Kidney

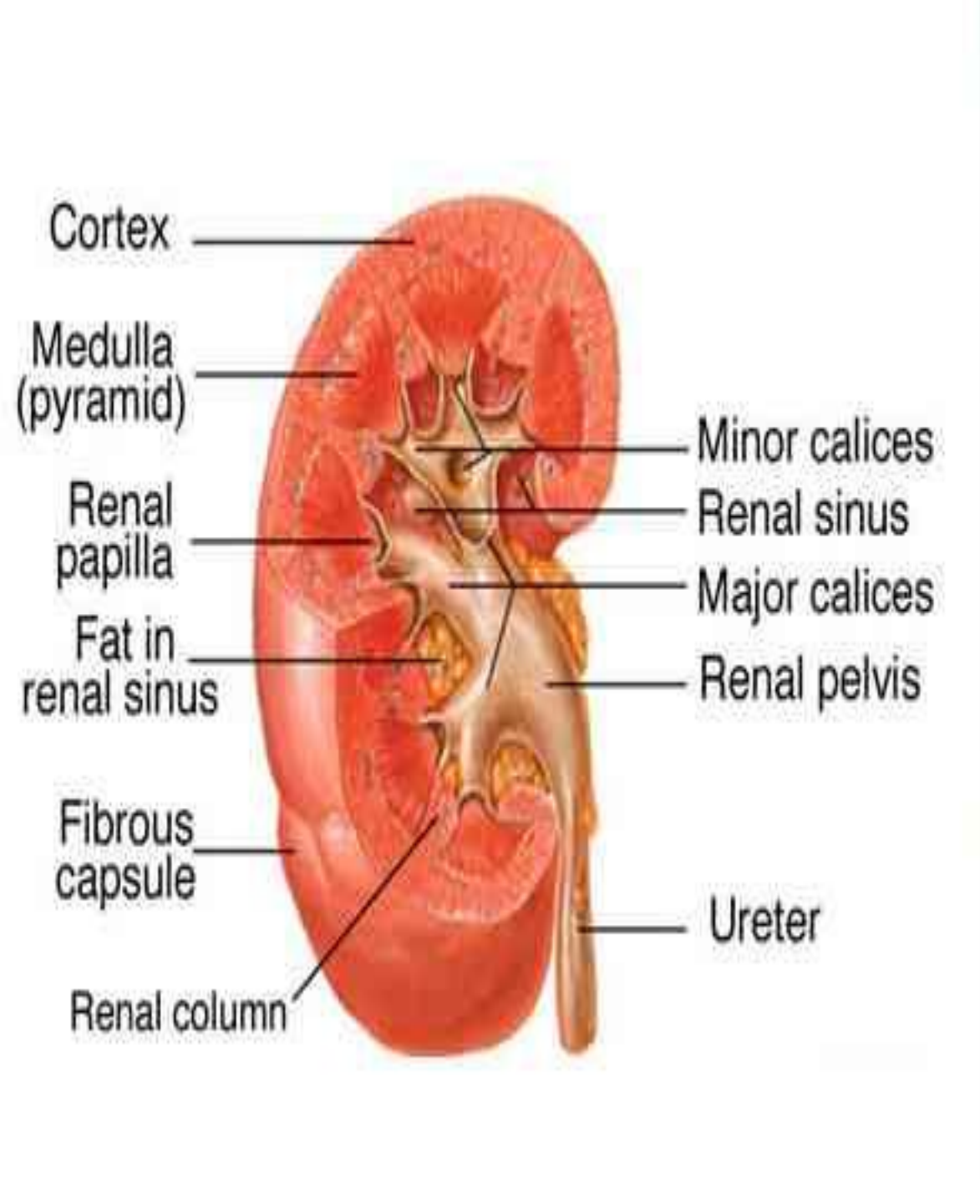
- The kidney is structurally complex organ that has evolved to serve a number of important functions such as excretion of the waste products of metabolism, regulation of body water and salts, maintenance of appropriate acid balance and secretion of a variety of hormones.

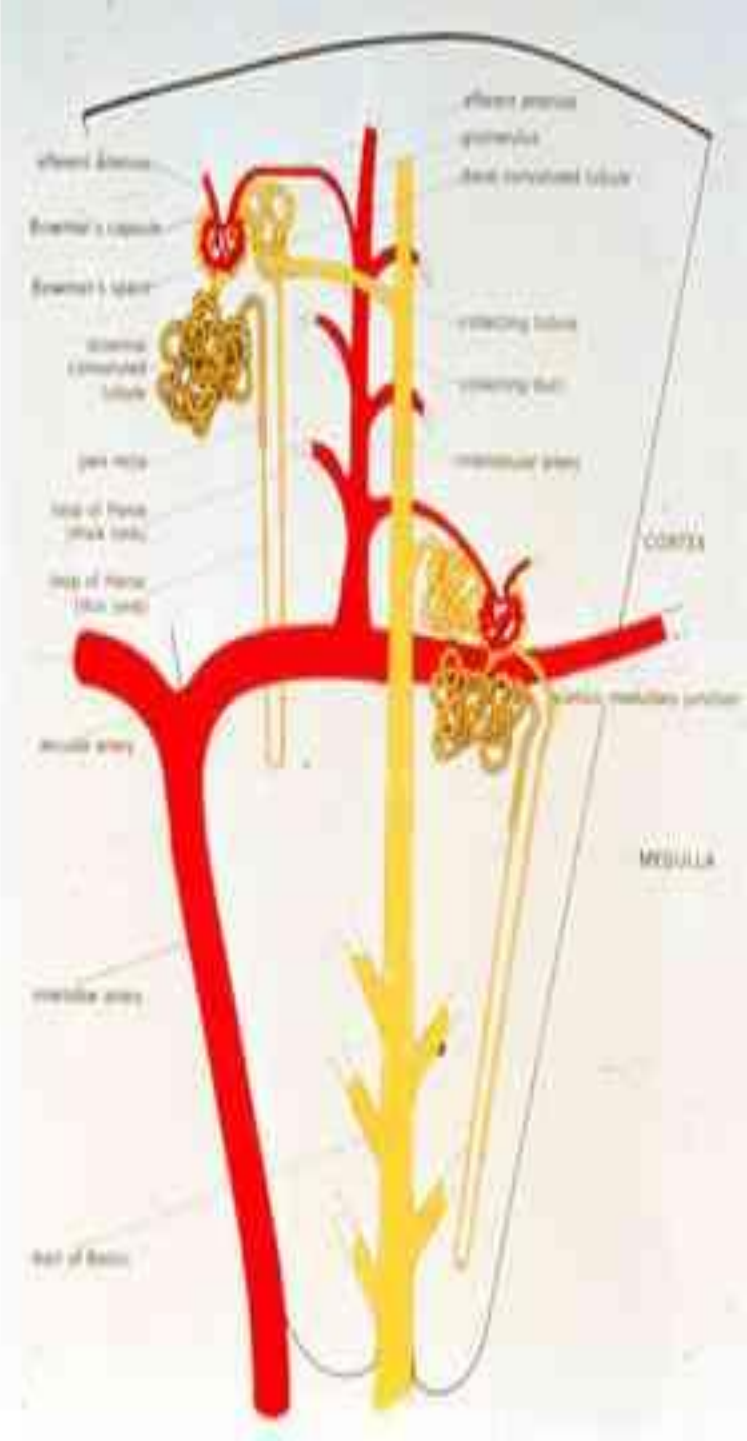
Each kidney is composed of 1-4 million nephrons.

