
بسم الله الرحمن الرحيم

Urinary system

Presented by

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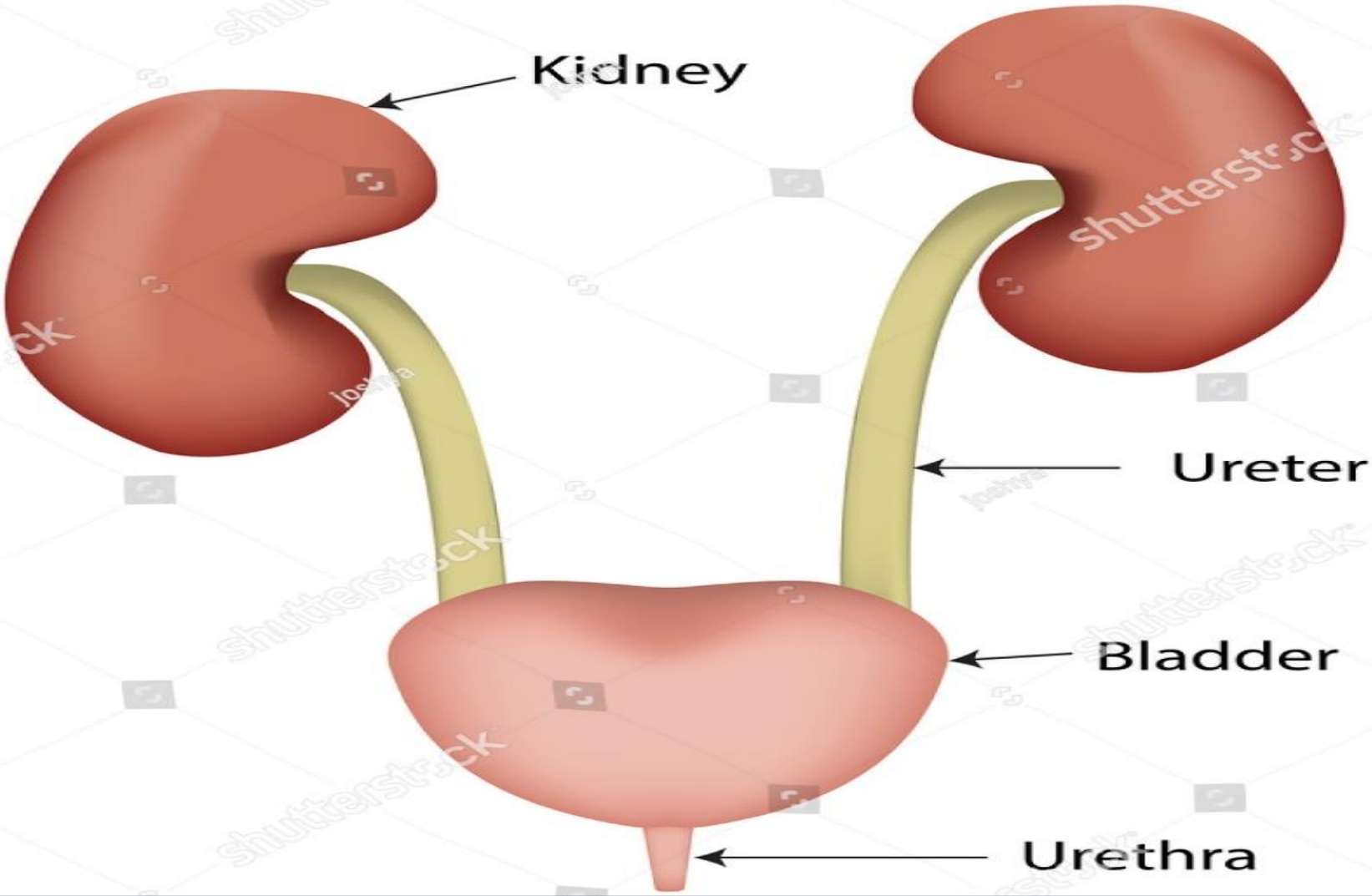
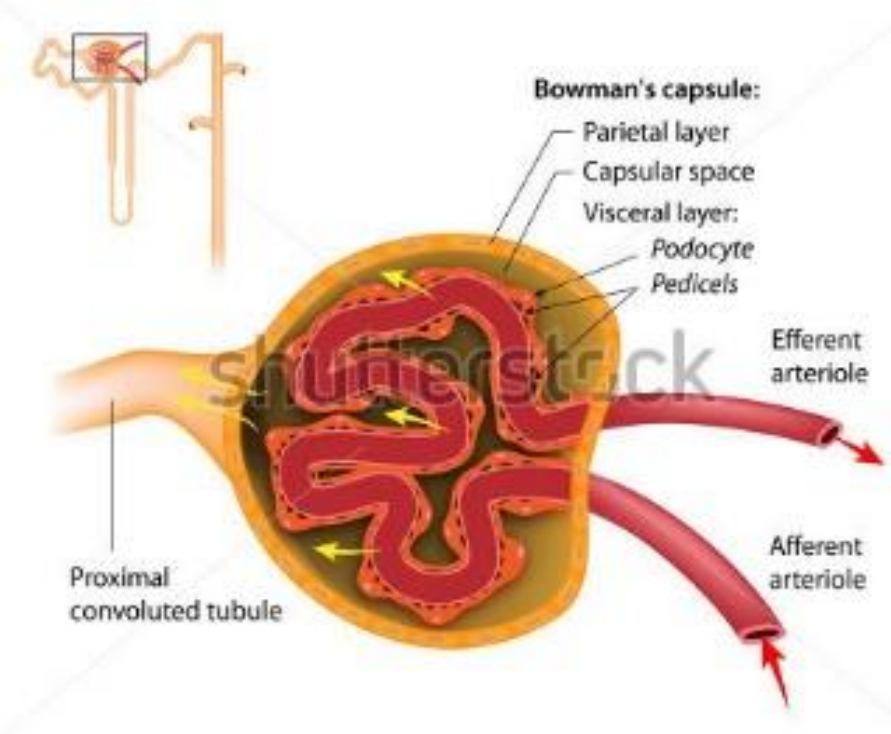
Urinary system



General considerations:

- Urinary system consists of 2 kidneys, 2 ureters, urinary bladder and urethra.
- Kidneys formed from:
- glomeruli
- Proximal & distal convoluted tubules
- loop of henel.



Function of nephron:-

- **Filtration and resorption device**
  - Filtration of blood occurs under influence of special pressure which allows for all crystalloids and prevents colloidal substance.
- ⅓ of volume of blood which push from heart enter kidneys with each blood cycle.

Function of nephron:-

- So, if this blood pressure decrease as in case of **hemorrhage, diarrhea or vomition** → leading to kidneys become unable to perform its function resulting in accumulation of non protein nitrogenous (N.P.N) as **ammonia, creatinine & creatine.**
- Determination of non protein nitrogen (N.P.N) in blood is very important to detect if kidneys is healthy or not.

Injuries to the glomeruli or renal tubules resulting in the following pathological condition:

- **Anurea** → complete absence of urine.
- **Oligurea** → decrease in the amount of urine.
- **Polyurea** → increase in the amount of urine, due to interference with resorption of fluid and electrolytes
- **Albumin urea** → increase in the amount of albumin in urine
- **Haemto-urea** → due to presence of erythrocytes in urine

Abnormalities OF DEVELOPMENT

Renal APLASIA OR AGENESIS

- It is the failure of one or both kidneys to be formed.
- Familial tendency for renal Aplasia has been observed in dogs.

MALPOSITION (ECTOPIC) OF THE KIDNEY:

- The kidneys are seen in abnormal position (most commonly displaced caudally).
- The swine is mostly affected animals and may be associated with Vit A def.

Abnormalities OF DEVELOPMENT

- **HORSESHOE (FUSED) KIDNEY: (normal function)**
  - **It results from a fusion of the anterior or the posterior poles of the kidneys (*like that of a horseshoe*)**

Abnormalities OF DEVELOPMENT

HYPOPLASIA OF THE KIDNEYS

- It is the failure of a kidney to develop to its normal size.
- It is either unilateral or bilateral (in the unilateral there is hypertrophy of other kidney).

Macroscopical Pictures:

- The hypoplastic kidney is smaller than normal (uniform reduction in size).
- The capsule is usually thickened, tense and adhered to the cortex.
- The parenchyma is usually grayish in color and tends to be firm.

Abnormalities OF DEVELOPMENT

Microscopical Pictures:

- There is a reduction in the number of nephrons (evident by fewer glomeruli).
- The remaining nephrons are usually hypertrophic
- The tubules in the medulla are often dilated and their epithelial cells are flattened.
- Interstitial fibrosis which causes separation of the tubules.

Abnormalities OF DEVELOPMENT

RENAL DYSPLASIA:

- It is disorganized development of the renal parenchyma due to lack of differentiation.
- It is either unilateral or bilateral
- Detected mainly in foals

Macroscopical Pictures:

- The affected kidneys are usually smaller than normal. (Misdiagnosis with hypoplasia).
- They are usually misshapen and fibrosed.
- They often contain thick-walled cysts.
- Their ureters are usually tortuous and dilated

Abnormalities OF DEVELOPMENT

- **RENAL DYSPLASIA:**
- **Microscopical Pictures:**
  - Immature tissues (glomeruli and tubules) are evident in adult kidneys.
  - Increased mitosis
  - Production of abnormal cells in varying numbers with tendency to be in abnormal arrangement
- **RENAL Atrophy:** very common
- **RENAL hypertrophy:** very common
- **RENAL hyperplasia:** un-common
- **RENAL metaplasia:** un-common

Abnormalities OF DEVELOPMENT

Renal cyst:

- The renal cysts are divided into
- 1) congenital
- 2) acquired.

The acquired cyst:

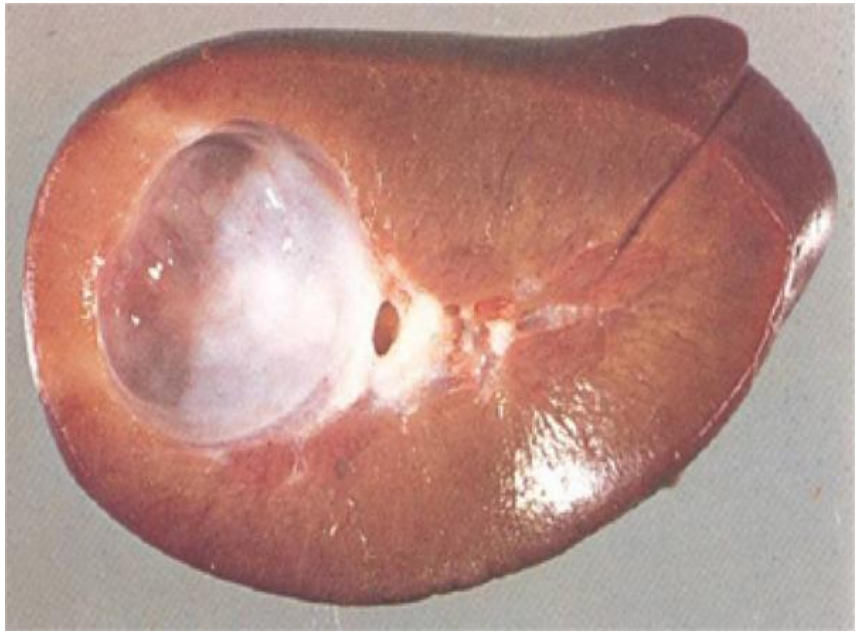
- The acquired cysts usually smaller than congenital cyst
- Arise due to obstruction of the urine flow as in case of disease causing inter-tubular obstruction or interstitial fibrosis

Abnormalities OF DEVELOPMENT

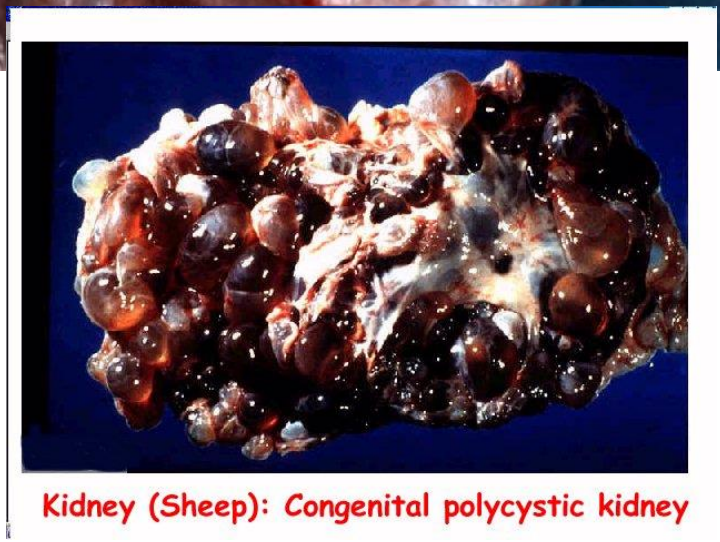
- The congenital cyst includes:
  - solitary large cyst involving the cortex or medulla
  - multiple small cysts (congenital polycystic kidney)
  - where the kidney has a numerous small size cysts containing clear fluid.
  - Cyst usually spherical with thin wall and lined with flattened epithelium
  - In cut section it appears like an irregular honeycomb.
  - described mainly in lambs, dogs and pigs
  - Enlarged cyst compress on the adjacent renal tissue resulting in disturbance in renal function

Abnormalities OF DEVELOPMENT

- The congenital cyst:
  solitary large cyst



polycystic kidney

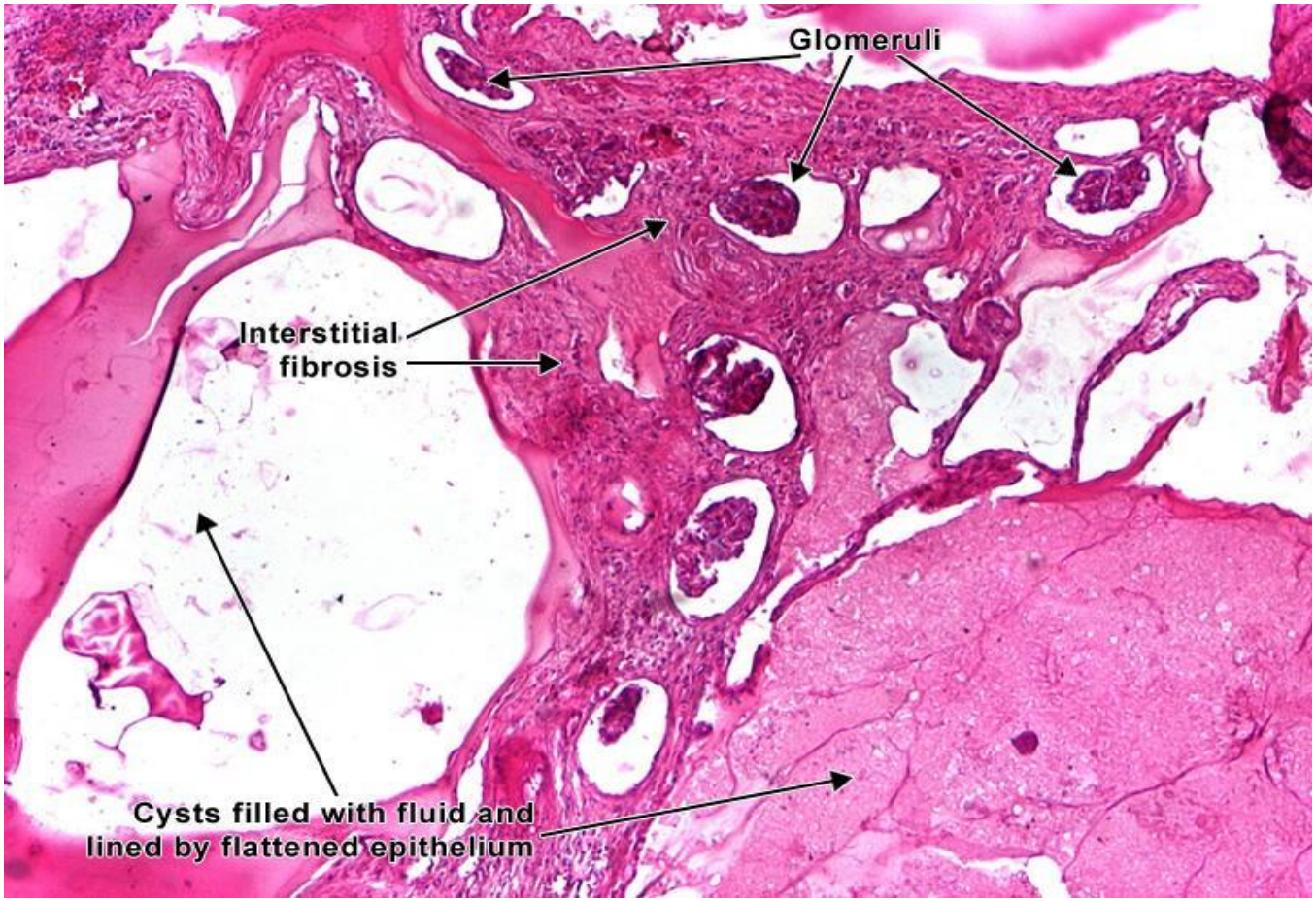


Kidney (Sheep): Congenital polycystic kidney

Abnormalities OF DEVELOPMENT

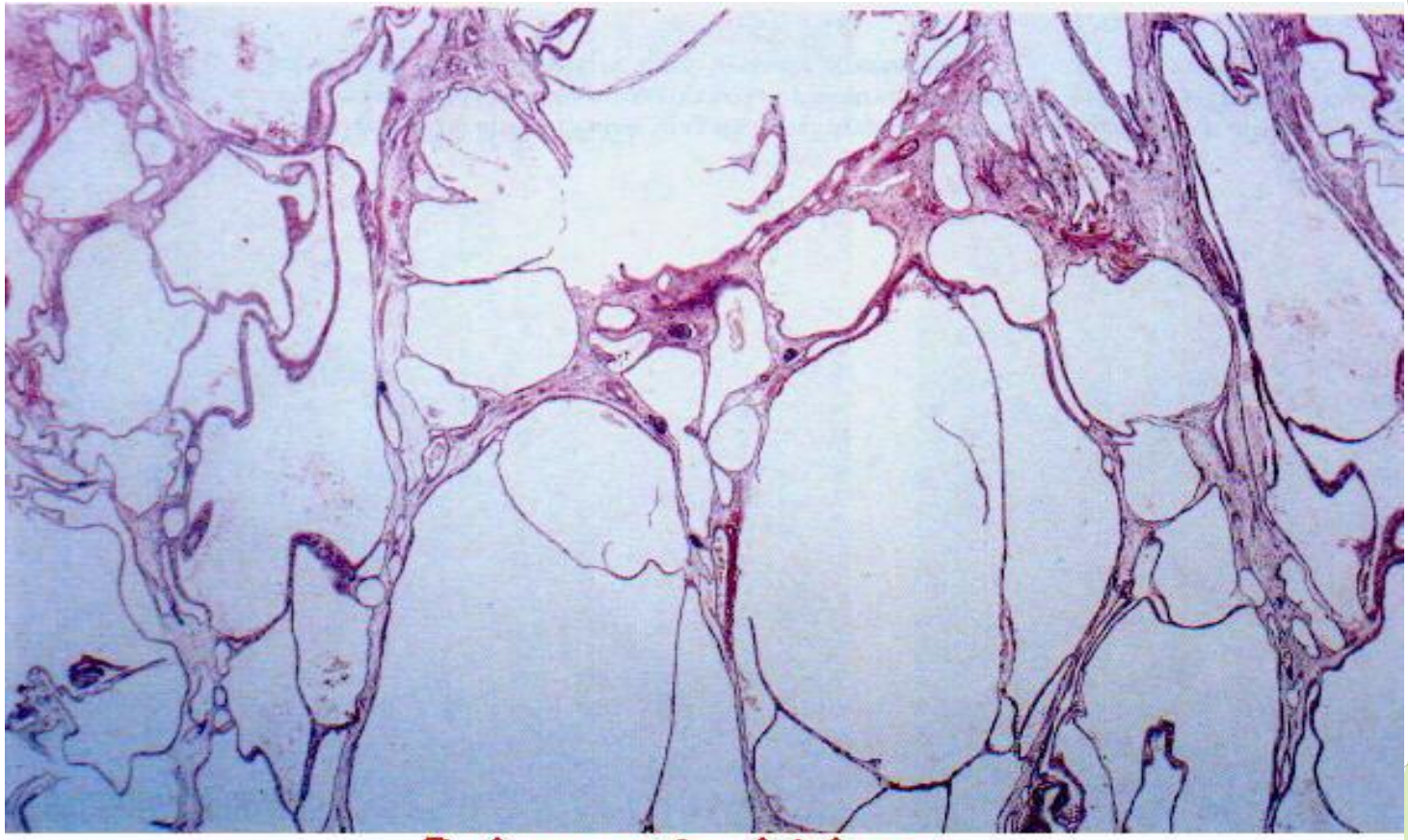
- The congenital cyst:

solitary large cyst polycystic kidney



Abnormalities OF DEVELOPMENT

- The congenital cyst:



Polycystic kidney

numerous large cysts replacing the cortex and medulla

CIRCULATORY DISTURBANCES OF KIDNEY:

RENAL HYPEREMIA:

- **A-Active Hyperemia: associated with inflammation (nephritis).**
- **B-Passive Hyperemia: associated with general venous congestion.**

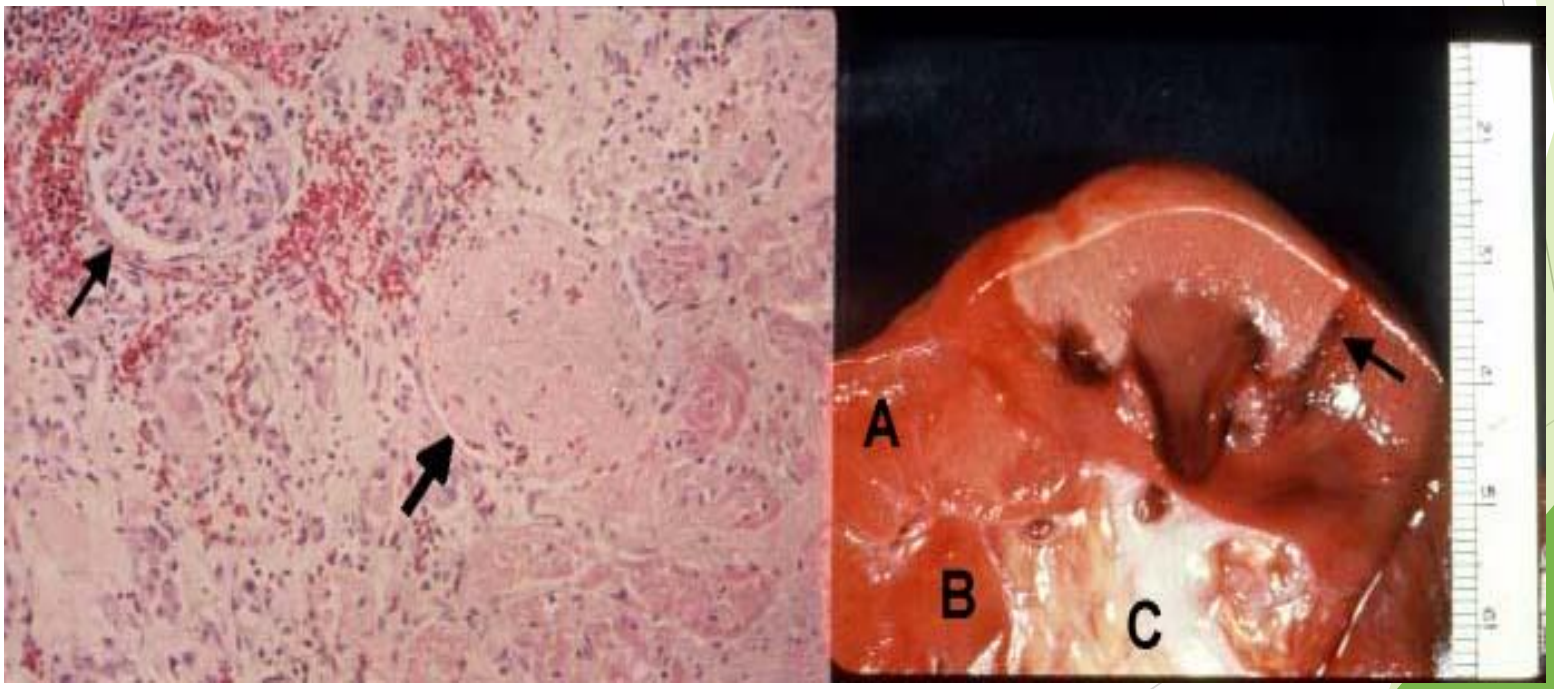
RENAL HEMORRHAGES:

- **Petechial or ecchymotic hemorrhages are seen in cortex and medulla associated with septicemic and some viral diseases.**

CIRCULATORY DISTURBANCES OF KIDNEY:

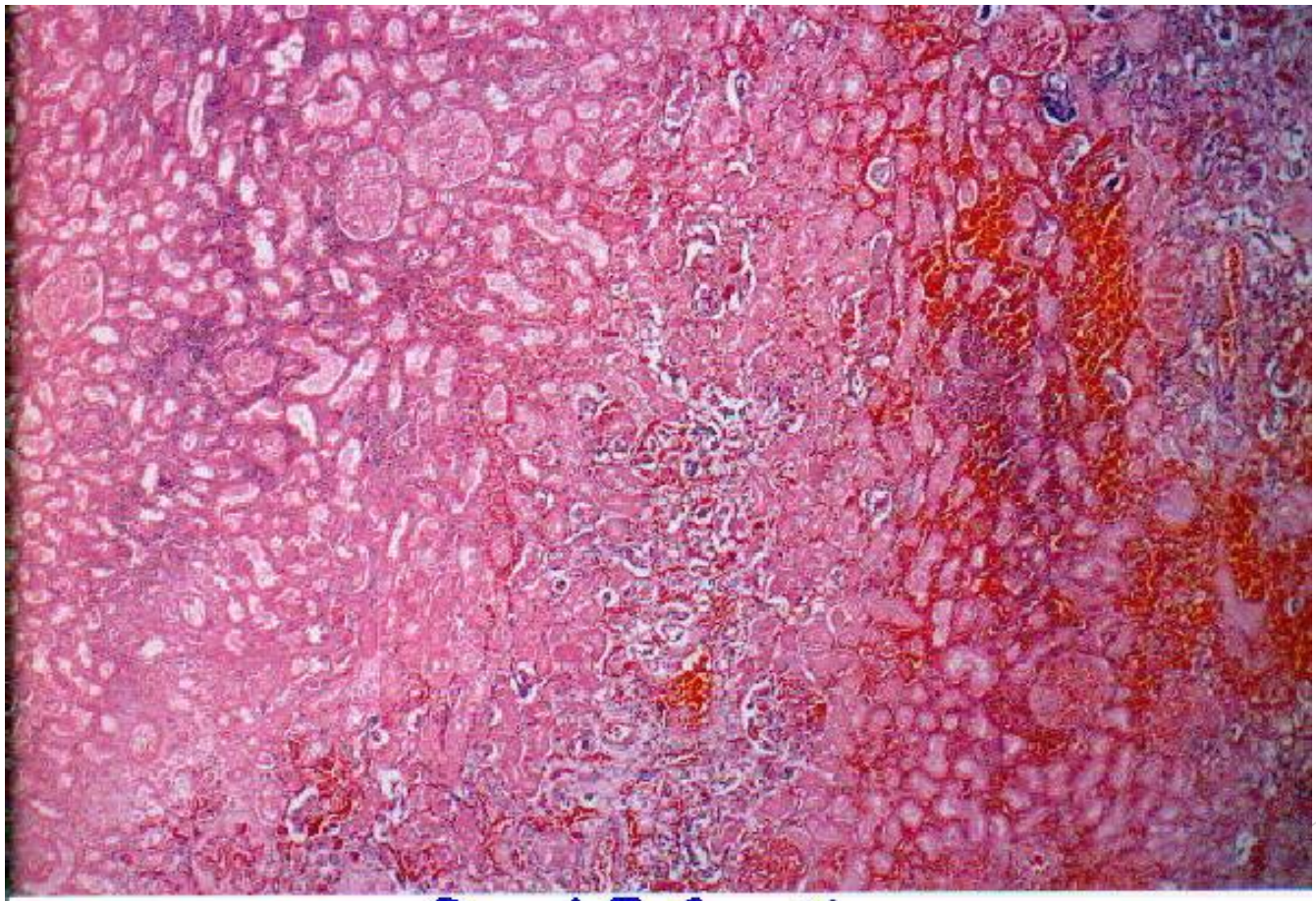
INFARCTION OF THE KIDNEY:

- Appeared as pale or red wedge shape area of ischemic necrosis with the apex near the point of obstruction and the base at the capsule.



CIRCULATORY DISTURBANCES OF KIDNEY:

- INFARCTION OF THE KIDNEY:



Renal Infarction

Area of coagulative necrosis surounded by hyperaemic zone

Degenerations and infiltrations (Nephrosis)

- **Nephrosis:** It is refer to any pathological changes except inflammation
  - If these non inflammatory condition occurred
  - In one kidney, leading to compensatory hypertrophy in second one,

  but if occur in two kidneys, lead to uremia & death.
- Nephrosis is including cloudy swelling, hydropic degeneration, fatty change, amyloid infiltration, gout, glycogen infiltration.
- Moreover, deposition of pigments such as hemosiderosis, melanin, hemoglobin and bile pigments

HYDRONEPHROSIS

- It is gradual accumulation of urine inside the renal pelvis which extend gradually to the renal parenchyma.

Causes:

- Incomplete (partial) or intermittent obstruction of the following:
- i-One or two ureters by calculi.
- ii-Urinary bladder by stones or tumors.
- iii-Urethera by enlarged prostate or stones.
- Cystitis
- Cystic or polycystic kidney

Pathogenesis:

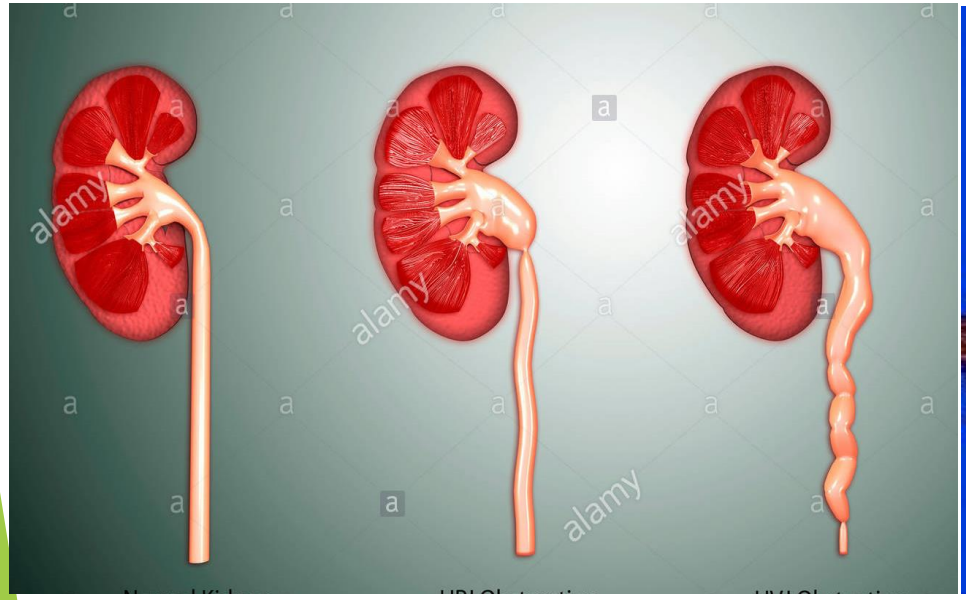
- Hydronephrosis can be unilateral or bilateral depend on the place of obstruction.
- Unilateral usually results from obstruction of ureters throughout its length.
- Bilateral is associated with both ureters, urinary bladder or urethral obstruction
- Increase intrapelvic pressure due to obstruction lead to dilatation of pelvis and calyces and blunting of renal papilla.
- Then leading to accumulation of fluid in renal tubules resulting in Increase intratubular pressure leading to microscopic tubular dilatation besides flattening and necrosis of tubular epithelium.

Pathogenesis:

- Increased in the intra-pelvic pressure lead to collapse of the interstitial vessels and renal flow decreased results in hypoxia and degeneration of renal epithelium.
- Much of glomerular filtrate diffuse into the interstitium which initiate hyperplasia of interstitial fibrous tissue
- The glomeruli remain functional for prolonged period but finally they atrophied and sclerotic.
- In most advanced case the kidney appears as thin walled fluid filled sac. If the bacterial contamination occurs, the kidney appear as a sac filled with pus (pyonephrosis)

Macroscopical Pictures

- The renal pelvis is distended with urine (early stage).
- The kidney become sac-like filled with urine (late stage).
- Tiny cysts appear in the medulla.
- Necrosis or ulceration of the wall of the renal pelvis.



Microscopical Pictures:

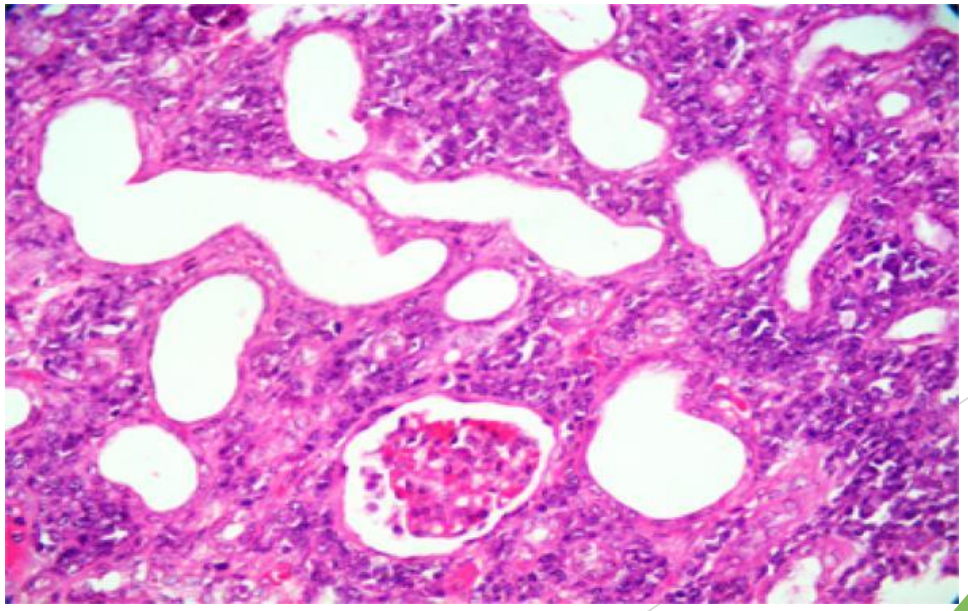
i-Atrophy of renal tubular epithelium.

ii-The glomeruli are compressed or collapsed.

iii-Fibrous connective tissue proliferation (may be hyalinized).

iv-Cystic dilation of the renal tubules.

V-Few lymphocytes infiltrations with plasma cells an macrophages



Fate of hydronephrosis:

- **i-Bilateral** Uremia Death.
- **ii-Unilateral** Hydronephrosis and hypertrophy of the other.

Uremia (Azotemia, Renal failure)

- Kidney fail to perform its normal function
- It is a pathological condition in which blood urea and harmful waste product (NPN), which are normally eliminated in the urine as urea, creatinine, uric acid and ammonia. remain in circulating blood, as a result of compete or partial failure of urinary excretion.

Uremia (Azotemia, Renal failure)

Uremia may be

- **(A) Acute (intra-renal) → e.g in acute glomerulonephritis e.g hypercellularity of endothelial lining of bl.vs (tuft) & podocytes within glomerular tuft enlarged glomerulus become avascular & filled with large number of lymphocytes.**

- **(B) Chronic (extra-renal) → factors that reduce the renal blood flow as in case of circulatory collapse, shock (severe loss of blood), or as in chronic inflammation of the kidneys.**

Causes of Uremia (Azotemia, Renal failure)

[A] Extra renal

- Ischemia → hydrostatic pressures not enough.
- Shock → (severe loss of blood), as sudden dilation of mesenteric blood vessels. So, filled with blood & blood not reach to brain & kidneys leading to Ischemia of kidney.
- Extensive hemorrhage.
- Severe diarrhea & vomition:-resulting in reduction in the volume of blood due to:-
- excessive fluid loss excreted & fluid draw from cells or tissues then from blood lead to dehydration → decrease hydrostatic pressure so, no infiltration & no urination.

Causes of Uremia (Azotemia, Renal failure)

[B] Post renal:-

- Including obstruction of urine outflow via lower urinary tract resulting in retention of constituents in the plasma that are normally removed by kidney

(1)-Complete obstruction of both ureters →

- which lead to atrophy of the kidneys.

(2)- Partial obstruction of both urters →

- Resulting in hydronephrosis as in case of stones which lead to collection of fluid in renal pelvis causing pressure atrophy on renal parenchyma leading to changes without inflammation. It appears as a sac contain fluid.

[C] Renal causes:-

- As any inflammation of kidneys

Pathogenesis of Uremia

- The lesions in uremia could be attributed to vascular degeneration and necrosis due to toxic effect of N.P.N. resulting in increased vascular permeability, thrombosis, and infarction
- Also, high concentration of ammonia leads to ulceration of mucous membrane

Symptoms of uremia

1. The animals not drink or eat
2. Vomiting.
3. Oligouria.
4. Skin dry rough and less elastic basal mucous membrane resulted in uremic stomatitis
5. urinferous odour represented by foul smelling of oral cavity especially in man and dog.

Gross lesions of uremia

- Lesions are dependent on the duration of the uremic state

- congestion of the mucous membrane.
- Urinous bad odor in skin, the buccal cavity because the excretory function of the kidney diminish the other excretory organ take a part in excreting the urea as intestine, skin, sweat gland then uremia result.
- Inflammation of the mucous membrane of the alimentary tract sometime with ulcer formation. Hemorrhagic gastritis and enteritis.
- Oral mucosa showing ulcerative and necrotic stomatitis

Gross lesions of uremia

- Ulcers are commonly present on the underside of the tongue
- Ulceration and hemorrhage in the stomach and colon
- Associated with mucoid material that adhere to the mucus membrane
- Fibrinous pericarditis or hydropericardium
- Pulmonary edema
- **Ascites**
- **Edematous limb.**
- **Ostermalacia: Softening of bone (known as renal rickets),** as uremia is associated with Ca-deficiency so, Ca. moved from bone lead to Osteomalacia.

Microscopic appearance of uremia

- In early stages → hyperemia in blood vessels.
- degenerative changes in the parenchymatous organs
- Then coagulative necrosis and ulceration in digestive system associated with leukocytic cellular infiltration mainly neutrophils
- Pulmonary edema in which alveoli are with inflammatory edematous fluid that mixed with fibrin rich exudates with leukocytic infiltration mainly neutrophils and macrophages.

❑ In acute (extra-renal) → kidneys is healthy & cause from outside kidney while (intra- renal) → kidneys is affected.

Microscopic appearance of uremia

- **Urea** has irritating effect cause hyperemia in all internal organs. In prolonged uremia, toxic injury of the bone marrow may occur resulting in Toxic aplastic anemia.

