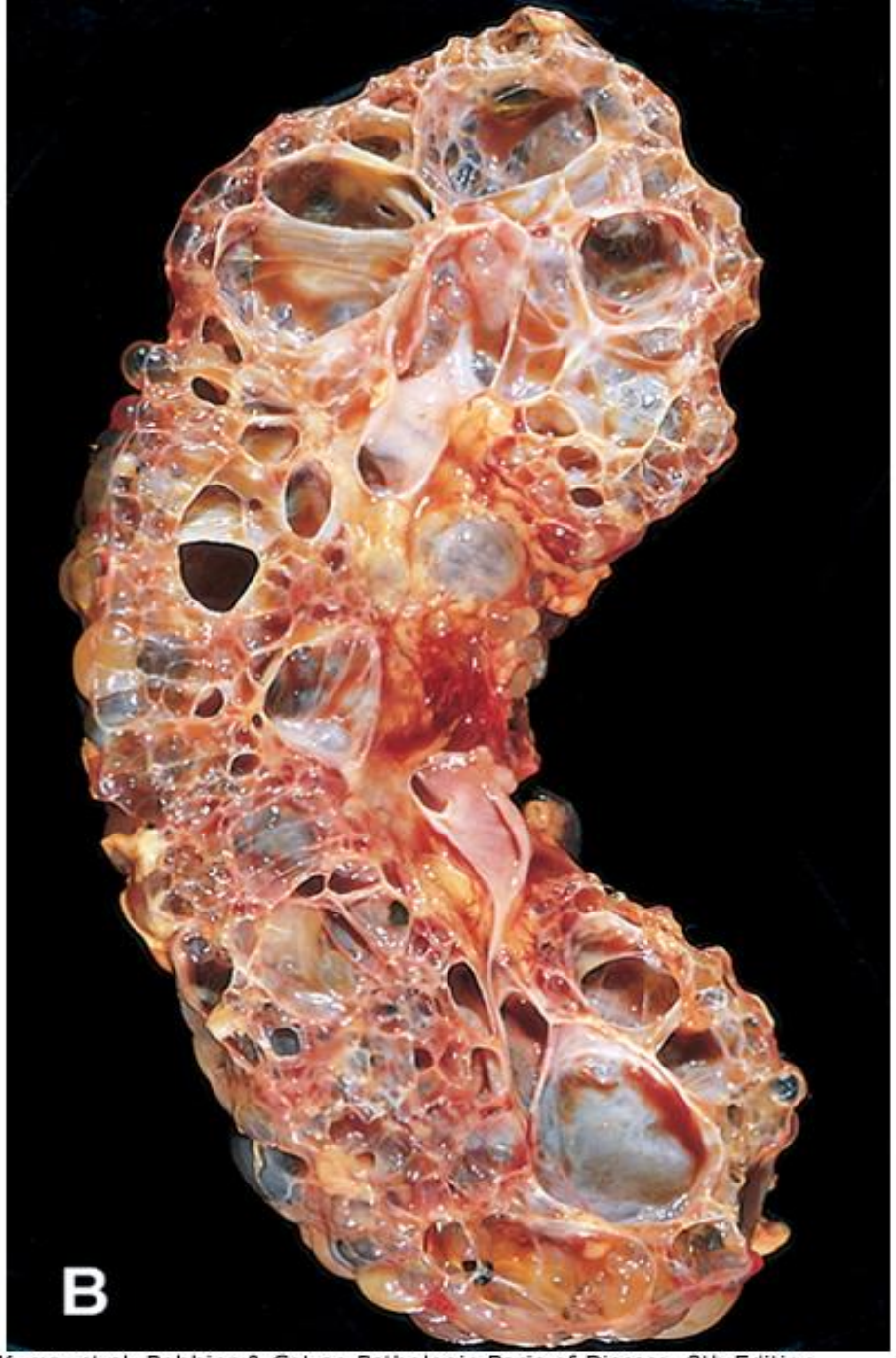
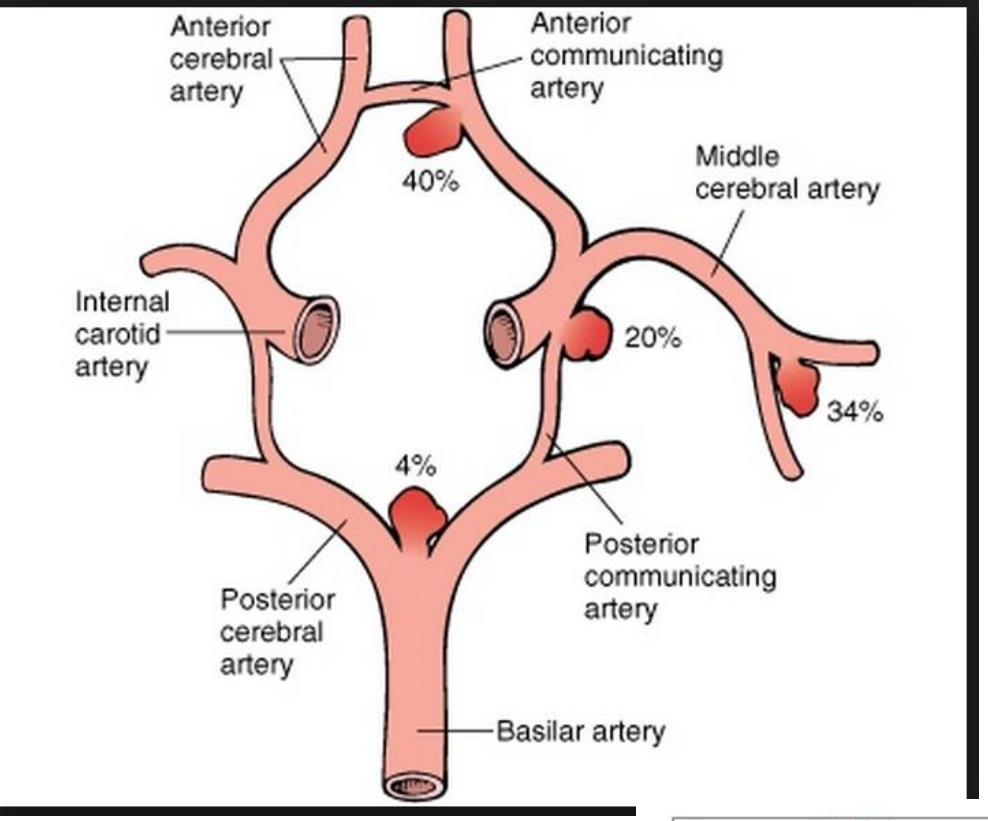
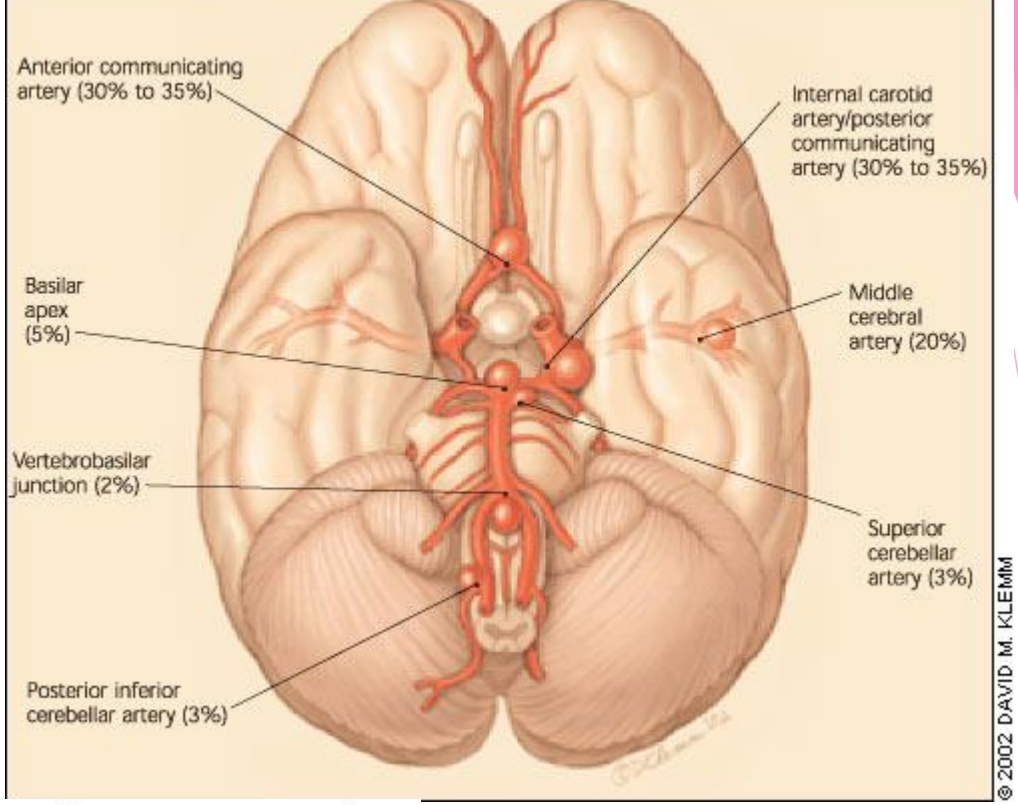




Autosomal Dominant Polycystic Kidney Disease

- Clinical:
- Many of these patients remain asymptomatic until about the 4th
- or 5th decade of life when renal insufficiency occurs because
- the cysts initially involve only portions of the nephrones, so renal
- function is retained .
- Presentation (variable): flank pain, hypertension, hematuria,
  - progressive renal failure
  - Large lesions are palpable
  - 40% have cystic disease of the liver (most common), spleen, pancreas, brain
  - Berry aneurysms in circle of Willis and can cause death in about 4-
  - 10% of patients due to subarachnoid hemorrhage.
  - Death due to uremia or hypertension
  - 40% of patients die of coronary or hypertensive heart disease, 25% of
  - infection, 15% of ruptured Berry aneurysm or hypertensive
  - intracranial hemorrhage.


Anterior cerebral artery
Anterior communicating artery
40%
Middle cerebral artery
Internal carotid artery
20%
34%
4%
Posterior communicating artery
Posterior cerebral artery
Basilar artery


