

# PATHOLOGY OF DIGESTIVE SYSTEM

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# MALFORMATIONS

- Cleft lip (**Chelioschisis**) cleft palate (**Palatoschisis**) Clefts of the mouth are midline fusion defects which occur during embryonic development
- Ingesta is likely to enter the respiratory passages and cause aspiration pneumonia and death



Cleft palate in a calf

# S

- ❖ Caries means decay of teeth in which the enamel is decalcified followed by softening and discoloration
- ❖ Caries is rare in domestic animals
- ❖ Occurs occasionally in pet dogs with imbalanced and inadequate diets

## Etiology

- ❖ Disturbances in calcium and phosphorus metabolism as well as dietary deficiencies of these minerals
- ❖ It is frequently seen in fluorine poisoning

## Pathogenesis

- ❖ The organic acids especially lactic acid, that are formed due to the action of bacteria on carbohydrates dissolve the salts of the enamel
- ❖ Same acids corrode into the less strong dentine, which contains in its structure 30% of protein
- ❖ The damage to the dentine is deeper and most widespread

## Gross Pathology

- ❖ Affected teeth usually have one or more depressed areas brown or black in colour
- ❖ In the early incipient stages, “Enamel Flecks” which are yellow stained spots on the enamel can be seen

# STOMATITIS

- This is diffuse inflammation of the mucous membrane of the mouth
  - **Gingivitis** for inflammation of the gums
  - Glossitis for inflammation of the tongue
  - **Lampas** for inflammation of the palate
  - **Cheilitis** for inflammation of the lips
  - **Pharyngitis** for inflammation of the pharynx
  - **Tonsillitis** for inflammation of the tonsils

## Occurrence

Stomatitis is common in animals

## Aetiology

It may be a primary affection or may occur as secondary to other diseases

### Physical

- Trauma by awns, thorns, burrs, wood pieces, glass pieces, sharp bits, irregular sharp teeth, sharp edged feeding utensils
- Thermal injuries: Hot drenches and eating frozen foods

### Chemical

- Caustic alkalies, corrosive acids, fertilizers

### Deficiency of vitamins

- Hypovitaminosis A especially in fowl
- Niacin deficiency : Black tongue in dogs

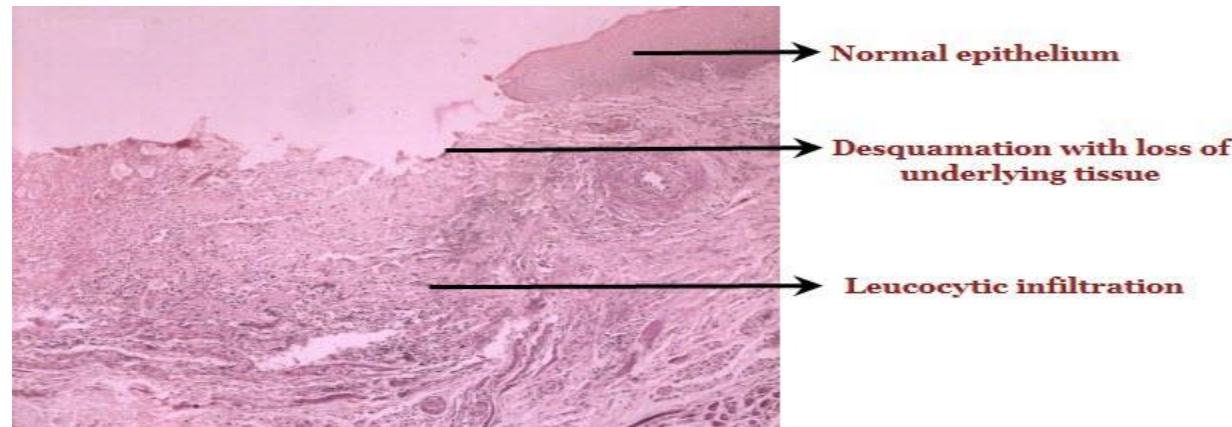
### Microorganisms

- **Bacteria:** *Actinomyces bovis*; *Actinobacillus lignieresii*; *Fusobacterium necrophorum*; *Pseudomonas aeruginosa*; *Corynebacterium pyogenes*; *Streptococci* and *Staphylococci*
- **Fungi:** *Candida albicans*
- **Viruses:** Foot and Mouth disease; Rinderpest; Virus diarrhoea -mucosal disease; Infectious canine hepatitis; contagious ecthyma; vesicular exanthema; fowl pox and blue tongue



Oral ulcers in a piglet

- The lesion starts as catarrhal inflammation of the mouth and pharynx with reddening and swelling of the mucosa which is covered by small, whitish raised spots (**Aphthous stomatitis**)
- These spots may latter develop into small crusts or into ulcers



Ulcer in the tongue

# **TYPES OF STOMATITIS**

## **Vesicular Stomatitis**

- ❖ Vesicles, blebs or blisters containing clear fluid are formed on the mucosa
- ❖ Seen in Foot and mouth disease, infectious vesicular exanthema and infectious vesicular stomatitis

## **Fibrinous and Necrotic Stomatitis**

- ❖ This is seen in infection by *Fusobacterium nerophorum*

## **Diphtheritic Stomatitis**

- ❖ Fowl pox produces diphtheritic stomatitis and pharyngitis in which a grayish membrane is found

## **Gangrenous Stomatitis**

- ❖ Very severe irritants may cause gangrenous stomatitis

## Thrush

- Thrush is found in birds
- Grey or yellowish thick tenacious material gets attached to the mucous membranes
- Caused by *Candida albicans*

# PATHOLOGICAL CONDITIONS IN SALIVARY GLANDS

## Foreign bodies

- ❖ Occasionally found in the ducts especially of the parotid and sub maxillary glands
- ❖ These are usually awns, wood pieces and kernels of grain causing inflammation
- ❖ Sometimes, these may produce obstruction and consequent dilatation of the ducts

## Dilatation

- ❖ Dilatation of the salivary ducts may occur when the flow of saliva is obstructed by foreign bodies, inflammatory exudates etc.

## Ranula

- ❖ Dilatation of the salivary duct and gland occurs as cyst on the floor of the mouth it is called a ranula
- ❖ This is smooth, rounded cyst containing a clear fluid
- ❖ This can be easily ruptured

## Sialoliths

- ❖ Sialoliths are salivary calculi
- ❖ These are common in horses
- ❖ Salivary calculi are usually single and sometimes may be very large preventing the flow of saliva
- ❖ These calculi produce stasis, distension of ducts and finally atrophy of the gland

## Sialadentitis

- ❖ Inflammation of the salivary glands
- ❖ It is very rare in animals
- ❖ It may be due to traumatic injury or due to infection by bacteria

## Gross pathology

- ❖ The glands are swollen and red
- ❖ Abscesses may be found in glands
- ❖ Sometimes, cystic dilatations may occur

## Sequelae

- ❖ Stasis of saliva in the ducts facilitates infection

Neoplasms of salivary glands are not common in animals

# OESOPHAGEAL CHOKE

Choke is obstruction of the oesophagus

It occurs in horses and cattle, but more common in the former

## Aetiology

- ❖ In cattle, large objects of food- beet root, carrot, apples, potatoes, fetal membranes, sticks and wire
- ❖ In dogs, large bones, Impacted masses of feed due to improper chewing, bad teeth and rapid gulping of dry feed
- ❖ Lesions of oesophagus: stenosis or diverticulum cause repeated choking
- ❖ Enlarged lymph nodes- mediastinal and cervical
- ❖ Neoplasms of adjacent tissue especially thymus - thymoma in new-born animals

## Gross pathology

- ❖ In the horses choke occurs in the thoracic area while in cattle and dogs the pharynx is obstructed

Choke may be complete or incomplete

### *Complete choke*

- ❖ In complete choke, feed will be returned and water will flow through the nostrils when animal is watered
- ❖ Aspiration of the feed will cause secondary foreign-body pneumonia
- ❖ In cattle, complete obstruction will cause dangerous tympany
- ❖ Because of pressure, ischemia and resultant necrosis and gangrene may develop

### *Partial choke*

- ❖ Partial obstruction will give rise to dilatation of esophagus above the obstruction - the esophageal diverticulum

### **Sequelae**

Death due to gangrenous pneumonia, bloat, cellulitis or asphyxiation

Esophageal diverticulum

Rupture of esophagus

### **Ectasia**

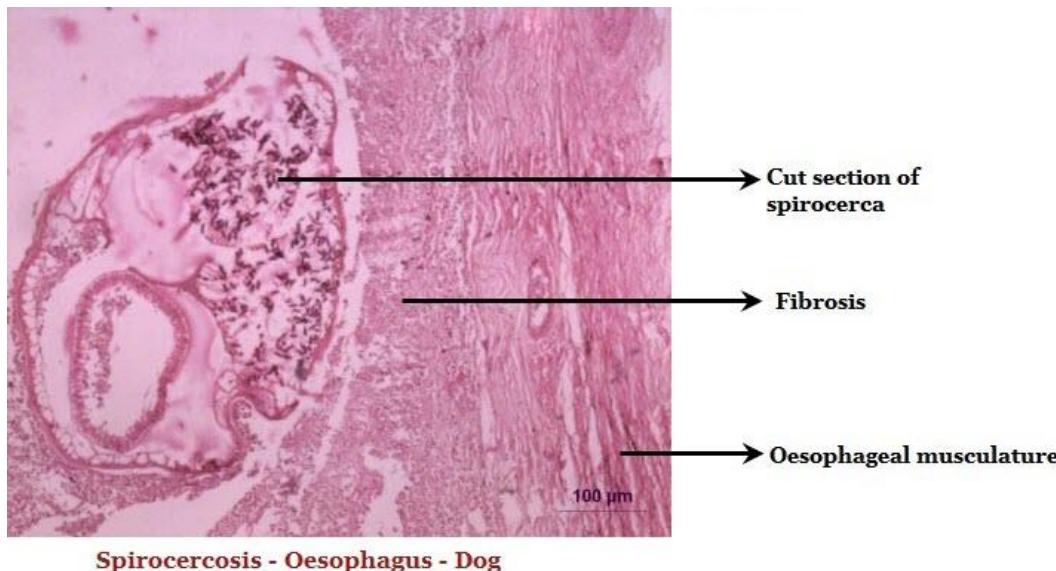
Dilatation of oesophagus

### **Oesophagitis**

This is inflammation of the oesophageal mucosa

# NEOPLASMS OF THE OESOPHAGUS

- ❖ Neoplasms of the oesophagus are not common
- ❖ In the thoracic portion of the oesophagus, fibrosarcomas and osteogenic sarcomas are found in connection with *Spirocercus lupi* infection
- ❖ The osteogenic sarcoma is evidently a metaplastic manifestation of the fibrosarcoma
- ❖ Metastases of these tumors are sometimes found in the lungs and other tissues
- ❖ Carcinoma in cat and horse and papilloma in cattle



# INGLUVITIS

Inflammation of crop is called **ingluvitis**

**Acute catarrhal ingluvitis**

*Aetiology*

Trauma by foreign bodies

Chemical agents : phosphorus, fertilizers

Toxins from decomposed food

Infectious diseases

Parasites – *Acuaria sp* ; *Capillaria sp*

*Gross pathology*

Lesions include cogestion, edema and tympanites

**Diphtheritic ingluvitis**

This is found in fowl pox

# TYMPANITES OR BLOAT

Bloat (or accumulation of gas) can occur

- When the gas is produced at too rapid a rate than can be eructated
- When the eructation mechanism is faulty

## Types

- ❖ Based on course, bloat may be acute or chronic
- ❖ Based on nature of gas, bloat may be dry or frothy

## Acute bloat

- ❖ This may be due to complete choke in esophagus
- ❖ It may also be due to sudden changes of feed
- ❖ Excessive feeding on legumes that are wet with dew or rain

## Chronic bloat

- ❖ The chronic variety occurs whenever there is any hindrance to eructation in the esophagus either within or without pressure by tumors, foreign bodies, enlarged lymph nodes, abscesses, constrictions or diverticula
- ❖ It may occur in lesions of the rumen causing decreased contractions of the ruminal wall as in atony, serosal adhesions, paresis, diffuse lymphomatosis

## Dry bloat

- ❖ The dry bloat is less harmful, since in this condition, the gases can be more easily got rid of by eructation

## Frothy bloat

- ❖ In the frothy bloat, the gas is trapped as small bubbles in the fluid forming a foamy mass which is not easily eructated

## Aetiology

- ❖ Saponin found in plants is a good saponifying agent
- ❖ Water-soluble proteins of the legumes are capable of forming froth
- ❖ Factors which increase the viscosity and lowers the surface tension of ruminal contents produce froth
- ❖ Normally, in rumen due to bacterial activity, fatty acids are produced which increase the surface tension
- ❖ If the production of these fatty acids is decreased, the surface tension will be lowered favouring froth production
- ❖ This is the theory behind the use of vegetable oils in the treatment of bloat

## Pathogenesis

- ❖ Some legumes contain HCN, which is toxic, causing paralysis of the ruminal or reticular musculature and so inhibits eructation
- ❖ Some legumes contain phosphatase which with arsenates accelerates fermentation producing a large quantity of CO<sub>2</sub>
- ❖ H<sub>2</sub>S, CO<sub>2</sub> and CO produced in large quantities causes paralysis of ruminal muscles
- ❖ If fed excessively on green plants only, which do not contain sufficient stiff fibres, the mucosa of the rumen is not adequately scratched to elicit the reflex contraction of the musculature
- ❖ Mechanical stimulation of cardia, especially by roughages, increases the rate of secretion of saliva
- ❖ Ruminal mucinolytic bacteria may destroy salivary mucin thereby producing frothy bloat
- ❖ Polysaccharides produced by capsulated ruminal bacteria may be another etiological factor in bloat
- ❖ Interference with the nerve pathways that are responsible for the eructation reflex may also lead to tympany
- ❖ The receptors for this reflex are in the reticulum and the afferent and efferent nerve fibres are in the vagus nerve
- ❖ Distended rumen compresses other abdominal organs and causes passive congestion since the pressure on thin-walled veins impedes circulation
- ❖ Along with this, there is forward thrust on the diaphragm, pressing on the lungs, which become smaller and sometimes atelectatic
- ❖ The result of this is hypoxia and ultimate asphyxia and death

## Gross pathology

- ❖ In animal that dies of bloat, besides congestion of the abdominal viscera, one may notice haemorrhages on the pleura, pericardium, tracheal mucosa and on bronchial lymph nodes as well as in the lymph nodes of head and neck
- ❖ Blood is tarry, as in Anthrax
- ❖ Liver is pale
- ❖ The rumen or diaphragm may be ruptured sometimes
- ❖ Within few hours of death, the ruminal epithelium peels off

# IMPACT OF THE RUMEN AND RETICULUM

- ❖ It occurs when rumen stops functioning, the musculature does not contract and so the food ingested stagnates
- ❖ This is a common condition in cattle

## Predisposing causes

- ❖ Lack of exercise and debility predispose the animal to atony of rumen

## Aetiology

- ❖ Overfeeding with large amounts of highly fermentable carbohydrate feeds
- ❖ Tight packing of the rumen leaves no room for bacterial growth and normal ruminal fermentation and digestion
- ❖ This leads to weak contractions of the ruminal and reticular walls and so the food does not get propelled
- ❖ Lack of water
- ❖ Defective mastication and salivation due to defects in teeth or lesions of the tongue
- ❖ Penetration of the wall of the rumen or reticulum by sharp objects like wire, nail etc.
- ❖ Paresis of rumen which may occur due to injury to vagus by pressure from abscesses, tumors, tubercular nodules, swollen lymph glands and ruminal displacements

## Pathogenesis

The pathogenesis of atony and impaction of the rumen after ingestion of large quantities of carbohydrate rich feeds is as follows

- ❖ The carbohydrates are fermented by gram positive organisms, notably *Streptococcus bovis*, with the formation of lactic acid, resulting in to **lowering of pH of the ruminal contents** to as low as 4 to 4.5 from a normal 5.5 to 7.5
- ❖ Due to the production of lactic acid the **osmotic pressure of ruminal contents increases** and so fluid is drawn into the rumen from the blood leading to hemoconcentration, anuria, dehydration and circulatory collapse
- ❖ As the pH of the ruminal constituents falls, the **motility of the rumen decreases** and there may even be complete **stasis**
- ❖ At the lowered pH, normal microfloras of the rumen are destroyed, the lactobacilli and streptococci thrive and the salivary secretion ceases so that **buffering action of the saliva is absent**
- ❖ Absorption of the lactate causes **acidosis**.