- **Mineralization:** alteration in plasma ion concentrations mainly in calcium, potassium and phosphate

❑ **Death from renal failure was attributed to:**

- **Cardiotoxicity** due to increase in the level of serum potassium
- **Pulmonary edema**
- **Metabolic acidosis** that resulted from reduction in the level of blood pH

Nephritis

- It is the inflammation of the kidneys.
- Nephritis can be classified according to:

1. Route of infection into:

- **A). Hematogenic (descending)**
- **b. Urinogenic (ascending)**

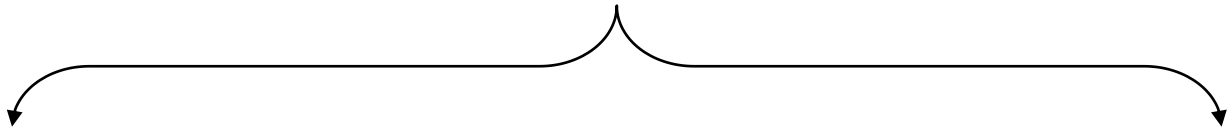
2. Anatomically into:

a. Glomerulonephritis

b. Interstitial nephritis

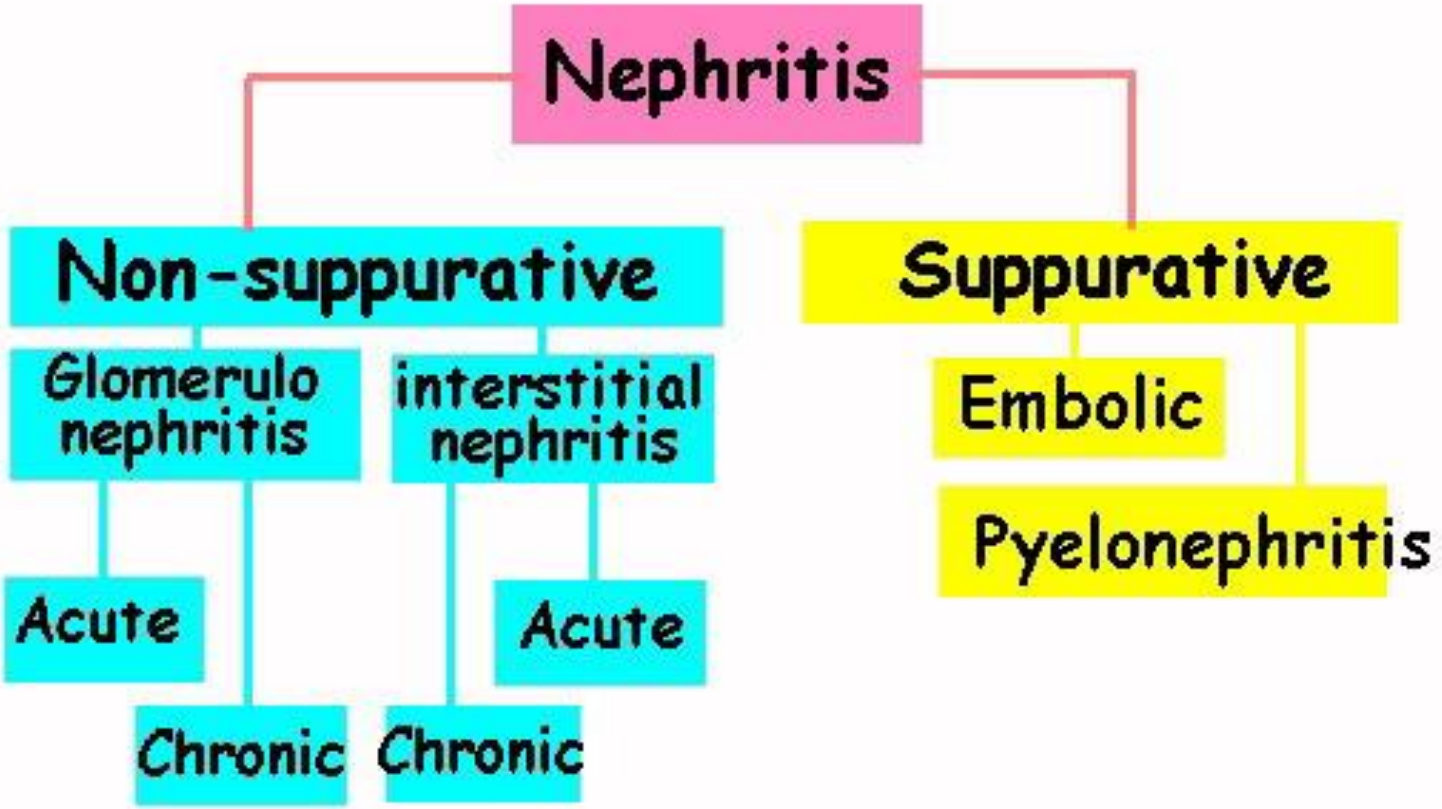
Nephritis

3. The type of exudate into:



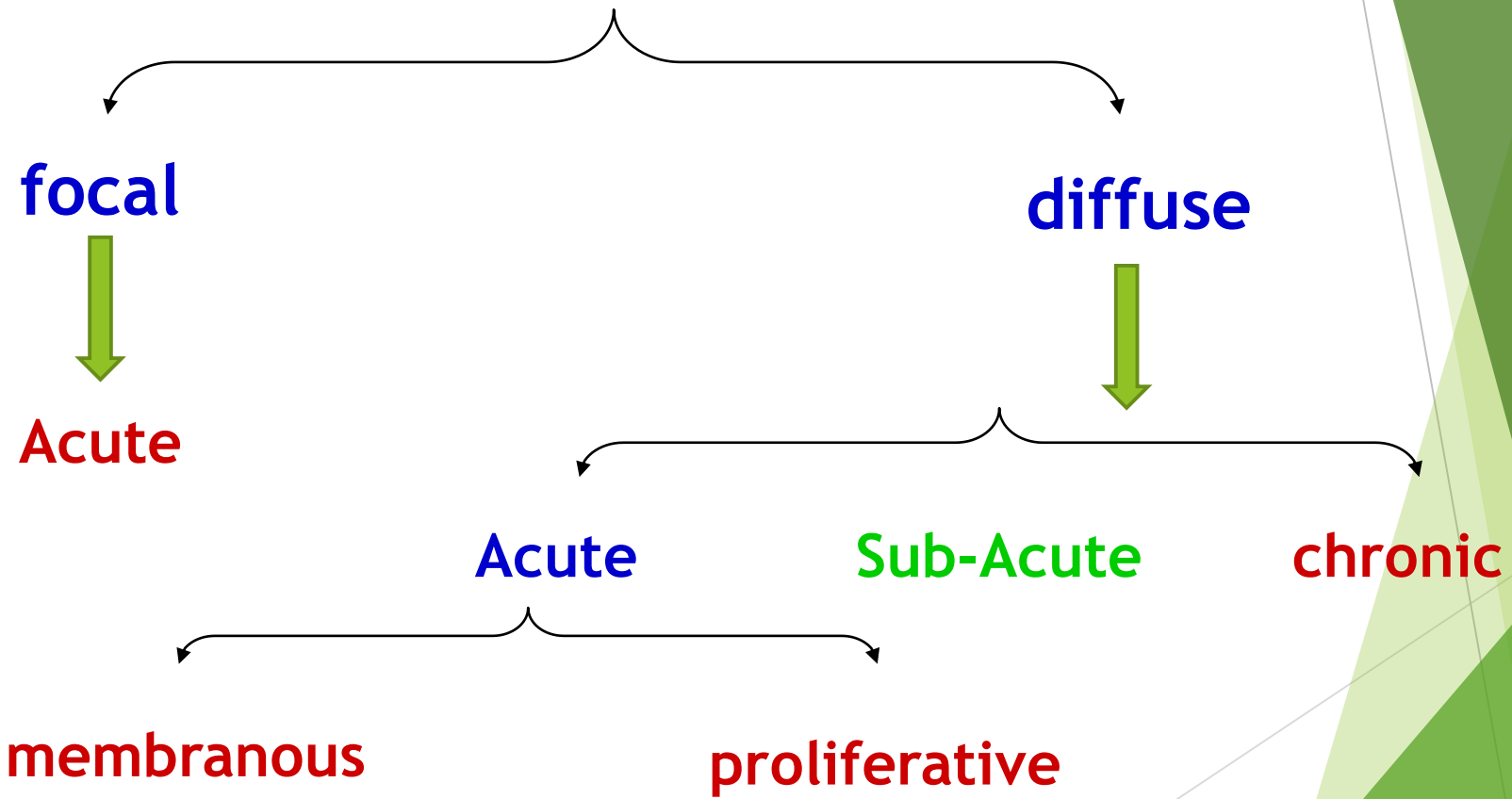
Suppurative nephritis

Non-Suppurative nephritis


Nephritis
Non-suppurative
Suppurative
Glomerulo nephritis
interstitial nephritis
Embolic
Pyelonephritis
Acute
Acute
Chronic
Chronic

Nephritis

- Non suppurative Glomerulonephritis



1- Acute diffuse glomerulo-nephritis

- Inflammation of kidneys including most or all glomeruli in both kidneys
- It is not common in animals ***(only reported in dogs and horses less than 3-years)***
- Its exact cause is not known but, it may be due to ***immune reaction or toxic agents***
- This type occur mainly in man than animal,
- Especially, in case of ***chronic tonsillitis, bronchitis, endocarditis, myocarditis, splenic abscess and pneumonia,*** where,the body begins to form antibodies against it

Pathogenesis:-

- antigen ***(bacteria or virus)*** may contain common antigen which are selectively deposited in the glomeruli

- The glomerular basement membrane may act as antigen to the body immune response ***(auto immunity)***

- Antigen-antibody complexes that may react with circulating cells to release some inflammatory mediators as histamine which change the permeability of blood vessels

Macroscopic picture:-

- The kidney may be slightly enlarged
- pale in colour, soft, edematous
- with the presence of petechial hemorrhage on the cortex
- The size cortex wider than normal

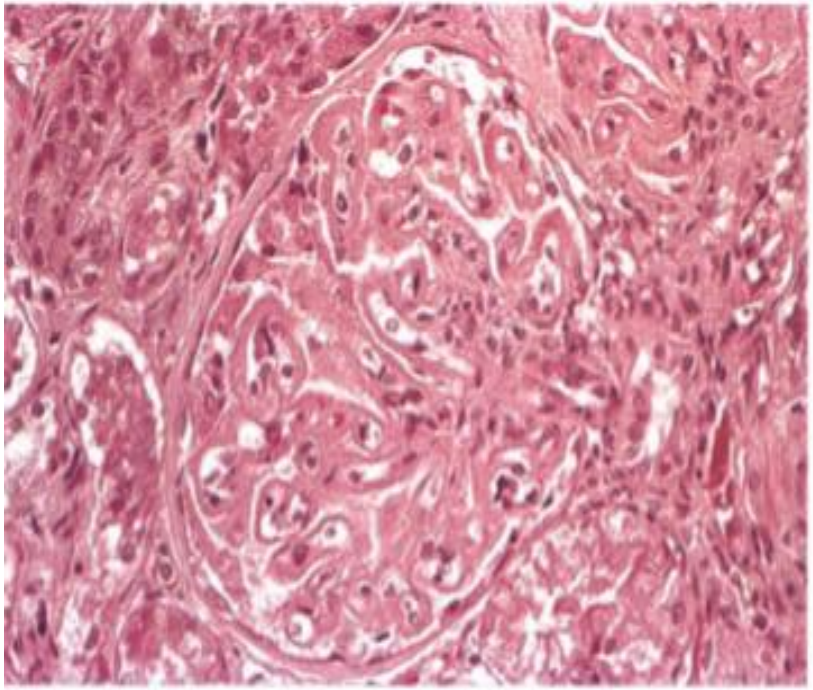
Microscopic picture:-

Acute membranous glomerulo-nephritis

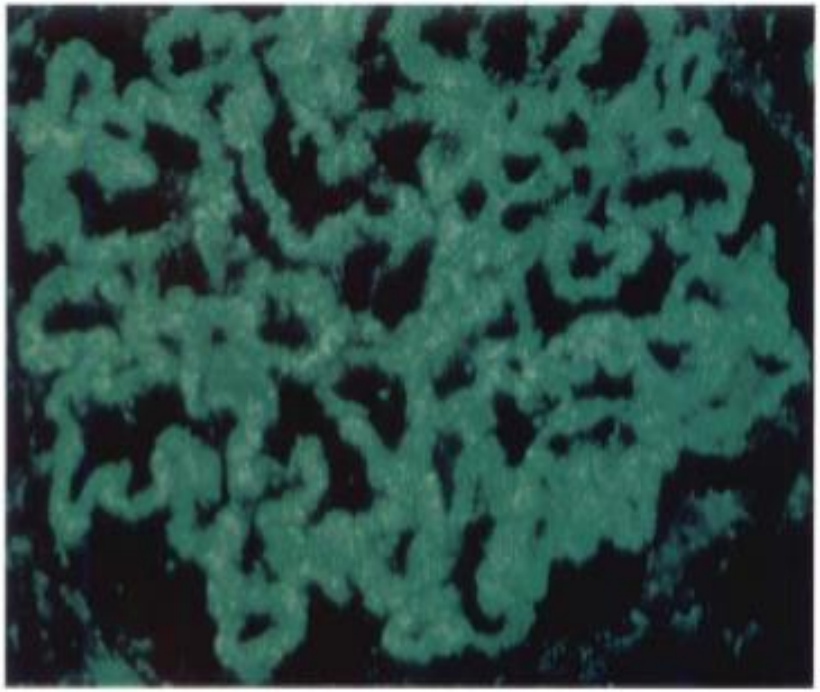
- Thickening of the glomerular basement membrane (due to due to deposition of immune (Ag-Ab) complex on the external side of glomerular basement membrane
- Thickening of the capillary wall of glomerular tuft due to deposition of immune complex
- The epithelial cells in the glomerulus become swollen due to presence of fat droplets.
- The epithelial cells of the renal tubules may also showing fatty change

Microscopic picture:-

Acute membranous glomerulo-nephritis



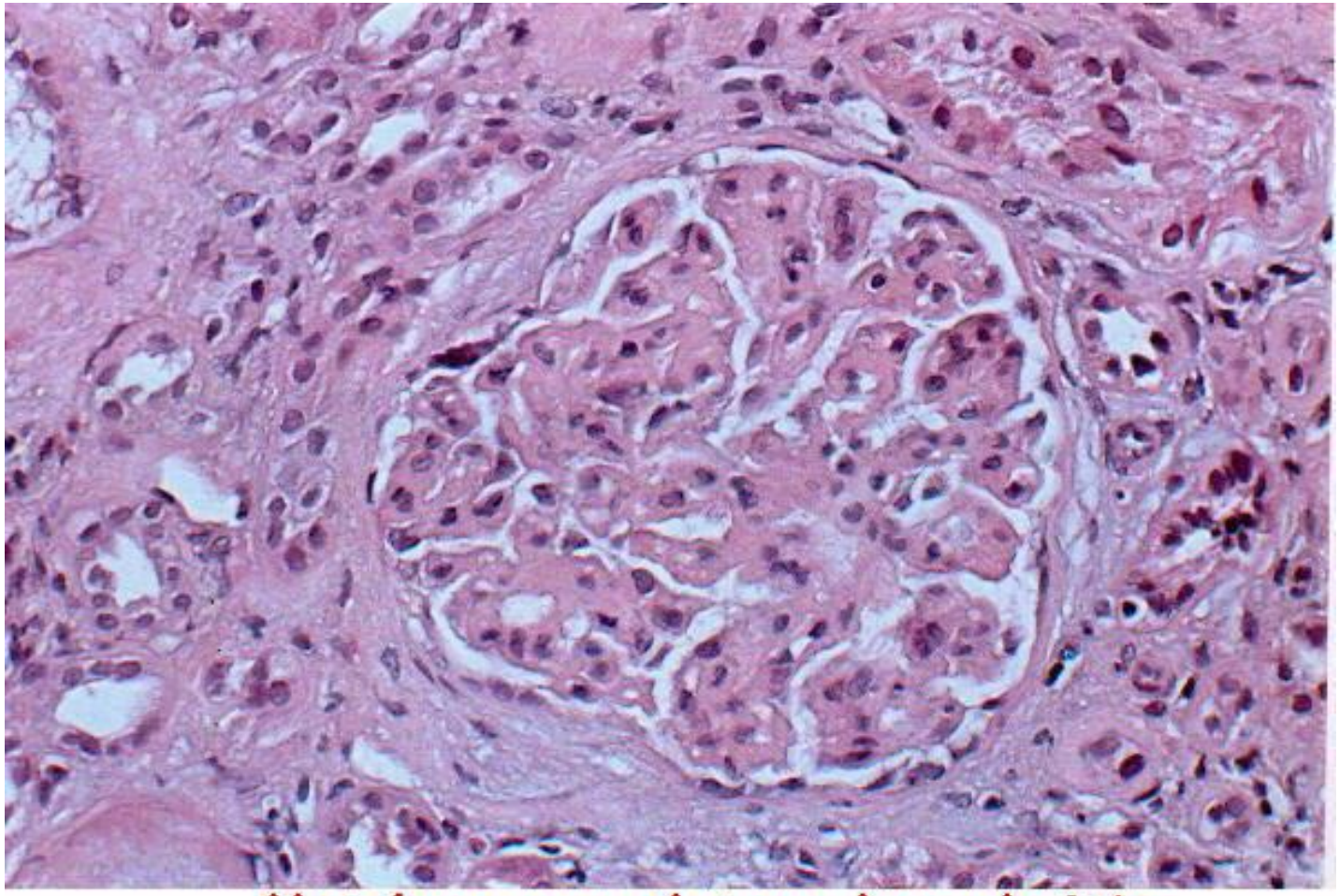
4–21 Membranous glomerulonephritis
Thickening of the capillary walls of a glomerulus due to deposition of immune complexes, and thickening of the glomerular basement membrane. Patent capillary lumina. Lack of hypercellularity. Dog. HE.



4–22 Membranous glomerulonephritis
Glomerulus. Granular fluorescence along the glomerular basement membrane due to deposition of immune complexes. In most spontaneous cases it represents an idiopathic lesion. Dog. Rabbit antidog IgG immunofluorescence.