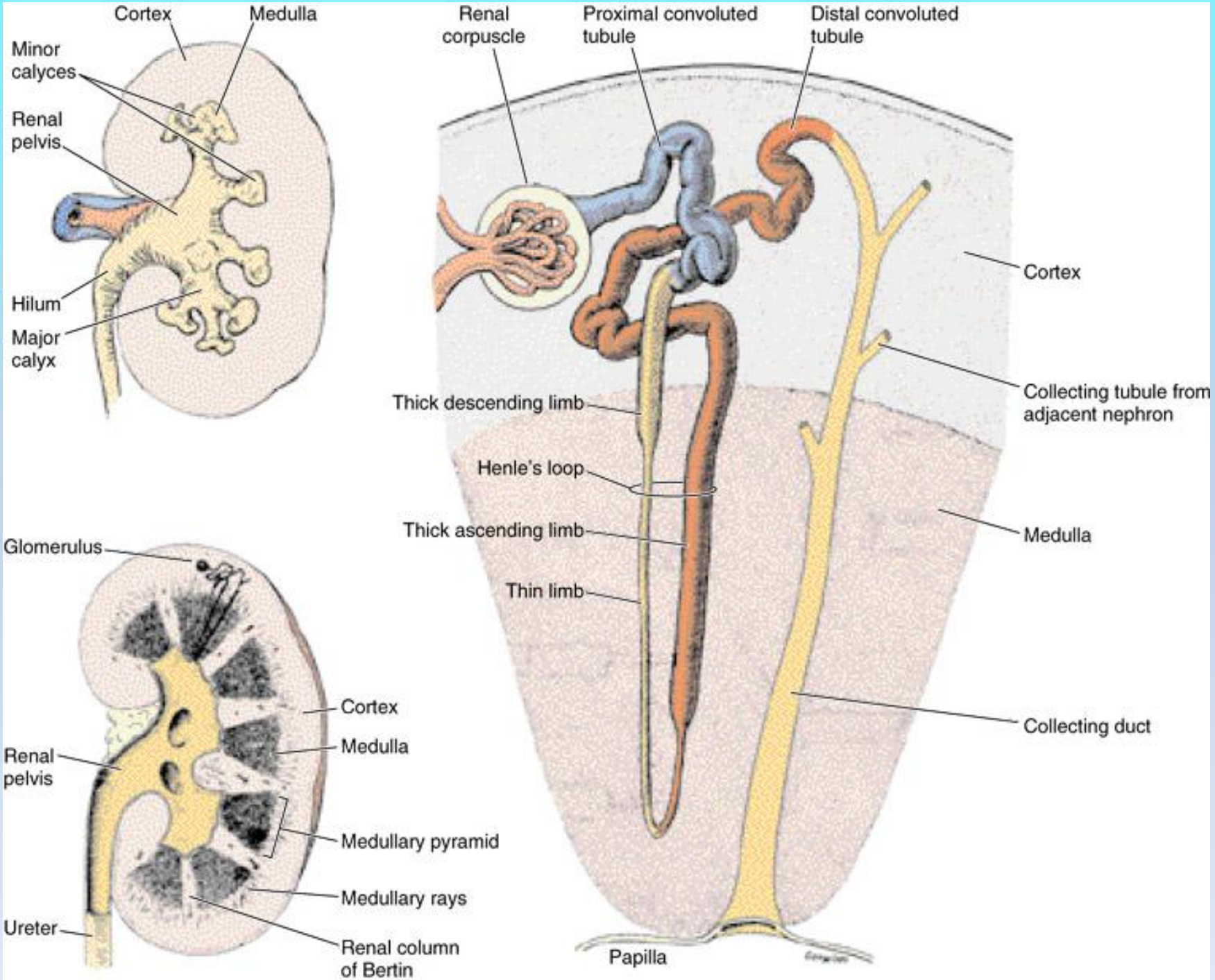
Each nephron is composed of:

- Glumerulus → Capsule (Bowmen's capsule)
  → Capillary tuft

- Tubules (proximal convoluted tubule, loop of Henle, distal convoluted tubule, and collecting tubule).