## Gross pathology

- ❖ At necropsy, the rumen will be found to contain hard, caked, undigested food with evil smelling odor
- ❖ In animals that die of acute atony, the contents of the rumen and reticulum are thin, porridge-like and bulky
- ❖ The cornified epithelium is soft and peels off easily, exposing hemorrhagic areas underneath
- ❖ The blood is dark and thick
- ❖ Heart musculature is flabby

## Histopathology

- ❖ In animals that survive for three days and more, demyelination of the nervous system may occur
- ❖ Sequelae
- ❖ In mild cases, if the primary cause is removed, normal state may be regained
- ❖ In severe cases, toxæmia will cause death
- ❖ Enteritis, peritonitis and ketosis

# TRAUMATIC RETICULITIS

This is a very common condition in older cattle

## Pathogenesis

- ❖ These animals ingest and swallow, along with their feed, a wide variety of sharp objects like needles, nails, pieces of fencing wire and screws
- ❖ Sometimes contraction of the rumen and reticulum during pregnancy cause the sharp object to pierce the wall of the reticulum
- ❖ Usually, it pierces the antero-ventral wall, which is near the diaphragm
- ❖ The passage through it is usually slow and so the track formed by the moving nail is thickened by a dense fibrous wall
- ❖ Piercing the diaphragm, the foreign body may enter the pericardium and even the heart, producing inflammation enroute
- ❖ Traumatic pericarditis with serofibrinous or purulent exudates may occur
- ❖ Sometimes, it may take a downward slope and pierce the chest wall near the xiphoid cartilage forming an abscess there

- ❖ At the point where the object pierces the reticulum, a localized peritonitis develops causing adhesions between the reticulum and the diaphragm at this place
- ❖ The sharp object may, sometimes, penetrate the lungs or the liver or the spleen causing abscesses in these organs
- ❖ Penetration of vagus nerve may result into vagal indigestion

### Gross Pathology

- ❖ The thick-walled blackened track followed by the foreign body and adhesions of the reticulum to the diaphragm may be clearly seen
- ❖ Along the track, fistulae connecting one abscess to the other may be found
- ❖ Fibrinous pericarditis with hypertrophied myocardium may be seen if foreign body has entered the pericardium

### Sequelae

- ❖ If the sharp object is not contaminated, only mechanical injury to the affected parts is seen
- ❖ Vagus indigestion, when the ventral branch of the vagus is affected by the inflammatory and scar tissue formed by the penetrating foreign body
- ❖ Diaphragmatic hernia may occur due to weakening of the diaphragm by lesions produced by the foreign body
- ❖ Abscesses of lung, liver and spleen
- ❖ Fatal because if the sharp object penetrates any larger artery

# MALPOSITIONS OF STOMACH

## Diaphragmatic hernia of stomach

- ❖ It may be met with in dogs and cats due to automobile accidents
- ❖ The diaphragm is ruptured and the stomach enters the thoracic cavity

## Abomasal displacement

This condition is met with more frequently after parturition

### Predisposing causes

- ❖ Atony of the abomasum due to feeding large quantities of concentrates
- ❖ Post- parturient diseases like milk fever, mastitis, metritis and ketosis may also cause atony of the abomasum

### Aetiology

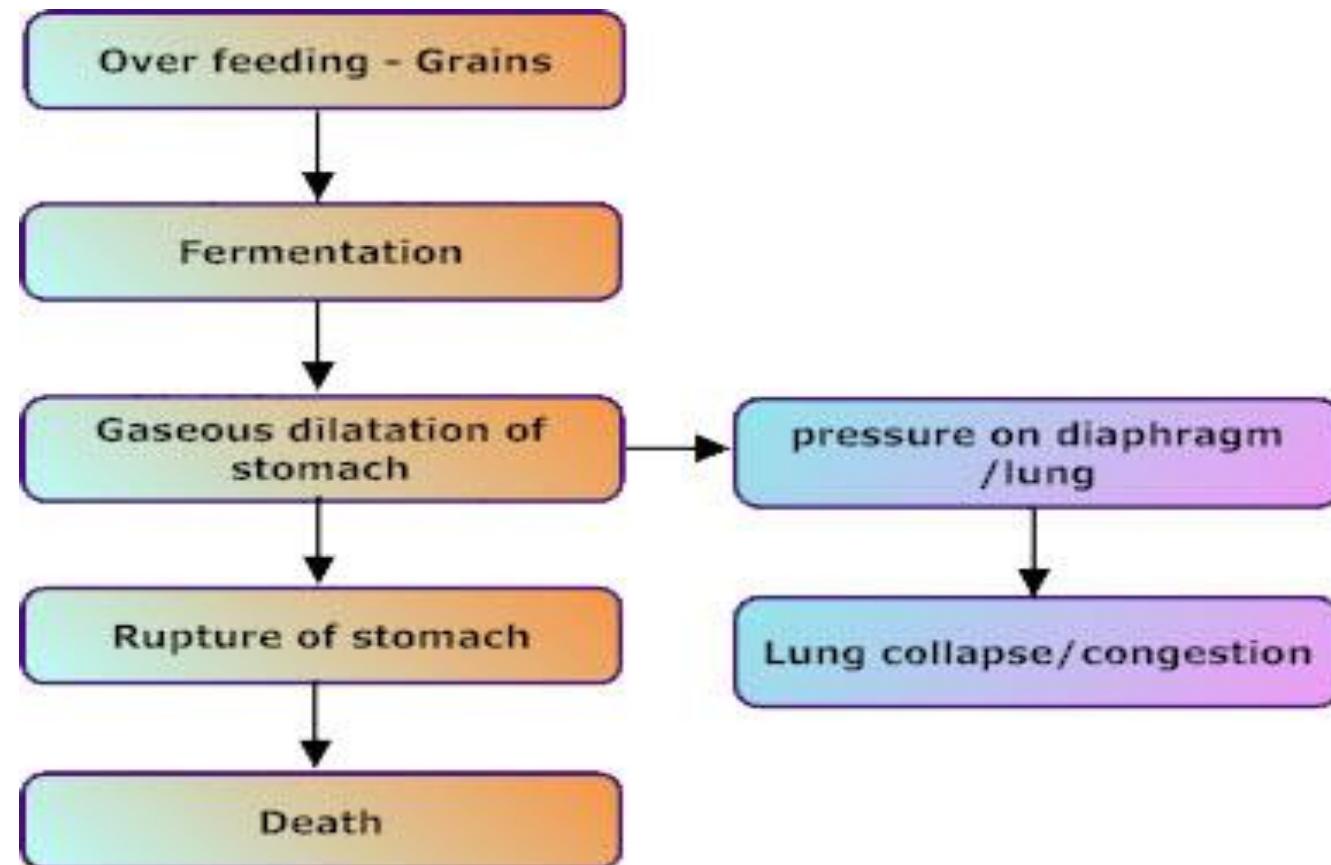
- ❖ A complication of surgical treatment for chronic indigestion
- ❖ Violent activity like jumping in estrus may be a cause in non-parturient cases
- ❖ A hereditary predisposition may exist

## Pathogenesis

- ❖ During pregnancy, the rumen may be lifted by the expanding gravid uterus and the abomasum may slip under the rumen
- ❖ After parturition, when the uterus recedes, the rumen drops to its normal position displacing the abomasum
- ❖ The greater curvature of the body of the abomasum which is more mobile slips under the ventral ruminal sac
- ❖ Atony of the abomasum due to feeding of concentrates after parturition prevents its correction

## Acute dilatation of the stomach

- It is due to excessive amounts of food or gas accumulating in the stomach
- Overeating; especially with grain in horse



# GASTRITIS

- ❖ Inflammation of the stomach is called gastritis
- ❖ Gastritis is a fairly common condition in animals
- ❖ Close confinement and unsanitary conditions where bacteria thrive contaminating feeds and feeding utensils

## Etiology

### Physical

- ❖ Faulty dentition prevents mastication
- ❖ Foreign bodies may traumatize the gastric mucosa
- ❖ Feeding very coarse material (eating bedding in horses and dogs)
- ❖ Feeding with frozen foods
- ❖ Overfeeding causing dilatation of stomach
- ❖ Feeding Spoiled, mouldy and fermented hay and silage or feeding easily fermentable foods
- ❖ When heavily fatigued animals are fed, the feed is not easily digested, stagnates, ferments and so produces irritation
- ❖ Too sudden changes of feed
- ❖ Toxic plants

## Chemicals

### Uremia

Caustic and corrosive chemicals like mercury, lead, copper, arsenic and phosphorus

### Stress

- ❖ In stress, adrenaline is produced in large quantities which is responsible for increased gastric secretion and gastritis.

### Bacterial

- ❖ In calves - enterotoxemia and colibacillosis; in pigs - erysipelas, vibrionic dysentery, salmonellosis and colibacillosis

### Viruses

- ❖ Pig- hog cholera; transmissible gastro-enteritis in baby pigs
- ❖ Cattle - rinderpest, mucosal disease
- ❖ In chicken - Ranikhet disease causes haemorrhagic proventriculitis

### Fungi

- ❖ Mucormycosis, moniliasis and aspergillosis cause gastritis in many animals

### Parasites

- ❖ Stomach worms - *Trichostrongylus sp.*, *Hemonchus sp.*, *Ostertagia sp.*, larval paramphistomes in ruminants
- ❖ Larvae of *Habronema sp.* and *Gastrophilus equi* in horses
- ❖ In pigs *Hyostrongylus rubidus*, *Ascarops strongylina* and *Physocephalus sexalatus*

# **TYPES OF GASTRITIS**

- ❖ **Acute gastritis**
- ❖ **Acute Catarrhal gastritis**
- ❖ **Acute hemorrhagic gastritis**
- ❖ **Chronic gastritis**
- ❖ **Parasitic Gastritis**

# **TYPES OF GASTRITIS**

## **Acute gastritis**

- ❖ Acute gastritis may be catarrhal, fibrinous, suppurative, haemorrhagic or necrotic, depending upon the cause and their severity
- ❖ By far the most common is the catarrhal and to a lesser extent, the hemorrhagic

## ***Pathogenesis***

- ❖ In gastritis, food does not get digested
- ❖ Motility of the gastric wall is retarded
- ❖ Irritation may produce pain and vomiting

## Catarrhal gastritis

### Gross pathology

- The gastric mucosa is covered with mucus
- The mucosa in some places may show ulceration
- The mucosa is thick and red

### Histopathology

- The mucosa shows catarrhal exudation, hyperemia and leucocytic infiltration
- Some of the gastric glands may be damaged and lost

## Acute hemorrhagic gastritis

This is a common condition

### Aetiology

- caustic chemical poisoning
- uremia
- acute infectious diseases like pasteurellosis, braxy, leptospirosis (in dogs)

### Gross pathology

- Due to haemorrhage, the mucosa is bright red in color and the gastric contents are blood stained
- Digested blood (acid haematin) imparts a brownish coloration to the contents

## Parasitic Gastritis

### Occurrence

- This is very common in animals

### Aetiology

- Cattle and sheep: *Hemonchus contortus*, *Ostertagia ostertagi*, *Trichostrongylus axei*
- Horses: *Habronema* larvae, *Trichostrongylus axei* and *Gastrophilus equi* larvae.
- Pig: *Hyostrongylus rubidus*, *Physocephalus sexalatus*, *Simondsia paradoxa*, *Ascarops strongylina*
- Cats: *Gnathostoma spinigerum*

### Pathogenesis

- The strongyles are blood suckers and they produce minute injuries on the mucosa
- The larvae may burrow into the mucosa for completion of their life cycle and thereby cause damage to the epithelium and glands
- Heavy infestation besides causing anemia will produce catarrhal gastritis

### Gross pathology

- *Gastrophilus* sp. in the stomach may produce ulcers
- *Habronema* larvae live in granulomatous nodules which may be infected by secondary bacteria and form abscesses

# Chronic gastritis

## Aetiology

- Usually the same causes as for the acute but operating for a longer time
- Sometimes it may be secondary to chronic gastric dilatation and cirrhosis

## Pathogenesis

- Ischemia in gastric dilatation and passive hyperemia and failure of detoxication in cirrhosis decrease the local resistance thereby facilitating infection

## Gross pathology

- The mucous membrane is thickened and covered with tenacious, viscid glassy mucus
- This condition is usually of a hypertrophic type with thickening of the gastric wall

## Histopathology

- There is exfoliation of the epithelium
- Hyperplasia of gastric glands and muscle fibres along with cellular infiltration and hyperplasia of basal lymphocytic nodules
- The mucosa may be thrown into polypoid folds (*polypoid gastritis*)
- The interstitial connective tissue hyperplasia exaggerates the mucosal foldings
- Occlusion of glands results into development of retention cysts

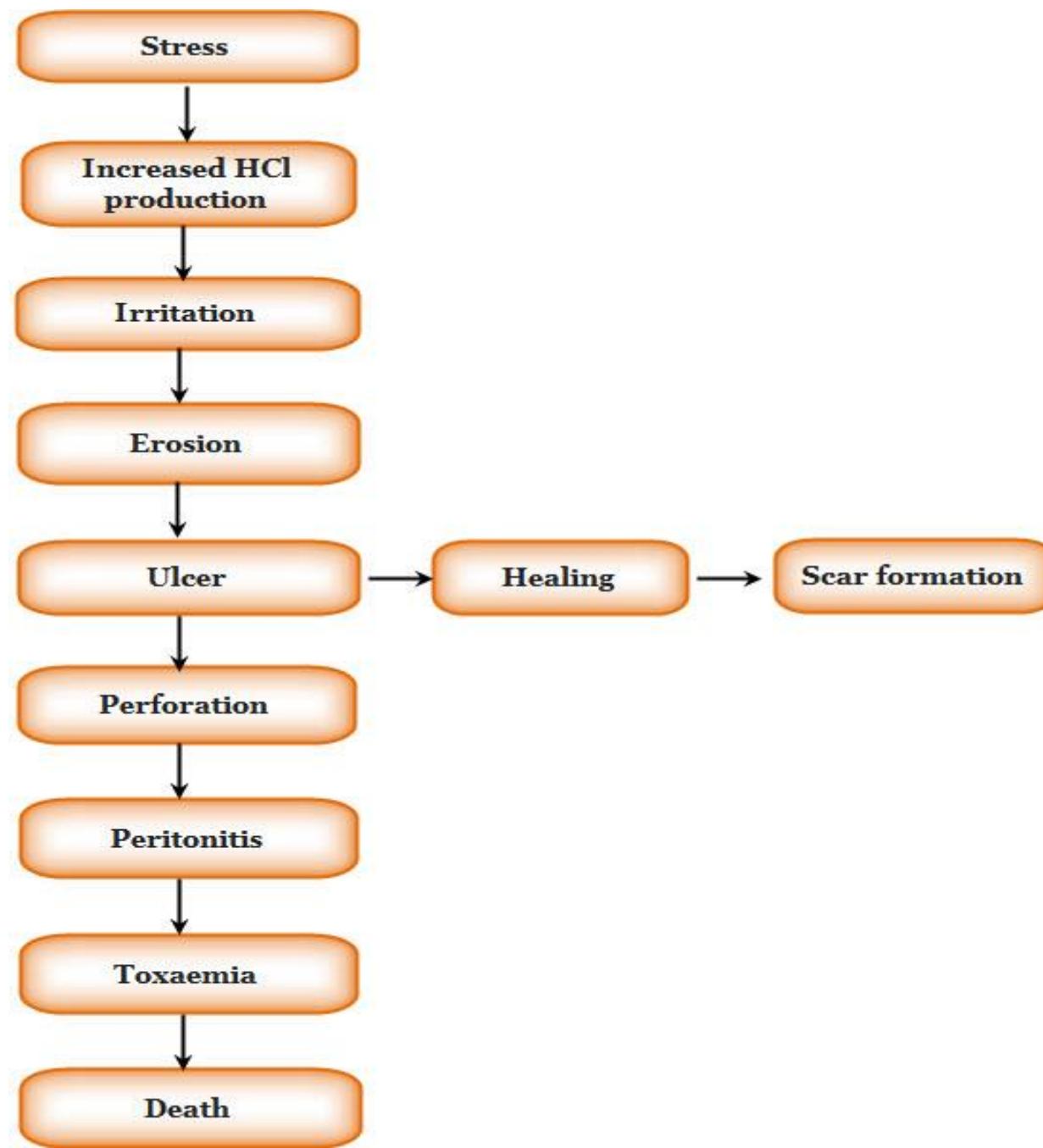
# GASTRIC ULCERS

## Occurrence

- ❖ Gastric ulcers are common among animals
- ❖ Small superficial defects are known as *erosions* which are common among ruminants
- ❖ Calves are more often affected

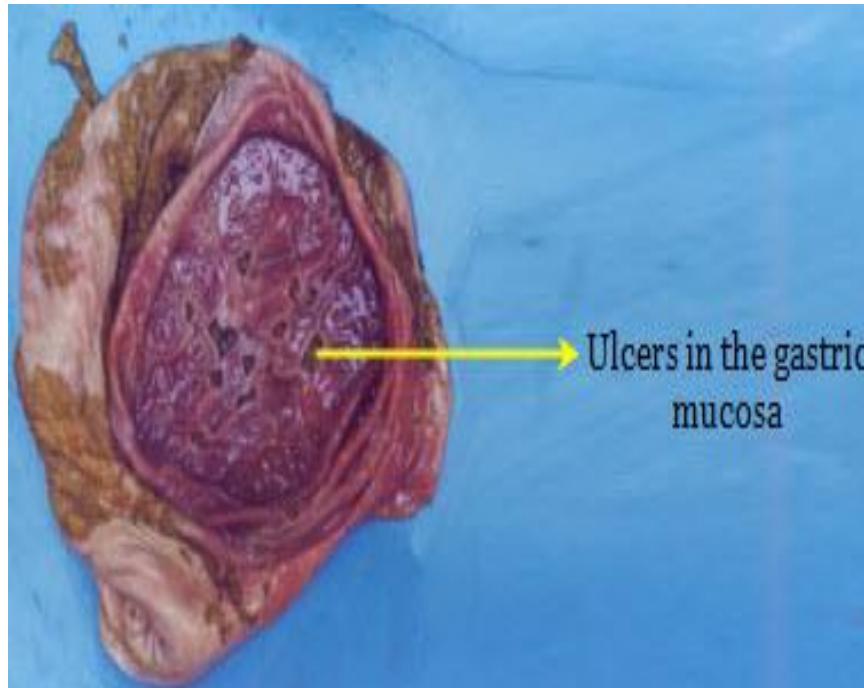
## Aetiology

- ❖ *Trauma*
- ❖ *Obstruction of pylorus in cattle*
- ❖ *Chemicals*
- ❖ *Nutrition*
  - Nutritional hepatic dystrophy (due to deficiency of vitamin E) in pigs
- ❖ *Infections*
  - *Viruses*
    - Erosions are common in the abomasum of cattle in rinderpest, mucosal disease, bovine malignant catarrh and pox
  - *Fungi*
    - *Mucor sp* and *Monilia sp* cause gastric ulcers in pigs
  - 
  - *Parasites*
    - In horses, larvae of *Gastrophilus* species and *Habronema megastoma*
- ❖ *Neoplasms of the stomach*

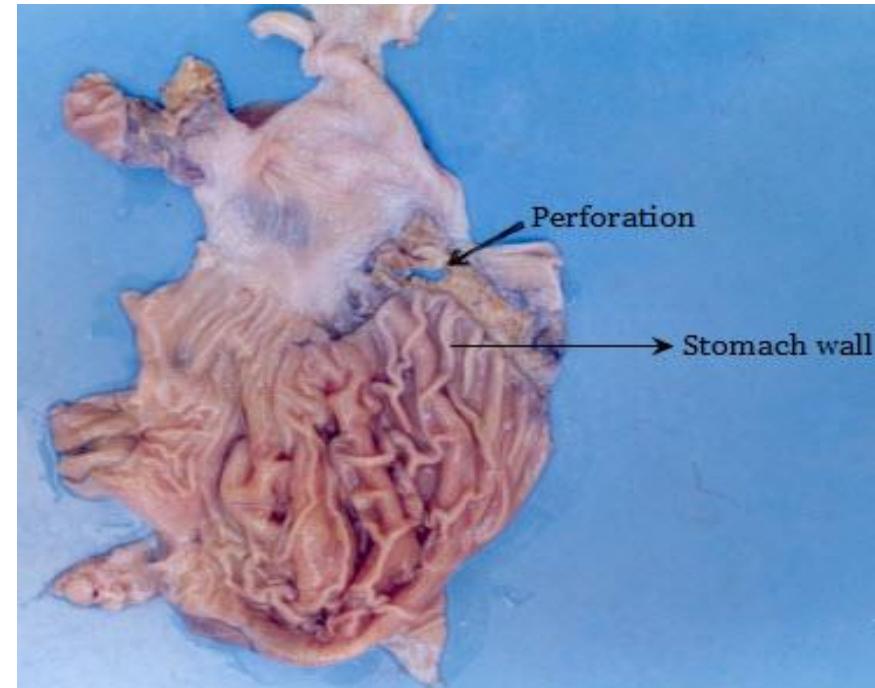


## Gross Pathology

- ❖ The mucosal erosions (in cattle) are of the size of a millet
- ❖ Slowly, by the action of the gastric juice the erosions may enlarge and become deeper to form ulcers
- ❖ The ulcers are usually demarcated, having raised borders with a punched-out appearance
- ❖ The base of the ulcers may be the sub mucosa or muscular coat or in some cases even the serosa
- ❖ When the serous coat is the base, perforations are likely to occur