Microscopic picture:-

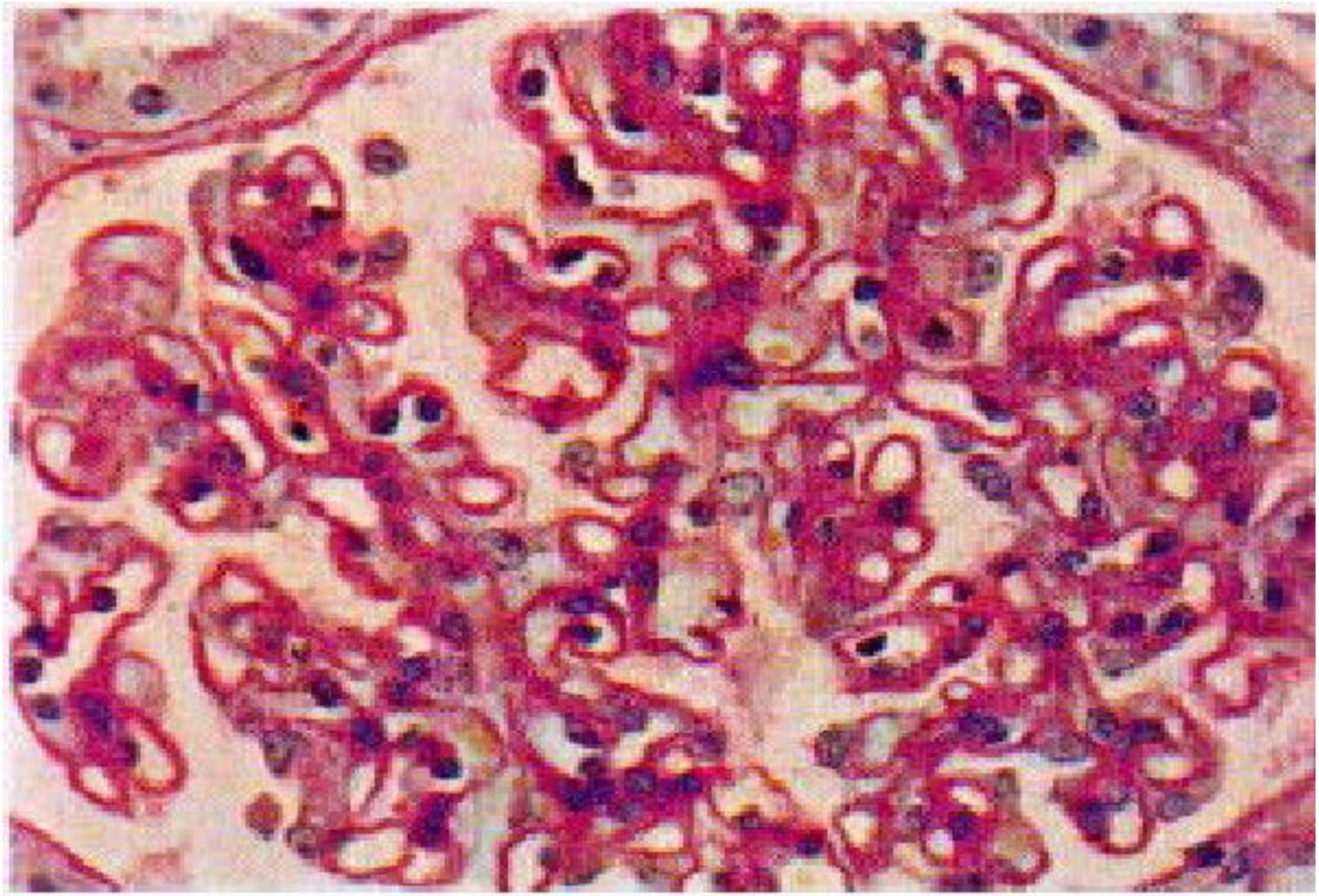
Acute membranous glomerulo-nephritis



Membranous glomerulonephritis

Diffuse thickening of the basement membrane in the glomerulus

Microscopic picture:-



Membranous glomerulonephritis
thickening of the basement membrane due to deposition of glucoploysaccharides (red-stained)

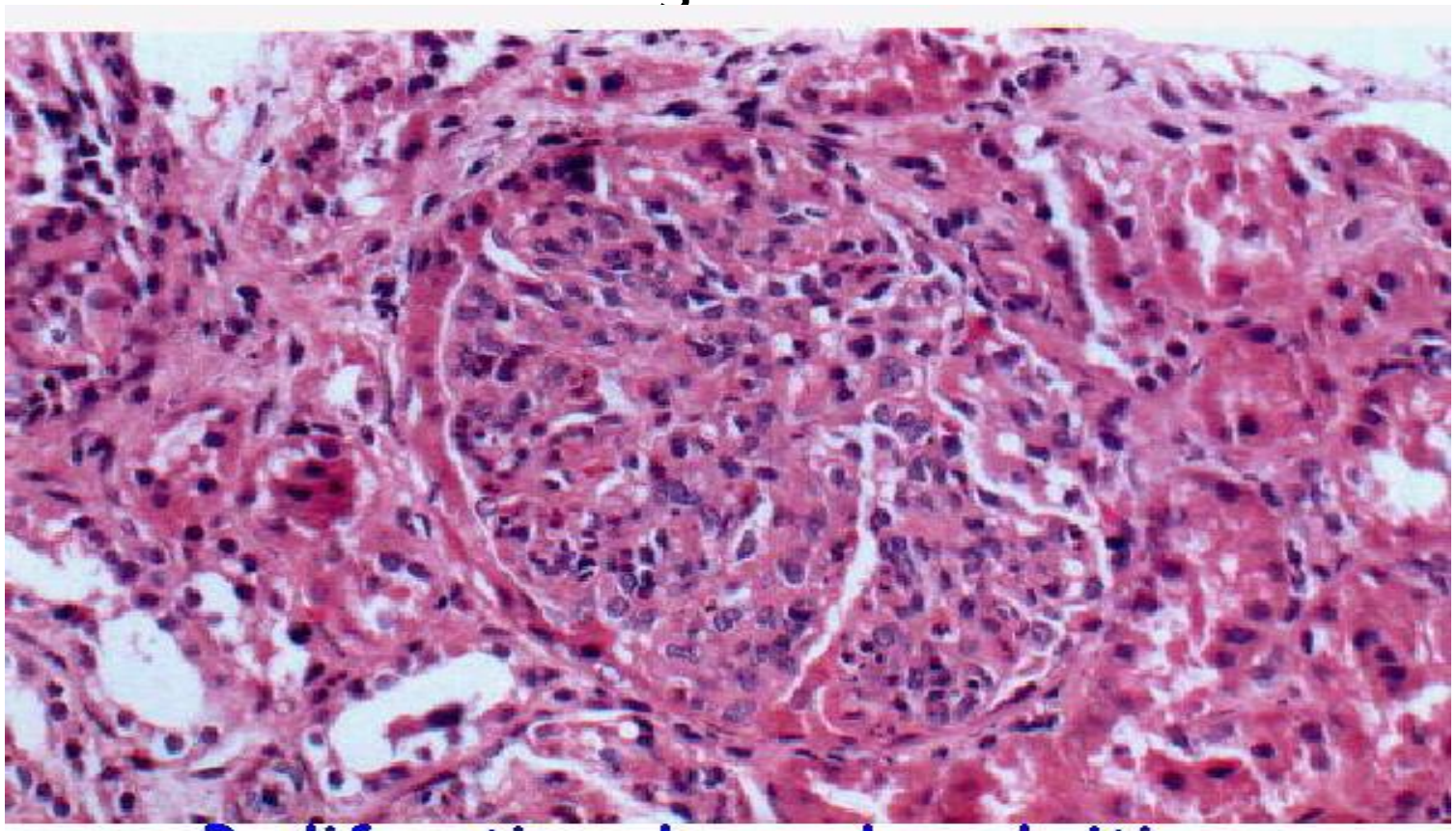
Microscopic picture:-

Acute proliferative glomerulo-nephritis

- The glomerular tuft appears as a group of cell formed from proliferated endothelial cells lining & leukocytic cellular infiltration mainly neutrophils resulting in enlarged glomeruli.
- This cellular proliferation result in compression of the capillaries and absence of red blood cells
- So, the glomeruli enlarged & avascular. So, No infiltration of blood occur & this lead to oligurea or even anurea followed by Uremia & death.
- As the disease progress, epithelial cells proliferate and sometimes adhere to the parital layer of Bowman's capsule

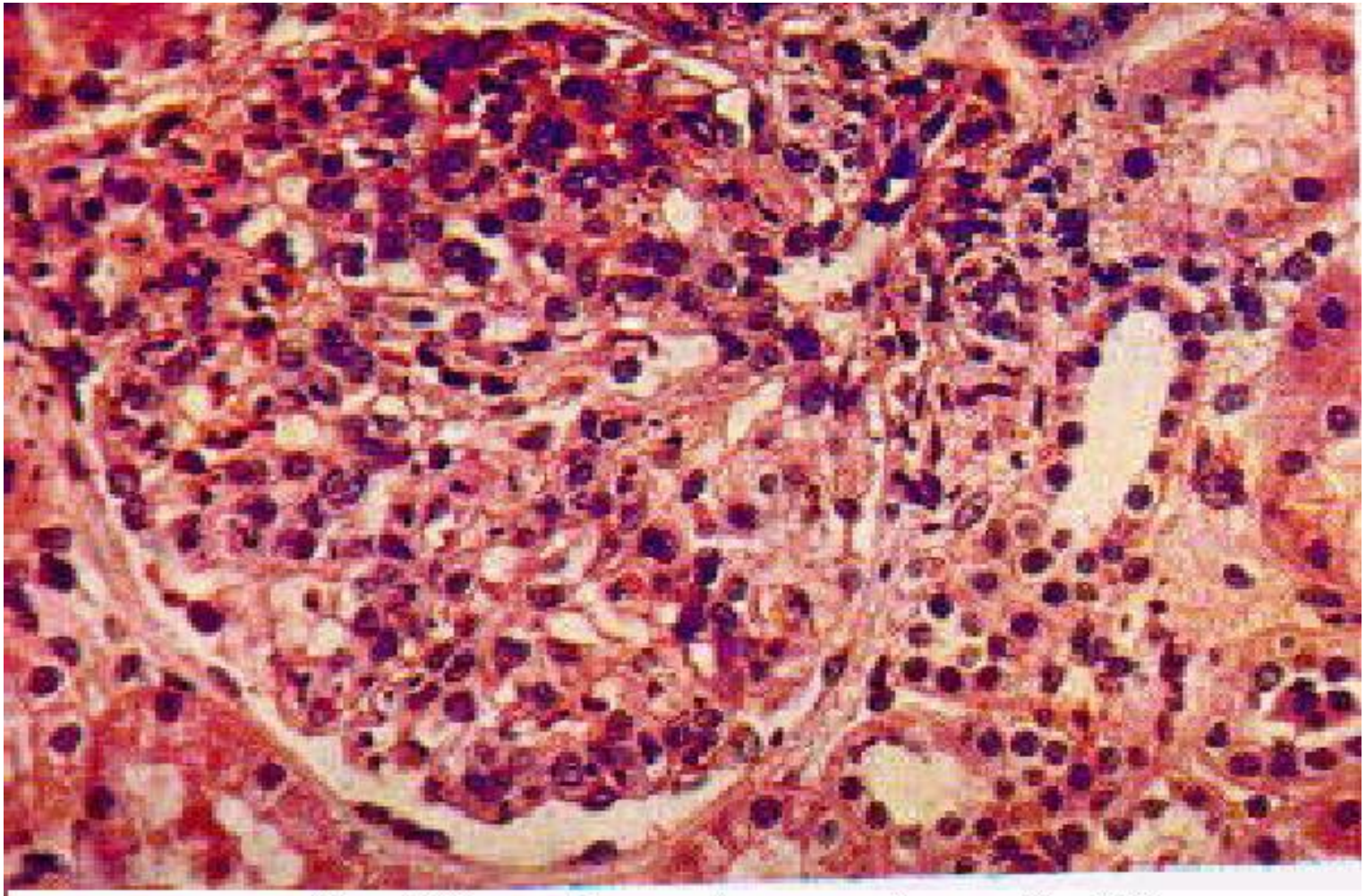
Microscopic picture:-

- Renal tubules have secondary changes as red blood cell casts or fatty changes in the lining epithelial of renal tubules due to interference with blood flow from glomeruli



Proliferative glomerulonephritis
Glomeruli are enlarged and hypercellular

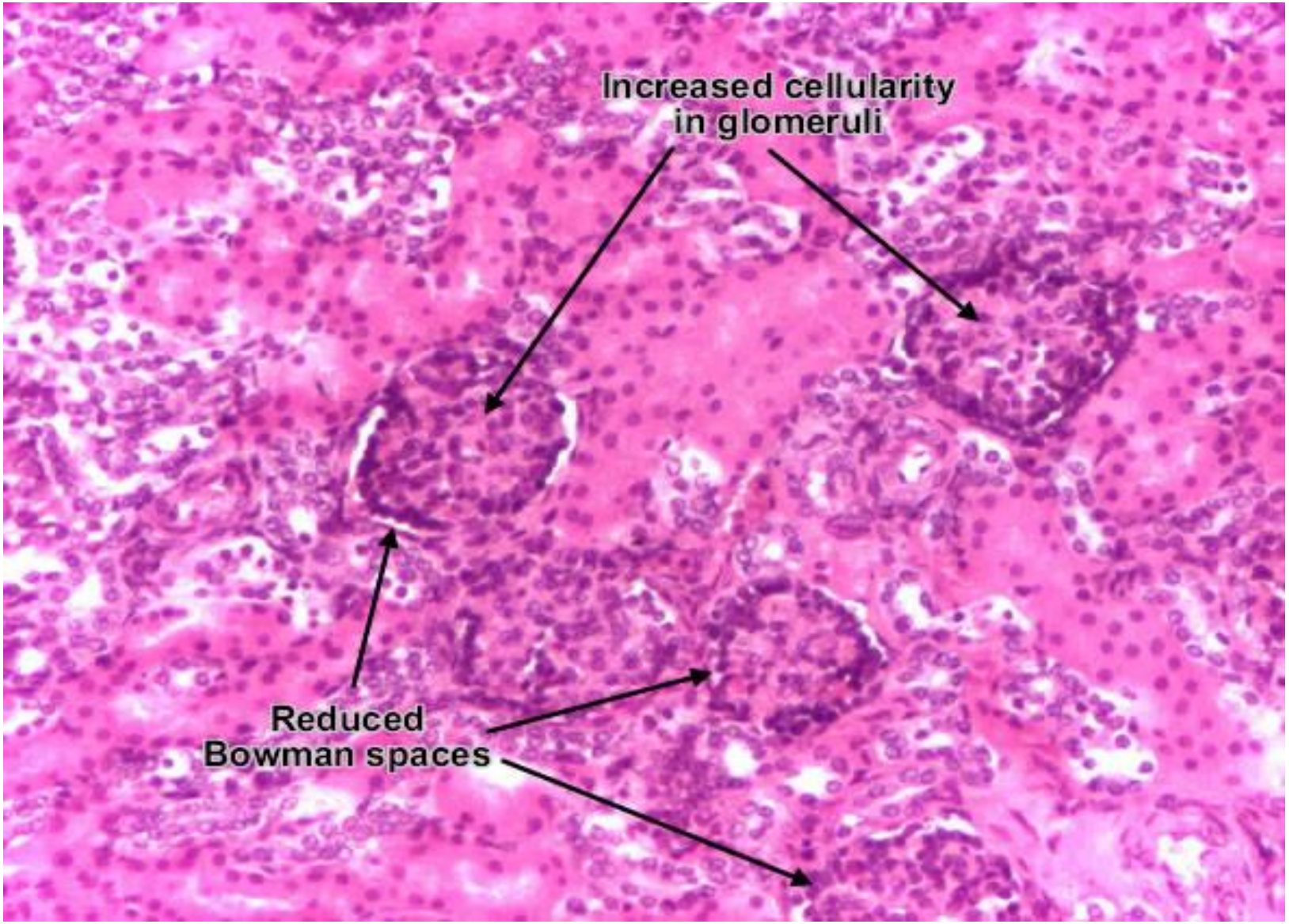
Microscopic picture:-



Proliferative glomerulonephritis

increased cellularity of the glomerulus

Microscopic picture:-



1- sub-acute diffuse glomerulo-nephritis

In acute form → The animal eithr die or go to →

sub-acute glomerulonephritis

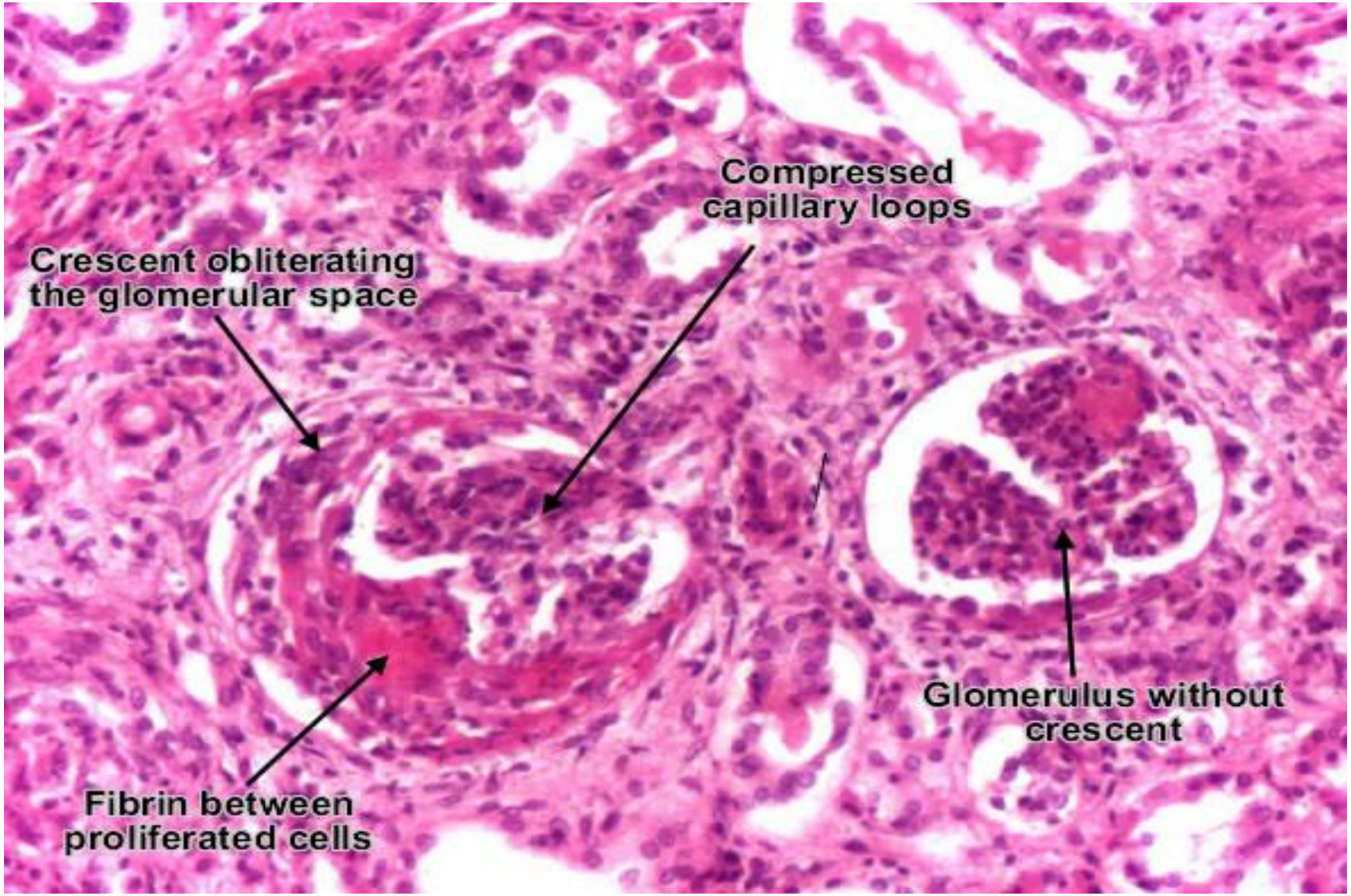
Macroscopic picture:-

- Large pale kidney with petechiae (brownish white)