Anterior communicating artery (30% to 35%)
Internal carotid artery/posterior communicating artery (30% to 35%)
Basilar apex (5%)
Middle cerebral artery (20%)
Vertebrobasilar junction (2%)
Superior cerebellar artery (3%)
Posterior inferior cerebellar artery (3%)
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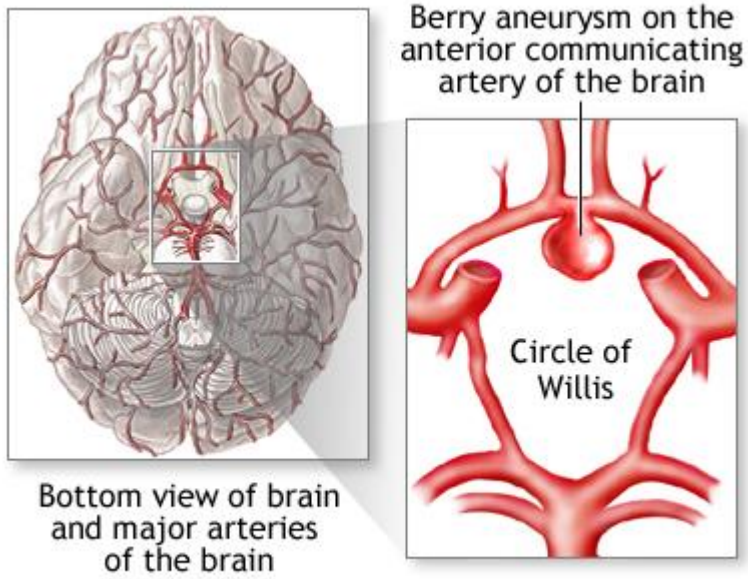
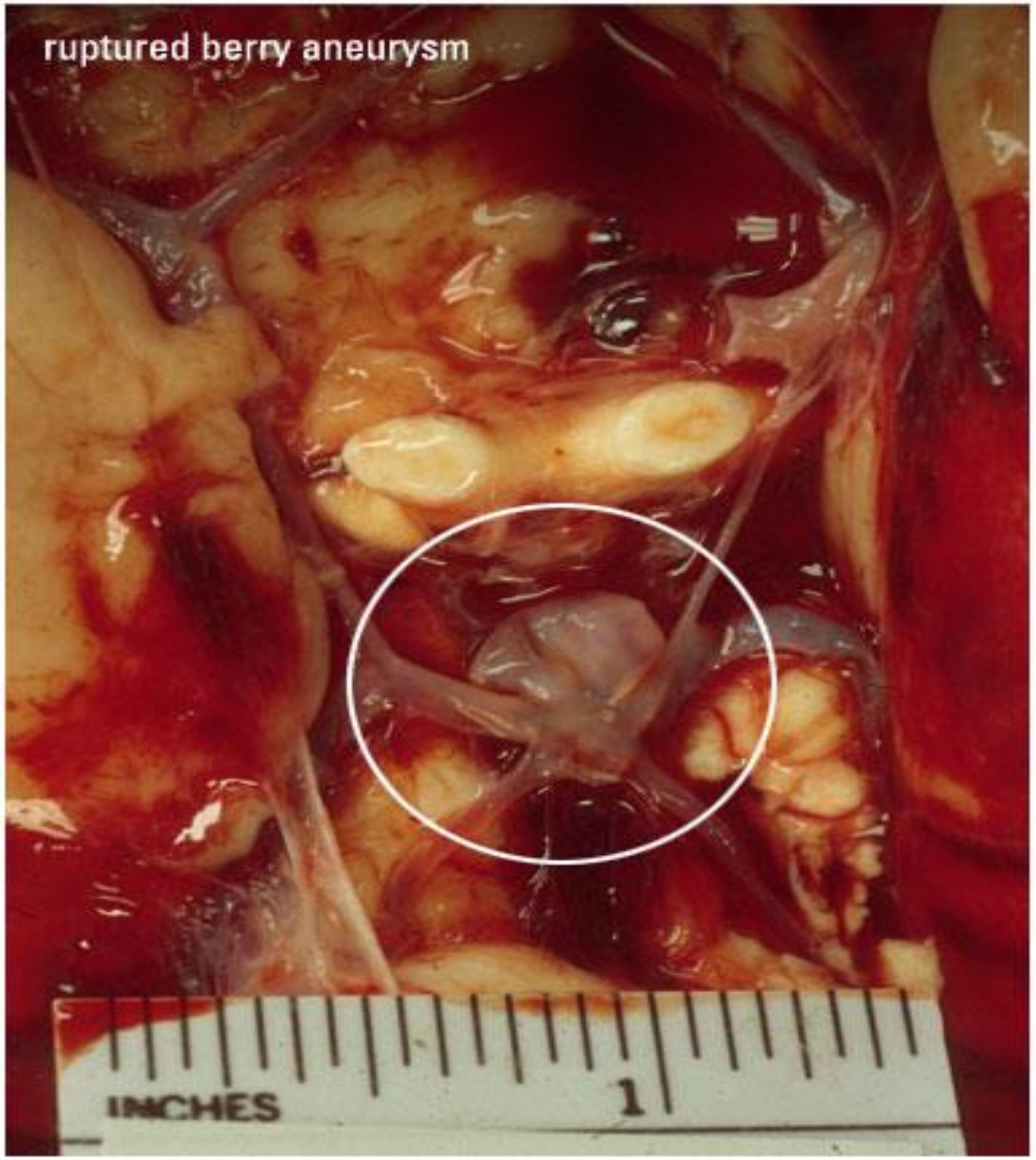
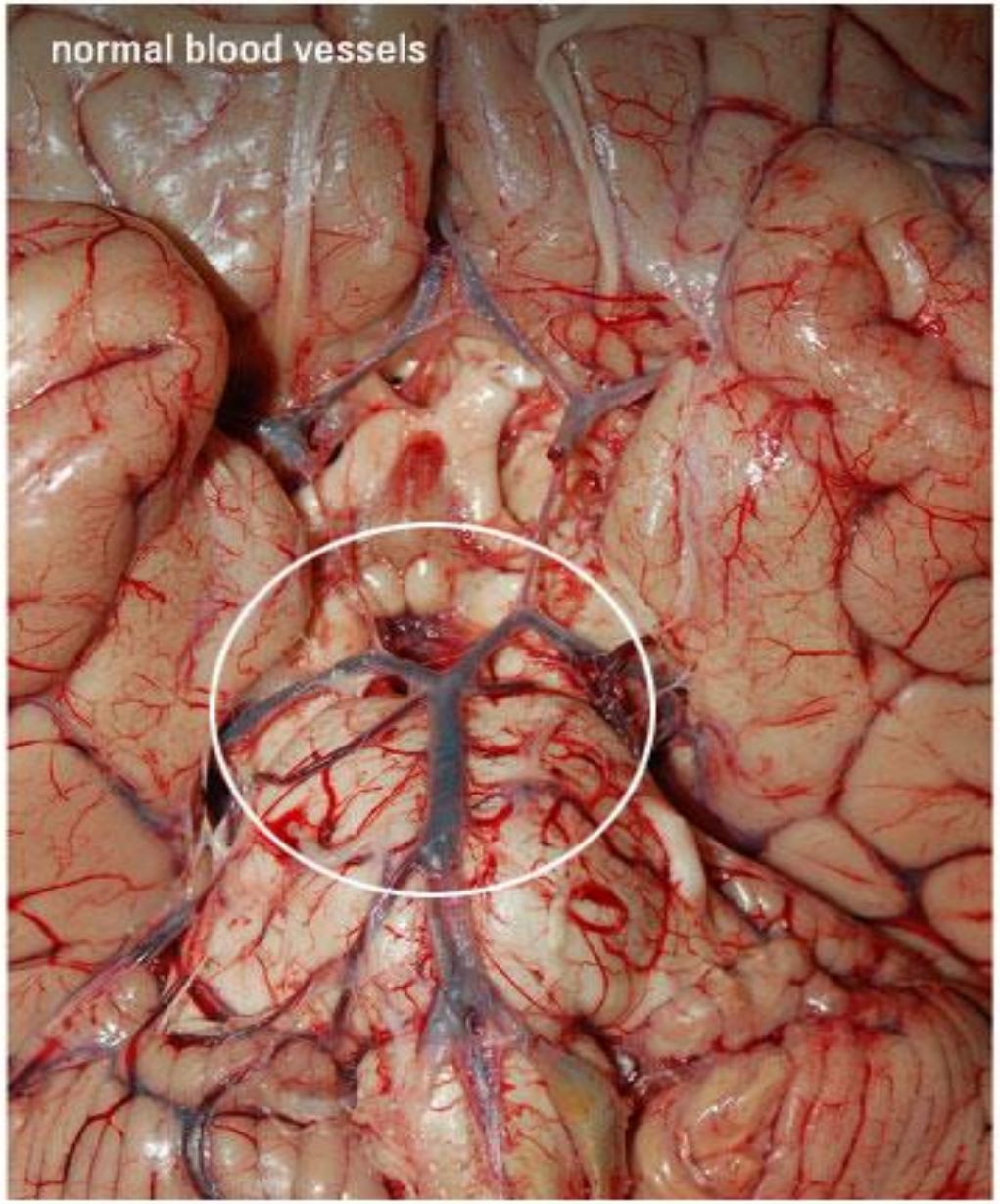
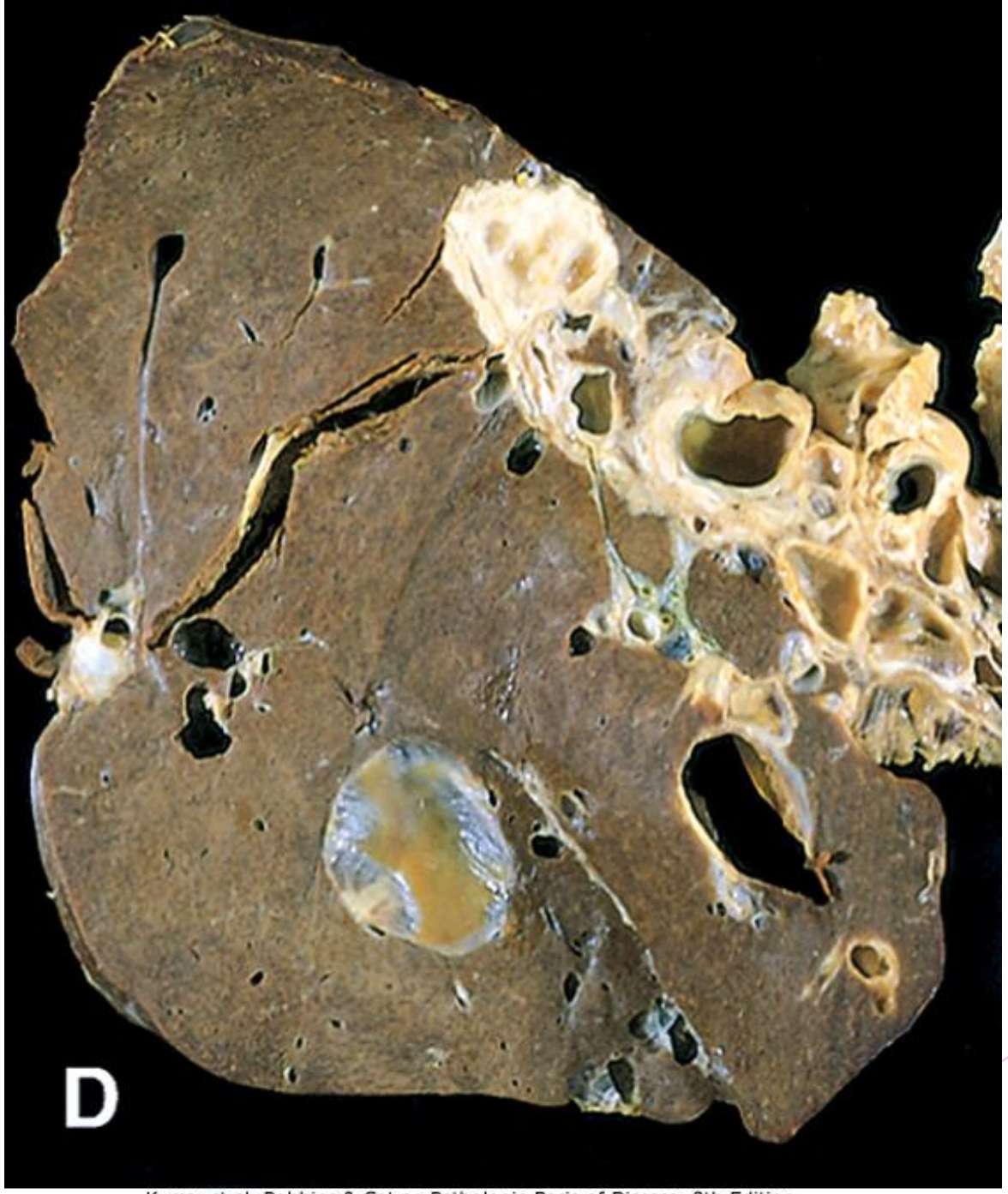

Berry aneurysm on the anterior communicating artery of the brain
Circle of Willis
Bottom view of brain and major arteries of the brain
A.D.A.M.


ruptured berry aneurysm
INCHES
1


normal blood vessels



Autosomal Recessive (Childhood) Polycystic Kidney Disease

- Is rare anomaly due to mutation of gene **PKHD1** on chromosome **6**
- Perinatal, neonatal, infantile and juvenile types.
- The first 2 are the most common
- In all cases are associated with liver cysts (congenital liver cirrhosis).

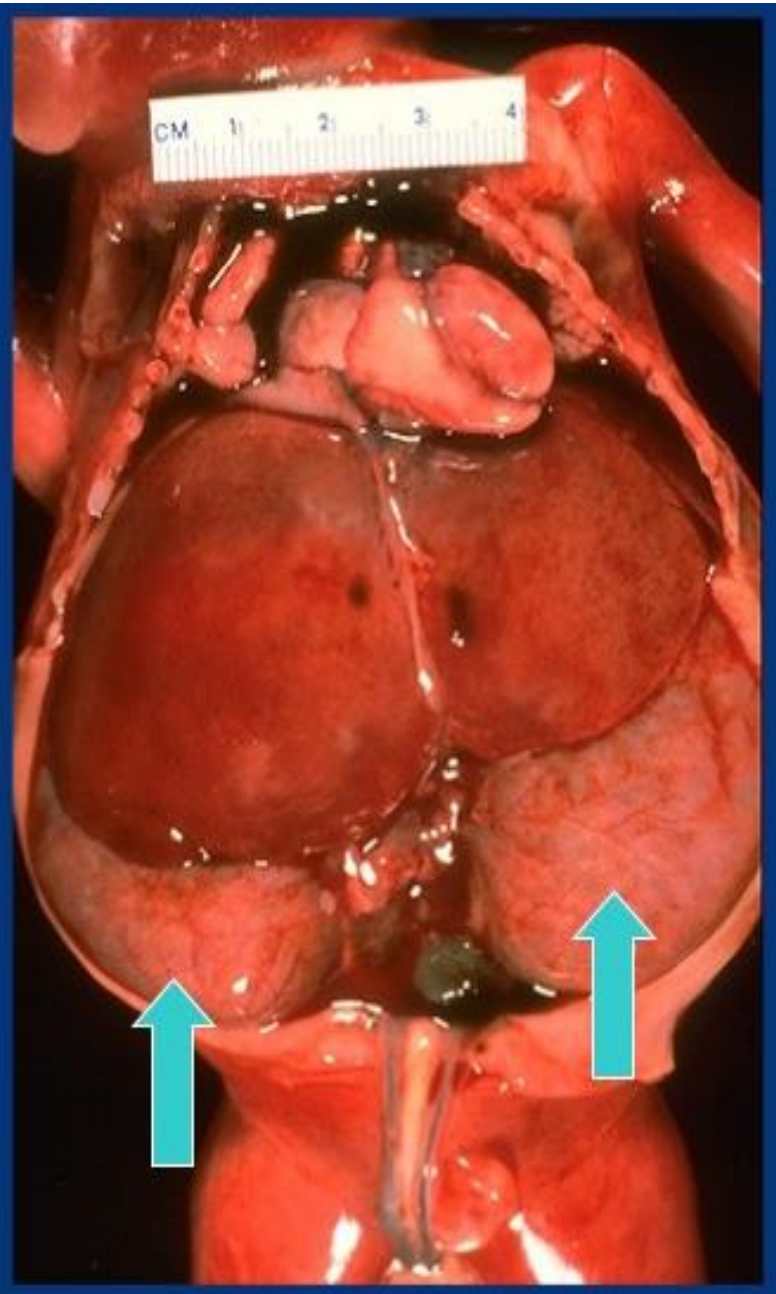
- **Gross:**
- bilateral Small cysts in the cortex & medulla (sponge like appearance).
- **Elongated channels** at **right angles** to the cortical surface.
- Mic:
- Uniform lining of cuboidal cells (Originate from **collecting ducts**).