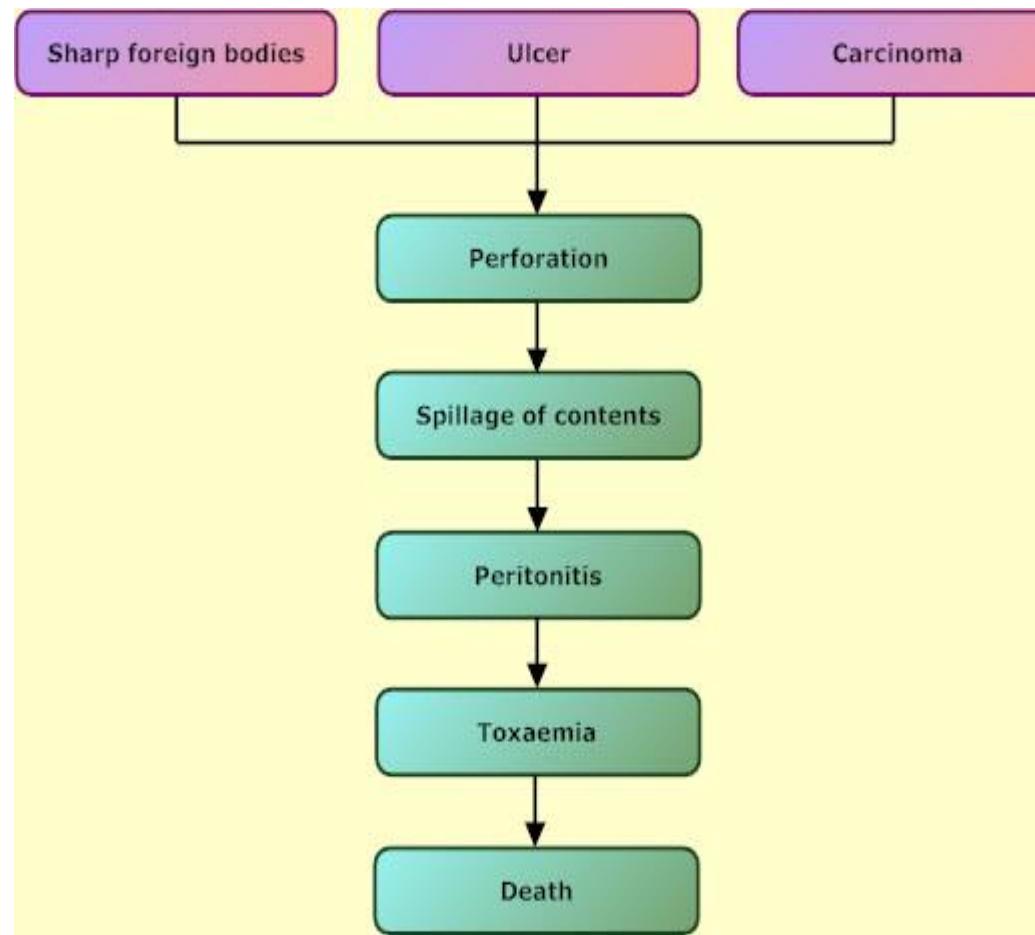


Ulcers in the gastric mucosa



## Histopathology

- ❖ The erosion is covered by an exudate consisting of mucus, fibrin and inflammatory cells
- ❖ The epithelium is exfoliated
- ❖ The sub-mucosa may be infiltrated by large number of leucocytes



# CONGENITAL ANOMALIES OF INTESTINE

## Atresia of the intestine

- ❖ It may be found as a hereditary defect among cattle

## Atresia of rectum

- ❖ In pigs and foals, atresia of rectum is an inherited lethal characteristic

## Imperforate anus

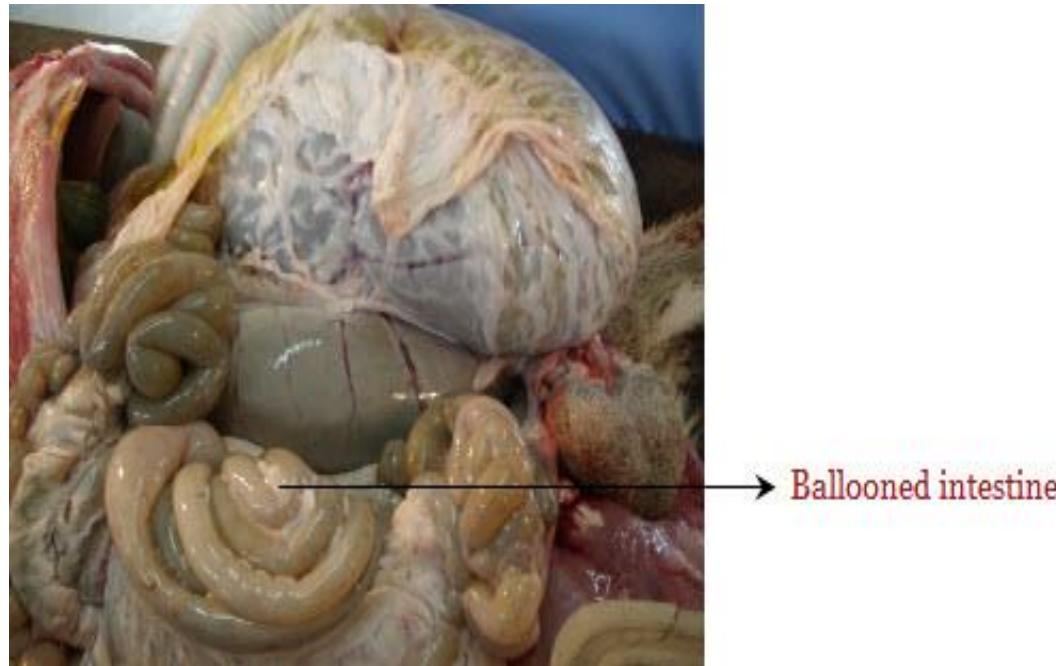
- ❖ In cattle, imperforate anus at birth is a semi-lethal character

## Persistent omphalomesenteric duct (Meckel's diverticulum )

- ❖ In pigs and horses, it is probably a hereditary defect
- ❖ It may become obstructed or inflamed or even ruptured resulting in peritonitis

# POSTMORTEM CHANGES IN INTESTINE

- ❖ Post-mortem changes in intestine include ballooning of intestine, thickening of intestinal mucus, blackening of intestinal mucosa (pseudomelanosis coli) and sloughing of intestinal mucosa



→ Ballooned intestine

# MECHANICAL OBSTRUCTION

- Congenital
- Atresia and imperforation of intestine
- Acquired
- *Stenosis*
  - Due to pathological lesions: hematoma, neoplasms, abscesses, chronic inflammatory scars, displacements like torsion, volvulus, intussusception and hernia
- *Impaction*
  - Foreign bodies like bone, stones, cartilage, rubber ball, rags and golf balls
  - Hair balls in cats
  - Impacted undigested coarse food, especially in the horse
  - Dogs- coproliths
  - Impacted meconium in new born animals
  - Parasites- masses of round worms in pigs and fowls and tape worms in sheep
  - Enteroliths
  - Neoplasms- lipoma in horses
- Accessory causes
- Sudden changes in feeds
- Faulty dentition

## **Pathogenesis**

- ❖ Obstruction causes weak peristaltic movements of the bowel above the points of obstruction, resulting in dehydration of the contents at that place
- ❖ Spasms with violent contractions of the gut above the place of obstruction causes intense pain (colic) and sometimes rupture may occur

## **Gross Pathology**

- ❖ The place of obstruction is usually distended
- ❖ The contents are hard, which pressing on the mucosa may cause necrosis and erosion
- ❖ Ultimately stenosis may develop at this part

## **Sequelae**

- ❖ Rupture and peritonitis leading to death may result if the obstruction is not relieved

# Torsion

Torsion is a twisting of intestines on its axis

## Occurrence

- ❖ It is frequent in horses
- ❖ This may also be seen in other animals

## Etiology

- ❖ Torsion occurs more often in the small intestines which have a long mesenteric attachment
- ❖ In the horse, the right colon is fixed by ligaments and so torsion occurs in the left and transverse colon
- ❖ In the cattle torsion of caecum is more common

## Pathogenesis

- ❖ The changes that occur in torsion are acute passive congestion leading to oedema, haemorrhage, gangrene, peritonitis and death

# VOLVULUS

## Definition

- ❖ Volvulus is a twisting of the bowel on itself as occurs when it passes through a tear in the mesentery

## Occurrence

- ❖ This condition is more frequently seen in horses
- ❖ May also be met with in other animals

## Etiology

- ❖ Violent movements as in rolling and struggling
- ❖ Violent peristaltic movements
- ❖ *Foreign bodies*: sand or enteroliths by their weight make the part heavy and aid in its winding around other parts
- ❖ Gas: Accumulation of gas makes the part bulge and twist round other viscera

## Gross pathology

- ❖ The affected portion is swollen and darkened in color
- ❖ The wall is very easily torn
- ❖ Peritonitis may be evident in some cases



Twisted small intestine on mesenteric axis

# INTUSSUSCEPTION

## Definition

- ❖ Intussusception is telescoping of a portion of intestine into another, usually the anterior into the posterior

## Occurrence

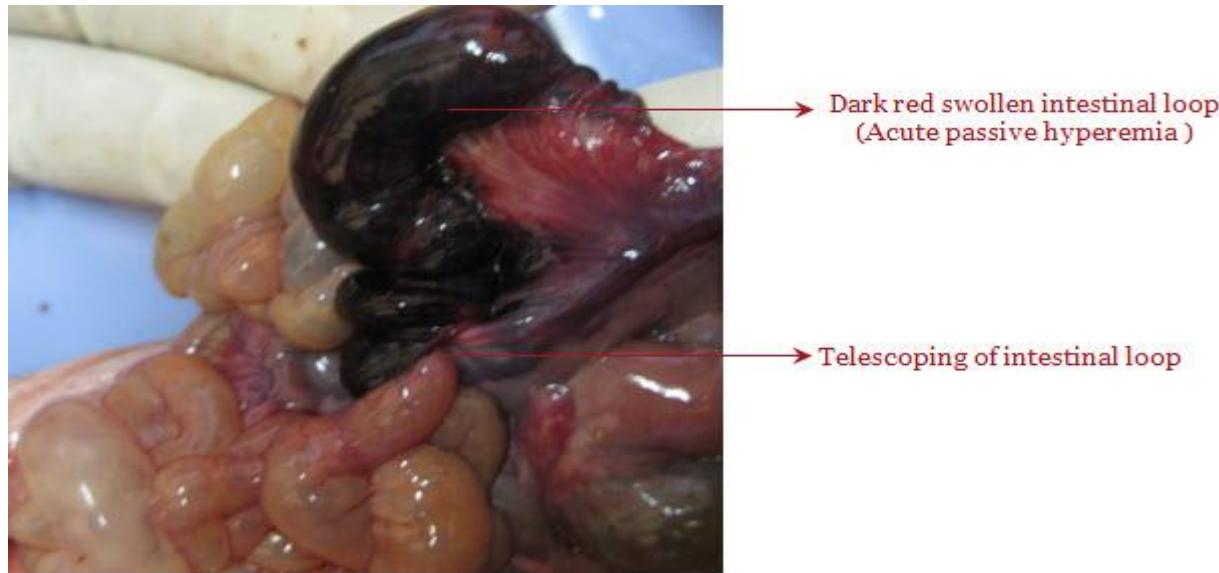
- ❖ It occurs mostly in the jejunum and cecum in dogs and cattle

## Pathogenesis

- ❖ Along with the portion of intestines, its mesentery is also dragged along and so there is compression of the thin-walled veins resulting in acute local passive hyperemia

## Gross pathology

- ❖ The affected part is dark-red or bluish and swollen



Intussusception of intestine in a piglet

# **HERNIA**

## **Definition**

- ❖ Hernia of the abdominal organs is the protrusion of the abdominal viscera through a natural or artificial opening

## **Occurrence**

- ❖ Hernia of intestines is commonly seen in domestic animals, especially the pig and horse

## **Etiology**

- ❖ The intestines may pass through a natural opening, the internal inguinal opening which is patent in the males
- ❖ The umbilicus, if not healed is another site of hernia
- ❖ Trauma when the abdominal muscles may rupture or even the diaphragm may tear resulting in the intestines passing through the opening
- ❖ Violent straining during parturition or defecation

## Types

Depending upon location, hernia may be *external* or *internal* (diaphragmatic, pelvic)

- ❖ If the hernial contents can be returned in to the abdominal cavity, it is called ***reducible hernia***
- ❖ If the hernial contents cannot be so returned it is called ***irreducible hernia***
- ❖ The causes of the latter are
  - ❖ Adhesions between the visceral mass and the hernial sac (The adhesions arise due to inflammation of the peritoneum)
  - ❖ Accumulation of ingesta in the loop of intestines making it too bulky to be reducible and
  - ❖ Venous stasis, oedema and incarceration, whereby the volume is so increased and the bowel cannot be reduced
- ❖ If the hernia does not have a parietal peritoneal covering of the viscous, it is called a *false hernia*
- ❖ In such cases, opening of the skin will reveal the bowel. The condition is called ***eventration***

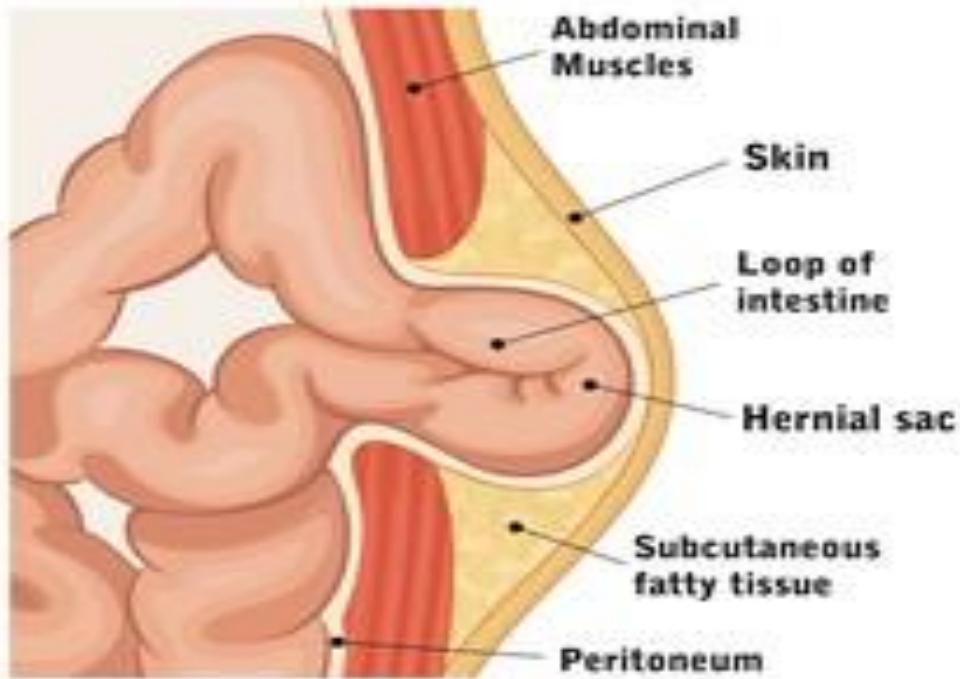
## **External hernia consists of**

A hernial sac formed by the parietal peritoneum and the covering skin

A hernial ring which is the opening in the abdominal wall and

The hernial contents

### **UMBILICAL HERNIA**



## Types of External Hernia

### Ventral hernia

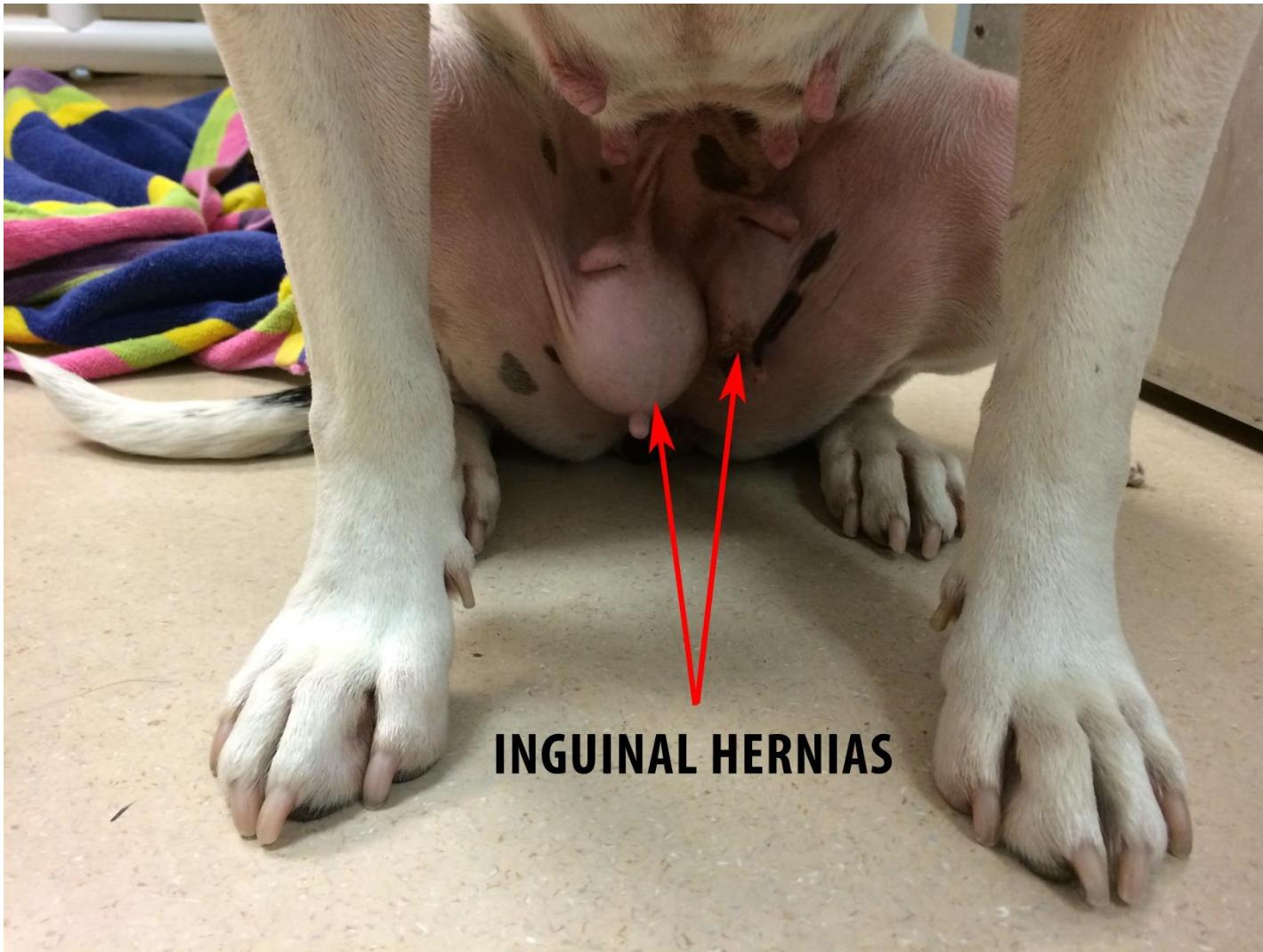
- ❖ The ventral hernia occurs when the abdominal muscles are ruptured
- ❖ This is common in horses (spontaneous in pregnant mares) and occasionally in cattle
- ❖ Trauma - Horn injuries, kicks, automobile accidents, laparotomy and castration scars
- ❖ In pregnant ewes, this may be due to muscular degeneration of nutritional origin