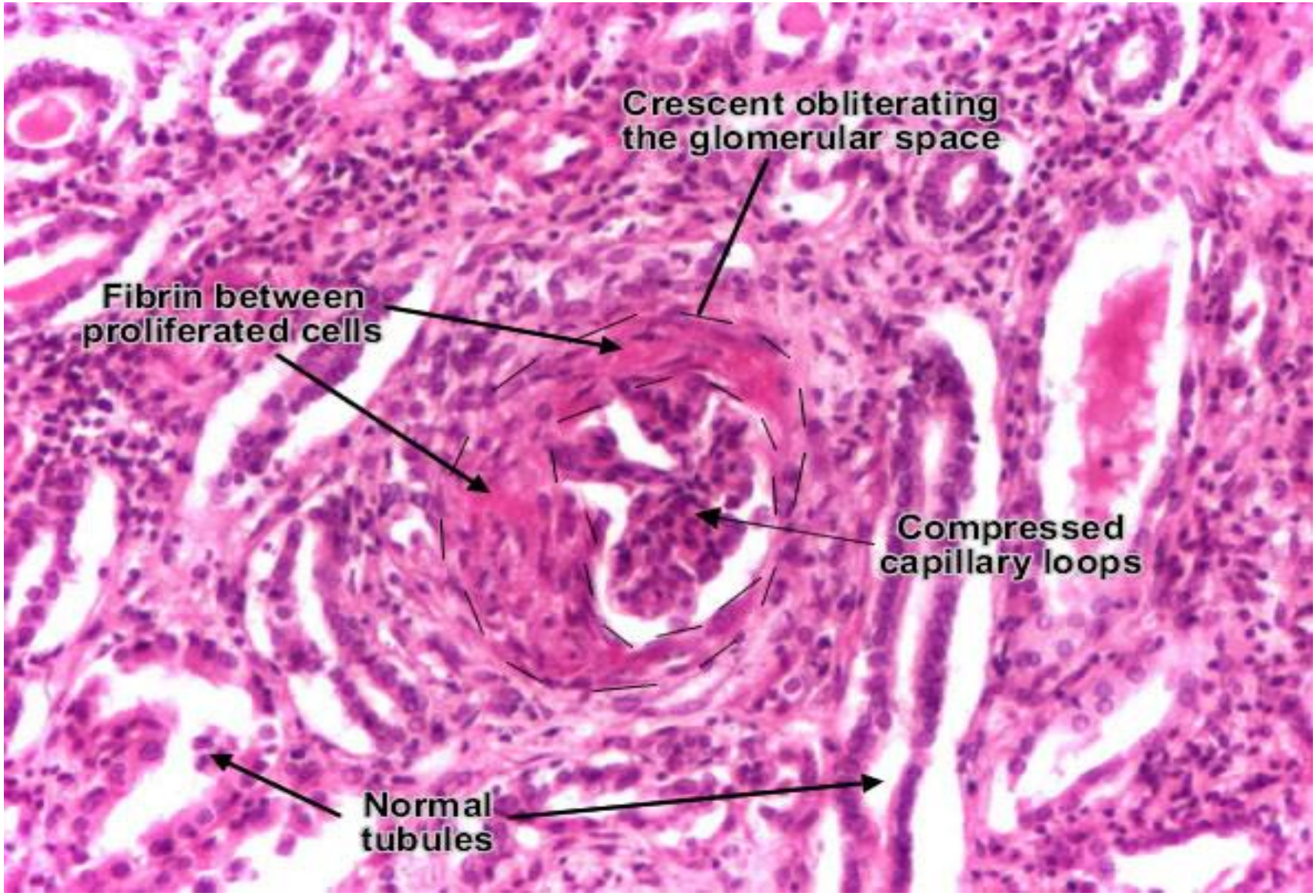
Microscopically:-

- the proliferated epithelium of the Bowman's capsule adds to the glomerular hypercellularity, and adhesions between visceral and parietal layers with the formation of ***epithelial crescents (crescentic glomerulo-nephritis).***
- ***Renal tubules suffered from cloudy swelling, necrosis of the lining epithelium with hyaline casts in their lumen***

1- sub-acute diffuse glomerulo-nephritis



1- sub-acute diffuse glomerulo-nephritis



2- sub-acute diffuse glomerulo-nephritis

❑ *Mechanism of crescentic glomerulo-nephritis*

- Formation of crescents is initiated by passage of fibrin into the Bowman space as a result of increased permeability of glomerular basement membrane.
- Fibrin stimulates the proliferation of parietal cells of Bowman capsule, and an influx of monocytes.
- Rapid growing and fibrosis of crescents compresses the capillary loops and decreases the Bowman space which leads to renal failure within weeks or months

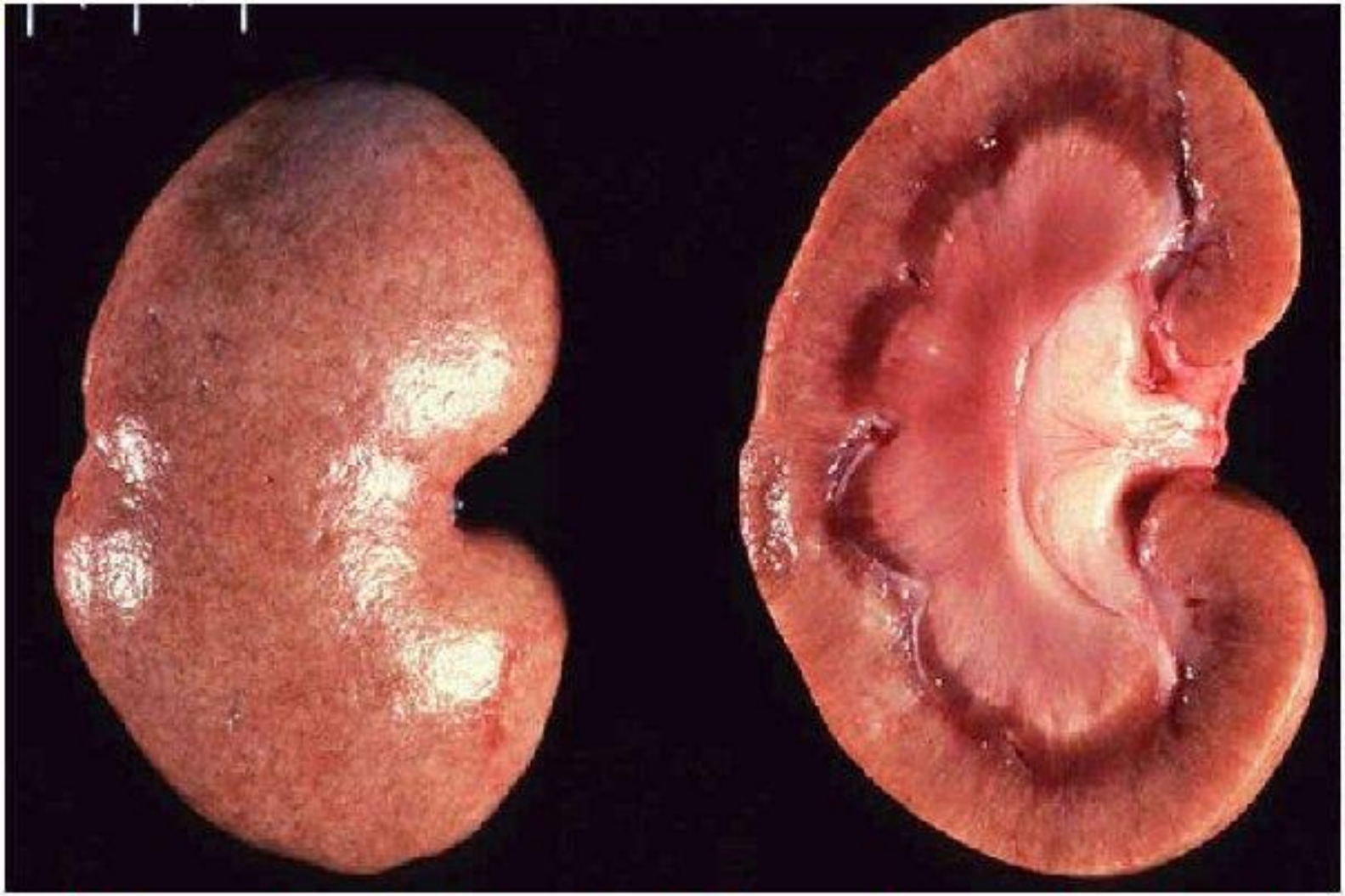
3- Chronic diffuse glomerulonephritis

- It occurs in late stage of sub-acute form

Macroscopic picture:-

- The kidneys are smaller than normal, rough, or pitted on the surface and tough to be cut
- In cut-surface the kidneys were coarse, granular or cystic (tiny cyst scattered on the cortex)

3- Chronic diffuse glomerulonephritis



Kidney (Dog): Chronic diffuse glomerulonephritis

The cortical surface is finely granular

3- Chronic diffuse glomerulonephritis

Microscopic picture:-

- Majority of the glomeruli affected depending on the stage of the diseased condition.
- Increase in the number of cells in the glomeruli
- Occlusion of the lumen of glomerular capillaries
- Obliteration of Bowman's space due to adhesion of epithelial cells with parietal layer of Bowman's capsule

3- Chronic diffuse glomerulonephritis

Microscopic picture:-

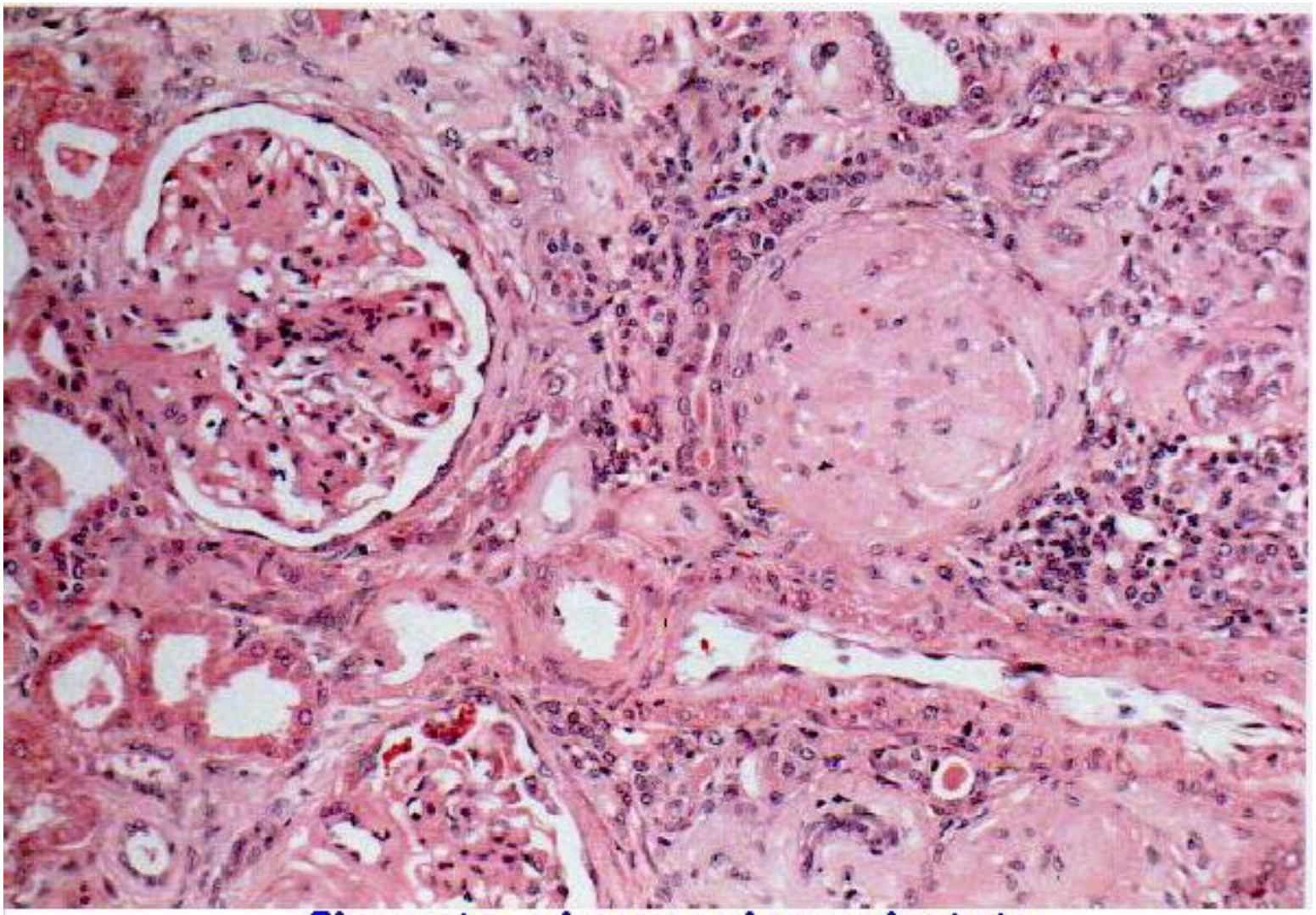
- Presence of chronic inflammatory cells as a large number of macrophages & plasma cells with fibrous connective tissue proliferation.
- So, partial or the entire glomeruli replaced by fibroblast & collagen fibers (sclerotic glomeruli).
- Fibrous C.T replaced the renal tissues leads to shrinkage and contraction of the kidney.
- different degrees of hyalinization (hyalinosclerosis - total replacement of glomeruli and Bowmann's space with hyaline).

3- Chronic diffuse glomerulonephritis

Microscopic picture:-

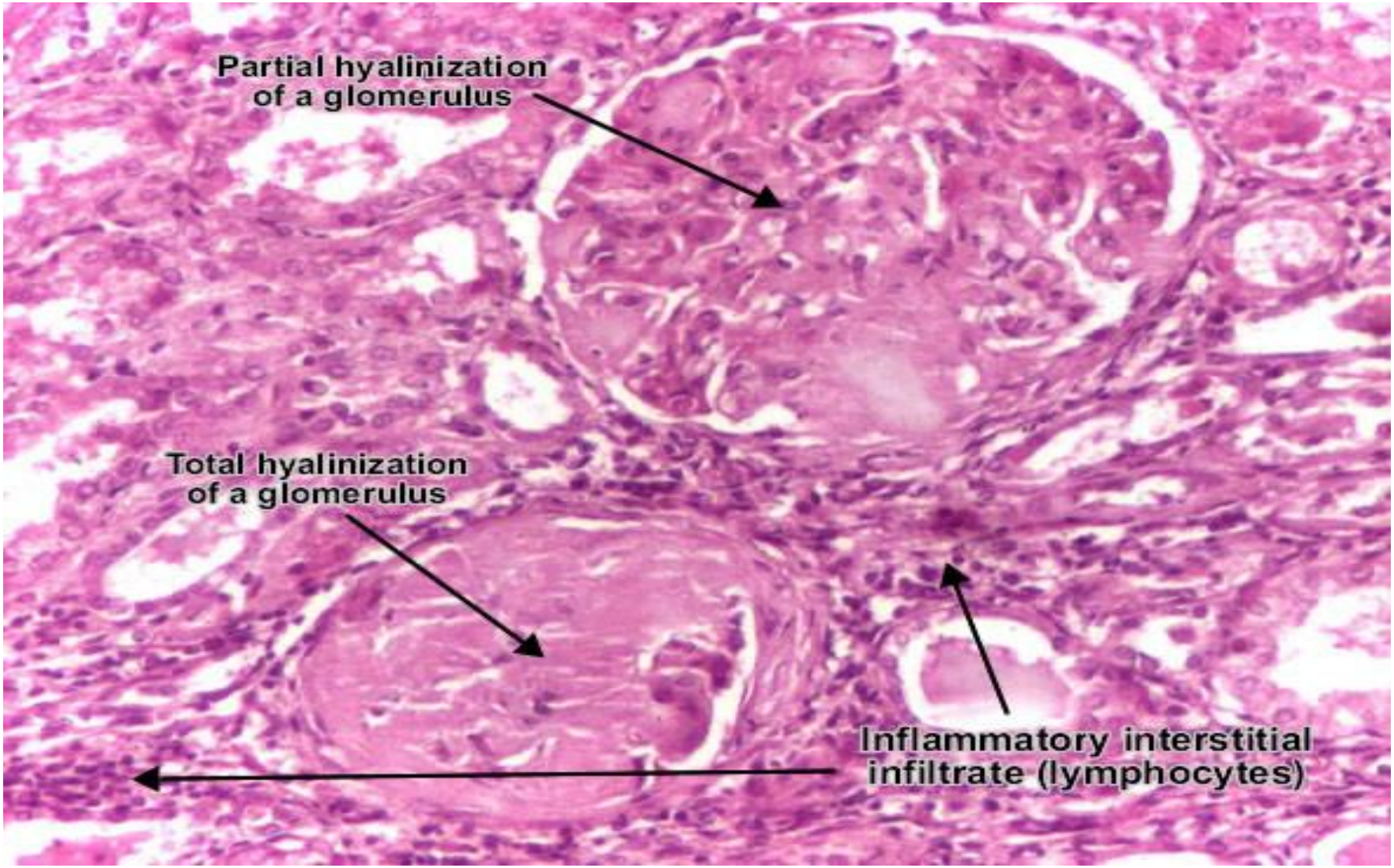
- The hyaline is an amorphous material, pink, homogenous, resulted from combination of plasma proteins, increased mesangial matrix and collagen.
- Totally hyalinized glomeruli are atrophic (smaller), lacking capillaries, hence these glomeruli are non-functional.
- Obstruction of blood flow will produce secondary tubular atrophy, interstitial fibrosis and thickening of the arterial wall by hyaline deposits
- In the interstitium is present an abundant inflammatory infiltrate (mostly with lymphocytes).
- In Function nephron, glomeruli suffered from hypertrophy with cystic dilatation of R.T.