Cortex
Medulla (pyramid)
Renal papilla
Fat in renal sinus
Fibrous capsule
Renal column
Minor calices
Renal sinus
Major calices
Renal pelvis
Ureter


CORTEX
MEDULLA


Cortex
Medulla
Minor calyces
Renal pelvis
Hilum
Major calyx
Glomerulus
Cortex
Medulla
Renal pelvis
Medullary pyramid
Medullary rays
Ureter
Renal column of Bertin
Renal corpuscle
Proximal convoluted tubule
Distal convoluted tubule
Cortex
Collecting tubule from adjacent nephron
Thick descending limb
Henle's loop
Thick ascending limb
Medulla
Thin limb
Collecting duct
Papilla

Renal Abnormalities

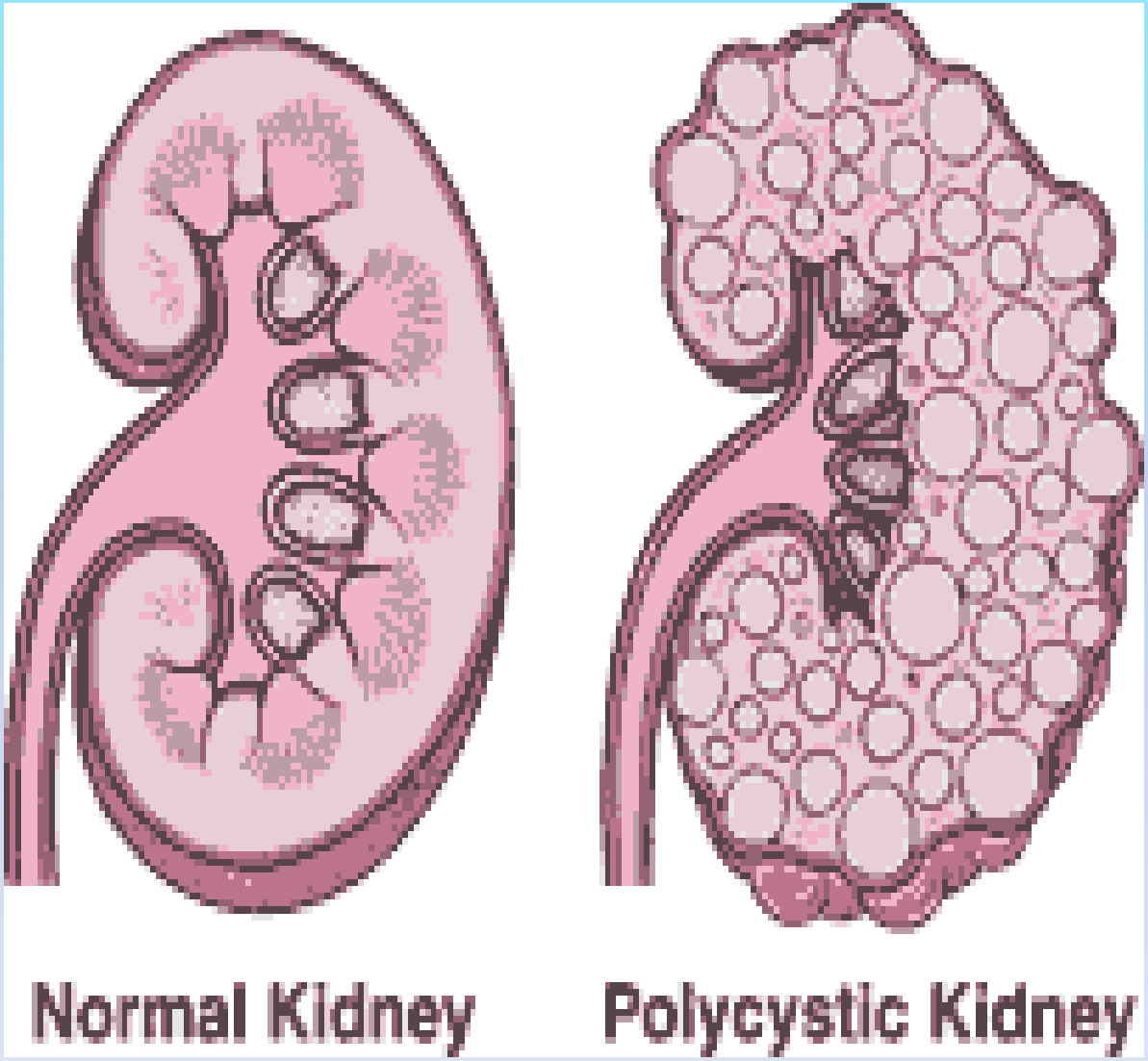
Congenital Abnormalities:

1. Agenesis and Hypoplasia
2. Alteration in kidney position
3. Alteration in kidney Form
4. Polycystic Kidney disease

Polycystic Kidney disease:

- These are a group of kidney disorders characterized by fluid filled sacs or segments that have their origin in the tubular structures of the kidney.

- The cyst may be single or multiple and can vary in size from microscopic to several centimeters. They may arise as a developmental abnormality or be acquired later in life, most from are hereditary.