### Umbilical hernia

- ❖ *The umbilical when the bowel passes through a congenital or acquired defect of umbilicus*
- ❖ It is seen in foals, calves and pups

### Inguinal hernia

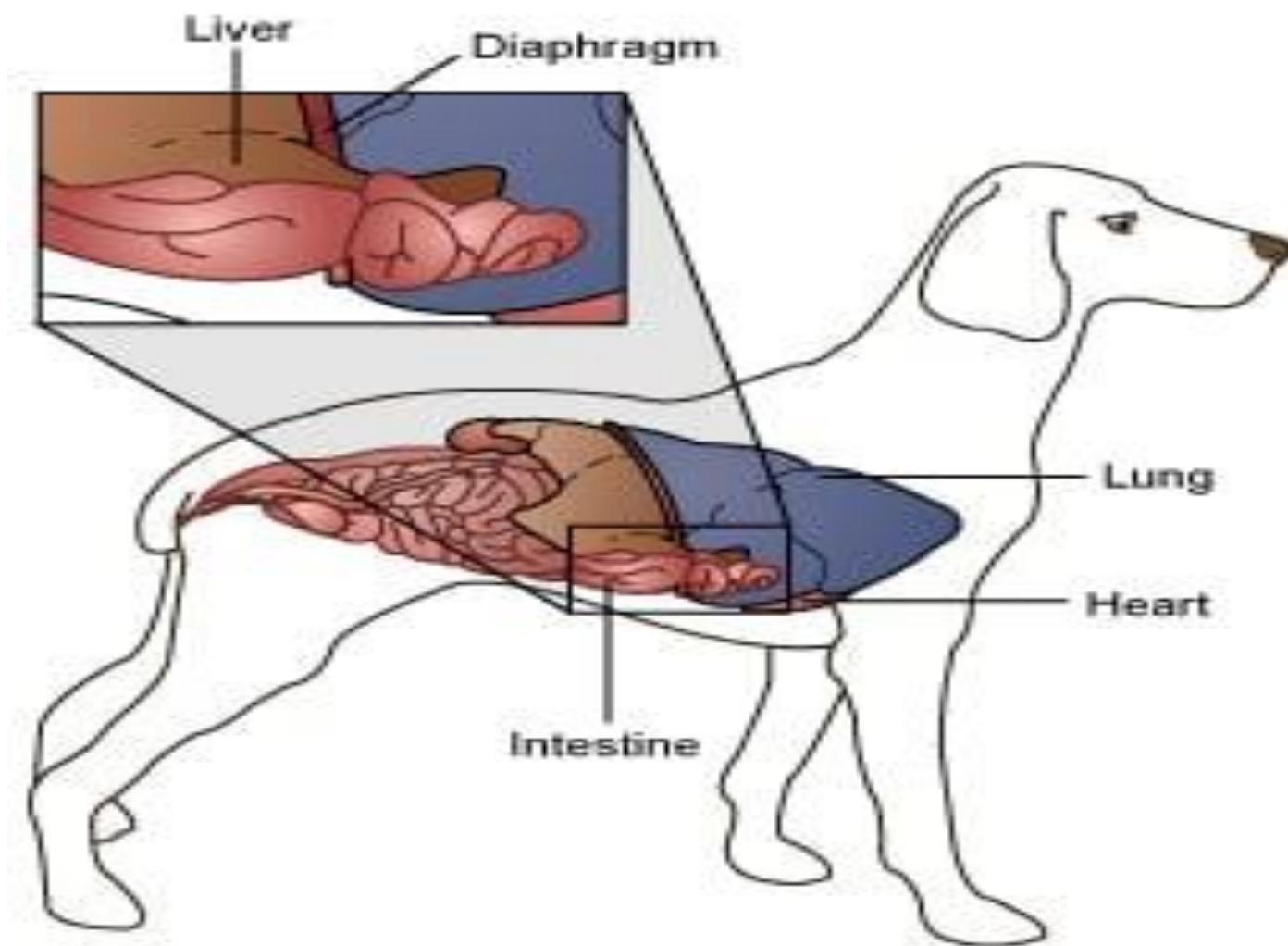
- ❖ In inguinal hernia, the bowel passes through the internal inguinal ring.
- ❖ *This is not so common in animals as in man, because of horizontal position: seen in colts and pigs*



**INGUINAL HERNIAS**

# Ventral Hernia





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## Scrotal hernia

- ❖ Here the intestines slide into the tunica vaginalis along the inguinal canal in contact with the spermatic cord
- ❖ The testes may undergo thermal atrophy as it is in contact with the intestines

## Femoral hernia

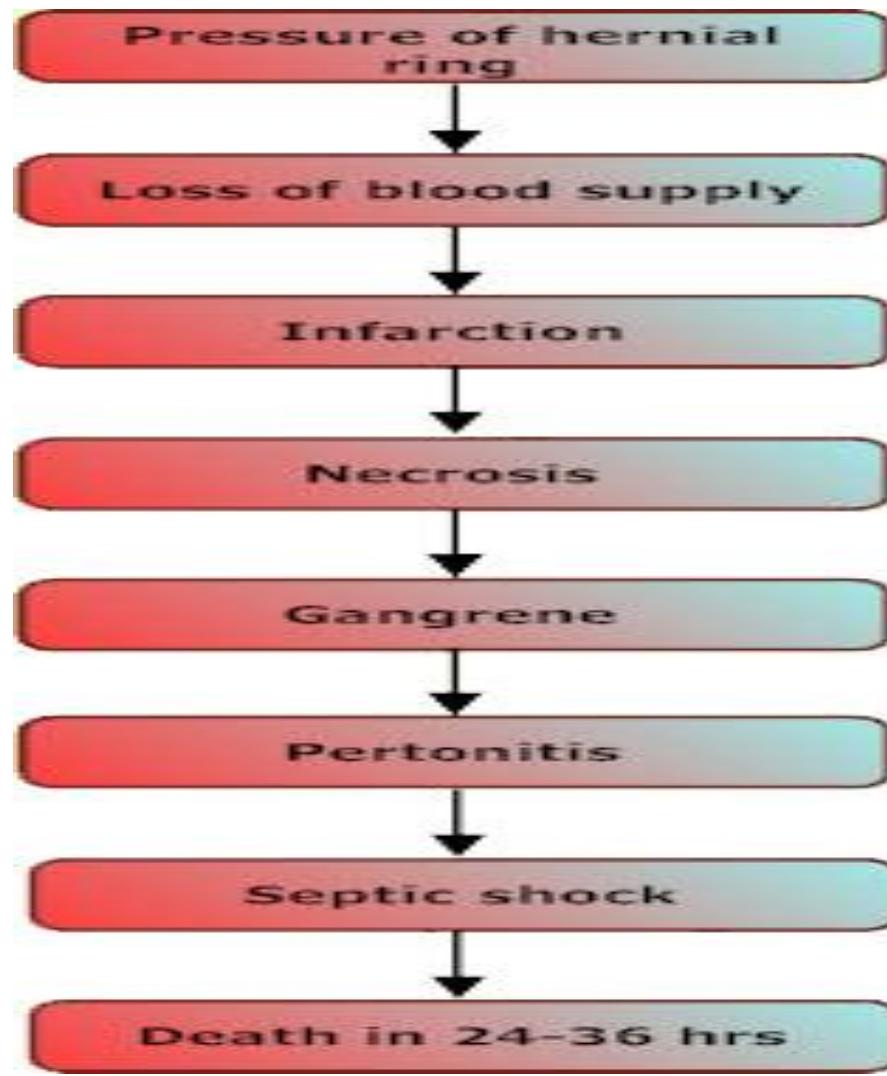
- ❖ Femoral hernia may develop when the omentum and intestines pass through the femoral triangle along the femoral artery
- ❖ Here the bowel is found on the inner surface of the thigh

## Perineal hernia

- ❖ The perineal hernia may occur in old dogs
- ❖ It may be due to violent straining as in case of enlarged prostate

## Strangulated hernia

- ❖ This is one in which the blood supply is cut off by the pressure of the hernial ring through which the intestines pass
- ❖ If not relieved in time, this condition is fatal since infarction, gangrene, peritonitis and shock will develop within 24 to 36 hours



## **Internal hernia**

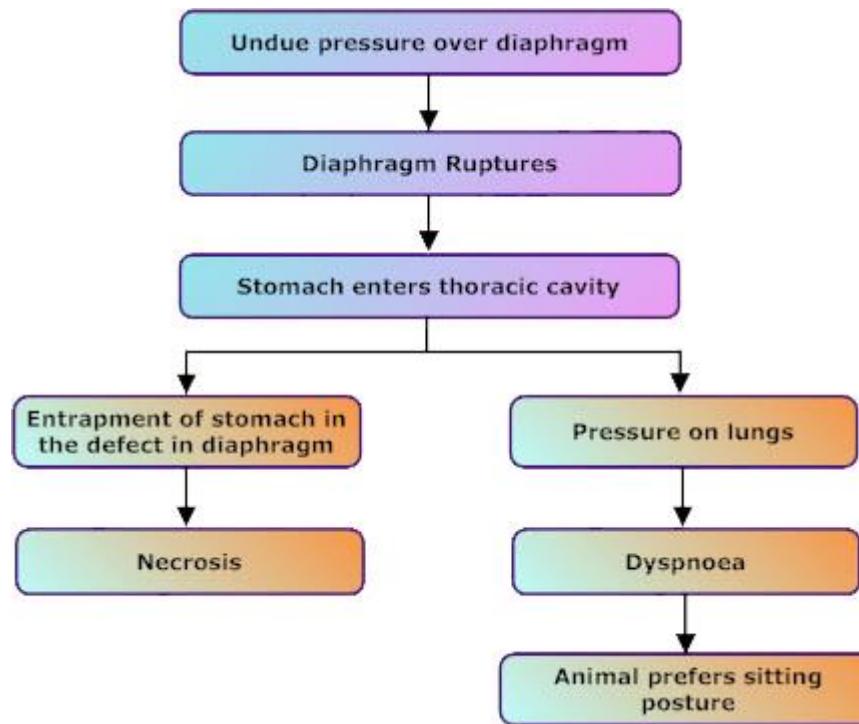
### **Diaphragmatic hernia**

#### *Aetiology*

Physical trauma from external side

Increased intra-abdominal pressure

#### *Pathogenesis*



# ENTERITIS

## Definition

- ❖ Enteritis is inflammation of the whole of the intestinal tract. But usually it is applied to the inflammation of the small intestines.
- ❖ The inflammation of the colon is called *colitis* , that of cecum *typhlitis*, that of rectum *proctitis* and of cloaca *cloacitis* or *vent gleet*

## Introduction

- ❖ Enteritis is of immense economic importance

## Occurrence

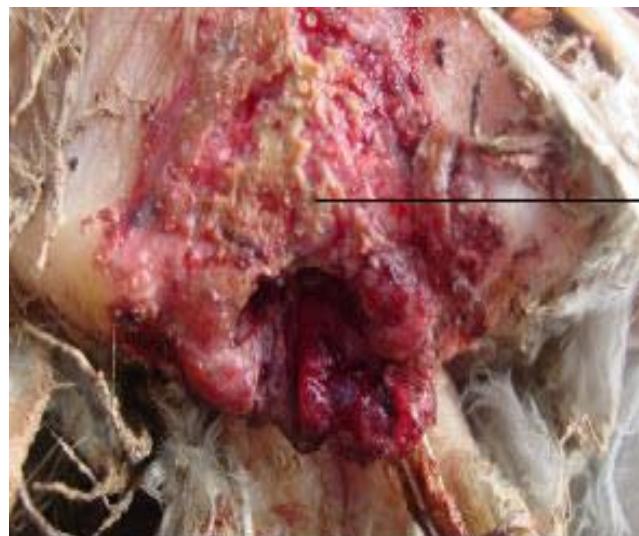
- ❖ Enteritis is very common in domesticated animals and fowls
- ❖ Since enteritis occurs along with gastritis (the same irritants causing gastritis passing on to intestines produces enteritis also) gastro-enteritis is a frequent condition met with

## Etiology

- ❖ Chemicals, bacteria, viruses, protozoa, rickettsia, helminths, fungi, disturbed metabolic processes as in ruminants, venous congestion as in portal hypertension and congestive cardiac failure, toxins of Clostridia, coliforms and spoiled or mouldy feeds and avitaminosis

## Gross pathology

- ❖ In enteritis, the whole length of the bowel may not be affected, inflammation localizing only at one part or the other



→ Swollen, ulcerated and congested vent and cloaca

Cloacitis (Vent gleet) in a chicken

# **TYPES OF ENTERITIS**

Enteritis is classified as follows:

- ❖ Catarrhal Enteritis
- ❖ Hemorrhagic Enteritis
- ❖ Fibrinous Enteritis
- ❖ Suppurative Enteritis
- ❖ Necrotic Enteritis

# CATARRHAL ENTERITIS

## Acute Catarrhal Enteritis

- ❖ This is the mildest of inflammation of the intestinal tract, occurring in a diffuse manner throughout the bowel

## Predisposing causes

- ❖ In calves and lambs avitaminosis A is a predisposing factor while in young pigs deficiency of animal proteins and trace elements predispose them to infections
- ❖ In such a state, the organisms are able to gain a foothold and thrive causing the disease

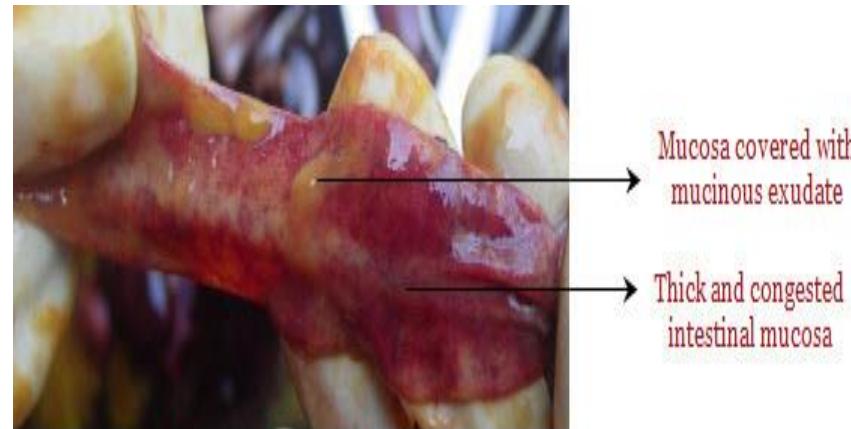
## Aetiology

- ❖ Causes include mild irritants like foreign bodies, sand, coarse feeds, chemicals, bites of parasites (hook worms), *Vibrio coli* (causing winter diarrhoea in cattle) and drugs

## Acute catarrhal enteritis may be noticed in

- ❖ Enteritis in sucklings – scours in calves, lambs, foals and piglets caused by *E.coli*, *Pasteurella*, *Salmonella*, *Proteus*, *Vibrio* and *Streptococci*
- ❖ Enterotoxemia in sheep
- ❖ Viral Diarrhoea -Mucosal Disease in cattle
- ❖ Virus gastroenteritis in pigs
- ❖ Salmon poisoning in dogs
- ❖ Salmonellosis, infectious cloacitis, pullet disease and ornithosis in fowls.
- ❖ Immature amphistomes

## Gross pathology



Catarrhal enteritis in a chicken

## Histopathology

- ❖ The intestinal contents consist of mucus, fibrin and exfoliated epithelial cells
- ❖ Goblet cells are numerous and produce large amounts of mucus



Catarrhal enteritis

- ❖ The tips of villi may be hyperaemic and edematous
- ❖ There is infiltration with leucocytes in the lamina propria and to a little extent in the submucosa

# Chronic Catarrhal Enteritis

## Aetiology

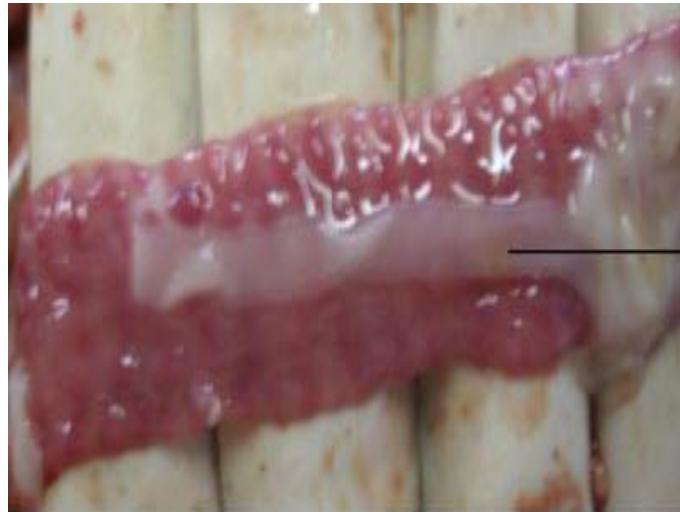
- ❖ It may develop from the acute condition or more usually it may arise gradually as in Johne's disease, intestinal helminthiasis, chronic venous congestion (due to congestive cardiac failure) and cirrhosis of liver