3- chronic diffuse glomerulo-nephritis

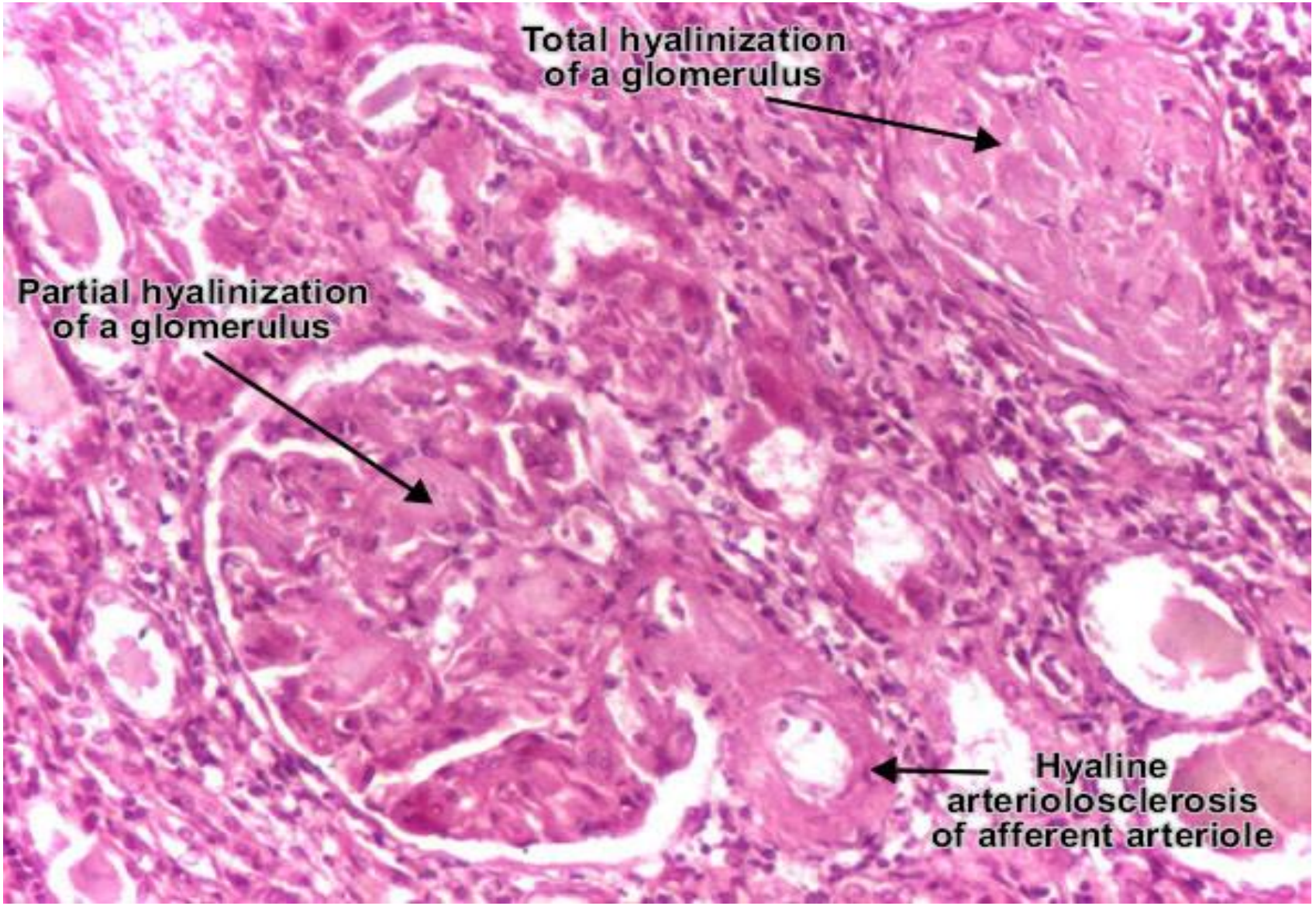


Chronic glomerulonephritis
glomeruli are fibrosed and hyalinized

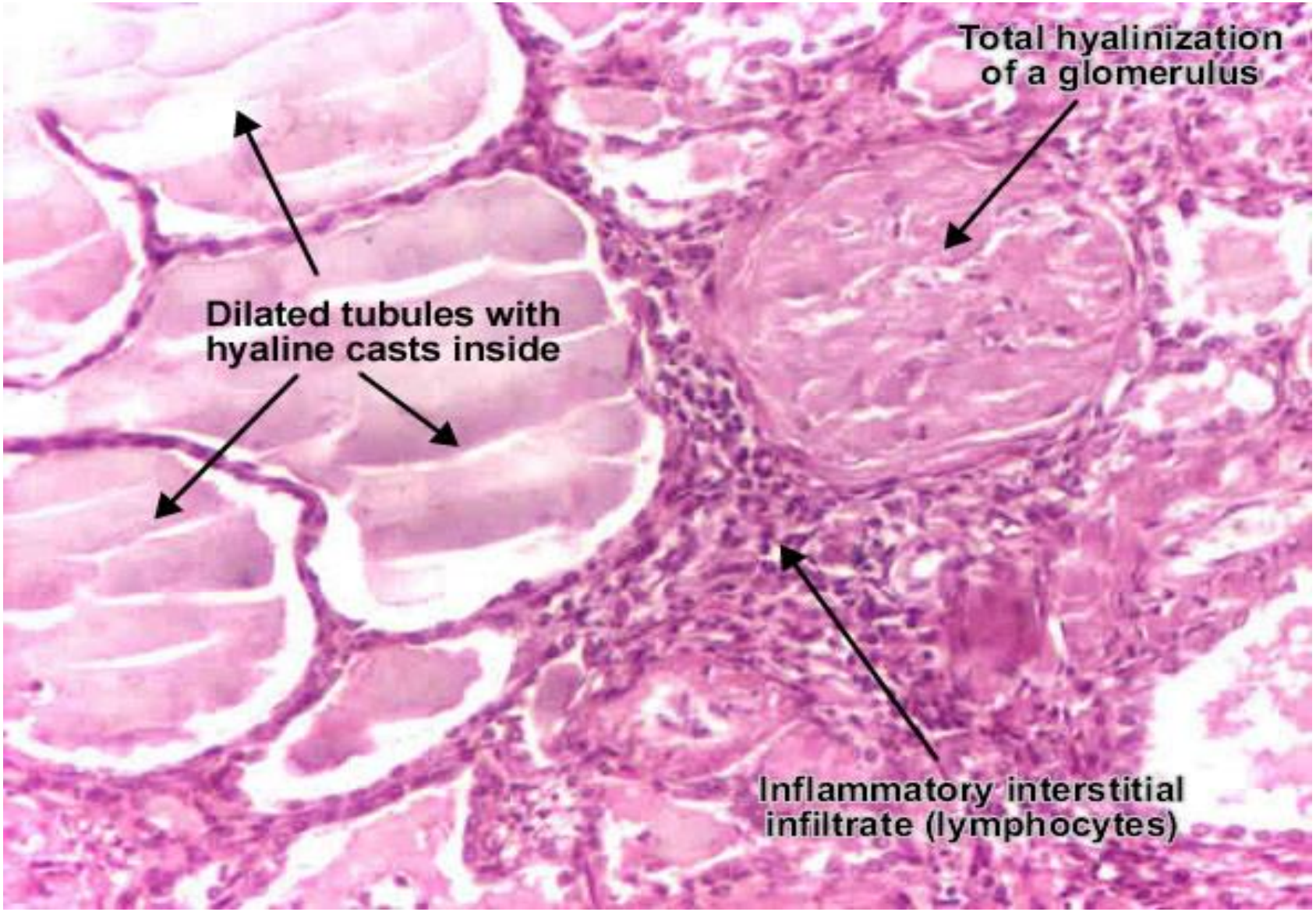
3- chronic diffuse glomerulo-nephritis



3- chronic diffuse glomerulo-nephritis



3- chronic diffuse glomerulo-nephritis

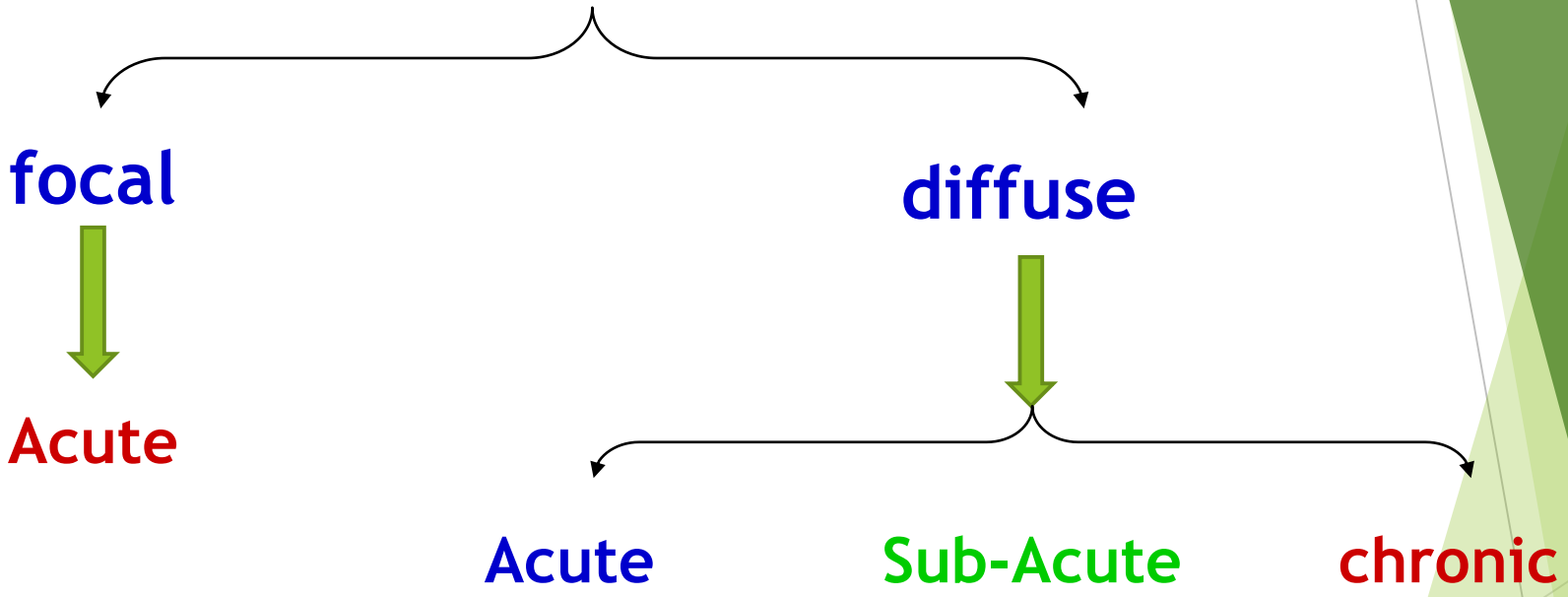


Acute focal glomerulo-nephritis:

- The changes occurred in scattered number of glomeruli
- This is the commonest type of glomerulo-nephritis in animals
- Causative agents may be leptospirosis, colibacillosis, pasteurellosis, salmonellosis)
- It is characterized by swelling and proliferation of endothelial cells lining of glomerular tuft in one or more lobules

Interstitial Nephritis

- Non suppurative interstitial nephritis



Acute diffuse Interstitial Nephritis :

- It is a diffuse inflammatory reaction mainly in the interstitial tissue
- It is common in dogs with leptospirosis

Causes:-

- Toxic → as toxic tubular nephritis.
- Chemical → as mercuric salts.
- Endogenous toxins → as burns toxins → necrosis of renal tubules.
- Leptospira pomona → causing non-supp-interstitial nephritis.

Acute diffuse Interstitial Nephritis :

Macroscopical findings:-

- Kidney may be of normal size or slightly enlarged
- The capsules strips easily
- The cortex showing mottling appearance of red-gray areas
- Cut section revealed greyish streaks or foci at cortico-medullary junction

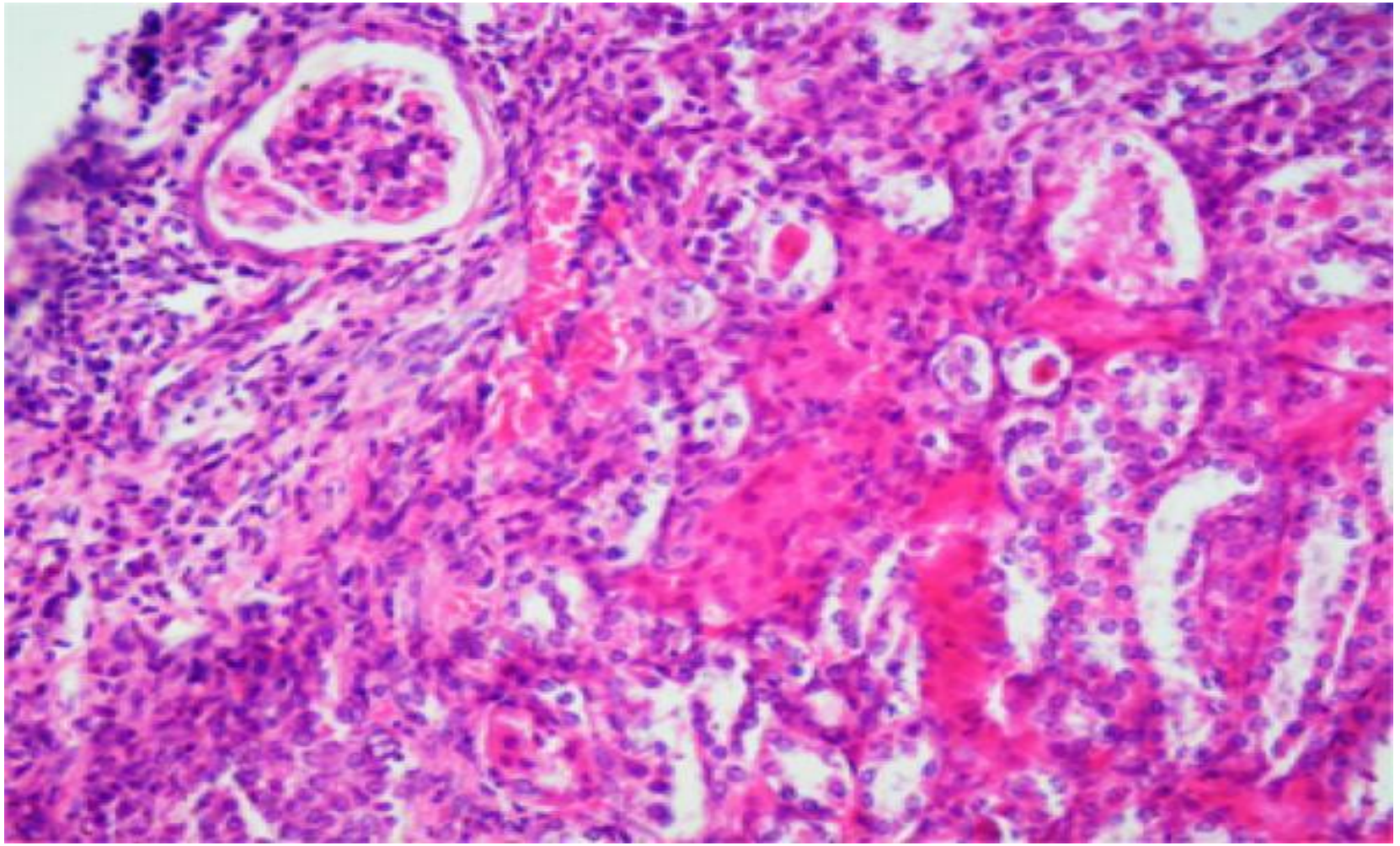
Acute diffuse Interstitial Nephritis :

Microscopical findings:-

- The interstitial tissues are edematous
- Aggregation of lymphocytes and plasma cells between or inside or replace renal tubules.
- Severe degenerative changes in the lining epithelium of renal tubules as cloudy swelling, fatty change or necrosis with hyaline casts in their lumen

Acute diffuse Interstitial Nephritis :

Microscopical findings:-



Sub-acute diffuse Interstitial Nephritis :

Causes:

- It is usually follows the acute as in Leptospirosis.
- It is usually associated with renal infection, pneumonia and enteritis in calves.

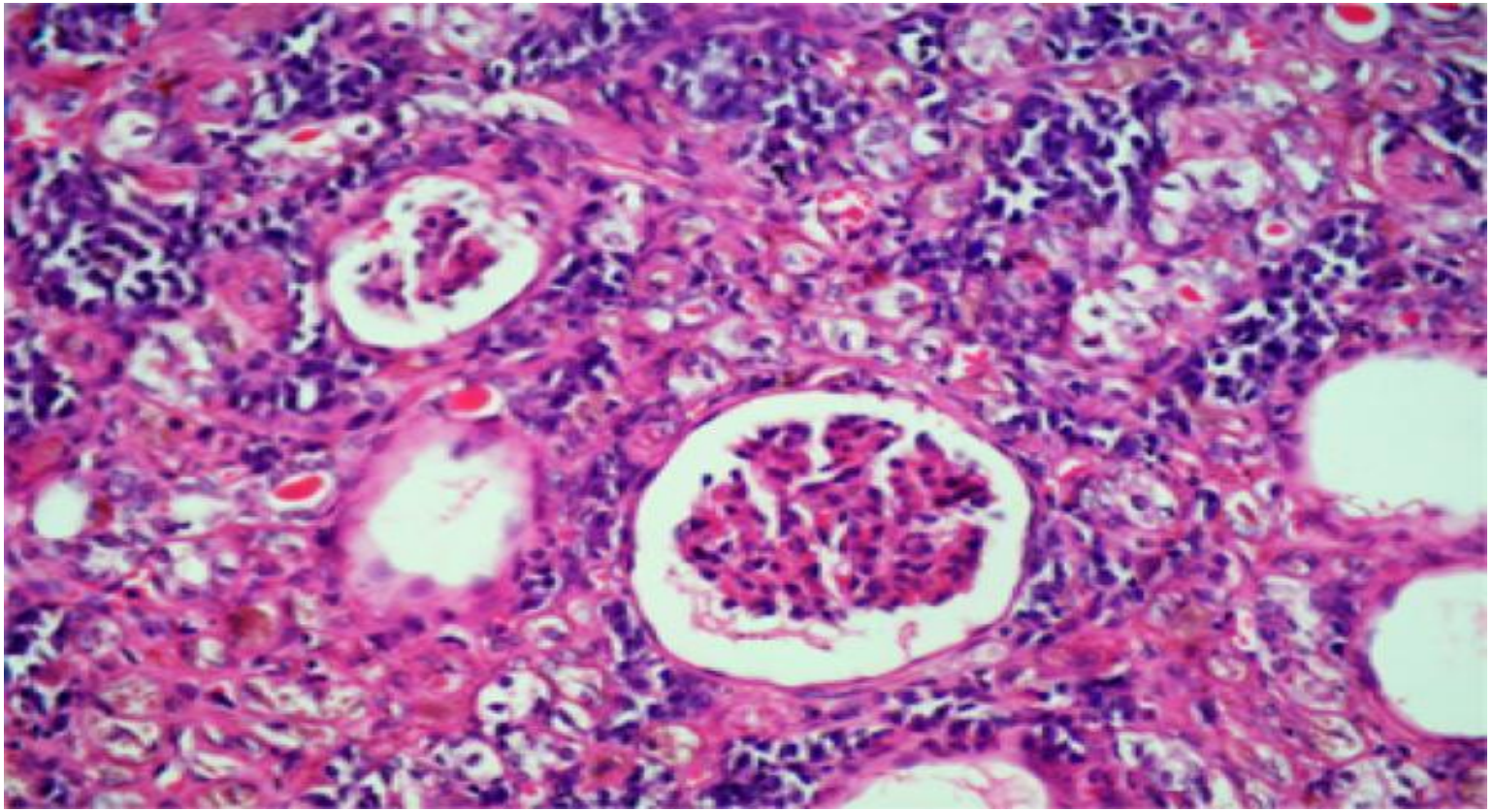
Macroscopical findings:-

- the kidneys are slightly atrophic and firm in consistency
- large white or grayish nodular masses in the outer medulla

Sub-acute diffuse Interstitial Nephritis :

Microscopical findings:-

- the nodules composed from lymphocytes with macrophages and few fibrous tissues
- Some renal tubules showed cystic dilatation



chronic diffuse Interstitial Nephritis :

Causes:

- After subacute form.
- It is common in dogs in its late stage of leptospirosis

Macroscopical findings:-

- Kidneys are shrunken, small in size, pale gray in colour, with irregular and nodular cut surface due to uneven contraction of fibrous connective tissue
- firm and difficult to be cut
- Cortex is shrunken and very narrow due to massive loss of nephrons
- The capsule is thickened and adherent to the under lying tissue

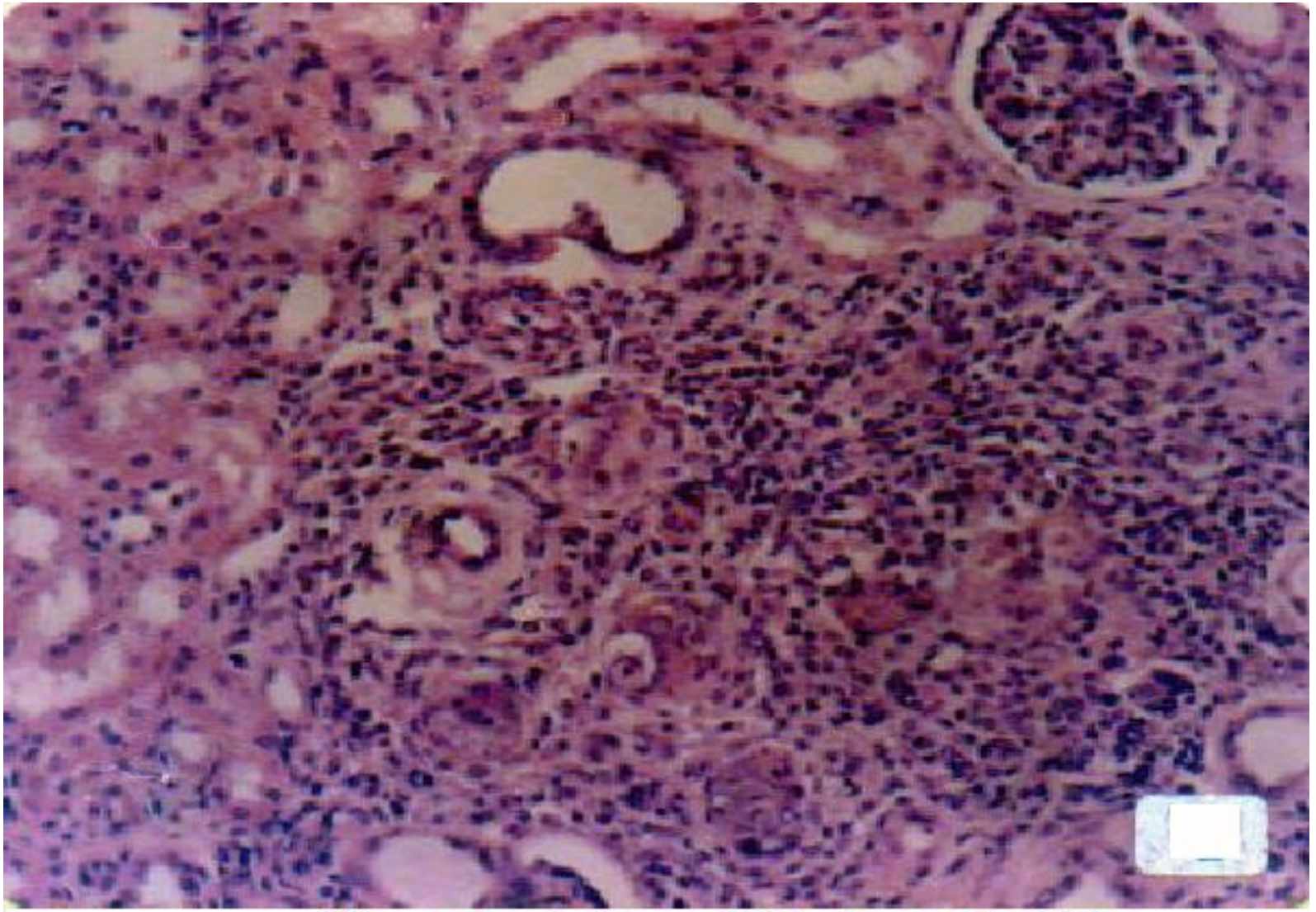
chronic diffuse Interstitial Nephritis :

chronic diffuse Interstitial Nephritis :