Morphology

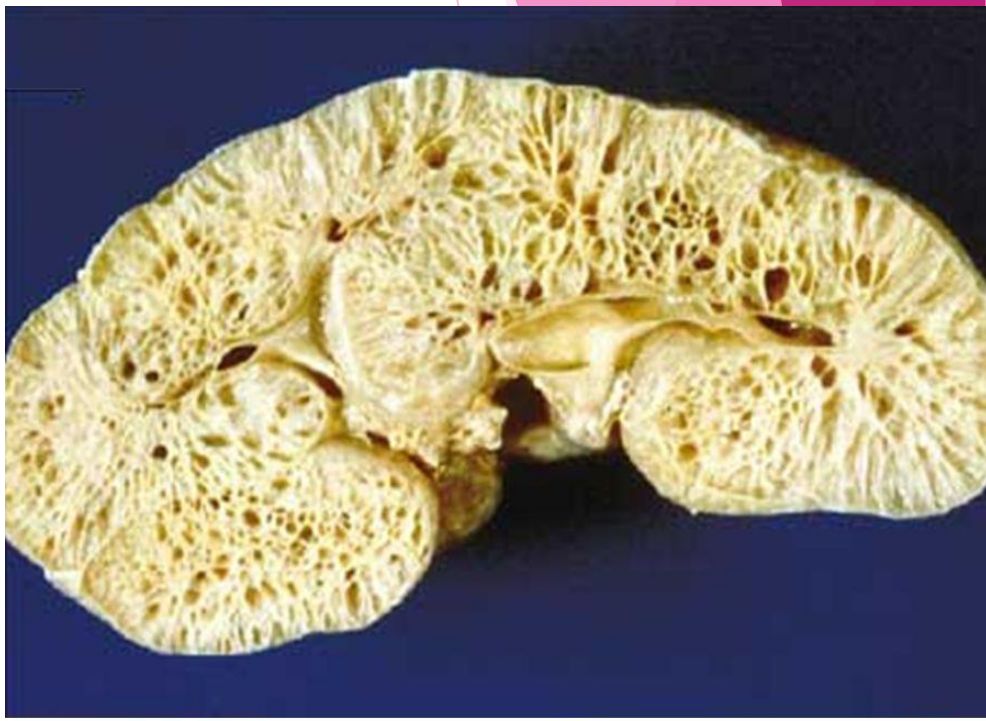
- The kidneys are enlarged (bilateral) and have a **smooth** external surface. On cut section, **numerous small cysts** in the cortex and medulla give the kidney a **sponge-like appearance**.

- **Smooth, Small, Sponge**

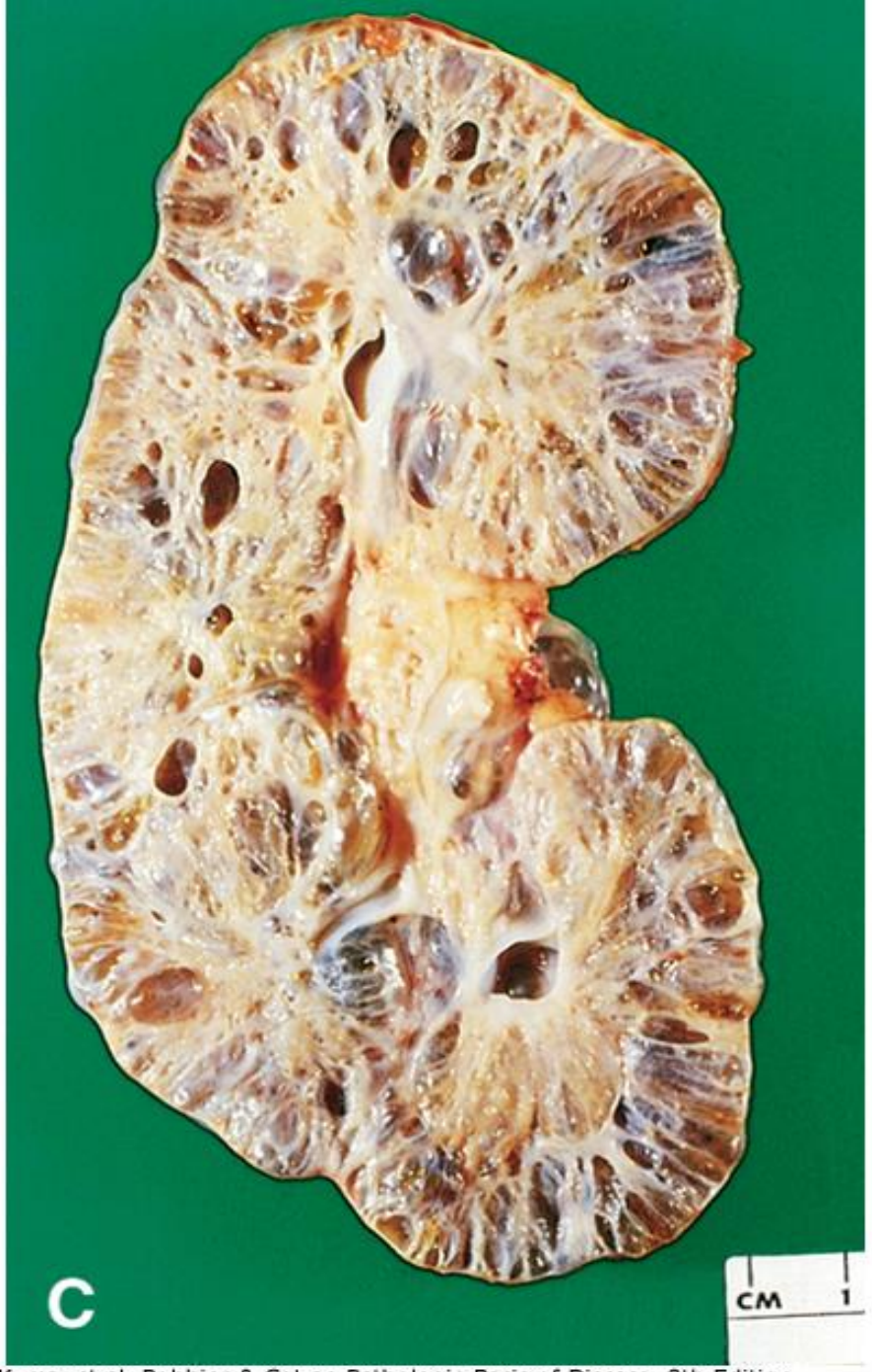
- The cysts are dilated channels **perpendicular** to the corticomedullary junction.
- Cysts originate from **collective tubules** and are lined by uniform cuboidal cells
- In almost all cases, the **liver has cysts with portal fibrosis** as well as proliferation of portal bile ducts.
- **Clinical Features;** serious manifestations are usually present at birth, and the young infant might succumb rapidly to renal failure. Patients, who survive infancy, may **develop congenital hepatic fibrosis.**
- Liver: epithelium lined cysts and proliferation of bile ducts

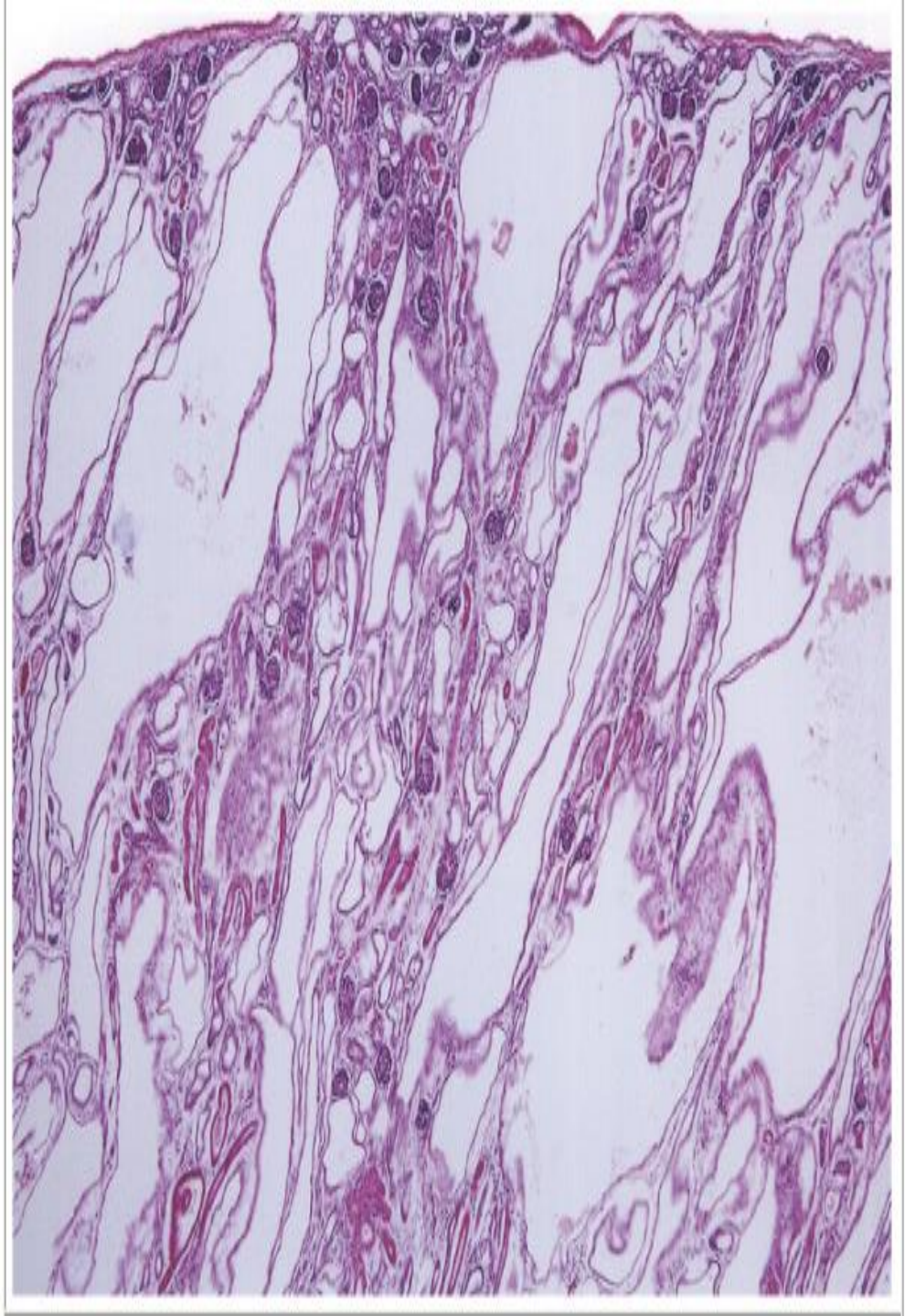


- This child died soon after premature birth at 23 weeks gestation resulted from markedly diminished fetal urine output as a consequence of polycystic kidney disease.
- Note the bilaterally enlarged kidneys that nearly fill the abdomen below the liver.
- The histological appearance in this case, coupled with the gross appearance., was consistent with recessive polycystic kidney disease (RPKD).