## Gross pathology

- ❖ The wall of the intestines is greatly thickened
- ❖ The mucosa is smooth (covered by thick mucus)
- ❖ The corrugations are sometimes present at right angles to the length of the intestines (*Chronic polypoid enteritis*)



Ascaridiasis in a chicken



→ Tapeworm in  
intestinal lumen

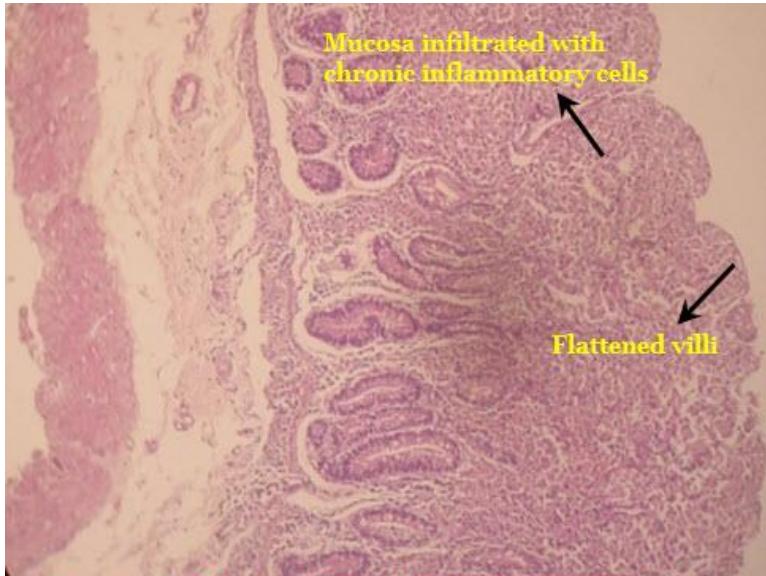
Cestodiasis in a goat



→ Thick and nodular intestinal  
mucosa

→ Congested intestinal mucosa

Chronic enteritis - Johne's disease in a sheep



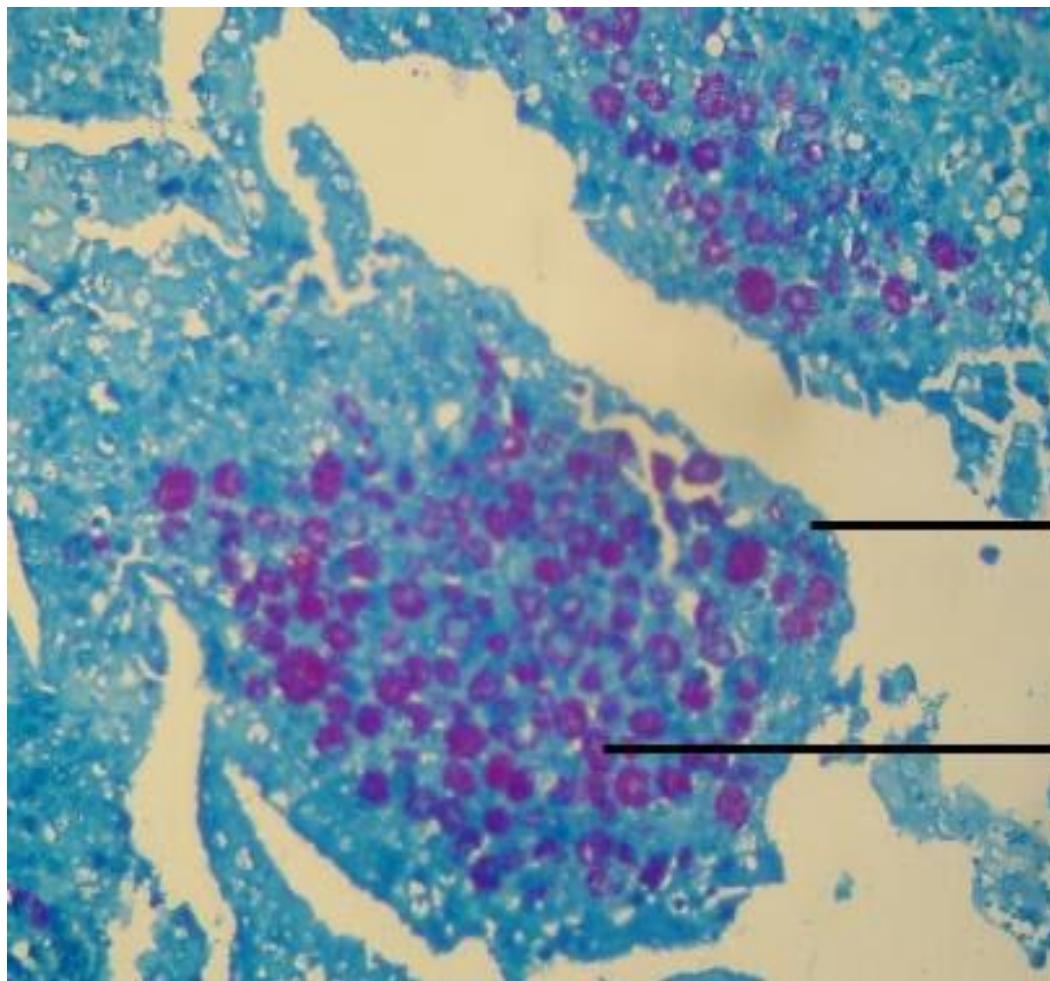
Chronic enteritis - Johne's disease - Intestine - Sheep



Johne's disease - Intestine - Sheep

The mucosa is covered with thick mucus

The characteristic appearance is the presence of numerous macrophages, plasma cells, lymphocytes and connective tissue cells in the lamina propria and even in the sub-mucosa



→ Villi of intestine

→ Acid fast bacilli in  
macrophages

Johne's disease - Intestine - Acid fast bacilli - Sheep

# HEMORRHAGIC ENTERITIS

- ❖ This is a more severe form of enteritis, characterized by the presence of erythrocytes in the exudate

## Aetiology

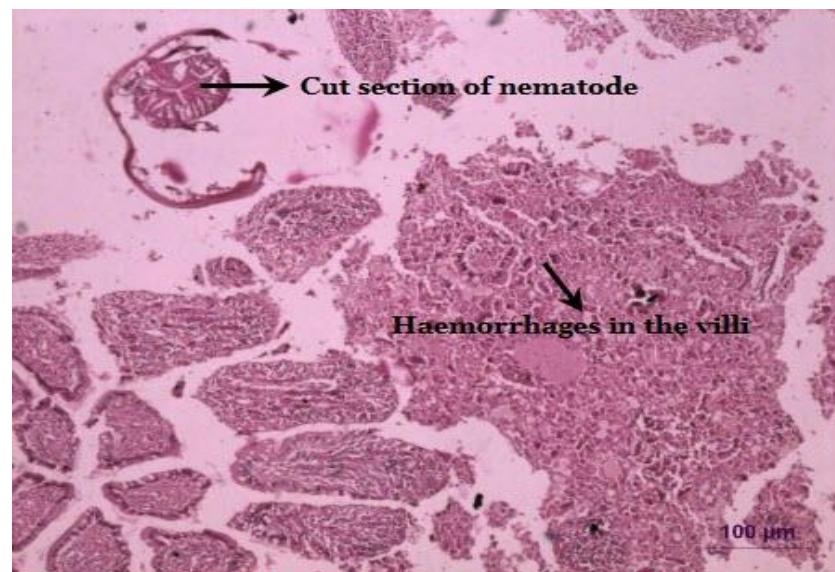
- ❖ This is mostly seen in septicemic bacterial and viral diseases e.g Anthrax and Rinderpest
- ❖ Poisoning by arsenic and croton oil
- ❖ Colibacillosis
- ❖ Enterotoxemia
- ❖ Coccidiosis
- ❖ manifested by hemorrhagic enteritis
- ❖ Vitamin B deficiency in dogs and pigs
- ❖ This condition may also be found in ancylostomiasis and uraemia of dogs

## Gross pathology

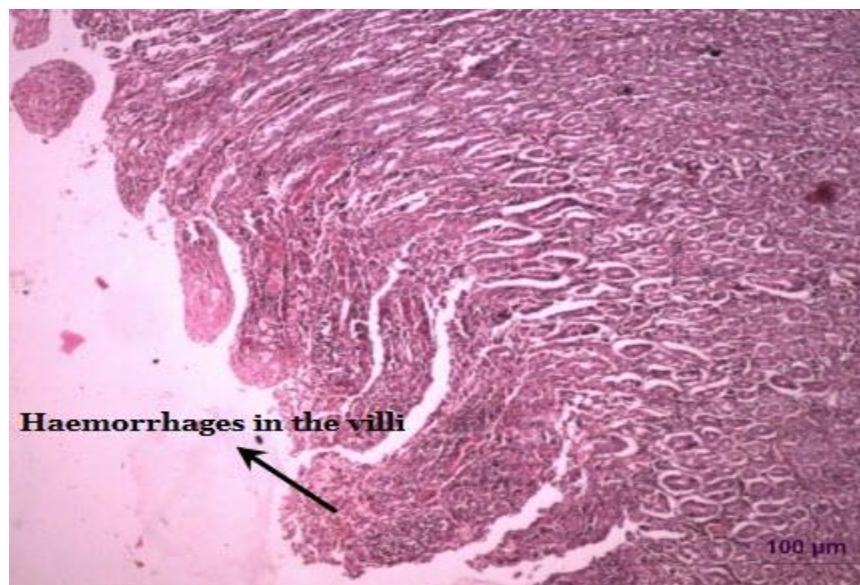
- ❖ Always patchy in distribution
- ❖ The intestinal contents are blood stained
- ❖ Blood found in the anterior portion of the intestines is digested and so is brown in color while in the posterior portion it is bright red

## Histopathology

- ❖ Red blood cells may be found in the exudate of the mucosa
- ❖ The villi may show necrotic changes
- ❖ The intestinal wall shows haemorrhages
- ❖ Thrombosis of some enteric vessels is evident



Parasitic enteritis - Ancylostome



Haemorrhagic enteritis - Dog

# FIBRINOUS ENTERITIS

This is of the diphtheritic type of enteritis

## Occurrence

- ❖ It occurs in cattle, pigs and cats, and rarely in horse and fowl

## Etiology

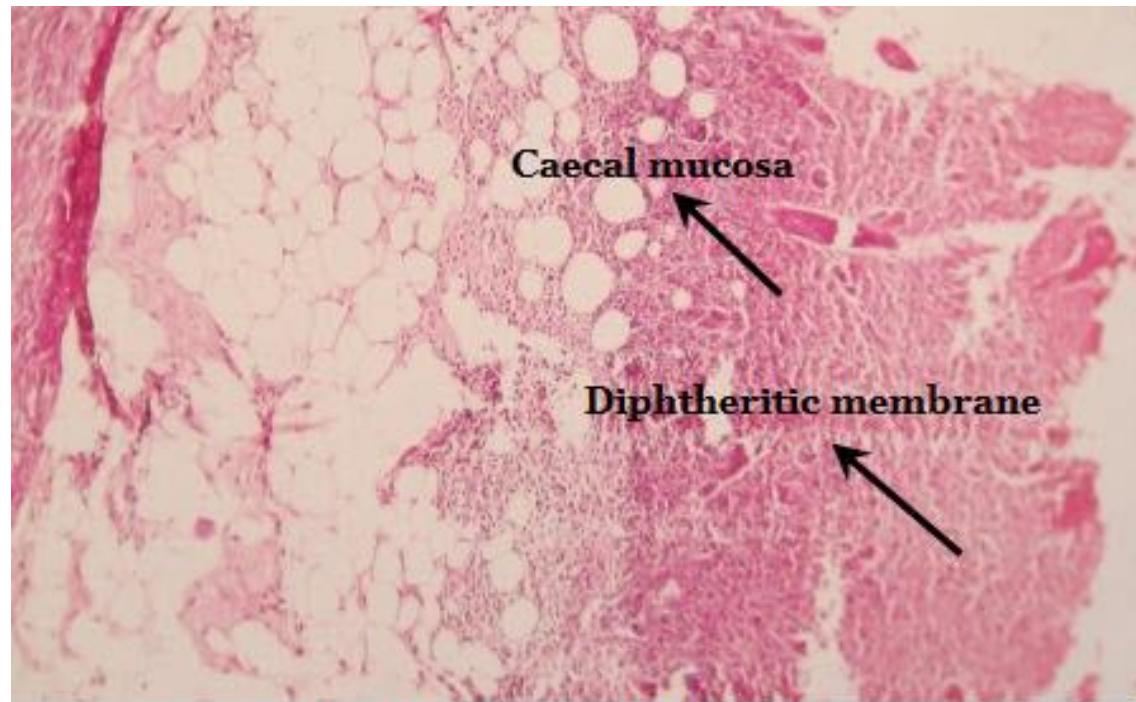
- ❖ *Chemicals:* Salts of mercury and arsenic
- ❖ *Bacteria:* Campylobacteriosis, *Salmonella cholerae suis* and *Escherichia coli*
- ❖ *Parasites:* *Echinostomum*, a fluke in turkeys

## Gross pathology

- ❖ The characteristic finding is the presence of strands of fibrin on the mucosa of intestine
- ❖ The wall of the intestine is edematous
- ❖ In more severe conditions, a thick, grayish or whitish-gray membrane may be covering the intestinal mucosa
- ❖ The inflammation may extend into the submucosa and petechial hemorrhages may be seen
- ❖ The mesenteric lymph nodes are swollen, haemorrhagic and juicy

## Histopathology

- ❖ The exudate consists of strands of fibrin containing in its meshes varying number of neutrophils and exfoliated epithelial cells
- ❖ Mucous membrane shows edema, hyperemia and infiltration by neutrophils
- ❖ Coagulative necrosis of the epithelium occurs in some places, which along with the exudate forms a membrane which is adherent to the intestine



Diphtheritic typhlitis - Pig

# SUPPURATIVE ENTERITIS

## Occurrence

- ❖ This is not a common condition in animals

## Aetiology

- ❖ It may result due to infection by pyogenic organisms (*Streptococci*, *Salmonella* and *Shigella*) infecting the wounds caused by helminths

## Gross pathology

- ❖ Macroscopically, the exudate contains pus

## Histopathology

- ❖ The exudate besides mucus contains exfoliated cells, neutrophils and bacteria

# NECROTIC ENTERITIS

## Etiology

- ❖ Severe irritants: Chemical- croton oil, mustard gas, wood preservatives; insecticides
- ❖ Bacteria- *Fusiformis necrophorum* and *Salmonella* sp.
- ❖ Viral diseases - Rinderpest, Viral Diarrhoea- Mucosal Disease, Hog cholera
- ❖ Protozoal diseases - Coccidiosis and histomoniasis
- ❖ Vitamin deficiency – Niacin deficiency in swine

## Gross pathology

- ❖ Patchy necrotic areas are seen
- ❖ Fibrin may be found on the necrotic mucosa
- ❖ When the necrotic material is removed, a red, raw, bleeding surface is seen
- ❖ The necrosis of the mucosa extends into the sub-mucosa also
- ❖ The mesenteric lymphatic nodes are swollen and juicy
- ❖ In hog cholera, the characteristic lesion is the “button ulcer”, which is a spherical ulcer in the mucosa of the colon



Intestinal coccidiosis a kid

Greyish spots in the  
mucosa seen through  
the serosa

## **Histopathology**

- ❖ Necrosis of enterocytes is seen
- ❖ The mucosa shows hyperemia and cellular infiltration
- ❖ The ulcer reveals a demarcated zone of necrosis in the mucosa and submucosa
- ❖ The button ulcers are tiny areas of infarction that arise by the occlusion of small arteries by the swollen and proliferated endothelium

## **Sequelae**

- ❖ The condition is mostly fatal
- ❖ If the condition is one of niacin deficiency, restitution of the deficiency may cure the condition

# CONCRETIONS

## Enteroliths

**Definition :** Calculi found in the intestine are called enteroliths.

### Etiology

- ❖ When animals are fed on wheat or bran which are rich in magnesium phosphate, intestinal calculi can occur.

### Genesis

- ❖ They are formed of triple phosphates which are deposited concentrically, layer after layer, over a nucleus/nidus of sand or a metal piece or an undigested vegetable fibre.
- ❖ When excessive amounts of magnesium phosphate are fed to an animal, and that too to one suffering from **chronic catarrhal gastritis** in which gastric juice is not secreted, much of the magnesium phosphate reaches the intestines in an undissolved state.
- ❖ This combines with ammonia that is formed from the decomposition of protein (which is also abundant in wheat and bran) to form triple phosphate.
- ❖ Enteroliths may sometimes attain a large size, some may weigh as much as 20 lbs and are usually round and smooth.

## PHYTOBEZOARS

- ❖ These are food balls (phyto= plant, bezoar=concretion) made of plant fibres.

### Occurrence

- ❖ They may also be found in the crops of birds and stomach of ruminants.

### Genesis

- ❖ These arise from plant fibres and awns which are impregnated with triple phosphate and rolled into balls.

### Gross pathology

- ❖ These have a velvety surface, light in weight and are brown in color.

## TRICHOBEZOARS

### Synonym

- ❖ Piliconcretions; Hair balls

### Definition

- ❖ Tricho= hair; bezoars= concretions

### Occurrence

- ❖ Hair balls are found mostly in the rumen of calves.

### Etiology

- ❖ Animals having itching skin conditions (animals infected with mange or lice) may lick each other when the loose hair may be swallowed.
- ❖ Similarly, calves kept together in enclosures, suck and lick each other's ears, tails etc., and swallow hairs.

## Genesis

- ❖ The hair is rolled into balls during ruminal contractions.
- ❖ Mucus of rumen may form a smooth coat over such balls.

## Sequelae of concretions

- ❖ More often these concretions are found only at post-mortem examination.
- ❖ The enteroliths, phytobezoars and trichobezoars are usually of no consequence unless they obstruct the passage, when, sometimes, even rupture may occur.
- ❖ Cattle may regurgitate a food ball into the oesophagus which may be choked.

# LIVER

## Functions of Liver

- **Metabolism** – Carbohydrate, Fat & Protein
- **Secretary** – Bile, Bile acids, salts & pigments
- **Excretory** – Bilirubin, drugs, toxins
- **Synthesis** – Albumin, coagulation factors
- **Storage** – Vitamins, carbohydrates etc.
- **Detoxification** – toxins, ammonia, etc.

# **DEGENERATIVE CHANGES IN LIVER**

# DEGENERATIVE CHANGES IN LIVER

- ❖ In degenerations there is an alteration in cellular elements (metabolic or enzymatic disturbance). Some changes are reversible, some if the insult lasts a prolonged period are irreversible.