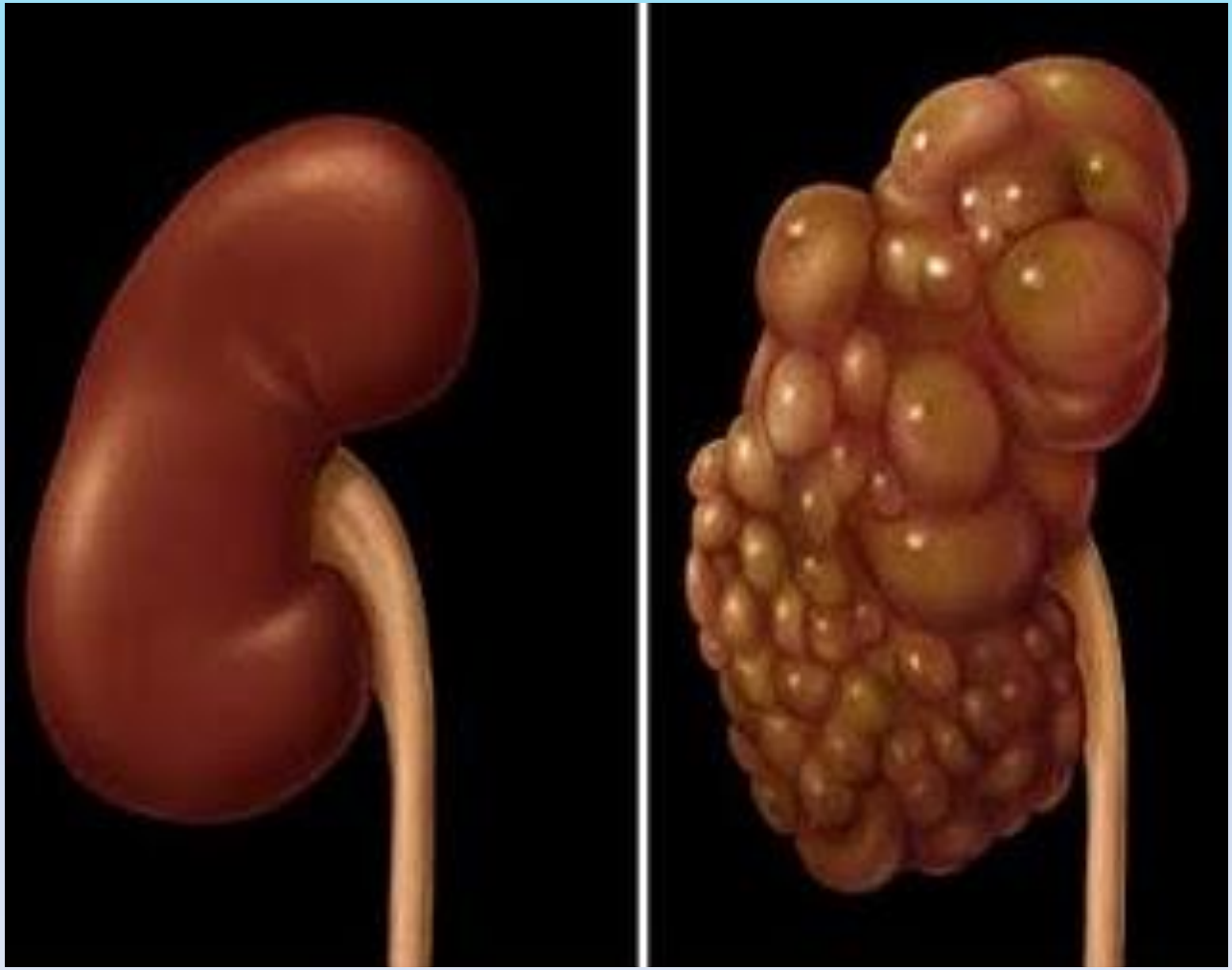

Normal Kidney
Polycystic Kidney

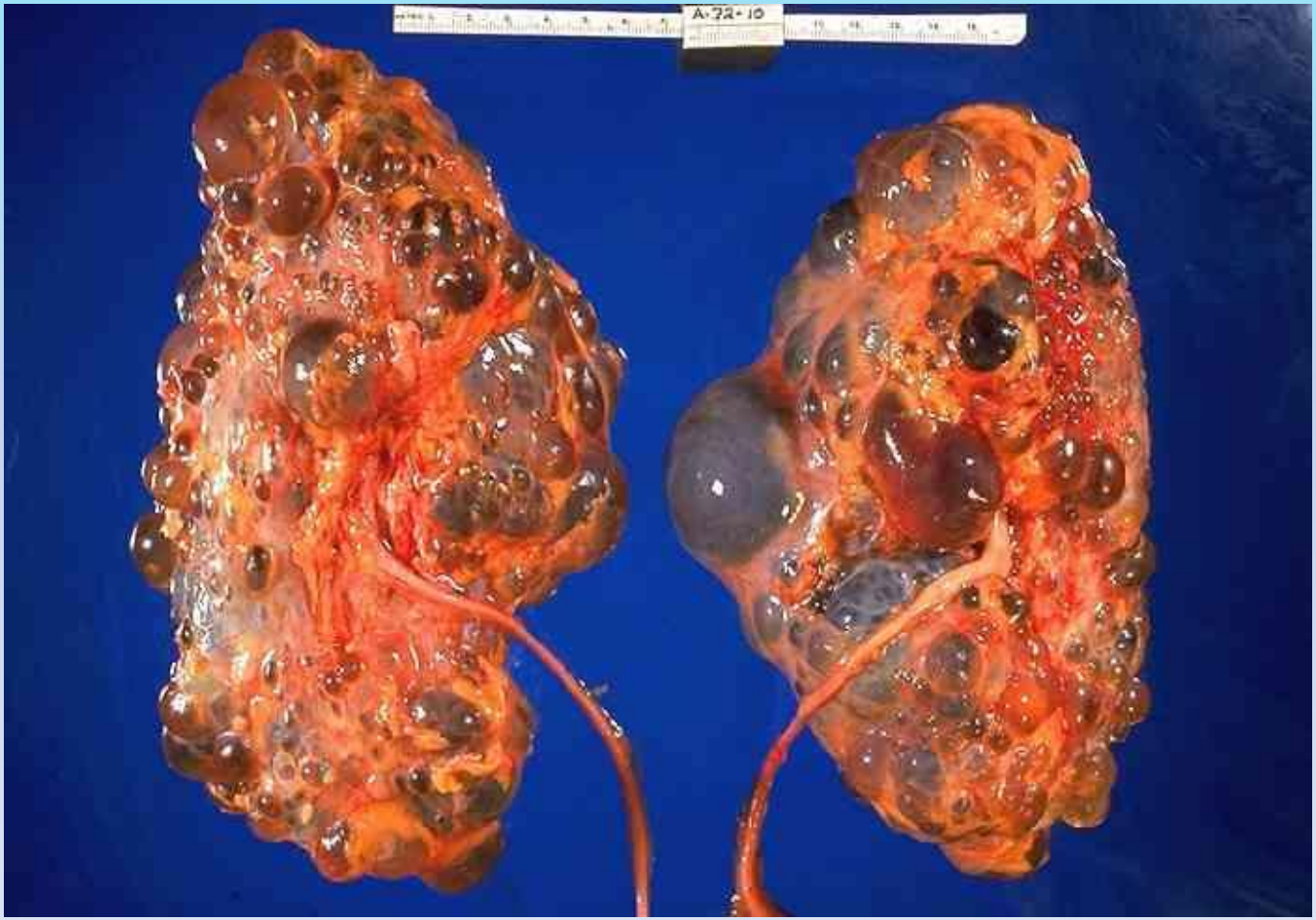
Inherited polycystic Kidney Disease:

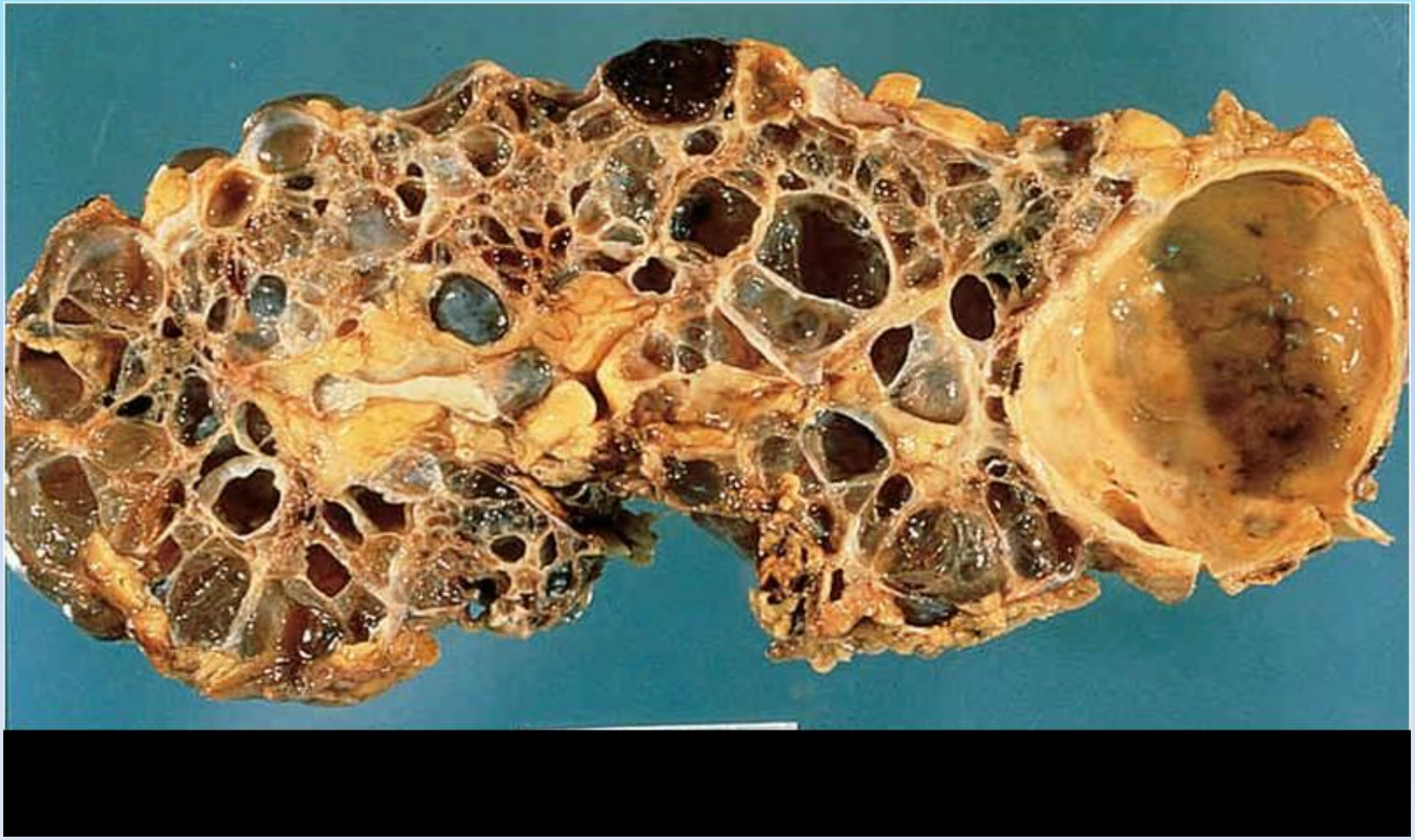
- Mainly there are two types of inherited cystic kidney diseases:

1). **Autosomal Dominant Polycystic Kidney Disease (ADPKD):**

This type appeared in adult and is characterized by numerous fluid filled cysts in the tubular structures of both kidneys with threat of progression to chronic renal failure. The progression of the disease is slow and end stage renal disease is seen mostly after 40 years age.




A-72-10



Clinical Signs:

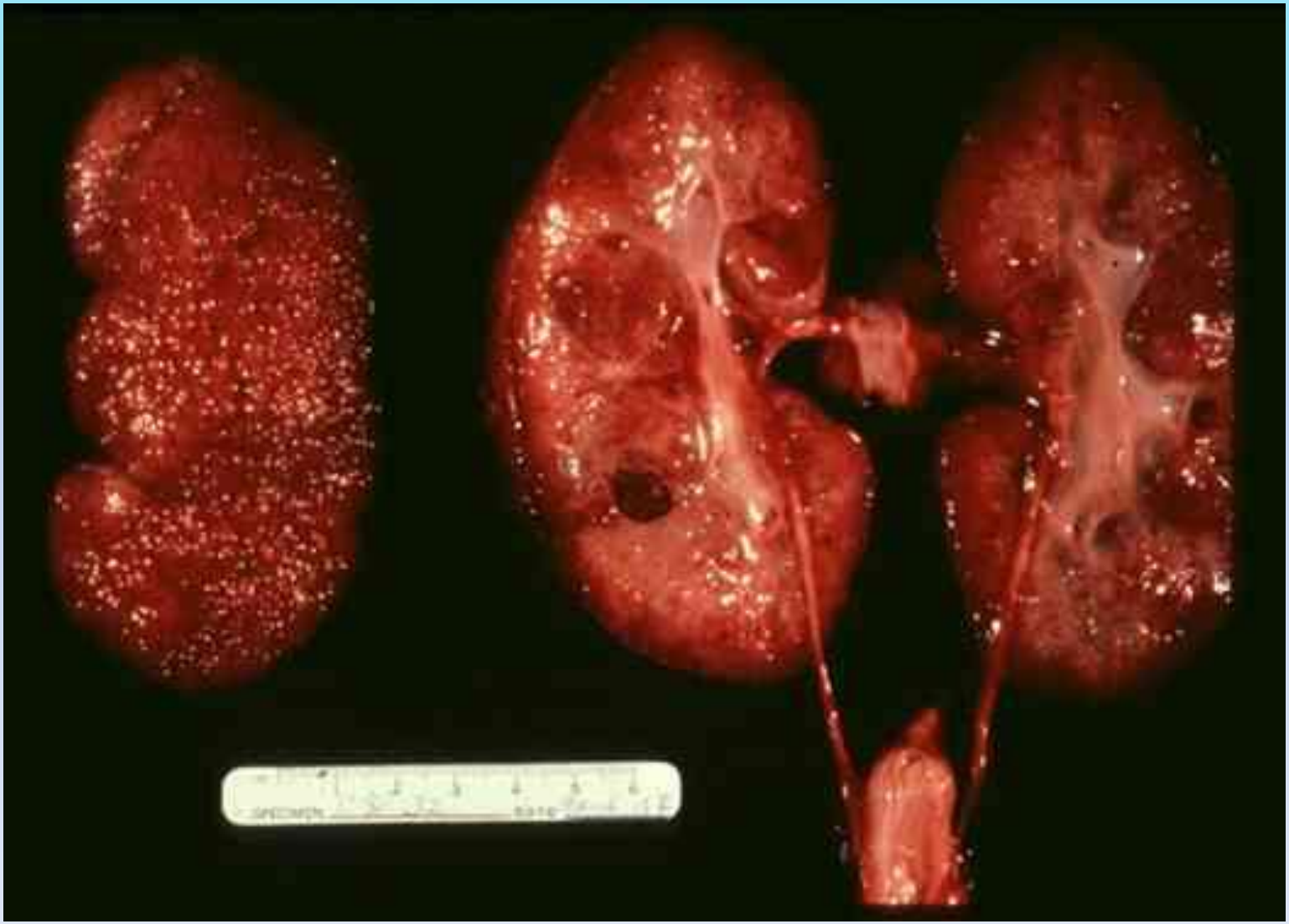
- Pain due to enlarging cyst.
- Ascending urinary tract infection.
- Hypertension due to compression of intra renal blood vessels with activation of the rennin-angiotensin mechanism.
- Nephrolethiasis.