Childhood polycystic kidney disease





Acquired (Dialysis-Associated) Cystic Disease

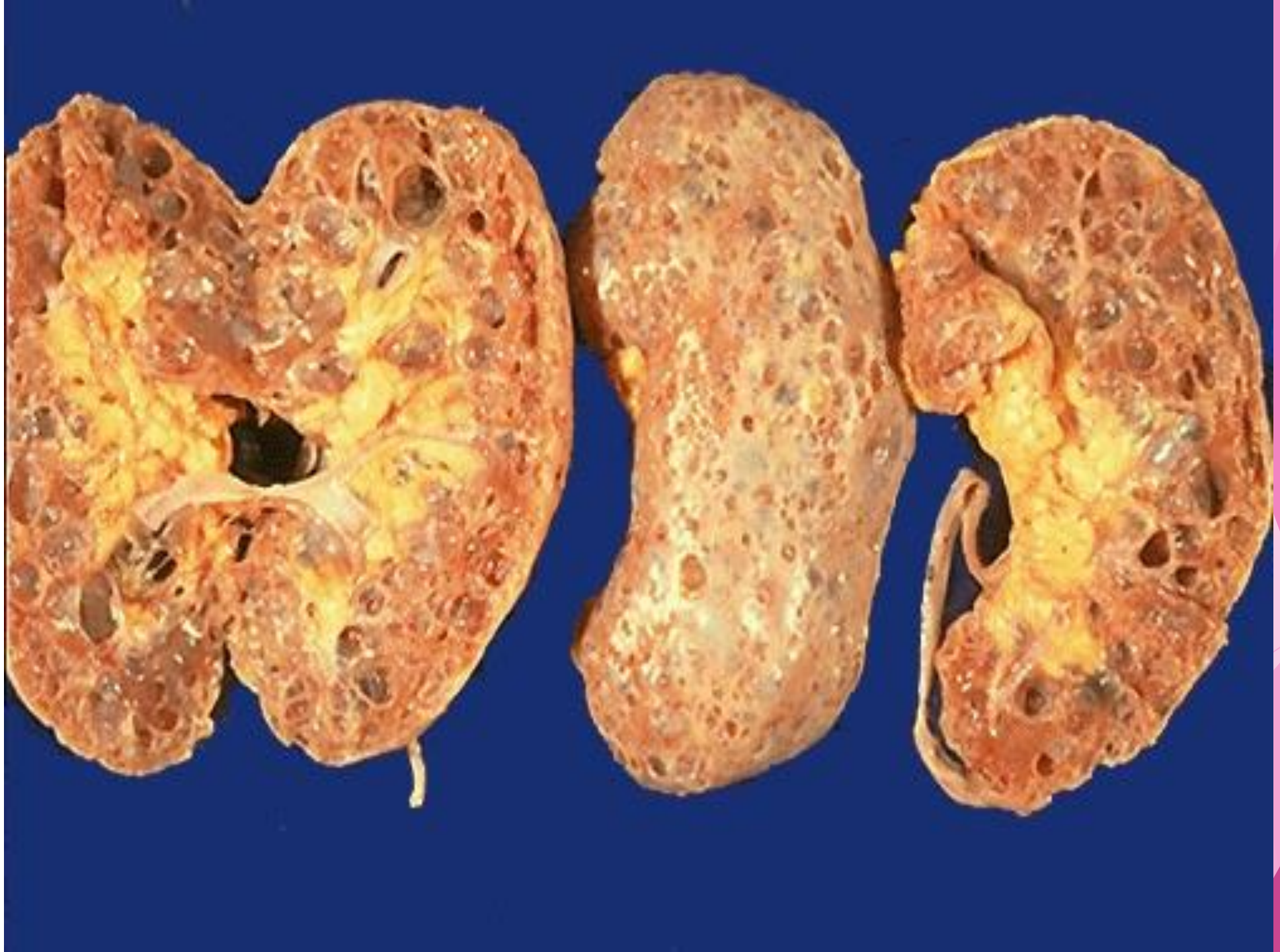
- The kidneys of patients on chronic dialysis, sometimes exhibit numerous cortical and medullary cysts.
- The cysts measure 0.5-2cm, contain clear fluid, are lined by either hyperplastic or flattened tubular epithelium and often contain calcium oxalate crystals.

- Cause: They probably form as a result of tubular obstruction due to interstitial fibrosis or by oxalate crystals.

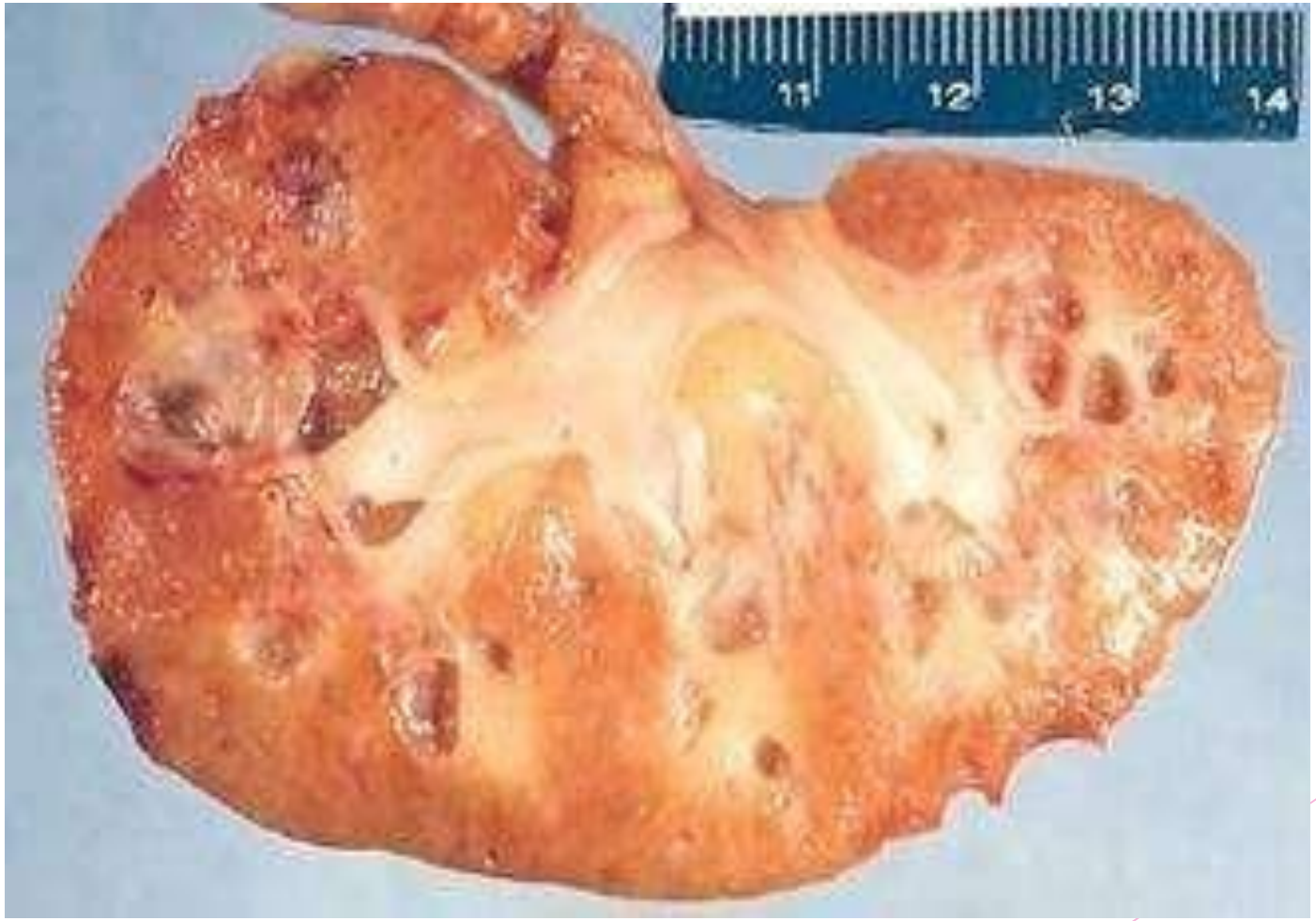
Acquired (Dialysis-Associated) Cystic Disease

- Most are asymptomatic, but sometimes the bleeding inside the cysts cause hematuria.

- The most important complication is the development of renal cell carcinoma in the walls of these cysts in about 7% of patients during 10 years period.

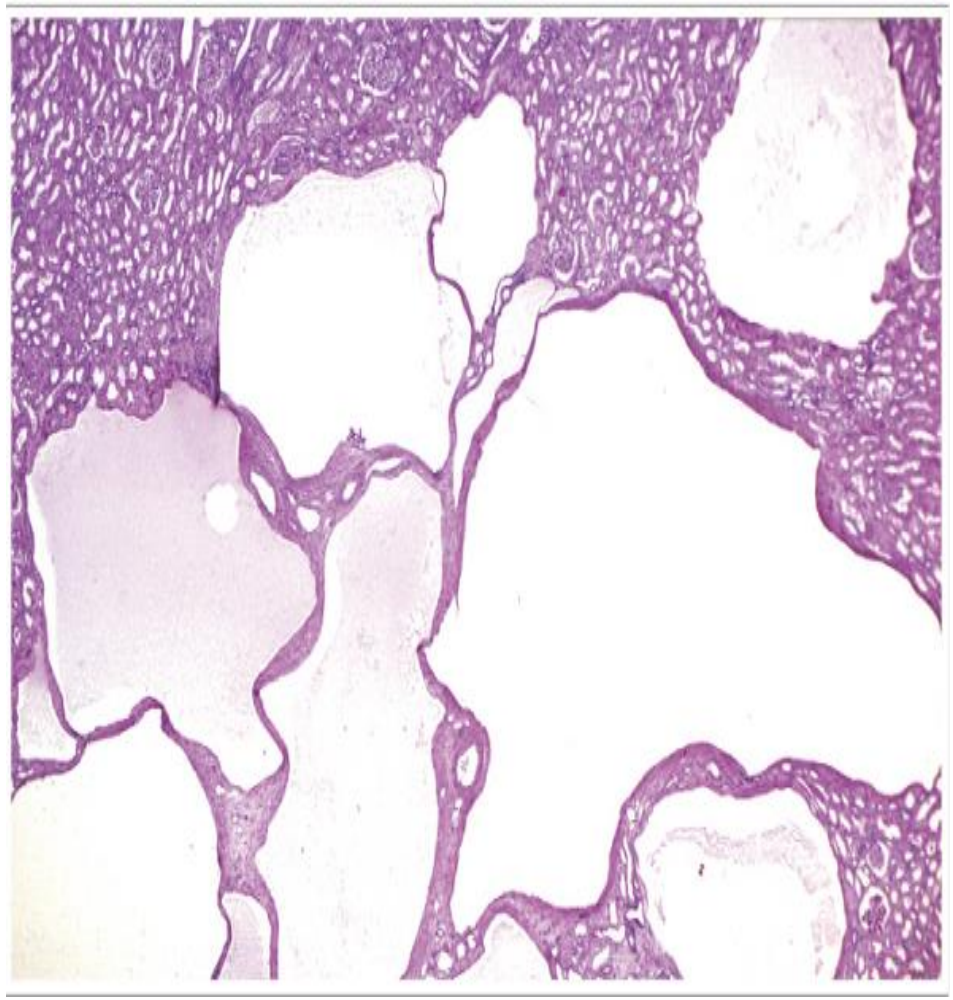


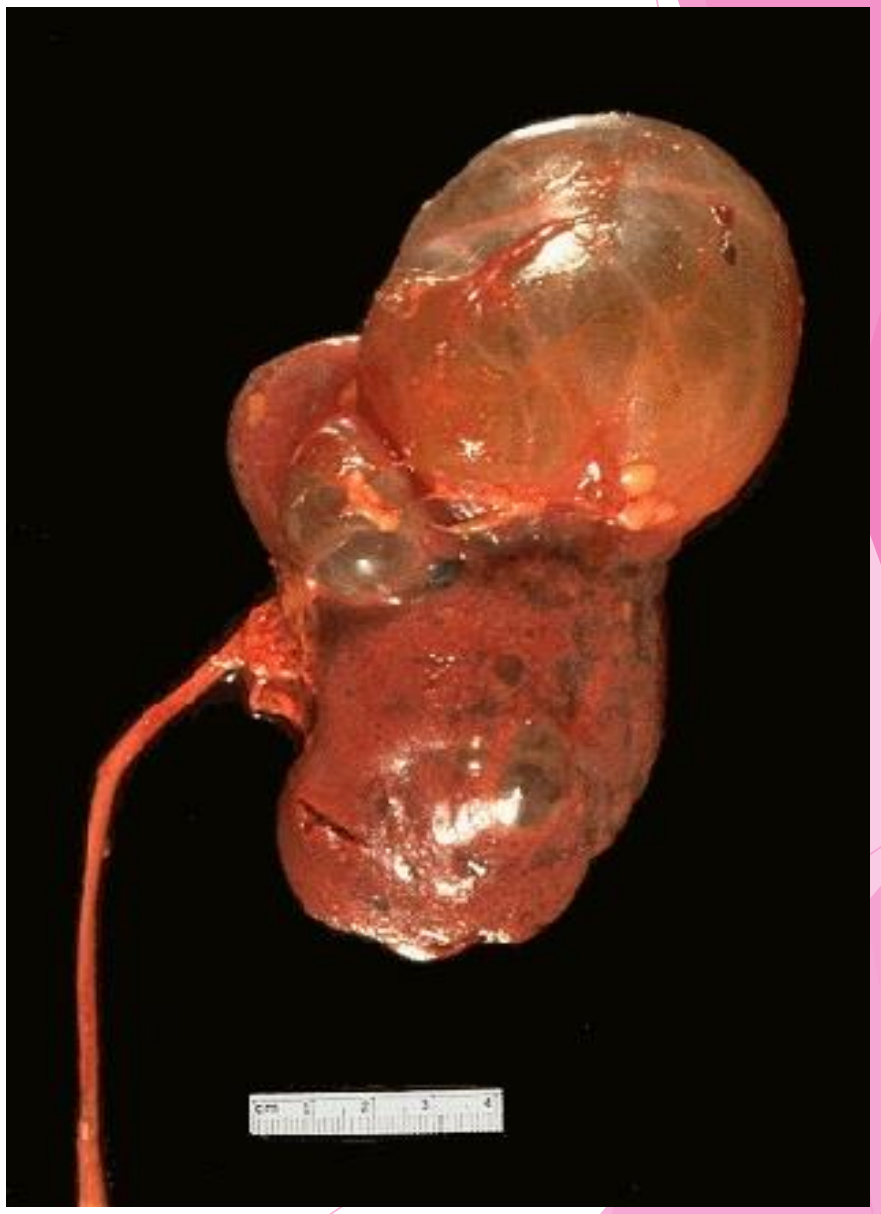
ACQUIRED (DIALYSIS)

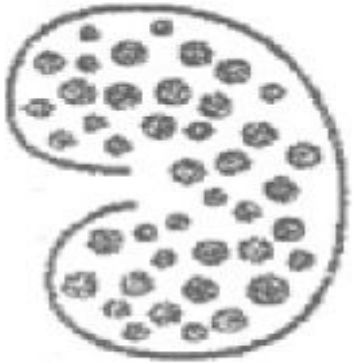


Simple Cysts

- Acquired, Incidental, very common
- These occur as single or multiple, usually cortical.
- The size range from 1-10 cm or more.
- They are translucent and filled with clear fluid.
- They are lined by a single layer of cuboidal or flattened epithelium.
- They are common postmortem findings. On occasion, hemorrhage into them may cause sudden pain, and calcification may be visible radiologically.
- The main importance of these cysts is in their differentiation from kidney tumors.