## Cloudy swelling

### Occurrence

- Cloudy swelling is common in the liver

### Etiology

- Hypoxia
- ❖ Poisons
  - ❖ Chemicals- salts of heavy metals – arsenic and lead
  - ❖ Plant toxins – glucosides, saponin
  - ❖ Drugs – carbon tetrachloride which was used as an anthelmintic
- ❖ Bacterial toxins seen in all infectious diseases
- ❖ Viruses

### Gross pathology

- ❖ The organ has a dull, **parboiled appearance**
- ❖ The liver is enlarged
- ❖ The capsule is tense
- ❖ Borders are rounded
- ❖ Consistency is softer
- ❖ On section, **it bulges at the cutting surface**
- ❖ Lobular markings are indistinct

### Histopathology

- ❖ Hepatocytes are swollen and have a pale granular cytoplasm due to swelling of mitochondria
- ❖ The nuclei may be indistinct

# Hydropic degeneration

## Etiology

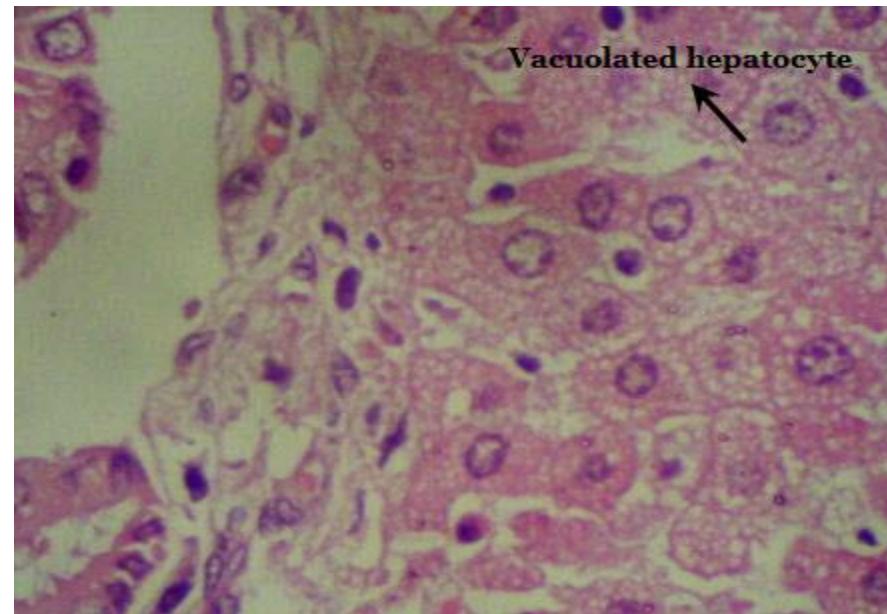
- Toxins and septicaemic infections
- Ether, chloroform and carbon tetra chloride poisoning
- Euthanasia

## Gross pathology

- Gross lesions are more or less similar to that of cloudy swelling

## Histopathology

- The cytoplasm of hepatocyte may contain one, two or more vacuoles in the cytoplasm.



Hydropic change - Liver - Sheep

# Fatty change

## Occurrence

- Fatty change in the liver is common in animals

## Etiology

- Hypoxia/anoxia: Chronic venous congestion
- Nutritional deficiency: Inadequate choline and apoproteins
- Metabolic diseases— Diabetes mellitus in dogs and cats, acetonemia/ketosis in cattle, pregnancy toxæmia in ewes, and deficiency of thyroxine and anterior pituitary hormones
- Bacterial toxins
- Poisons: Inorganic – phosphorus, arsenic, antimony; Organic - chloroform, carbon tetrachloride, tannic acid, tetrachlorethylene, alkaloids of phytotoxins, aflatoxin and Senecio

## Pathogenesis

- The liver is too sick to metabolize the dietary as well as the fat brought to it from the depots. Defect may be anywhere in the process from metabolism of fatty acids to the formation and release of lipoproteins

## Gross pathology

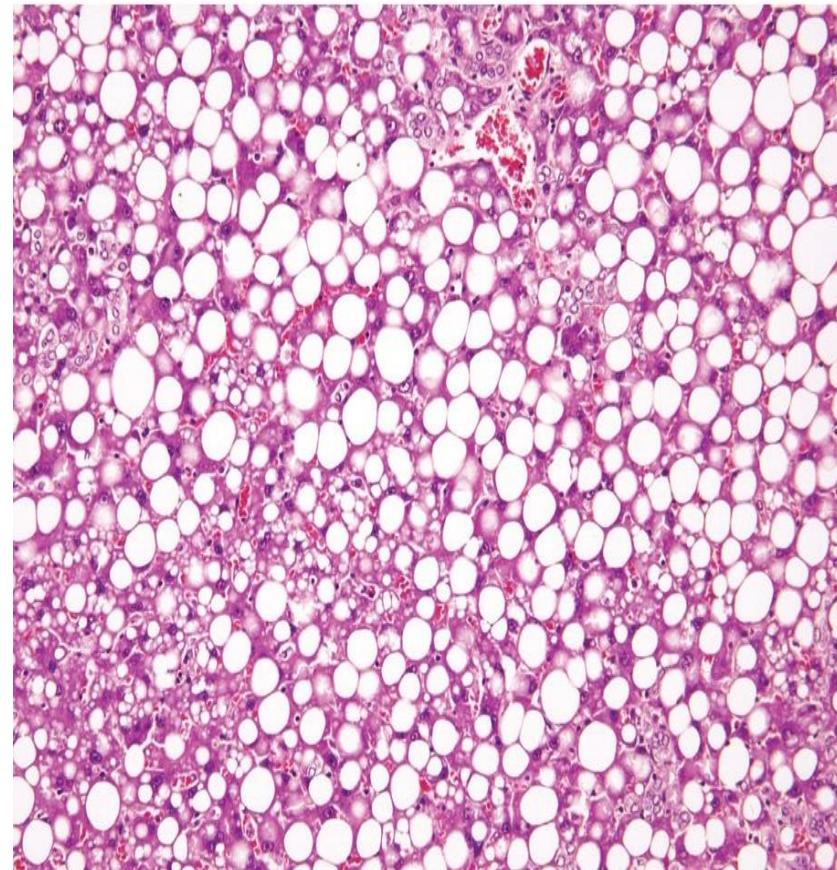
- The liver is enlarged with round margins, has a smooth surface and is pale or yellowish
- Consistency is friable
- On section it bulges on the cut surface
- **Fat droplets are seen on the blade**
- Very fatty livers as in pregnancy toxæmia of sheep, float in water

## Histopathology

- ❖ The hepatic parenchymal cells contain fat droplets, either as a single large globule or as multiple small globules.

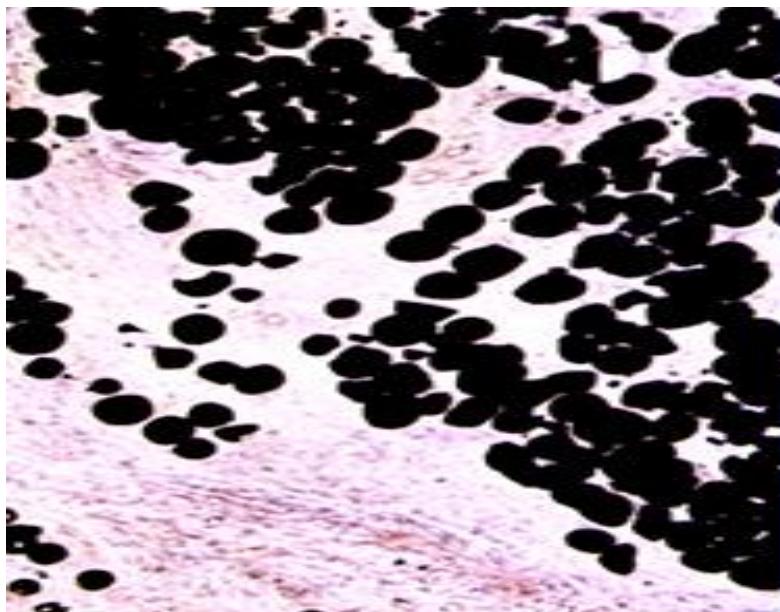


Fatty liver haemorrhagic syndrome - Chicken

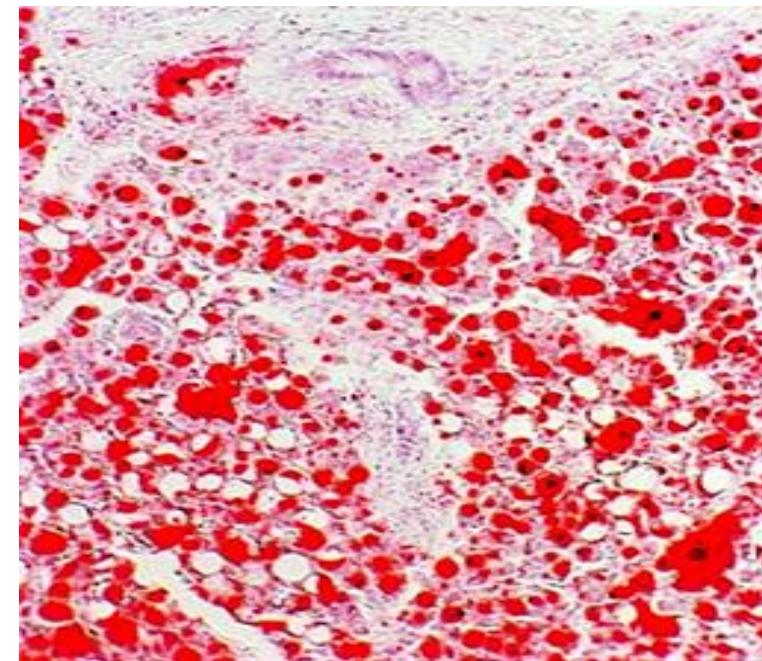


# Special staining for fat

1. Osmium tetroxide
- 2.Sudan Black
- 3.Oil Red O



Sudan Black Method  
Fat - Black



Oil Red O Method  
Fat - Red

# HEPATITIS

- Hepatitis is an **alterative inflammation of liver** in which the various degenerative processes like cloudy swelling, fatty change and necrosis are caused by irritants which also produce inflammation.
- Besides, in liver, these degenerative changes are accompanied by lymphocytic or leucocytic infiltrations typical of an inflammatory reaction.

# Types of Hepatitis

## ❖ Infectious Hepatitis

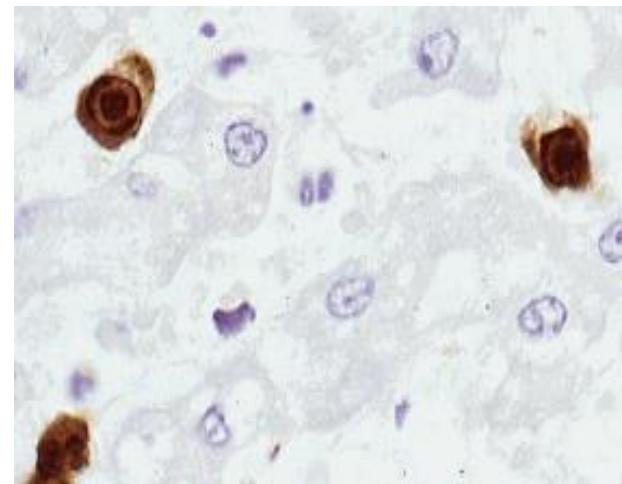
- ❖ Viral hepatitis
- ❖ Bacterial hepatitis
- ❖ Parasitic hepatitis

- Acute Toxic Hepatitis
- Chronic Hepatitis (Cirrhosis)
- Auto immune Hepatitis

# Viral Hepatitis

## Infectious Canine Hepatitis

- Canine adeno virus type 1
- Predilection site is vascular endothelium & hepatocytes
- Characterized by hepatic necrosis and widespread hemorrhages
- Basophilic intranuclear inclusion bodies and necrosis



# Rift valley fever

- Acute, arthropod borne disease
- Ruminants, calves & lambs extensive mortality
- Abortion in pregnant Ewes & Cows

## Lesions

- Hepatomegaly with orange brown discoloration
- Pale foci of necrosis
- Diffuse petechiae & ecchymoses

## Herpes viral infections

- ❖ EHV, IBRT, CHV, FVRT, pseudo rabies, yellow fever, mouse hepatitis, Wesselbron disease

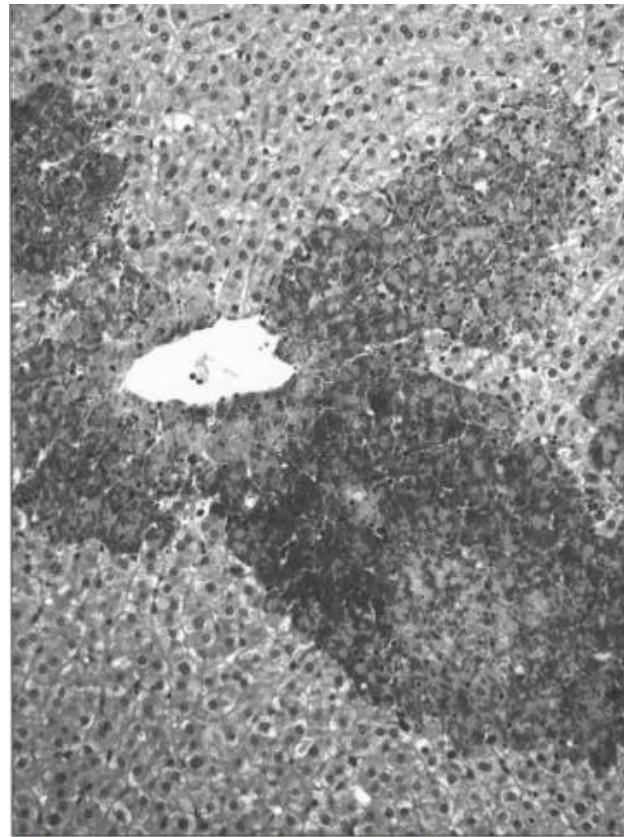


Figure 2.57 Focal necrosis with hemorrhage in Rift Valley fever in a lamb. (Courtesy of S Youssef)

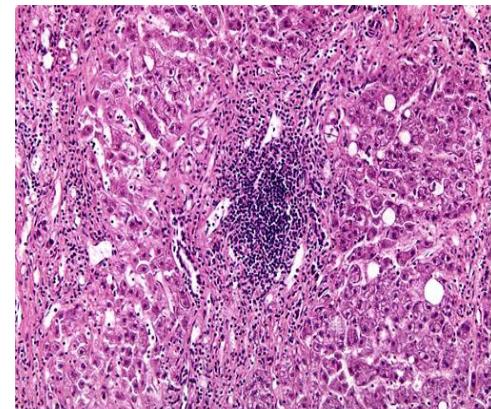
# Bacterial Hepatitis

Black disease	Listeriosis	Bacillary hemoglobinuria
Leptospirosis	Tyzzer's disease	Salmonellosis
Yersiniosis	Nocardiosis	Tularaemia
Necrobacillosis	Tuberculosis	Actinobacillosis
Liver abscess	Pasteurella Infections	



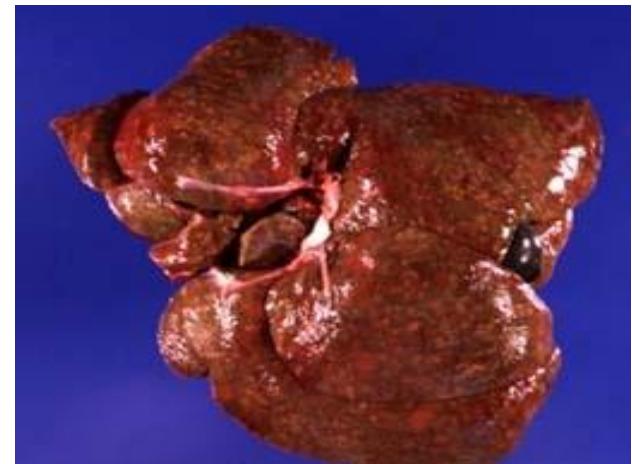
## Black disease

- ❖ *C. novyi*
- ❖ Multiple distinct foci of necrosis
- ❖ Zone of hyperemia surrounds the necrosis
- ❖ Parasitic migration tracts are seen



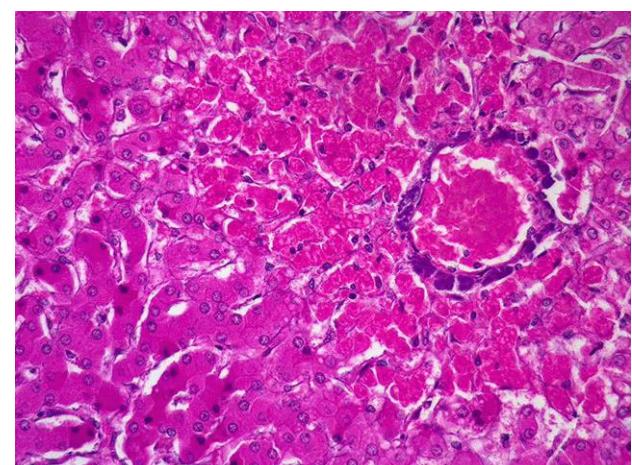
# Bacillary hemoglobinuria

- ❖ *C. haemolyticum*
- ❖ Large areas hepatocellular necrosis and almost solitary
- ❖ Sharply demarcated pale area , surrounded
- ❖ Hyperemic zone
- ❖ Parasitic migratory tracts visible



# Leptospirosis

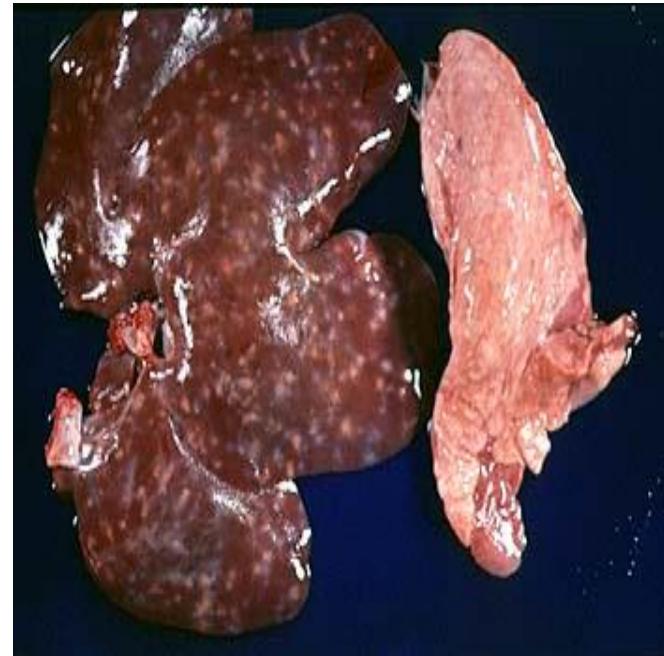
- ❖ Liver is involved in acute cases of all animals
- ❖ Ischemic injury
- ❖ Intravascular hemolytic anemia
- ❖ Centrilobular necrosis