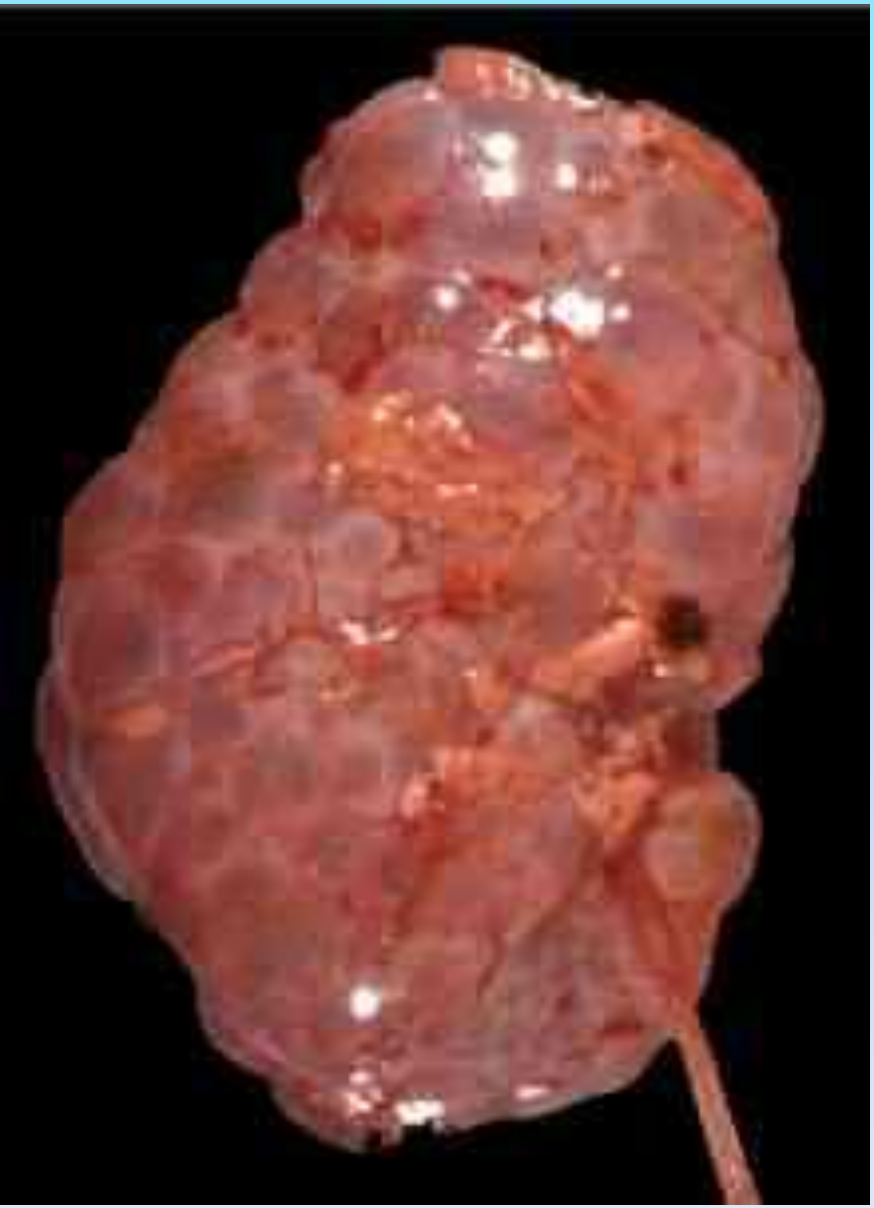
Treatment

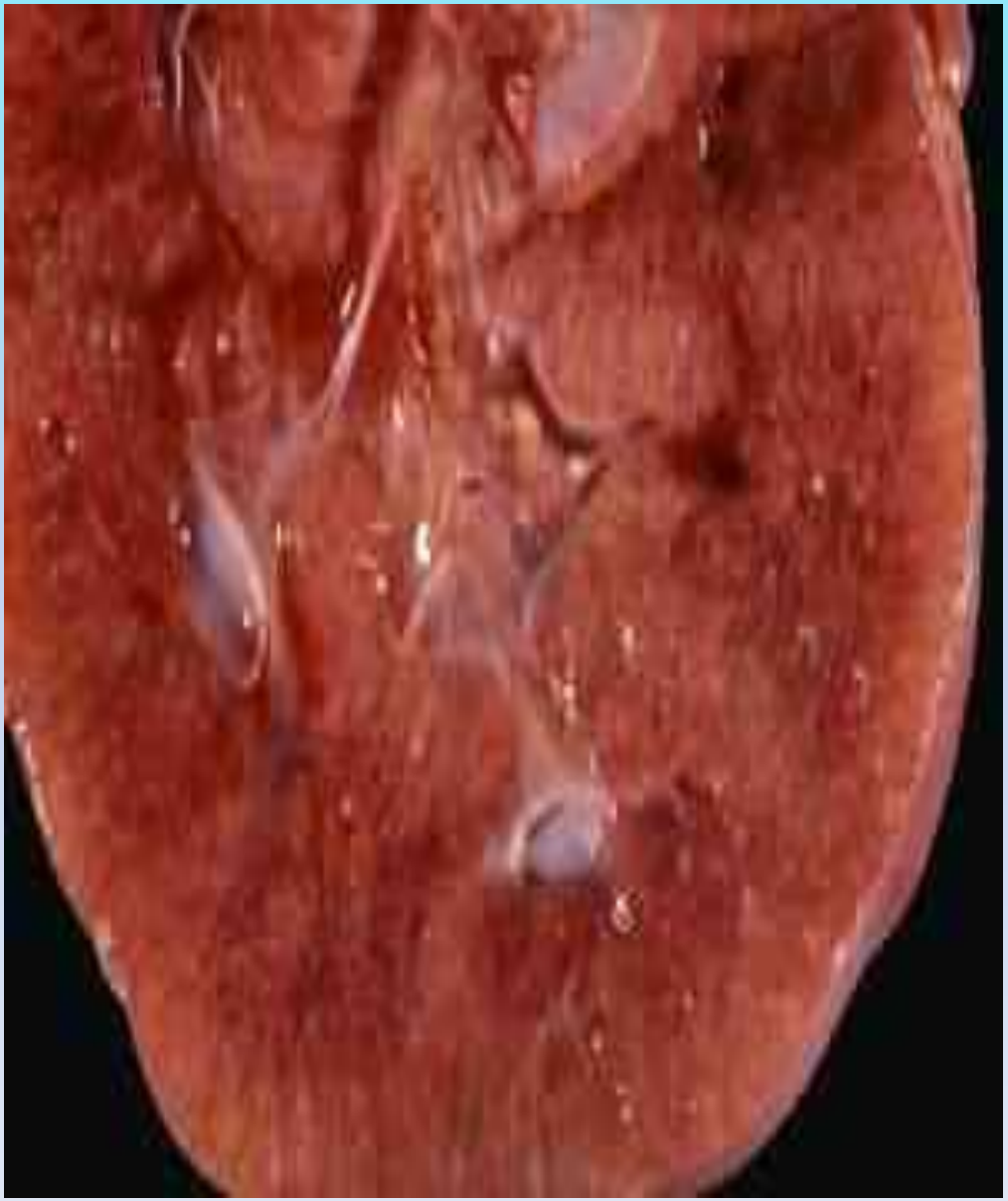
- It is largely supportive and aimed at delaying progression of the disease.