	Inheritance	Pathologic Features	Clinical Features or Complications	Typical Outcome	Diagrammatic Representation
Adult polycystic kidney disease	Autosomal dominant	Large multicystic kidneys, liver cysts, berry aneurysms	Hematuria, flank pain, urinary tract infection, renal stones, hypertension	Chronic renal failure beginning at age 40–60 years	
Childhood polycystic kidney disease	Autosomal recessive	Enlarged, cystic kidneys at birth	Hepatic fibrosis	Variable, death in infancy or childhood	
Simple cysts	None	Single or multiple cysts in normal-sized kidneys	Microscopic hematuria	Benign	
Acquired renal cystic disease	None	Cystic degeneration in end-stage kidney disease	Hemorrhage, erythrocytosis, neoplasia	Dependence on dialysis	

Renal diseases can be divided according to the parts of kidneys into

- 1. Glomerular diseases
- 2. Tubular diseases.
- 3. Interstitium diseases.
- 4. Blood vessels diseases.
-

This division is useful because

- a. The early manifestations of each group of diseases tend to be distinctive.
- b. These groups differ in their pathogenesis, for e.g., glomerular diseases are often immunologically mediated, whereas tubular and interstitial disorders are more likely to be caused by toxic or infectious agents. However, it should be noted that:
- 1. The interdependence of renal components translated into that damage to one component is almost always affects secondarily the others.
- 2. All forms of chronic renal disease tend ultimately to damage all four components of the kidney thus, eventuates in chronic renal failure (end-stage kidney disease ESKD).

GLOMERULAR DISEASES

Glomerular Diseases:

- They constitute some of the major problems in nephrology; in fact they are the most common causes of chronic renal failure in humans.
- **Clinical manifestations of renal disease;**

Acute Nephritic Syndrome: characterized by

- Gross hematuria (macroscopic).
- Mild to moderate proteinuria.
- Edema.
- Hypertension
- Typical example is ***Poststreptococcal glomerulonephritis.***

2. ***Nephrotic Syndrome:***

- Heavy Proteinuria (> 3.5 gram of protein / 24hours).
- Hypoalbuminemia.
- Severe edema.
- Hyperlipidemia & lipiduria.

3. ***Asymptomatic hematuria & / or Proteinuria:***

- Mild Glomerular abnormality.

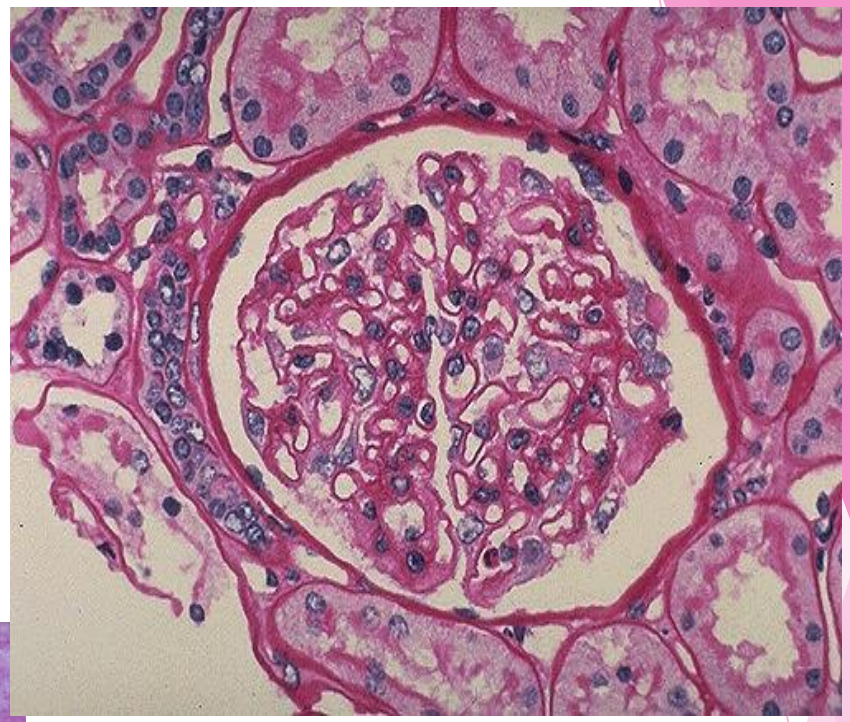
- 4. ***Rapidly Progressive Glomerulonephritis:***
- Loss of renal functions in a few days or weeks.
- Manifested by active urine sediment (hematuria, **Dysmorphic RBC_S, RBC_S Casts**).
- 5. ***Acute Renal failure:***
- Oliguria (< 500 cc) or Anuria (no urine flow).
- Recent onset of ***Azotemia.*** **(in Latin: nitrogen = azot)**
- 6. ***Chronic Renal failure:***
- Prolonged symptoms & signs of ***Uremia.***
- Can be the end result of all chronic renal diseases.
- 7. ***Urinary tract infection:***
- Characterized by ***Bacteruria & Pyuria*** *(*bacteria and leukocytes in the urine).
- **8. *Nephrolithiasis*:** Characterized by ***Renal colic.***
- ***Important notes:***
- **Azotemia**: is a biochemical abnormality that means elevation of blood urea nitrogen (BUN) and creatinine
- levels, and is related largely to a decreased glomerular filtration rate (GFR).
- Could be **Prerenal Azotemia** (Hypoperfusion of kidneys), **renal** (due to kidney diseases), **Postrenal Azotemia** (obstruction below the kidney).
- **Uremia:** characterized by
  - *Clinical Signs, Symptoms & Biochemical abnormalities*.
  - Renal damage (impair excretory, endocrine, & metabolic functions of kidneys). Uremic patients frequently
  - manifest secondary involvement of the gastrointestinal system (e.g., uremic gastroenteritis), peripheral nerves
  - (e.g., peripheral neuropathy), and heart (e.g., uremic fibrinous pericarditis).

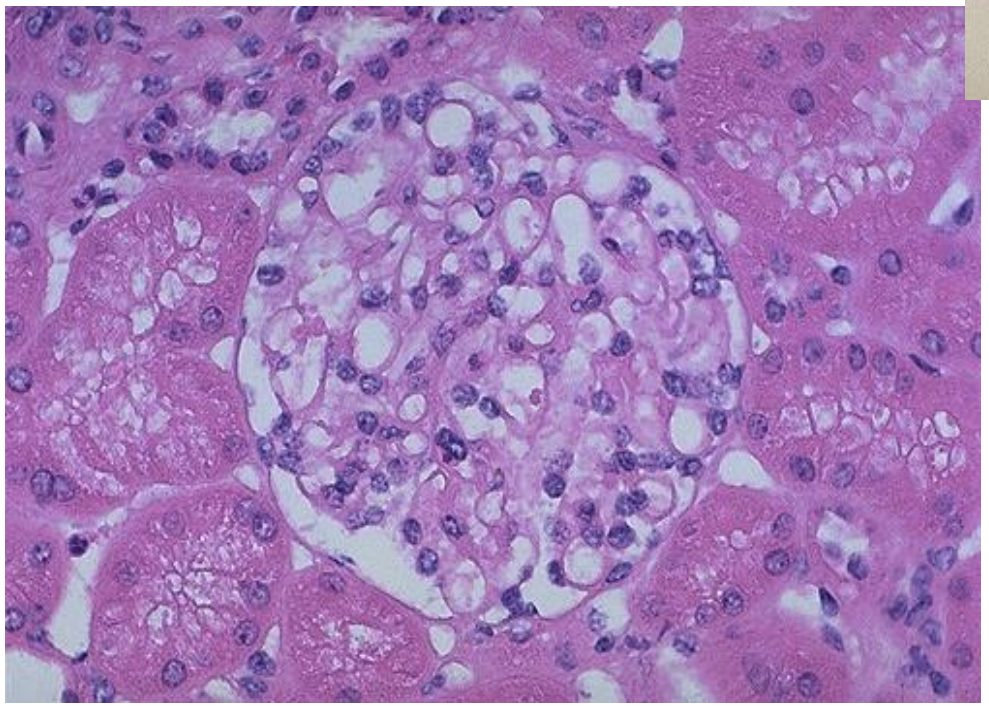
Principal Systemic Manifestations of Chronic Kidney Disease and Uremia

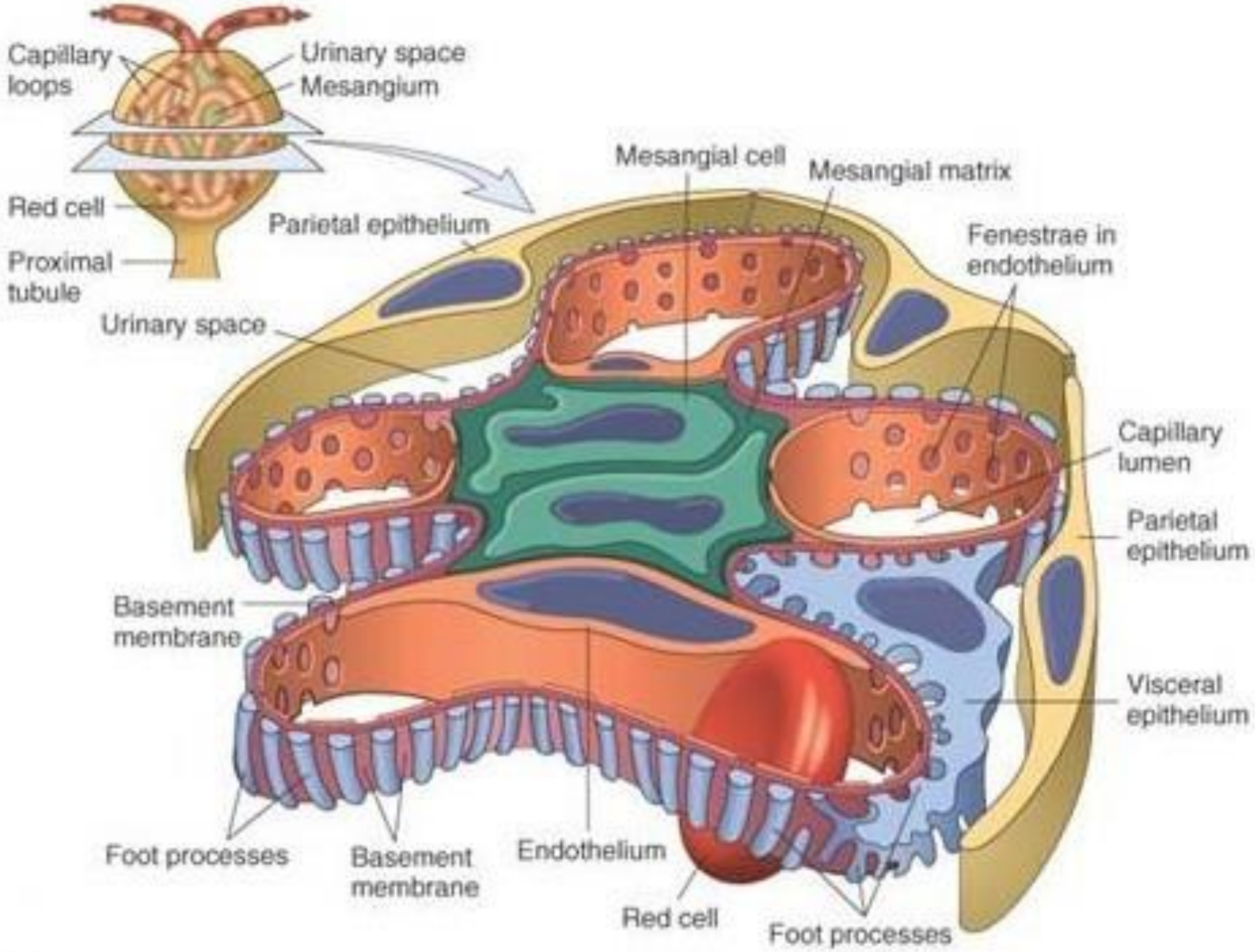
Fluid and Electrolytes
Dehydration
Edema
Hyperkalemia
Metabolic acidosis
Calcium Phosphate and Bone
Hyperphosphatemia
Hypocalcemia
Secondary hyperparathyroidism
Renal osteodystrophy
Hematologic
Anemia
Bleeding diathesis
Cardiopulmonary
Hypertension
Congestive heart failure
Cardiomyopathy
Pulmonary edema
Uremic pericarditis
Gastrointestinal
Nausea and vomiting
Bleeding
Esophagitis, gastritis, colitis
Neuromuscular
Myopathy
Peripheral neuropathy
Encephalopathy
Dermatologic
Sallow color
Pruritus
Dermatitis