# PARASITIC HEPATITIS

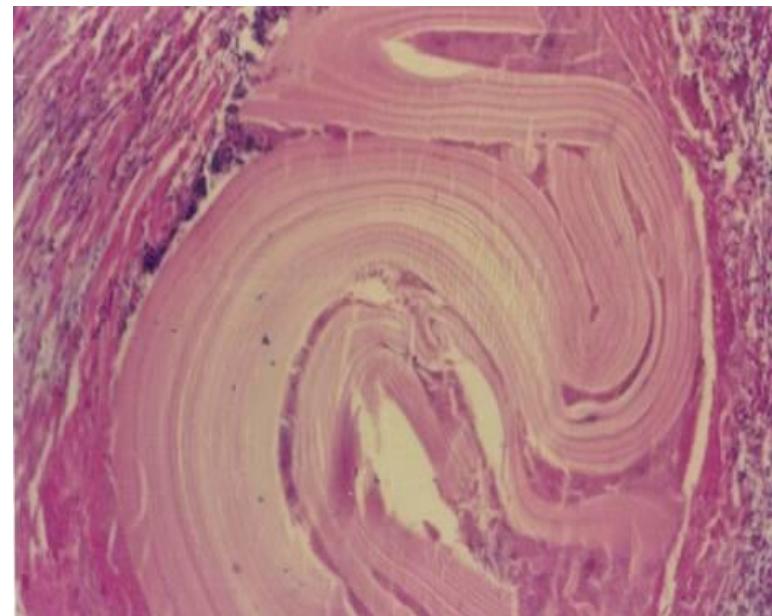
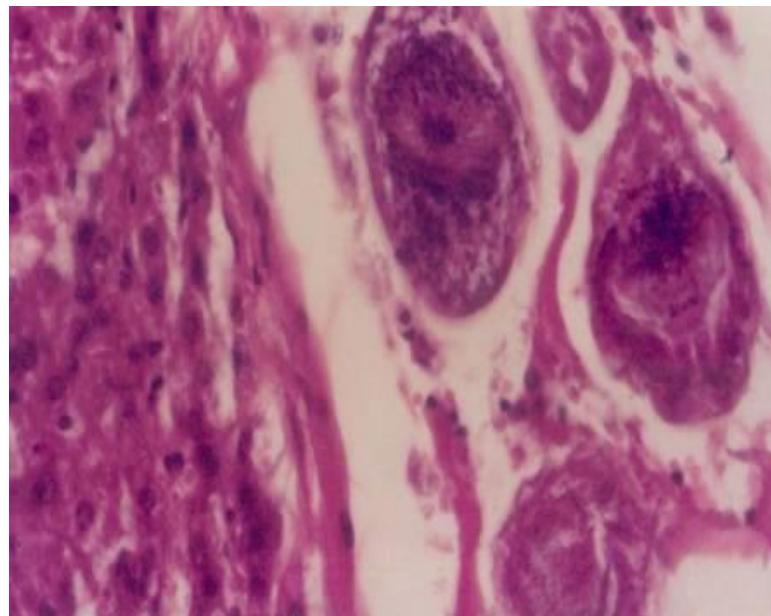
## NEMATODES

- ❖ Migration of larvae through the Liver produce tracts of hepatocellular necrosis and inflammation.
- ❖ Fibrous scar formation, small abscess or granuloma.
- ❖ Milk spotted liver – *Ascaris suum*.
- ❖ Hepatic scarring - *Stephanurus dentatus*, *Strongylus*, *Capillaria hepatica* sometimes in Dogs & Cat



## CESTODES

- ❖ *Echinococcus granulosus* –  
Hydatid Liver Disease
- ❖ Liver and lungs are affected
- ❖ *Stilesia hepatica*
- ❖ *Thysanosoma actinoides*  
Cholangio hepatitis



# TREMATODES

- Liver fluke disease of ruminants – *F. hepatica*, *F. gigantica* & *F. magna*
- *F. hepatica* causes hemorrhagic tracts & necrotic parenchyma
  - Hepatic abscess, necrotic hepatitis,
  - Infiltration immature flukes
  - Cholangitis, cholangio-hepatitis
- Immature *F. magna* causes extensive parenchyma damage, necrosis



# Toxic Hepatitis

- Drugs and toxins are cause for acute or chronic liver injury
- Some accompany inflammation- toxic hepatitis

## Mech. of Action

### Toxin

Direct toxicity (phosphorous)

Conversion of a liver xenobiotic to a toxin(detoxification)  
(eg- aflatoxins, carbon tetrachloride)

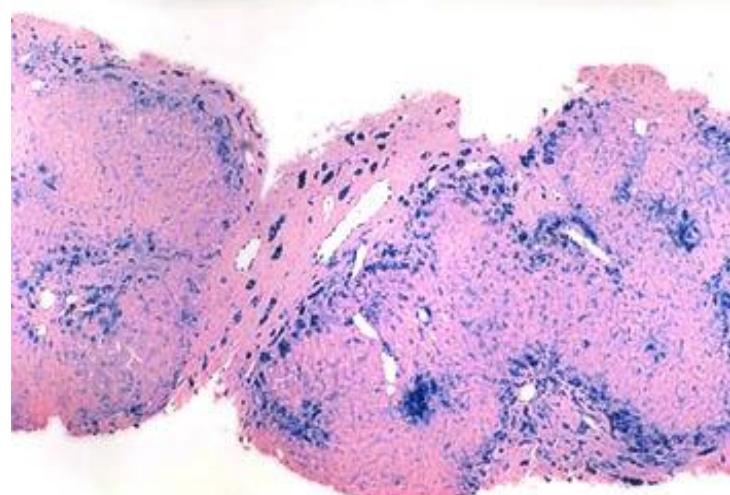
It may be predictable or unpredictable/idiosyncratic toxins

Predictable –e.g.: CCl<sub>4</sub>, Phosphorous and most hepatotoxic plants

Unpredictable/idiosyncratic toxins – eg: halothane toxicity.

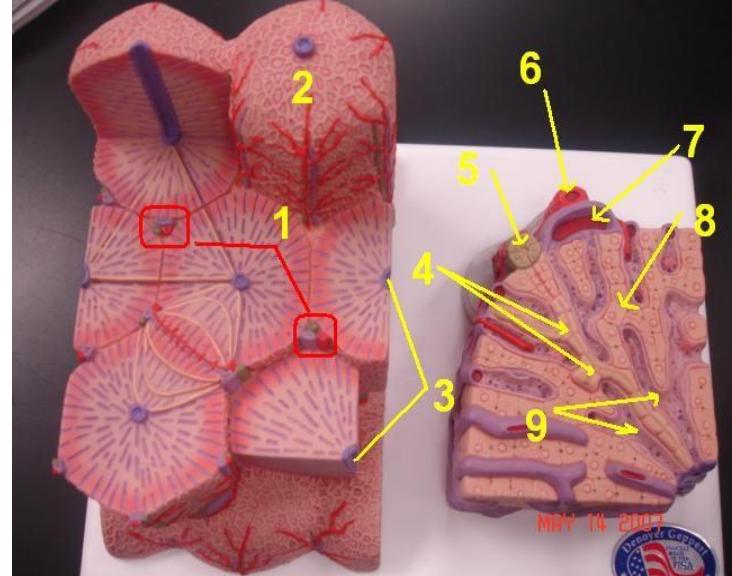
# Specific causes

- Gross and histopathological features varies with cause
- Hepatic injury vary with several factors like type, dose & duration
  - ❖ Chemical poisons – Cu, PO<sub>4</sub>, Hg, Iron, etc.
  - ❖ Plant poisons – Senecio, Crotalaria, Heliotropium - Alkaloids
  - ❖ Mycotoxins - Aflatoxin, Pomopsin & Sporidesmin



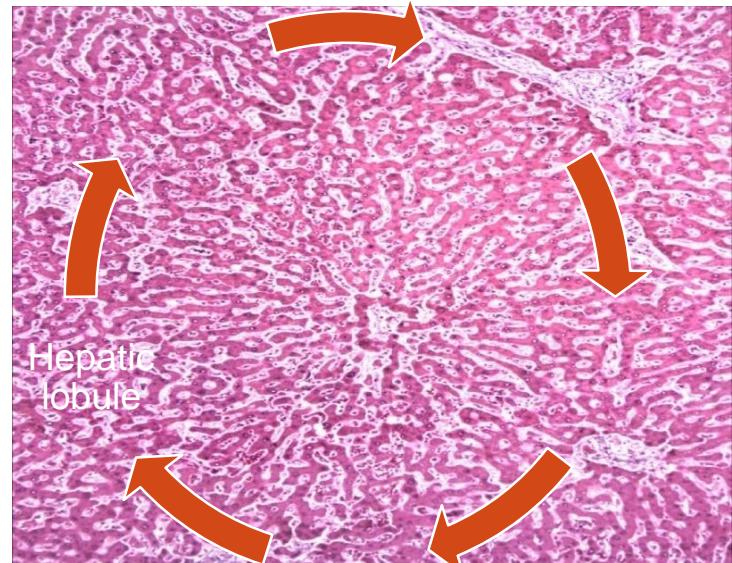
# Gross structure

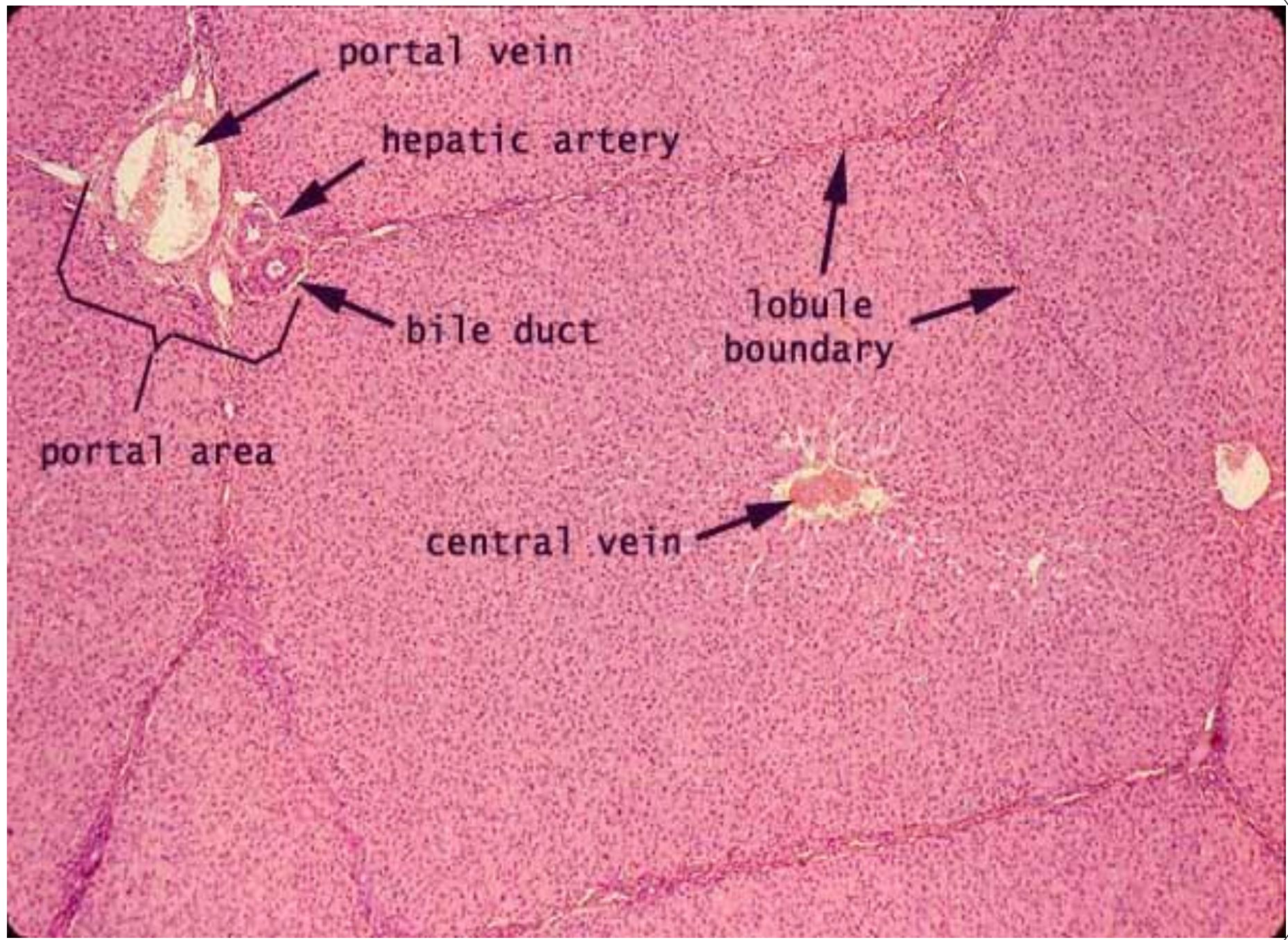
- ❖ Parenchymatous organ covered by Glisson's capsule



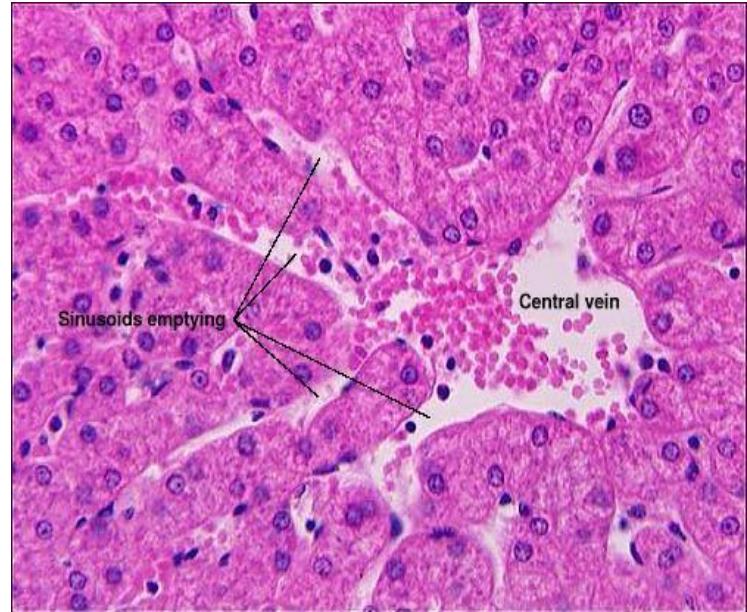
# Microscopic structure

- Hepatocytes are arranged in plates, seen as cords
- Classical functional unit of liver- hepatic lobules
- A hexagonal structure where hepatocytes arranged cords



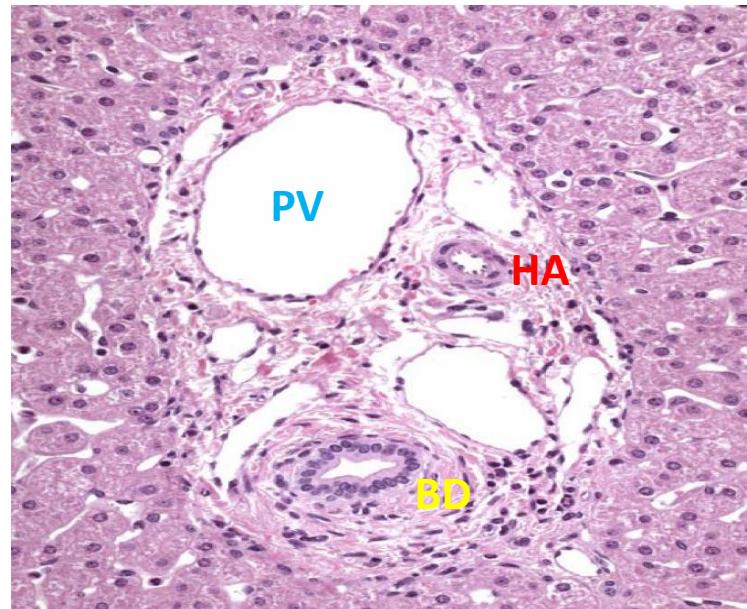


- At the center central vein
- Around periphery of each lobule is 4-5 portal triad