2). <u>Autosomal Recessive Polycystic Kidney Disease (ARPKD):</u>

It is infant disease characterized by cystic dilatation of collecting tubules.







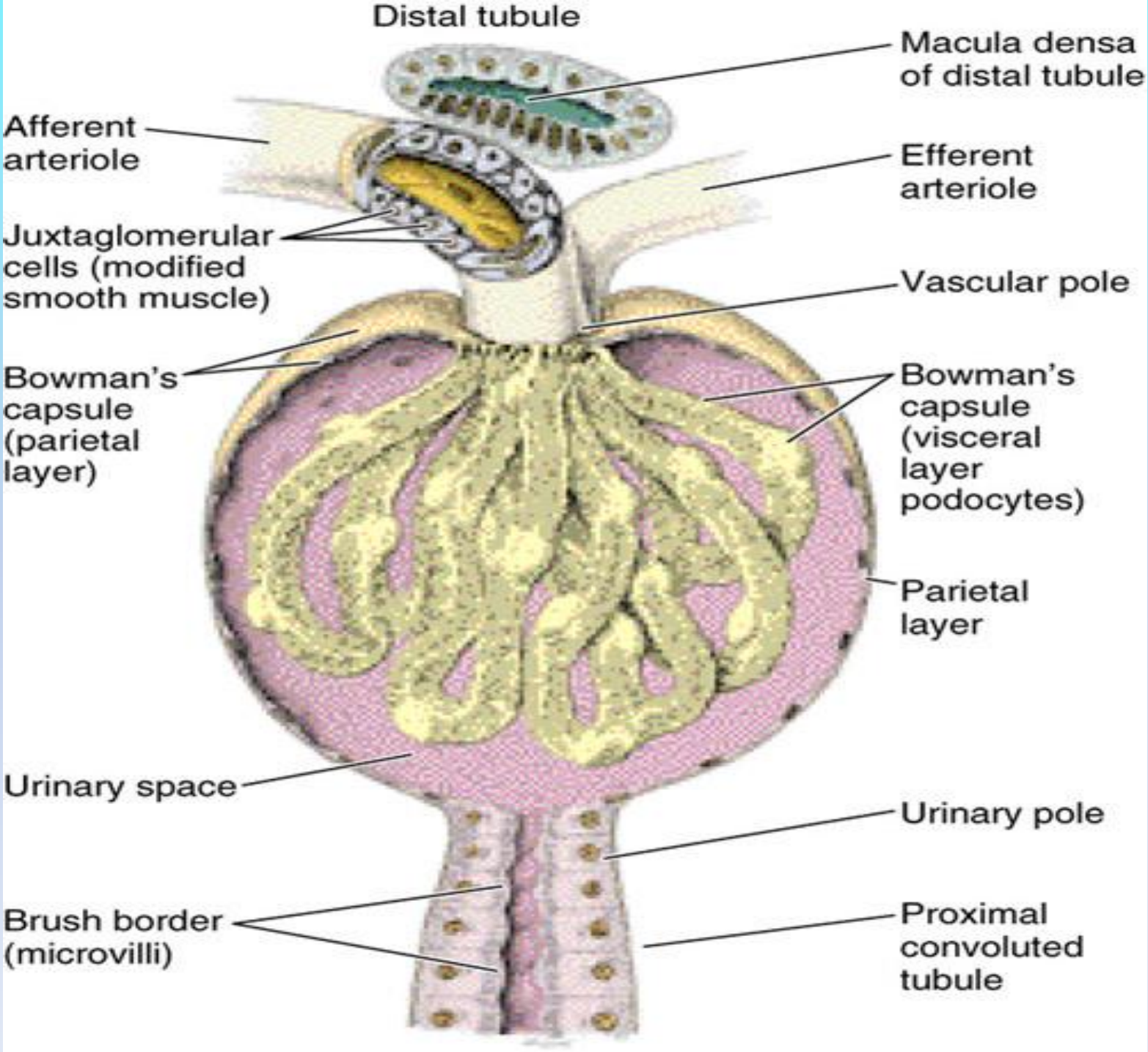
Clinical Signs:

- Bilateral flank masses accompany by sever renal failure.
- Signs of impaired lung development and variable degrees of liver fibrosis and portal hypertension is usually noted within the first few weeks of life.

Glomerular Disorders

- Mean injury to the glomerulus leads to renal disease.

- **Etiology of Glomerular Injury:**
- **Immunologic causes:** Most cases of primary and many cases of secondary glumerular disease probably have an immune origin.


Distal tubule
Macula densa of distal tubule
Afferent arteriole
Efferent arteriole
Juxtaglomerular cells (modified smooth muscle)
Vascular pole
Bowman's capsule (parietal layer)
Bowman's capsule (visceral layer podocytes)
Parietal layer
Urinary space
Urinary pole
Brush border (microvilli)
Proximal convoluted tubule

2. Non immunologic causes, include:

-metabolic disease e.g. diabetes.

-haemodynamic change e.g. hypertension.

-toxic e.g. drug, chemicals.

-stress

3. Hereditary glomerular diseases.

Nephritic Syndrome:

- It is an inflammation of glomeruli characterized by of hematuria (either microscopic or grossly visible), variable degrees of proteinuria, diminish glomerular filtration rate (GFR), oliguria.

- The inflammatory process leads to occlude the capillary lumen and damage the capillary permitting red blood cells to escape into the urine. Also hemodynamic changes produced by inflammation leads to decrease the GFR.

- Decrease GFR lead to hypertension and odema due to enhance the tubular reabsorption of salt and water.
- This type of glomerular disease occurs in many cases like systemic lupus erythrematosus (SLE) and it associated with acute proliferative glomerulonephritis such as postinfectious glomerular nephritis.

Acute Proliferative Glomerulo-nephritis:

- **<u>Acute Postinfectious Glomerulonephritis</u>:** It is an inflammation of glomeruli due to deposit of immune complex in the capillary tuft of the glomeruli leading to proliferation of capillary tuft cells.

- *Causes*: It occurs after infection with Streptococcus (β haeholytic). Also can occurs after infection with Staphylococcus, number of viral agents e.g. mumps, measles and chickenpox.

Clinical Manifestations:

- Occur after 10-15 days from infection with Streptococci, these are; oliguria, proteinuria, hematuria, discoloration of urine and odema of the face and hands with hypertension due to retention of sodium and water.

Nephrotic Syndrome

- It is characterized by massive proteinuria (> 3.5 g/day) and lipiduria associated with hypoalbunimia (< 3g/ 100ml), generalized odema and hyperlipidemia (cholesterol > 300mg/100ml).

- The glomerular membrane acts as a size and charge barrier through which the filtrate must pass. Any increase in permeability allows protein to escape from the plasma into the glomerular. Massive proteinuria results, generalized edema, as in following figure:

- **Glomerular damage**

↓

- **Increase permeability of glomerular capillary to the protein**

↓

Proteinuria (albumin < 3g/ 100ml)

Decrease in oncotic pressure

Compensatory synthesis of proteins (including lipoprotein) by liver.

Decrease plasma volume

Decrease GFR

Fluid escape into tissue

Increase Aldosterone secretion

Fluid retention

Edema

Hyperlipidemia

- Odema appears in many parts like lower extremities, lung, and abdominal cavity. So there is dyspnea due to pulmonary odema and pleural effusions, and there is diaphragmatic compromise due to ascites.
- Hyperlipidemia leads to increase risk for development of atherosclerosis.

Causes:

- The glomerular derangements that occur with nephrosis develop due to:

- 1). Systemic disease such as diabetes mellitus

- 2). Primary glomerular lesion leading to nephrosis such as focal segmental glomerulosclorisis and others.

Diabetic Nephropathy

- Diabetic nephropathy is a major cause of chronic disease and most common cause kidney failure.
- The effect of diabetes mellitus on kidneys as three important lesion these are:

1. Glomerular lesion:

   diabetes mellitus increases the thickness of basement membrane of capillary tuft (glomerulosclerosis).

2. Renal vascular lesions (arteriosclerosis)

3. pyelonephritis.

- Elevation in blood glucose produce increase in glomerular filtration rate (GFR) and increase in glumerular pressure that lead to enlargement of capillary pores by means mediated angiotensin II increase.
- Hypertension and cigarette smoking lead to increase the severity of diabetic nephropathy. Thus control of blood pressure (to level 130/80 mmHg or less) and smoking cessation are recommended in persons with diabetes.

Hypertensive glumerular disease

- Mild to moderate hypertension causes sclerotic changes in renal arterioles and small arteries which is called nephrosclerosis.

- Hypertensive nephropathy is associated with a number of changes in the structure and function of kidney:

1). In kidneys are smaller than the normal and are usually affected bilaterally.

2). There is narrowing of the arterioles and small arteries due to thickening of the blood vessel walls.

3). Blood flow to the nephron decreases causing tubular atrophy, interstitial fibrosis and changes in glomerular structure and function.

Tubular disorders