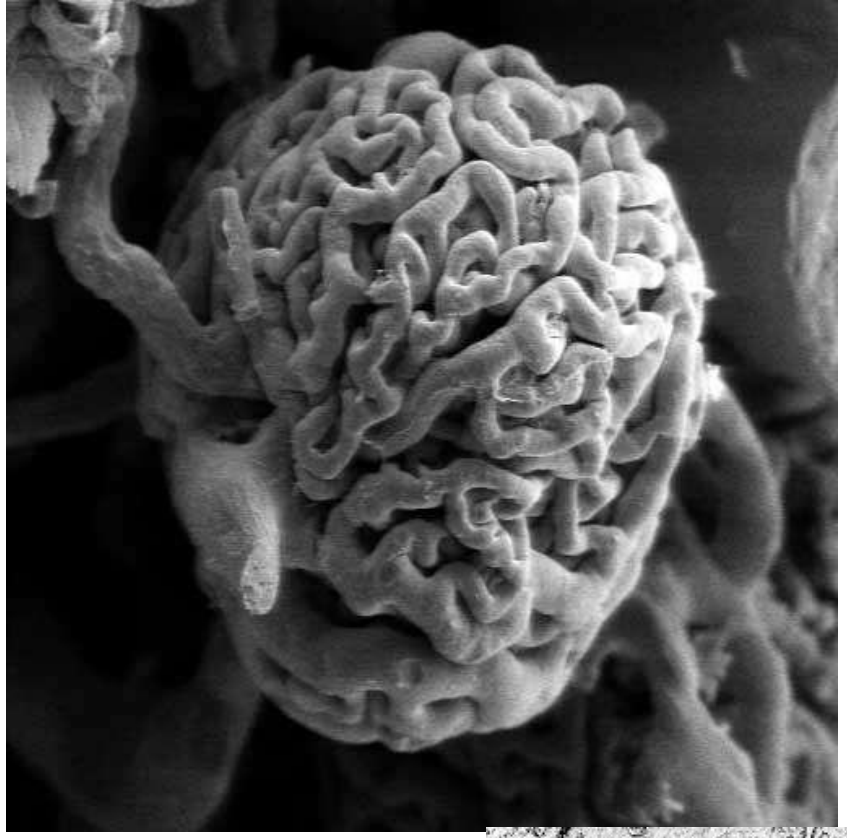
Clinical Presentations of GN

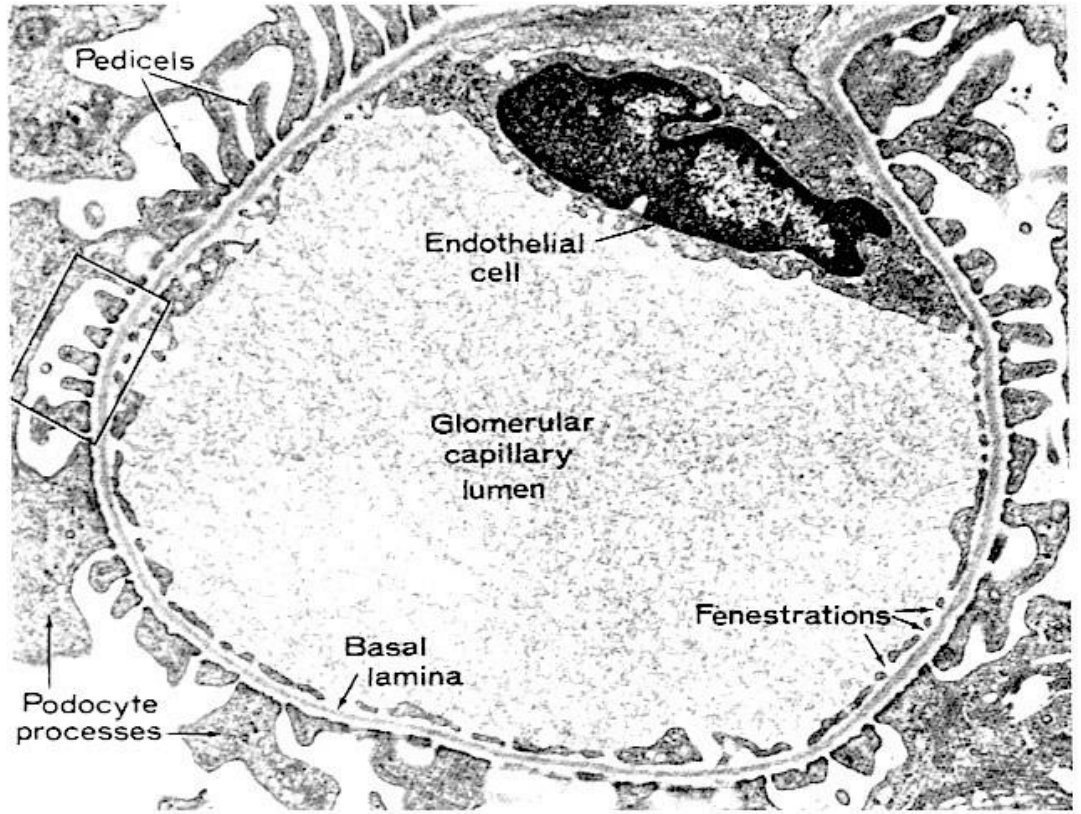
Minimal change disease	Nephrotic
Focal-segmental glom sclerosis	Nephrotic
Membranous gn	Nephrotic
Diabetic nephropathy	Nephrotic
Acute Post. Strept. gn	Nephritic
Crescentic gns	Nephritic
Membranoproliferative gns	Nephrotic/Nephritic
SLE	Nephrotic/Nephritic
IgA nephropathy	Nephrotic/Nephritic
Alports syndrome/thin BMS	Nephrotic/nephritic
Light chain-associated diseases	Nephrotic/acute renal failure

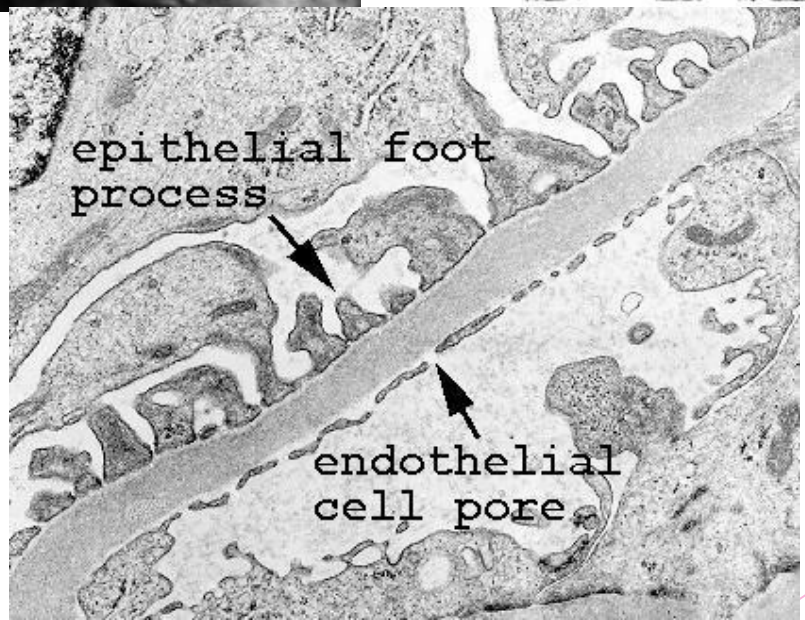





Capillary loops
Urinary space
Mesangium
Red cell
Parietal epithelium
Proximal tubule
Urinary space
Mesangial cell
Mesangial matrix
Fenestrae in endothelium
Capillary lumen
Parietal epithelium
Basement membrane
Visceral epithelium
Foot processes
Basement membrane
Endothelium
Red cell
Foot processes

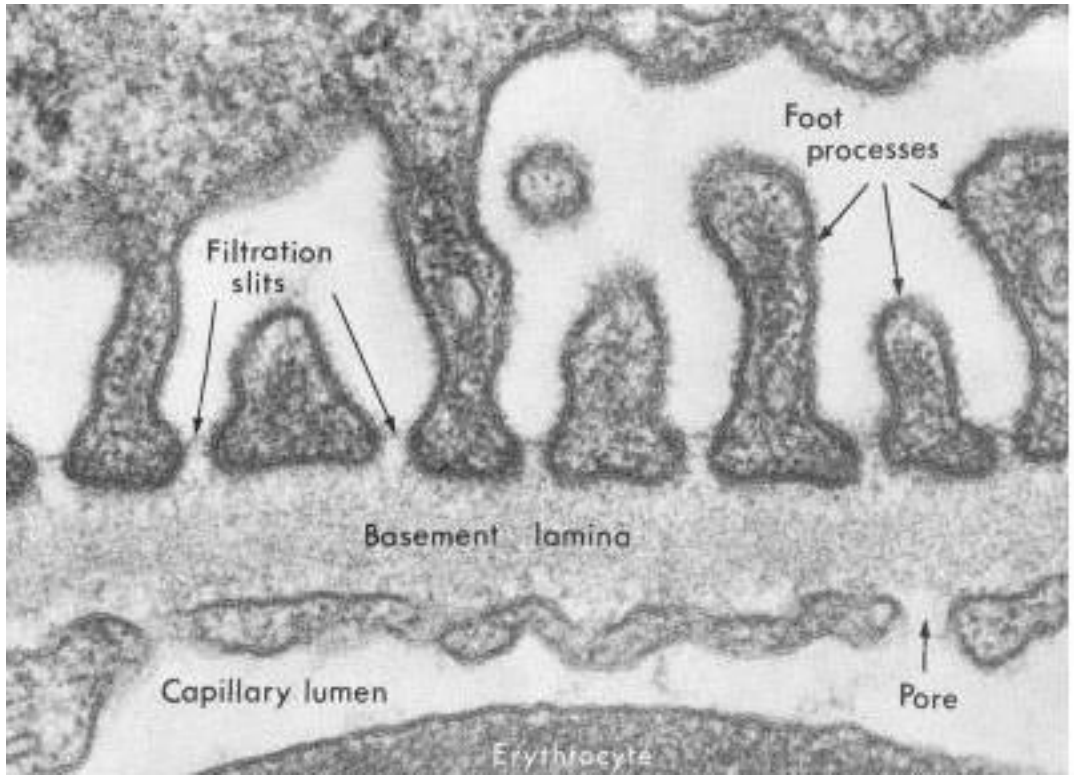


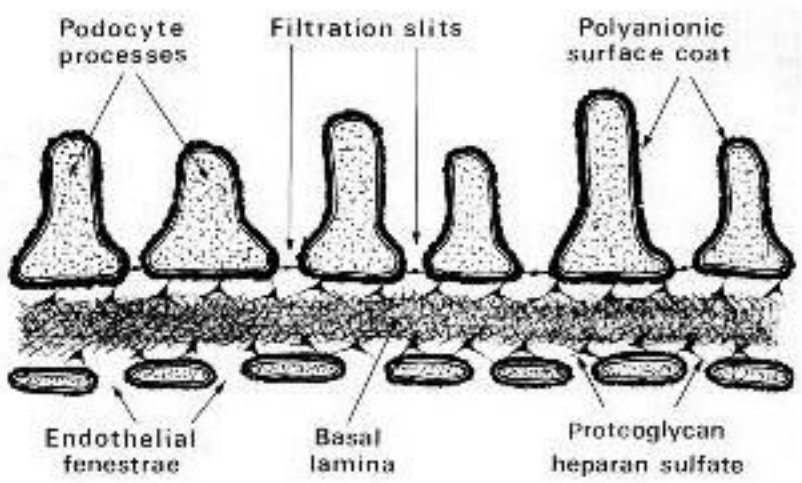

Pedicels
Endothelial cell
Glomerular capillary lumen
Fenestrations
Basal lamina
Podocyte processes


epithelial foot process
endothelial cell pore

S.E.M.

T.E.M.


Foot
processes
Filtration
slits
Basement lamina
Capillary lumen
Pore
Erythrocyte


Podocyte
processes
Filtration slits
Polyanionic
surface coat
Endothelial
fenestrae
Basal
lamina
Proteoglycan
heparan sulfate

- One of important function of Glomerular wall is Selective Permeability (high permeable to H2o & small solutes) while
- completely impermeable to molecules of size & molecular charge of Albumin.
- The podocyte is decisive to the glomerular barrier function by providing a distal resistance to the flow of water and a barrier to the filtration of proteins. It is also largely responsible for synthesis of GBM components

Histologic alterations

- **There are 5 basic tissue reactions**

- 1. **Increased glomerular cellularity**
  - a. Proliferation of mesangial or endothelial cells.
  - b. Leukocyte infiltration, including neutrophils, monocytes, and, in some diseases, lymphocytes.
  - c. Formation of crescents(proliferation of parietal epithelial cells
  - .