Microscopical findings:-

- Proliferation of interstitial fibrous connective tissue with leukocytic cellular infiltration
- Atrophy of renal tubules with necrosis of its lining epithelium
- Some of the remaining renal tubules showing cystic dilatation
- Many of these cystic tubules contain hyaline or granular casts
- The majority of glomeruli not affected
- The proliferation may extend to replace degenerated tubules
- Fibrosis replaces the renal parenchyma

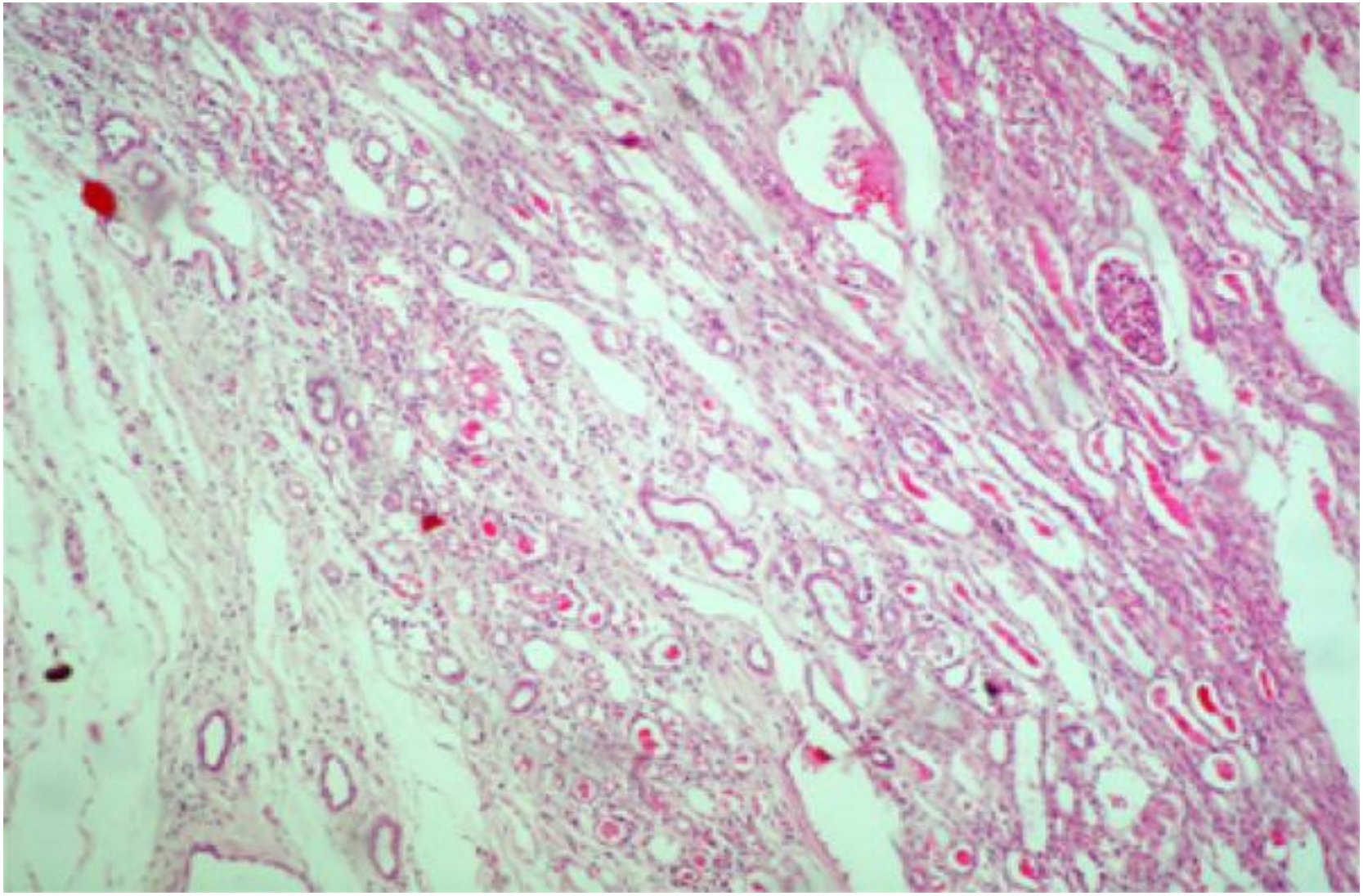
chronic diffuse Interstitial Nephritis :



Chronic interstitial nephritis

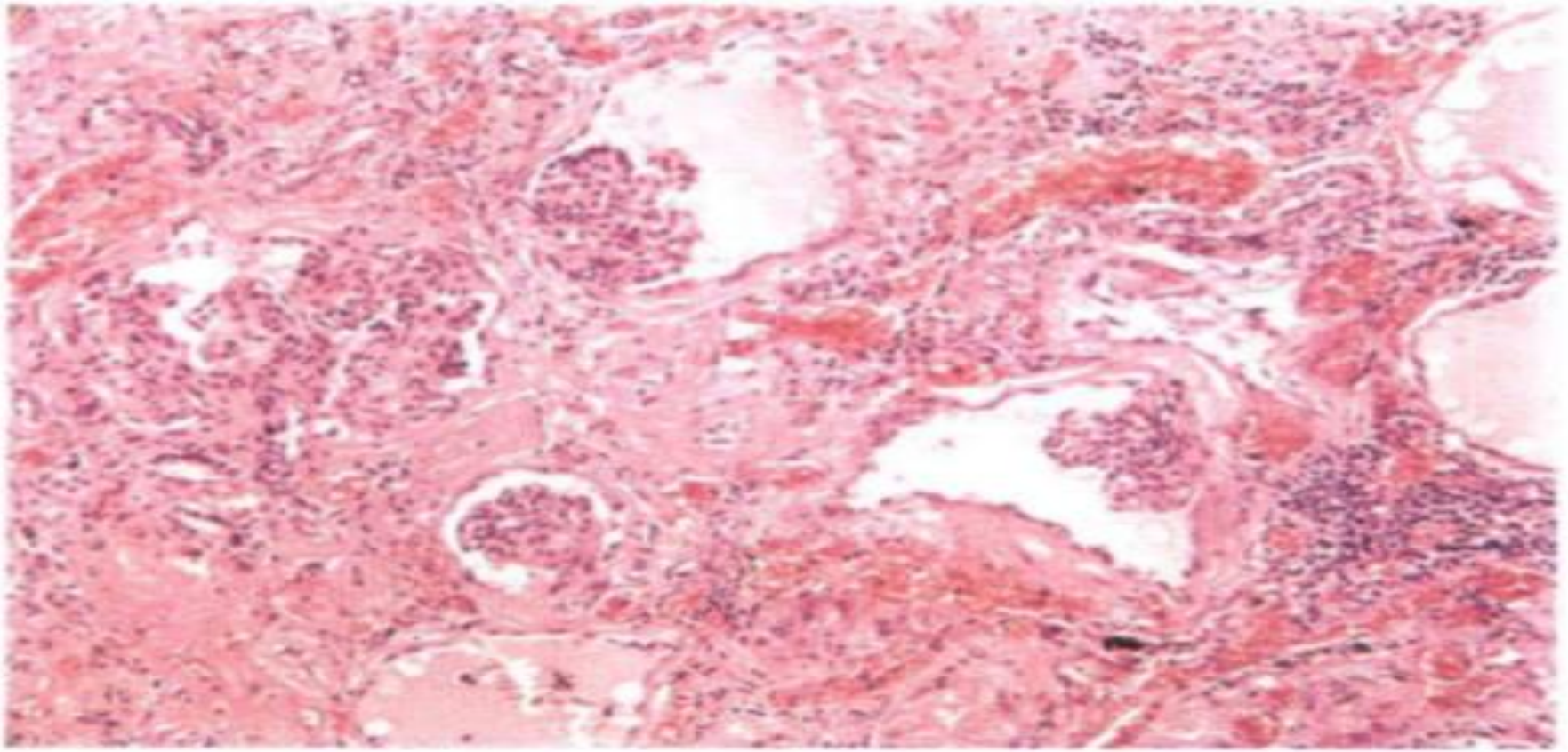
early stage with abundant infiltrating lymphocytes

chronic diffuse Interstitial Nephritis :



replacement of the renal tissue by fibrous connective tissue besides cystic dilatation of some renal tubules

chronic diffuse Interstitial Nephritis :



4–30 Chronic interstitial nephritis
Renal cortex. Almost complete replacement of nephrons by fibrous tissue, which contains random accumulations of mononuclear inflammatory cells. Remnants of tubules, glomeruli situated close together, dilated urinary spaces filled with proteinaceous material, and atrophic glomerular tufts are evident. Adhesions between Bowman's capsule and a tuft of a compensatory-hypertrophied glomerulus (left). Dog. HE.

Acute focal Interstitial Nephritis (White spotted kidneys) :

- It is a type of inflammatory condition occur in multiple foci in interstitial tissue
- It is very common in all domestic animals

  specially in calves

Causes:

- In calves may be related to E.coli infection
- While in adult cattle, it may be related to leptospirosis, malignant catarrhal fever, lumpy skin diseases
- In horse as in case of equine infectious anemia

Acute focal Interstitial Nephritis (White spotted kidneys) :

Macroscopical findings:-

- Multiple grayish-white nodules are visible through the capsule
- The nodules are usually 0.5-1.0cm in diameter
- The capsule may strip or there may be a fibrous adhesion to the surface of one or more nodules
- On cut surface, nodules are seen confined to cortex

Acute focal Interstitial Nephritis (White spotted kidneys) :

4–31 Focal interstitial nephritis
Renal surface. Multiple grayish-white prominent nodules (white-spotted kidney) in the renal cortex due to a proliferative inflammation. Histologically characterized by lymphocytic, plasmacellular and histiocytic infiltration, and fibrosis. May be caused by different bacteria e.g. *Escherichia coli*, *Salmonella*, *Brucella*. Veal calf.

Acute focal Interstitial Nephritis (White spotted kidneys) :

Microscopical findings:-

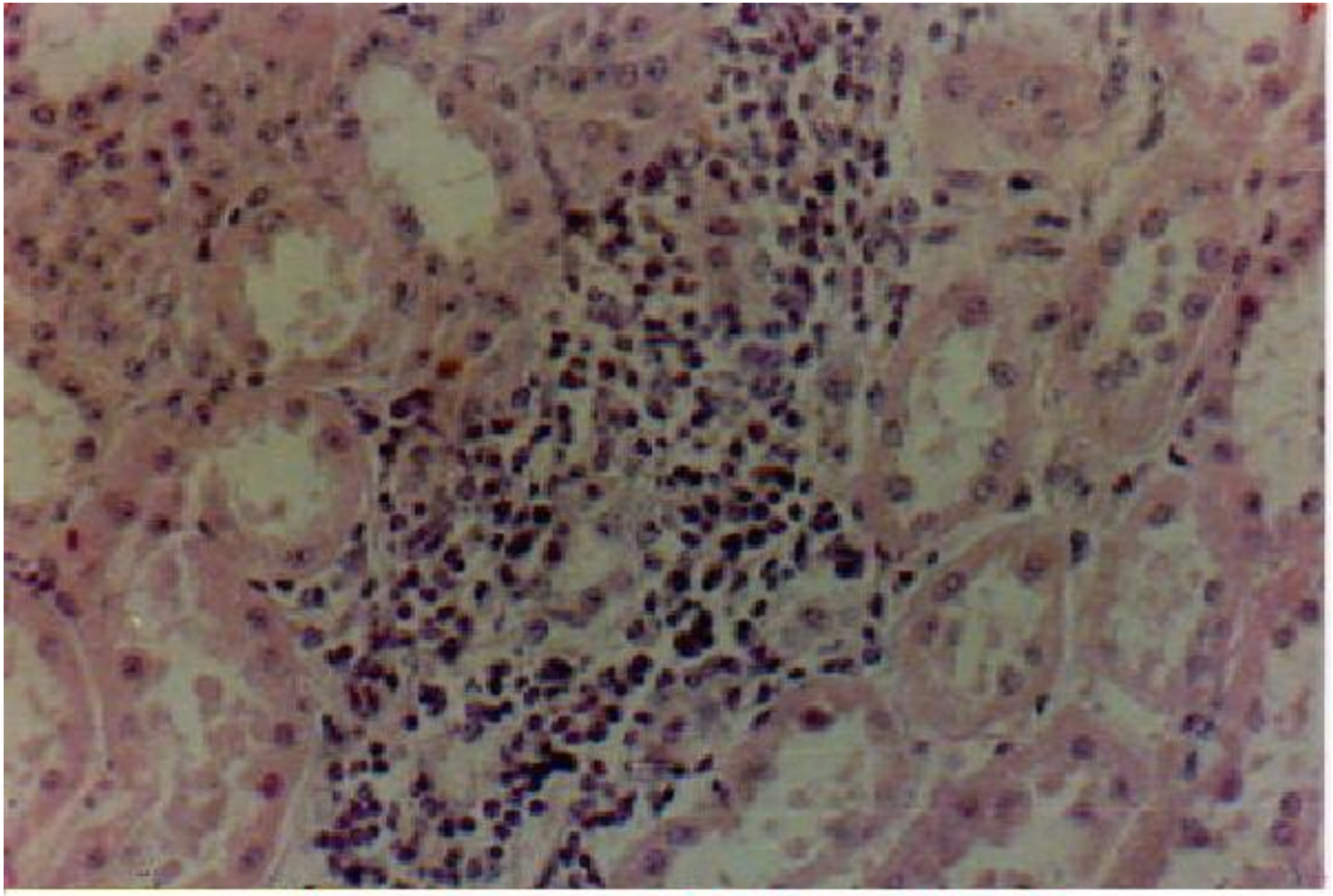
- The glomeruli are normal
- Interstitial tissues are edematous
- In early stage, leukocytic cellular infiltration mainly plasma cells and lymphocytes
- In late stage, histocytes appear and increased in number
- proliferation of interstitial fibrous connective tissue which surround many tubules in the renal cortex, these tubules become atrophied and disappear

Acute focal Interstitial Nephritis (White spotted kidneys) :

Microscopical findings:-

- The proliferation may extend to replace degenerated tubules
- Some other tubules are dilated and lined by degenerated epithelium
- Hyaline or granular casts are absent

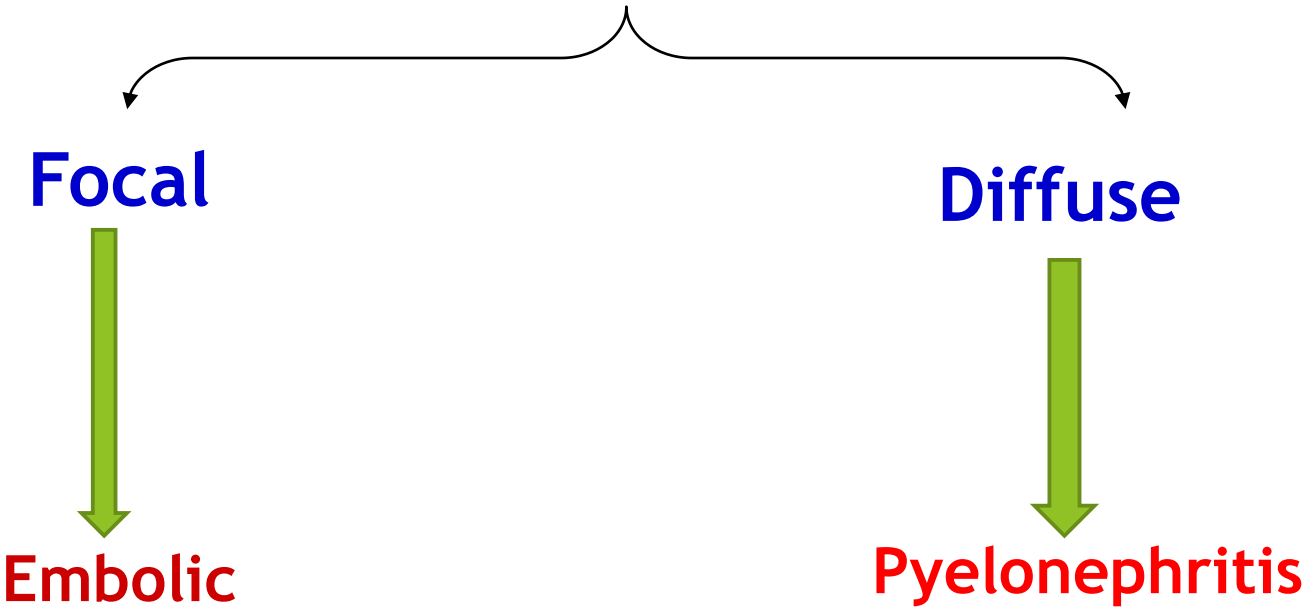
Acute focal Interstitial Nephritis (White spotted kidneys) :



Focal interstitial nephritis

Infiltration of lymphocytes in the interstitial tissue

Suppurative Nephritis



Focal (Embolic) Nephritis

- Infection is hematogenous due to presence of localized bacterial colony
- Specific pyogenic infections as Shigilla equiruilis, Streptococcus, Escherichia coli and Corynebacterium pyogenes. Which reach the kidneys via the blood stream
- It occurs during course of bacteremia or septicemia as in case of septic endocarditis, septic metritis, septic pneumonia

Focal (Embolic) Nephritis

Macroscopical findings:-

- Few or many abscesses of variable size are found in the cortex
- These abscesses have the size of pin headed or larger and surrounded by hyperemic zone
- Suppurative inflammation is usually cortical
- On cut section medulla is usually free only linear grayish extension from large cortical suppurative foci
- If healing occur, a scar area formed
- The capsule is adherent to the area of fibrosis

Focal (Embolic) Nephritis

Macroscopical findings:-

- Both kidneys are usually involved
- Few or many abscesses of variable size are found in the cortex
- These abscesses have the size of pin headed or larger and surrounded by hyperemic zone
- Suppurative inflammation is usually cortical
- On cut section medulla is usually free only linear grayish extension from large cortical suppurative foci
- If healing occur, a scar area formed
- The capsule is adherent to the area of fibrosis