- 2. **Basement membrane thickening**, best seen in sections stained with (PAS). By EM, it can be resolved as one of 2 alteration;

- a. Deposition of amorphous electron dense material, of immune complexes, on the **endothelial** or **epithelial** side of basement membrane, or within the **GBM itself**.
- b. Thickening of the BM proper, as occurs in diabetic glomerulosclerosis.

3. **Hyalinization and sclerosis,** made up of plasma proteins and collagen material deposited exracellularly.

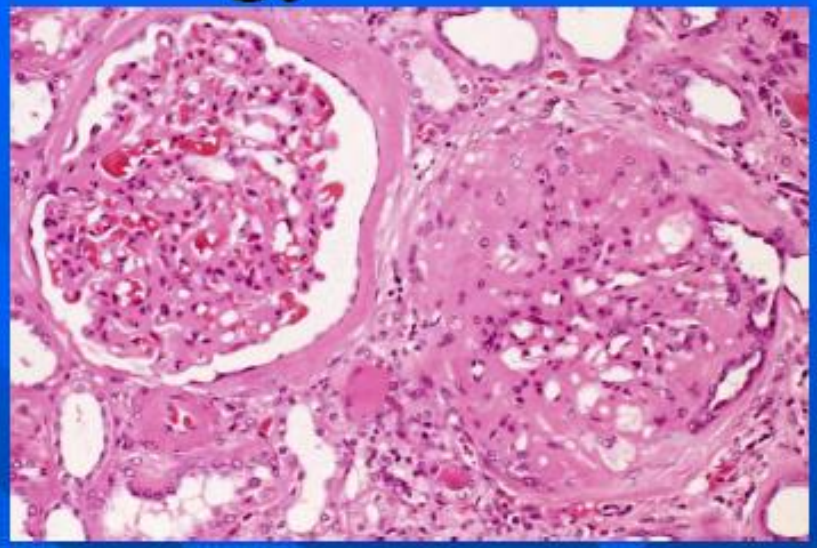
4. **Additional alterations** include; accumulation of lipids, fibrin or other metabolic materials.

5. Intraglomerular vascular thrombosis

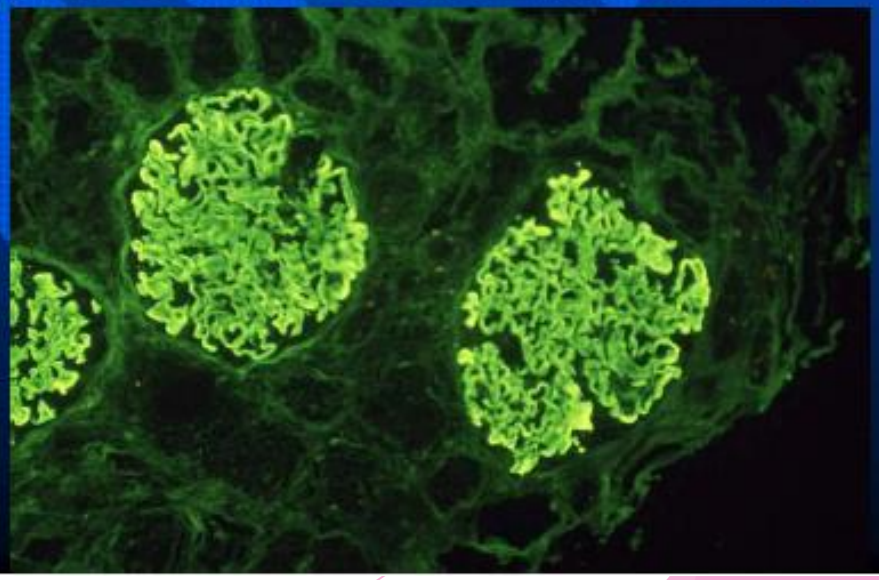
- The histologic changes can be further subdivided into;
  1. Diffuse 2. Focal. 3. Global. 4. Segmental. 5. Mesangial

Terminology

- Focal - some glomeruli (< 80%) involved

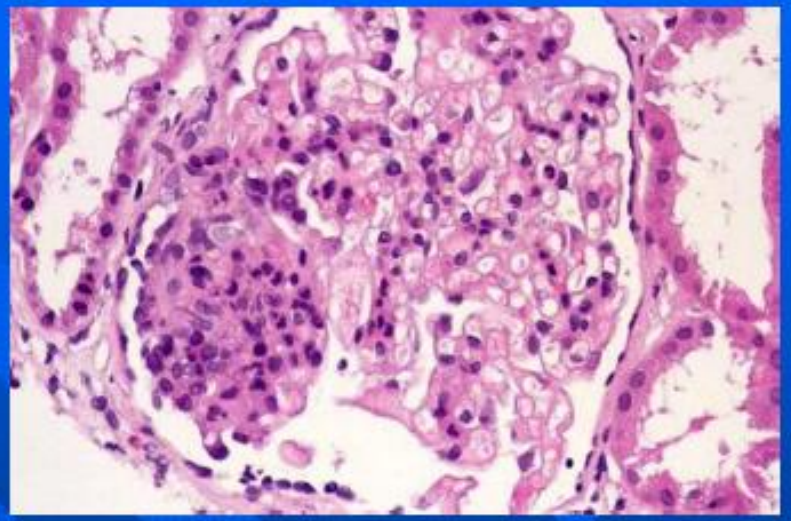


- Diffuse - most glomeruli (>80%) involved

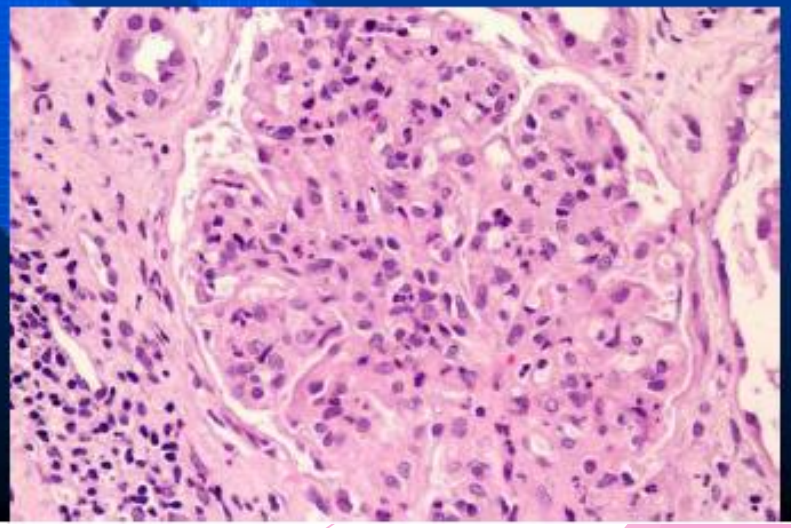


Terminology

- Segmental - portion of a glomerulus involved



- Global - entire glomerulus involved



Pathogenesis

- Although little is known about etiologic agents and triggering events, it is clear that immune mechanisms underlie most forms of primary glomerulopathies, and many of the secondary forms.

Immune-mediated mechanisms;

1. Ab-mediated,

a. Insitu immune complex deposition,

- In this form of injury, immune complexes are formed locally by antibodies that react with intrinsic tissue antigen or with extrinsic antigens "planted" in the glomerulus from the circulation.
  - **Fixed intrinsic antigens for e.g. (BM)like Goodpasterur syndrome (Ab against glomerular and pulmonary BM) The pattern of immune deposition by immunofluorescence microscopy is linear.**
  - **Planted antigens (exogenous as infectious agent or drug and endogenous as DNA, immunoglobulins) The pattern of immune deposition by immunofluorescence microscopy is granular**

b. Circulating immune complex deposition

- **Endogenous Ag (DNA, tumor)**
- **Exogenous Ag (infectious products)**

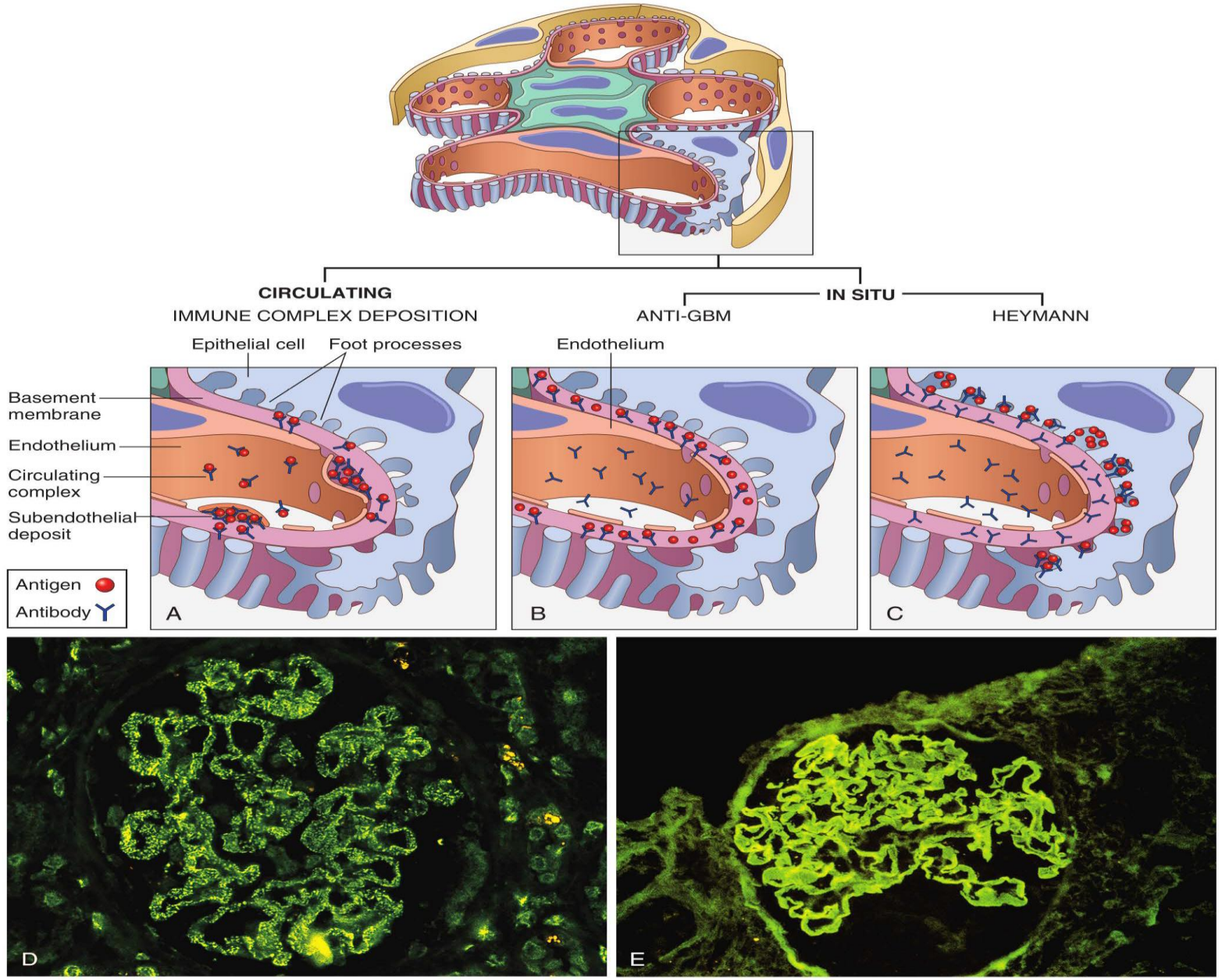


Figure 20-4 Antibody-mediated glomerular injury can result either from the deposition of circulating immune complexes **(A)** or, more commonly, from in situ formation of complexes exemplified by anti-GBM disease **(B)** or Heymann nephritis **(C). D** and **E,** Two patterns of deposition of immune complexes as seen by immunofluorescence microscopy: granular, characteristic of circulating and in situ immune complex nephritis **(D),** and linear, characteristic of classic anti-GBM disease **(E).**

Pathogenesis-cont

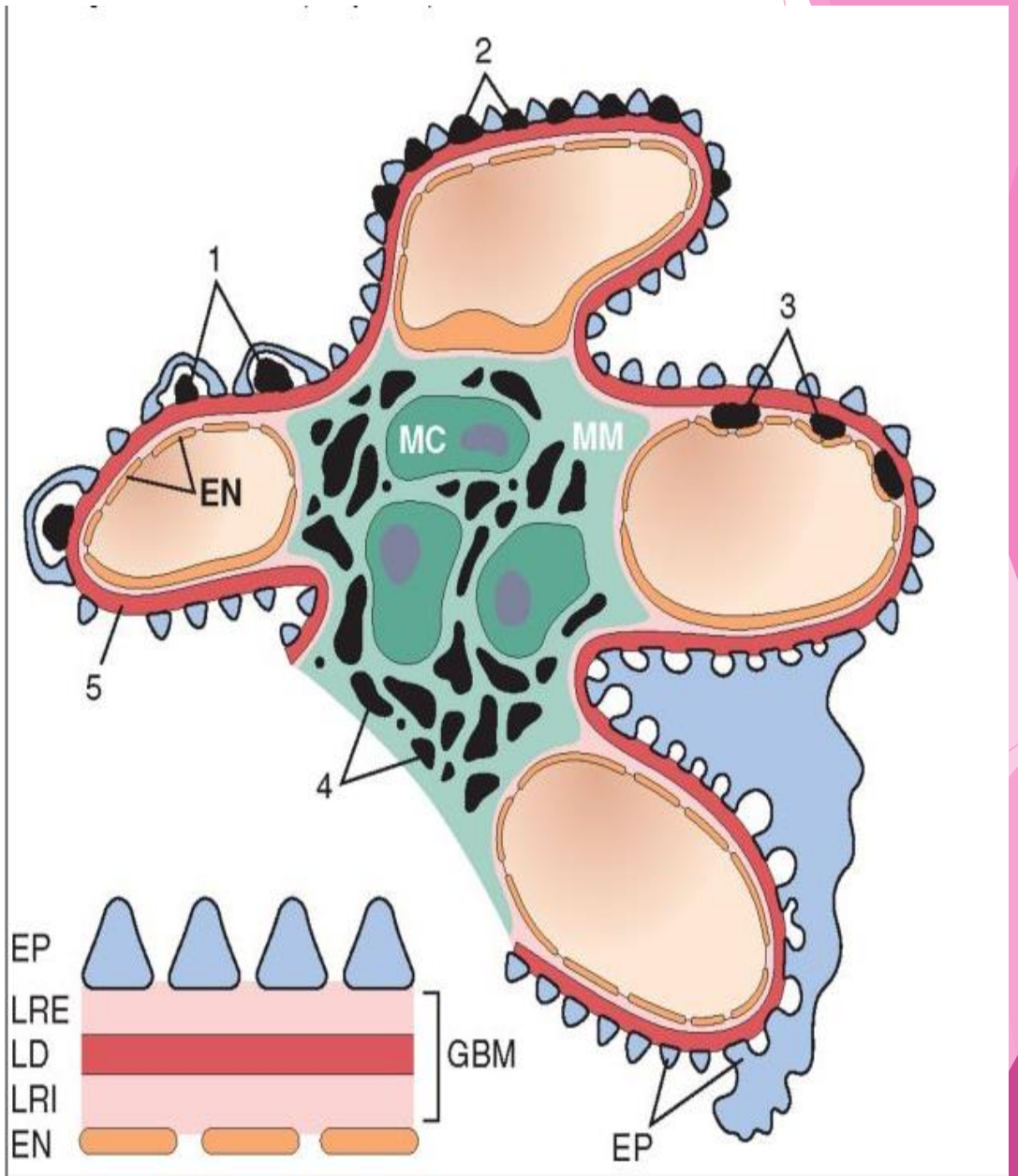
2. Cell-mediated. T cell-mediated injury may account for some cases of glomerulonephritis (GN)

3. Activation of alternative complement pathway.

Localization of immune complexes in the Glomerulus

1. Subepithelial humps,
2. Epimembranous deposits,
3. Subendothelial deposits,
4. Mesangial deposits,
5. Basement membrane.

- EN, endothelium; EP, epithelium; LD, lamina densa; LRE, lamina rara externa; LRI, lamina rara interna; MC, mesangial cell; MM, mesangial matrix.



GLOMERULAR DISEASES

- **Primary Glomerulonephritis**
  Acute diffuse proliferative GN
  Rapidly progressive GN
  Membranous GN
  Lipoid nephrosis (minimal change disease)
  Focal segmental glomerulosclerosis
  Membranoproliferative GN
  IgA Nephropathy
  Chronic GN

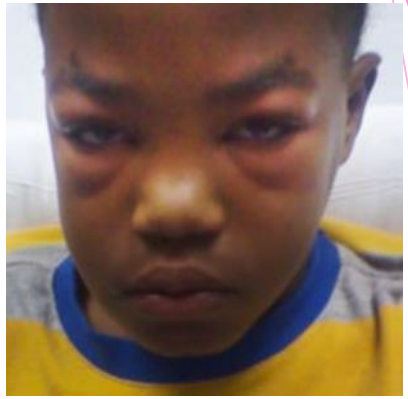
- **Secondary (Systemic) Diseases**
  Systemic lupus erythematosus
  Diabetes mellitus
  Amyloidosis
  Goodpasture's syndrome
  Polyarteritis nodosa
  Wagener's granulomatosis
  Henoch-Scholein purpura
  Bacterial endocarditis

. **Hereditary Disorders**
  Alport's syndrome
  Fabry's disease

Nephritic Syndrome

- Injury ….proliferation of the cells within the glomeruli(endothelium and mesangial cells), accompanied by leukocytic infiltrate…. This inflammatory reaction injures the capillary walls(formation of holes in the basement membrane),…. permitting escape of red cells into the urine, …and induces hemodynamic changes that lead to a ….reduction in the GFR. The reduced GFR
- is manifested clinically by oliguria, fluid retention, and azotemia. Hypertension is the result of both the fluid retention and excessive renin release
- **Clinical Symptoms:**
  - **Oliguria**(due to decreased GFR)
  - **Azotemia** (elevated creatnine and BUN)
  - **Hypertension** (due to retention of salt and excessive renin secretion)
  - **mild-moderate Protinuria** >150mg but <3.5g
  - **BUN/Creatinine** level of >15
  - **Hematuria** – best defined as *red cell casts* and RBC with dysmorphic membranes
    - Commonly defined as “smoky brown urine”

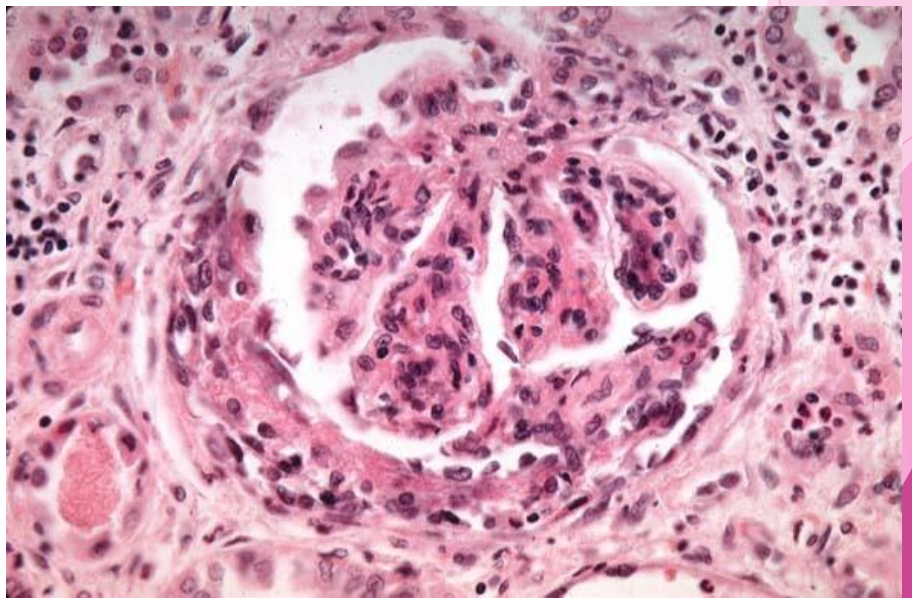
ACUTE GLOMERULONEPHRITIS



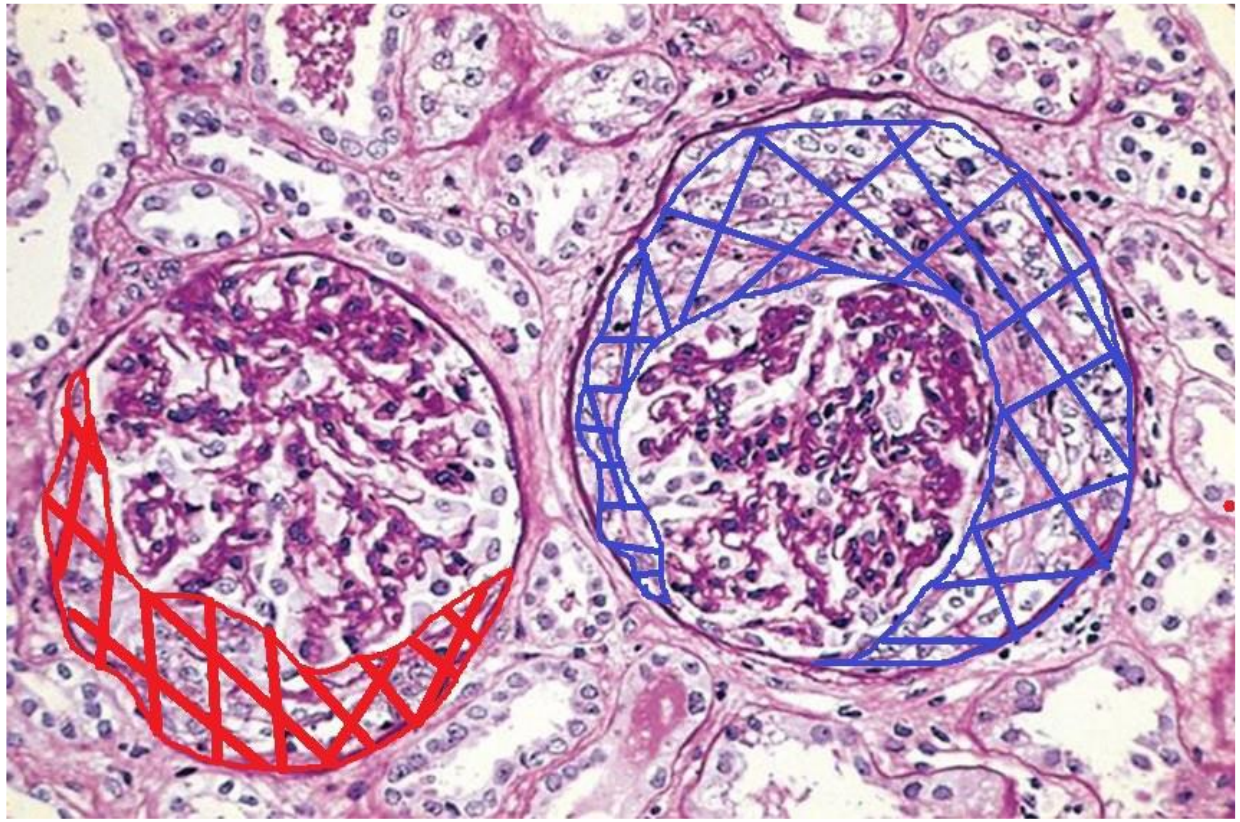
- Nephr**i**tic
- **H**oles in the membrane
- **H**ematuria, **H**ypertension,
- **H**ardly any urin(Oliguria), Azotemia
- in children following a strep infection
- POSTSTREPTOCOCCAL (old term)
- **H**YPERCELLULAR GLOMERULI
- INCREASED ENDOTHELIUM AND MESANGIUM
- IgG, IgM, C3 along GMB FOCALLY(**H**ump)
- 95% full recovery

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

- **Clinical definition, NOT a specific pathologic one**
- **“CRESCENTIC”**
- **Anti-GBM Ab**
- **IMMUN COMPLX**
- **Anti-Neut. Ab**

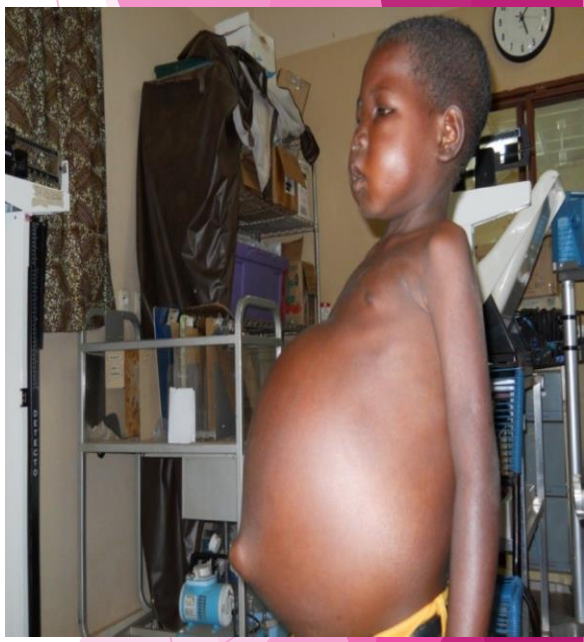


Crescentic glomerulonephritis (PAS stain)



NEPHROTIC SYNDROME

- Syndrome of Glomerular dysfunction that is characterized by *increased loss of proteins in the urine* due to increased basement membrane permeability
- **CLINICAL MANIFESTATIONS**
- Massive proteinuria without hematuria [>3.5g/ day]
- Hypoalbuminemia [<3g/dl]
- Periorbital edema then …
- Generalized edema – Due to ↓'d plasma oncotic pressure
- Hyperlipiduria and Oval Fat Bodies
- Hyperlidemia and Hypercholesterolemia – due to loss of lipoproteins and alterations in liver production of lipoproteins
- Increase in Infections due to loss of low weight globulins and complement
- Loss of anticoagulants → hypercoagulable state



NEPHROTIC SYNDROME

- **Only MASSIVE PROTEINURIA**
- **HYPOALBUMINEMIA**
- **EDEMA**
- **LIPIDEMIA/LIPIDURIA**
- **NUMEROUS CAUSES:**
  - **MEMBRANOUS, MINIMAL CHANGE, FOCAL SEGMTL.**
  - **DIABETES, AMYLOID, SLE, DRUGS**

End