Focal (Embolic) Nephritis

Macroscopical findings:-

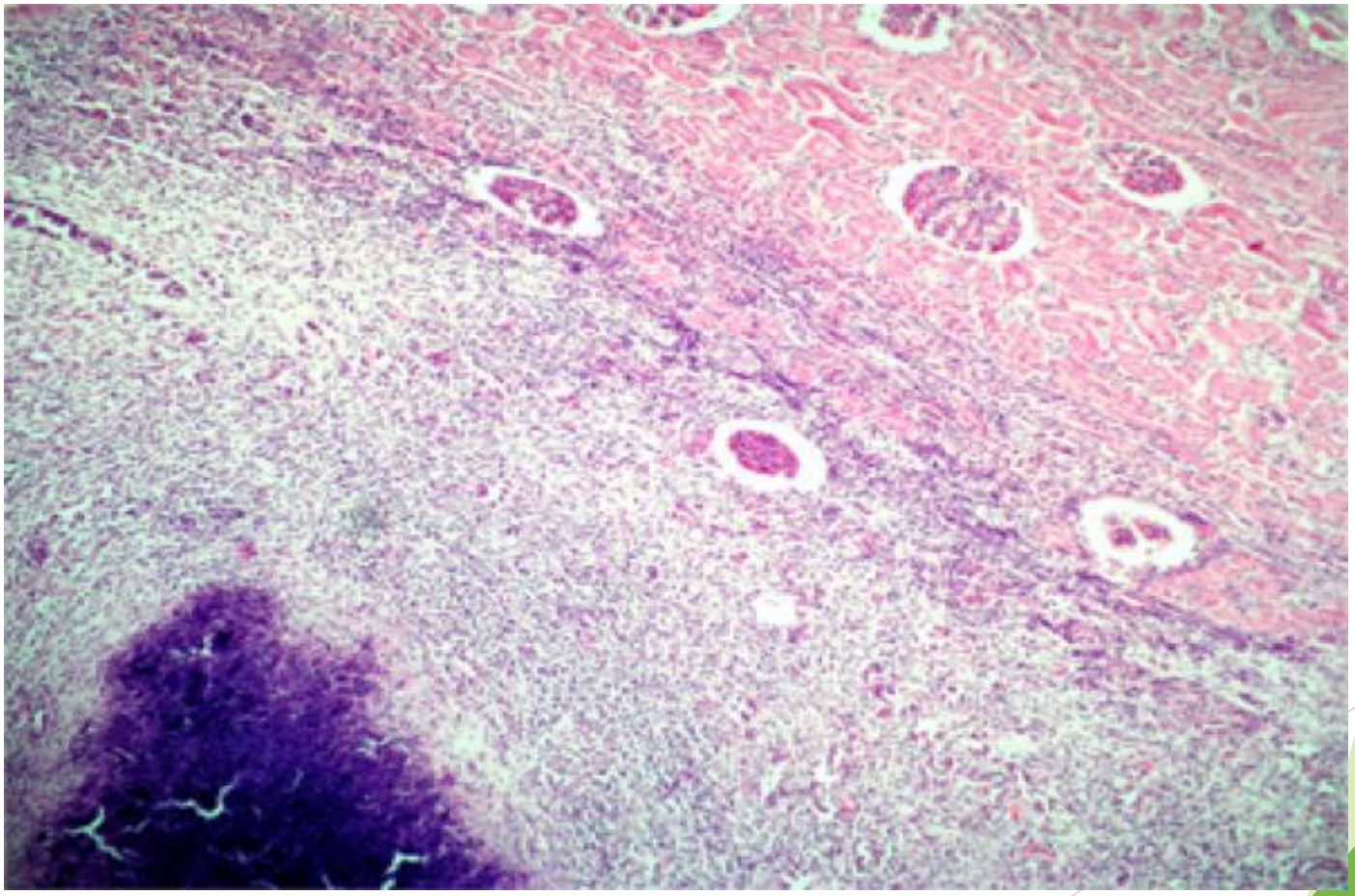
Focal (Embolic) Nephritis

Microscpical appearance:-

- Leukocytic infiltration in the interstitial tissue
- Aggregation of neutrophils in renal tubules with pus as a basophilic material around renal tubules or infiltrated lumen of renal tubules.
- Or abscess in renal parenchyma surrounded by line of defense (dilated blood vessels, fibrin threads & neutrophils).
- Tubular epithelium undergo necrosis
- Bacterial colonies could be detected in the lumen of renal tubules

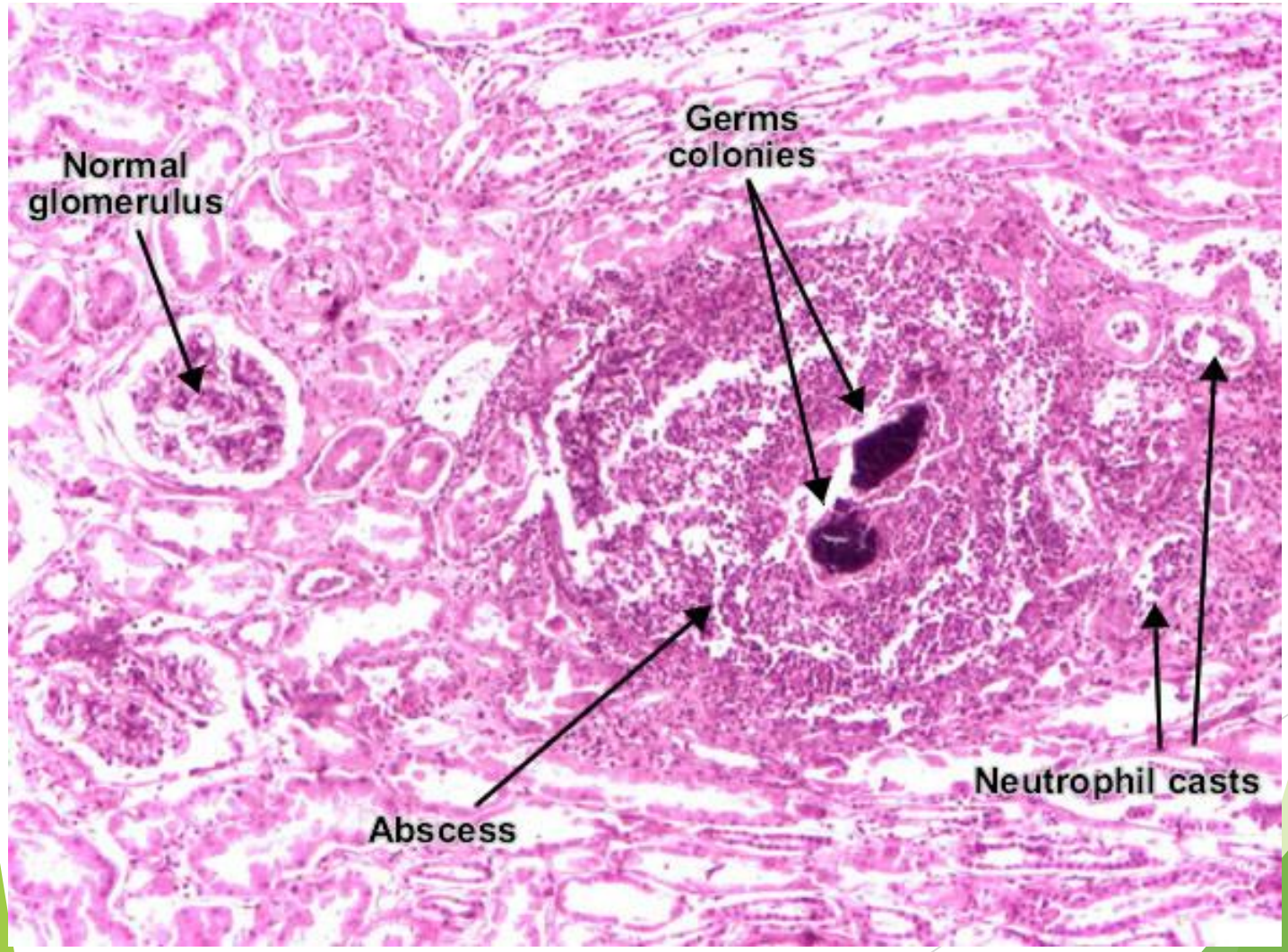
Focal (Embolic) Nephritis

Microscopical findings:-



Focal (Embolic) Nephritis

Microscopical findings:-



Diffuse (pyelonephritis) Nephritis

- It is an inflammatory condition which involve the renal pelvis (peylitis) and renal medulla (nephritis)
- Infection usually enters through the lower urinary tract specially after parturition.
- It is an ascending (Urogenic) from U.B. → through the ureter to the renal pelvis → kidneys
- It may be due to ***Corynebacterium renalis.***

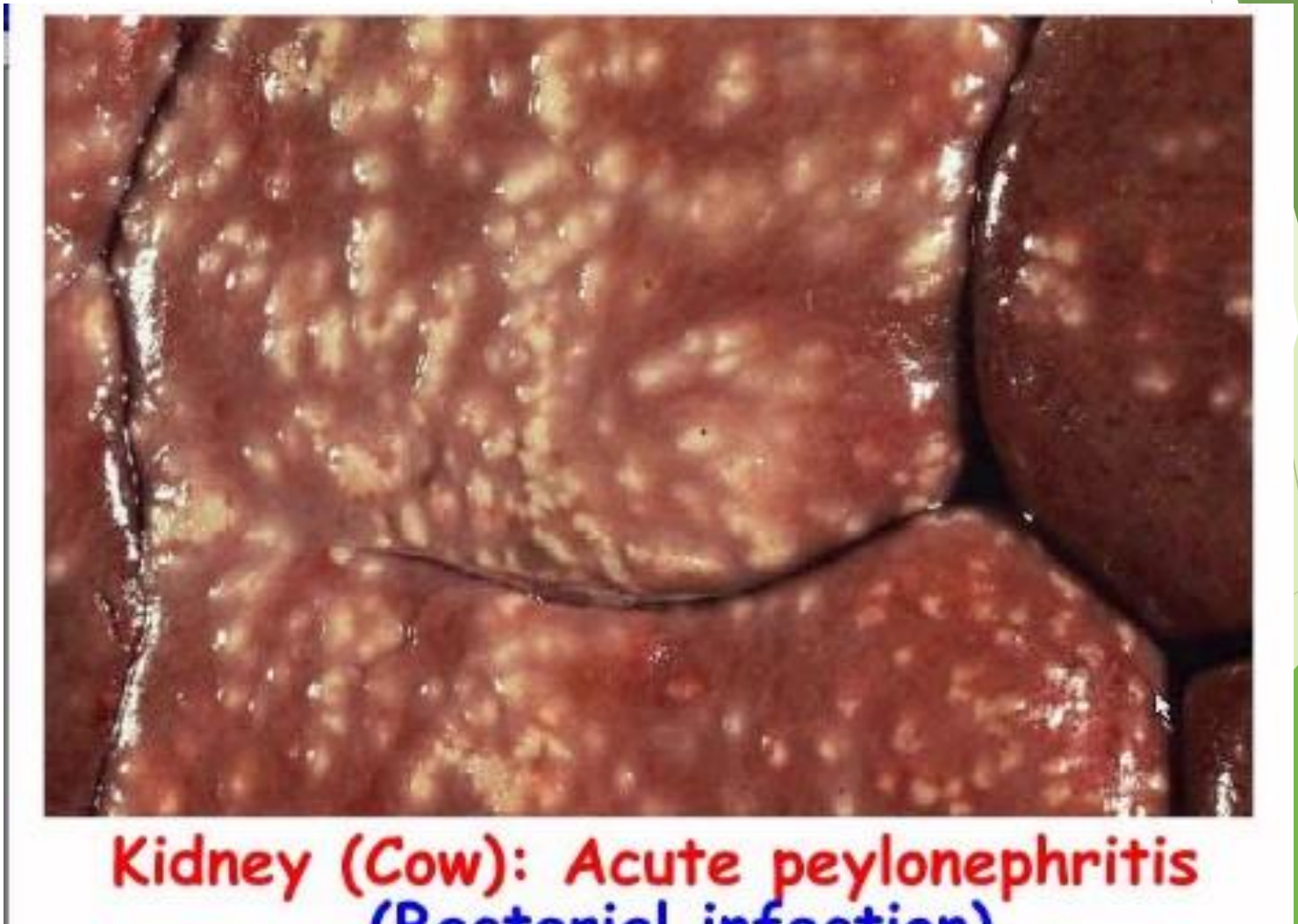
Diffuse (pyelonephritis) Nephritis

Macroscpical appearance:-

- One or both kidneys may be affected.
- Grayish white foci are seen on the renal surface.
- Cut surface shows sloughing and necrosis of the renal papillae.

Diffuse (pyelonephritis) Nephritis

Macroscpical appearance:-



Kidney (Cow): Acute peylonephritis

Diffuse (pyelonephritis) Nephritis

Kidney (Cow): Acute peylonephritis

White pale streaks through the cortex

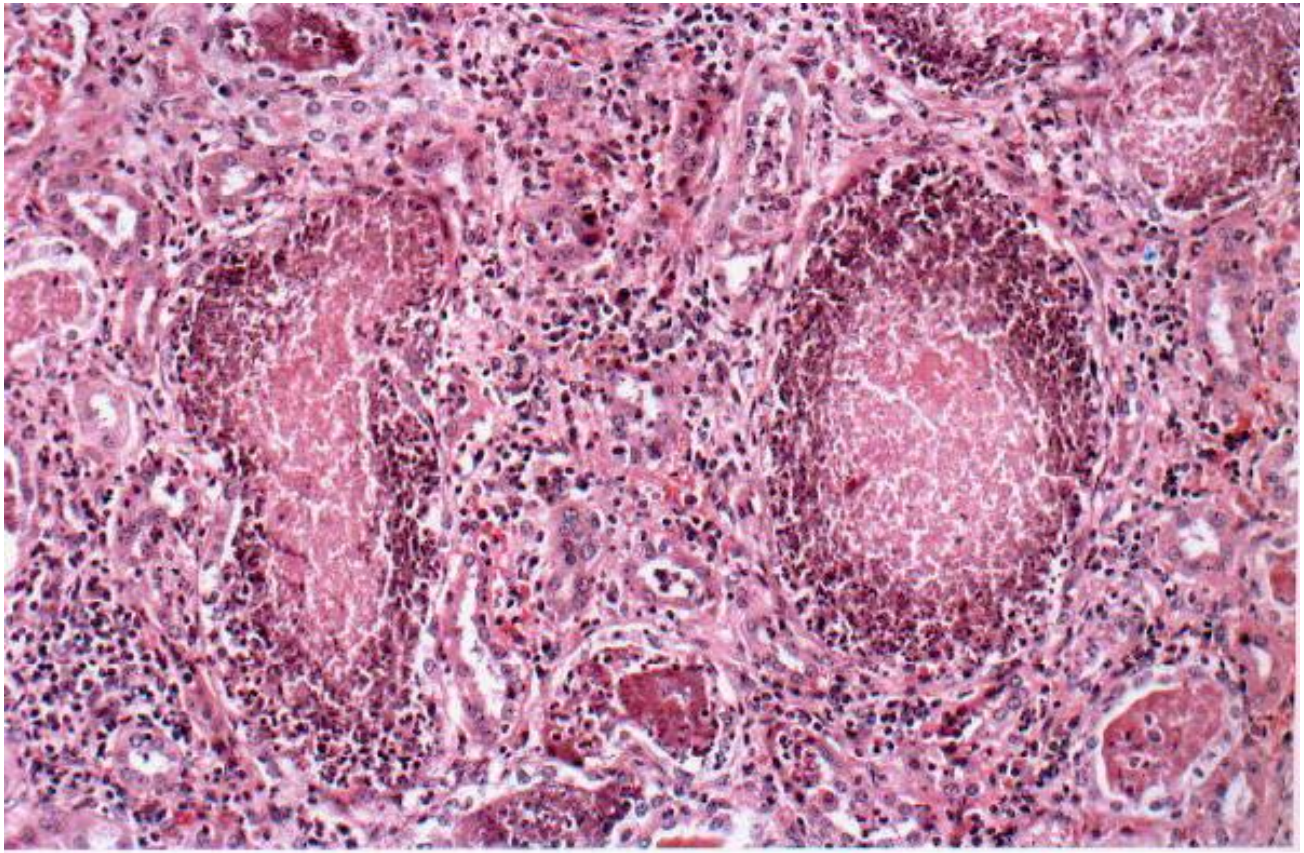
Diffuse (pyelonephritis) Nephritis

Microscpical appearance:-

- The glomerular tuft and Bowman's capsule are infiltrated with neutrophils
- Bacterial colonies could be detected in the glomerular tuft or in the lumen of renal tubules
- Lining epithelium especially of collecting tubules are necropsied and their lumen are filled with cellular casts

Diffuse (pyelonephritis) Nephritis

Microscpical appearance:-



Pyelonephritis
Infiltration of neutrophils and abscess formation in the interstitial tissue in the medullary region

Lower nephron nephritis (L.N.N)

1) Sulpha drug:-

- Precipitate in renal tubules as needle crystal inside renal tubules causing atrophy while the part above affected portion showing cystic dilatation.
- The most affected parts, ascending loop of Henel & distal convoluted tubules, so, it is called L.N.N.
- 2) Plant oxalate:- some plants contain excessive amount of oxalate.
- (3) Excessive hemorrhage: - causing Hb urea which closes renal tubules causing lower nephron nephritis.

Lower nephron nephritis (L.N.N)

[4] Myoglobin urea or Monday resting disease.

- In which occur crushing of muscles, then myoglobin release lead to lower nephron nephritis, it is called azot uria.
- The renal tubules are destructed & having dark brown color.

Mechanism →

- When horse work after rest, causing increase in glycogen metabolism of muscle lead to formation of excessive lactic acid which cause necrosis & destruction of muscle.

Renal Tumor

Primary tumors:

are rare and include

- Adenoma, renal carcinoma, fibroma, fibrosarcoma, hemangioma

- **Nephroblastoma:-**
- **is a malignant tumor occur during embryonic stage inside uterus special in pig & rabbits.**

  This tumor arising from renal tissue in embryonic stage

Renal Tumor

Secondary tumors

- They are due to spread of some malignant tumors as the kidney is a common site for metastasis of malignant neoplasm arising from other organs as in case of

lymphosarcoma:-

- Mainly in cattle
- Lymphosarcoma may be primary in kidney of poultry suffering from leukosis

Ureter

Aplasia

Inflammation: ureteritis

- may be a part of inflammation of the whole urinary tract.

Dilatation:

- in case of pyelonephritis.
- Or in case of partial obstruction of UB or urethera

Narrowing:

- may be congenital or inflammatory.

Blocked:

- May be blocked by calculi descending from the renal pelvis

Cystitis

- inflammation of Urinary bladder under normal condition is rare due to resistance to infection by continuous elimination of microorganism during normal urine outflow
- The predisposing cause is stagnation of urine or trauma by calculi
- Cystitis is higher in incidence in female than in male
- It may be acute or chronic
- Acute one may be, catarrhal, fibrinous, purulent or hemorrhagic cystitis

Cystitis

Chronic cystitis

- In which the wall is thickened and nodular and grayish-white in colour

Causes of cystitis: →

- E.coli, Staph, T.B., Corynebacterium and streptococcus spp.
- stagnation of urine or
- trauma by urolith

Urethra

Urethritis

- is common in animals due to urolith and corynebacterium renale infection.

Obstruction of urethra

- by urinary calculi is frequent in sigmoid flexure of steers and sheep

Urolithiasis or nephrolithiasis

- It is the formation of stony precipitate anywhere in the urinary passages
- The stone is called urolith or urinary calculus.
- The calculi are most common in the bladder and called cystic calculi.
- It is considered an important cause of urinary tract disease mainly in dog, cat, sheep and goats

Urolithiasis or nephrolithiasis

Factors important in the formation of calculi

- High level of oxalate, phosphate and magnesium
- Vitamin A deficiency
- Metabolic defect in uric acid metabolism resulting in uric acid calculi
- Hyperparathyrodism resulting in increase in ca and ph
- Urine pH
- Reduce water intake

Urolithiasis or nephrolithiasis

Characters of calculi

- Calculi may have smooth or rough surface
- It may be solid, soft or friable
- Their colour may be gray, white, brown or yellow depending on their composition
- Their effect depend on their size as large calculi may cause urinary obstruction which more common in male than female due to long and narrow urethera

Urolithiasis or nephrolithiasis

Characters of calculi

- **The chemical composition of calculi are varies greatly according to animal species**

1) In herbivorous animals:

- **The calculi formed mainly from *silicate* which may be conjugated with phosphate, carbonate,or oxalate of calcium or magnesium due to alkaline pH of urine**

2) In carnivorous animals:

Formed mainly from calcium oxalate due to acidic pH of urine, which is very hard and sharp edges causing damage to urinary epithelium

Urolithiasis or nephrolithiasis

Effect of calculi

- Calculi produce mechanical irritation to the urinary epithelium
- Partial obstruction lead to hydronephrosis
- Complete obstruction lead to uremia
- Obstruction of urine outflow resulting in dilatation or rupture of urinary bladder