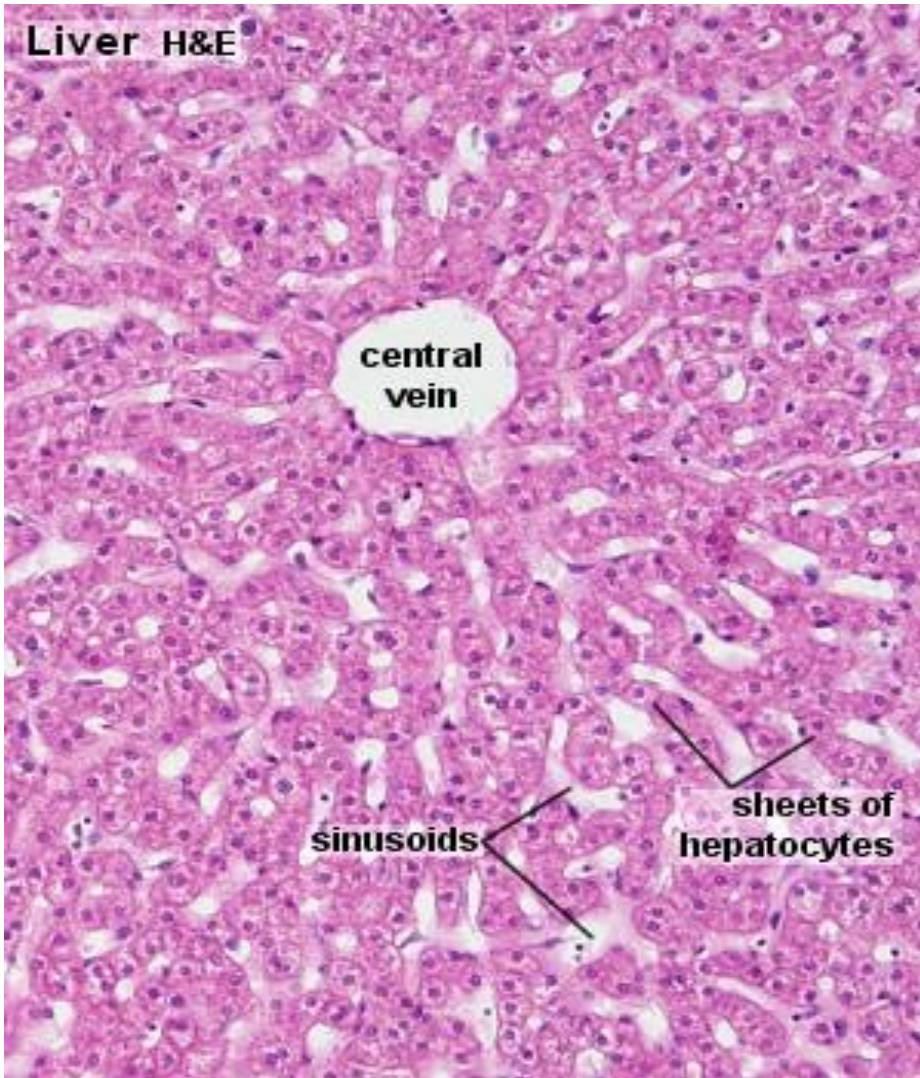


## Portal triad

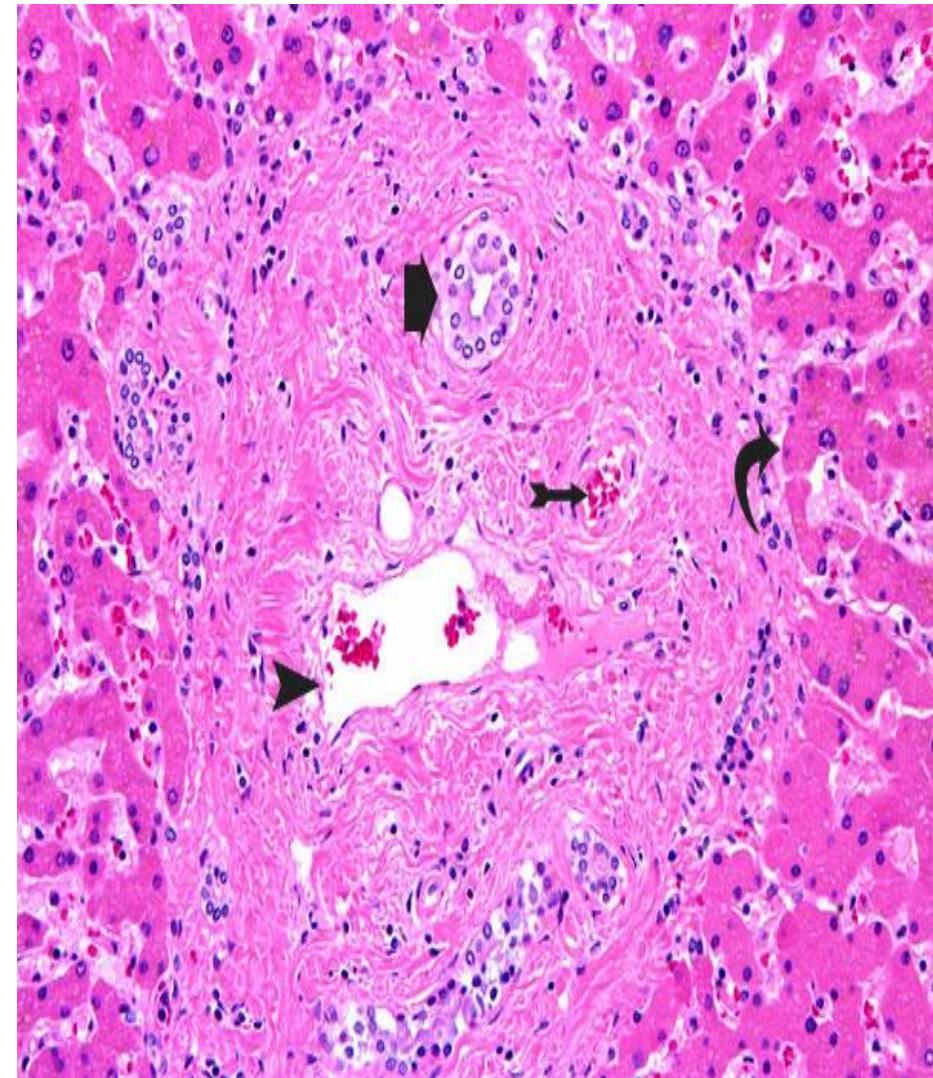
- Hepatic artery
- Portal vein
- Bile duct
- Several lymph vessels



Liver H&E

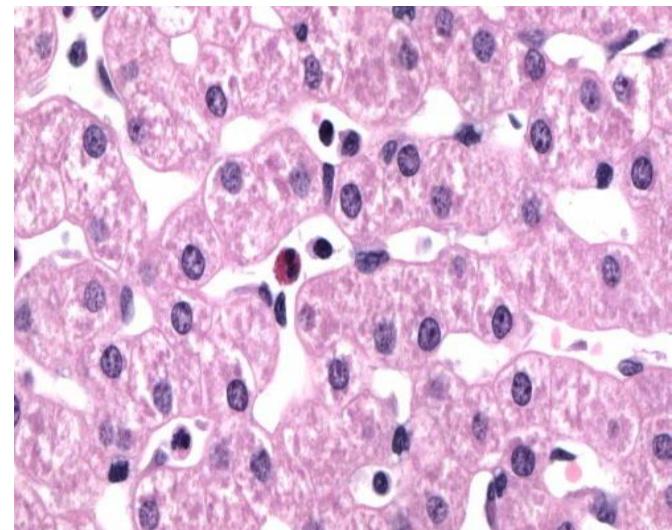
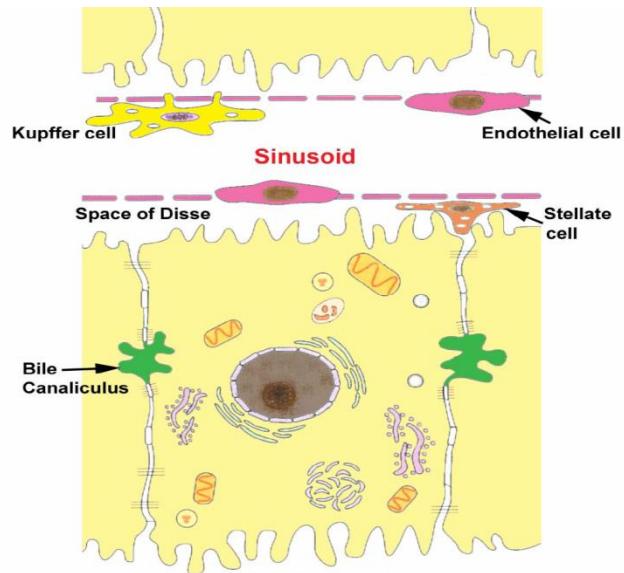


- ❖ Liver divides into lobules with a central vein
- ❖ The plates of the hepatic cells radiate from the central vein



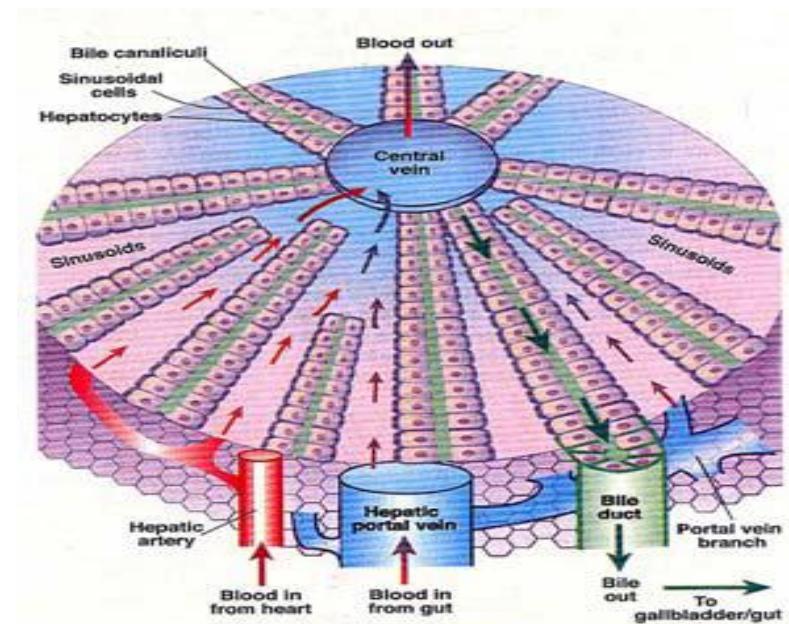
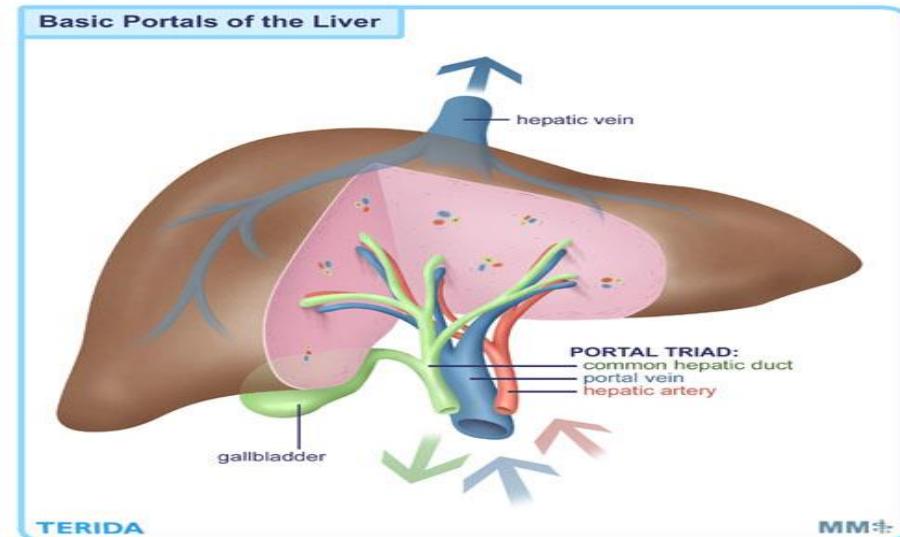
# Parenchymal microanatomy

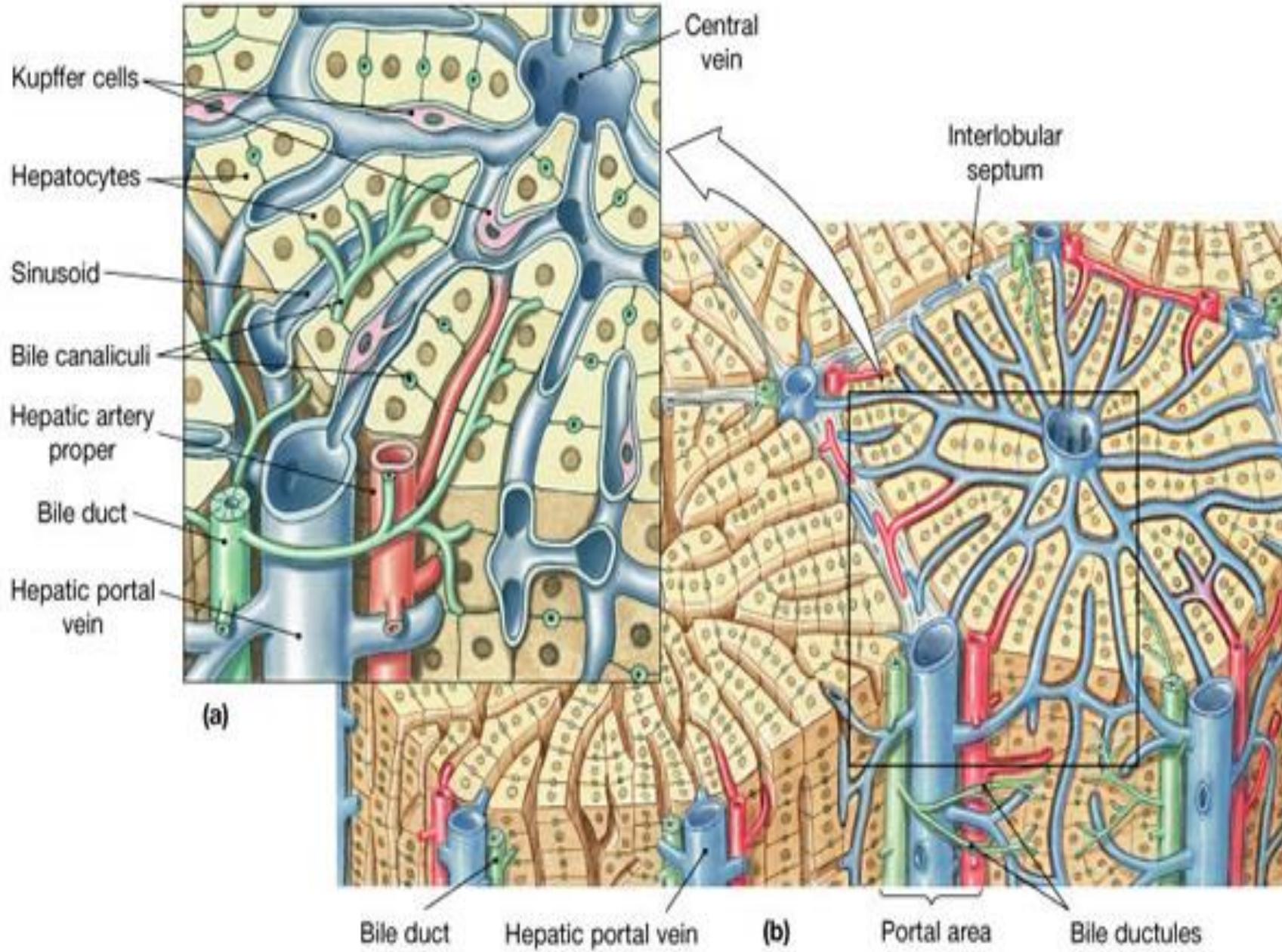
- Cords of hepatocytes are separated by sinusoids, lined by fenestrated endothelium and phagocytic **Kupffer's cells**
- **Space of Disse**- space between endothelium and hepatocytes
- **Ito cells/ lipocytes/ stellate cell**- fat containing cells- interspersed between hepatocytes



## Parenchymal microcirculation

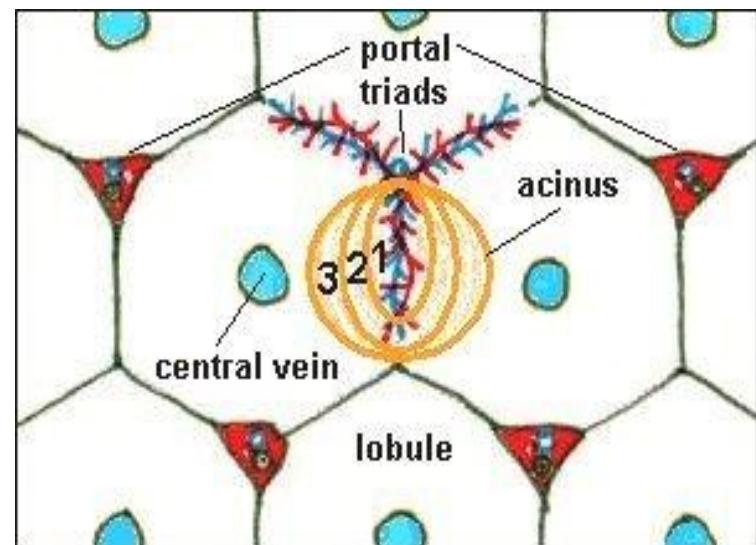
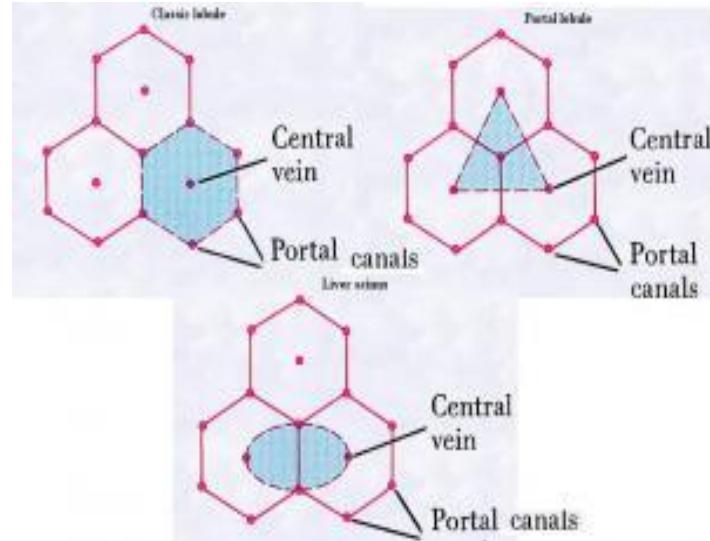
- ❖ Dual blood supply
- ❖ Portal blood and hepatic artery blood mix in the sinusoids
- ❖ Bile flow from central to towards the periphery of hepatic lobules

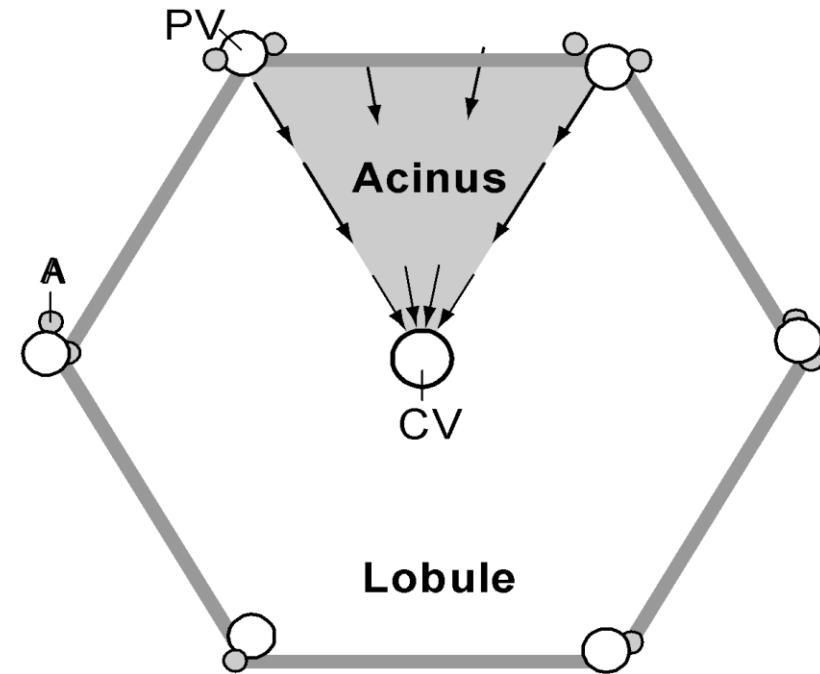
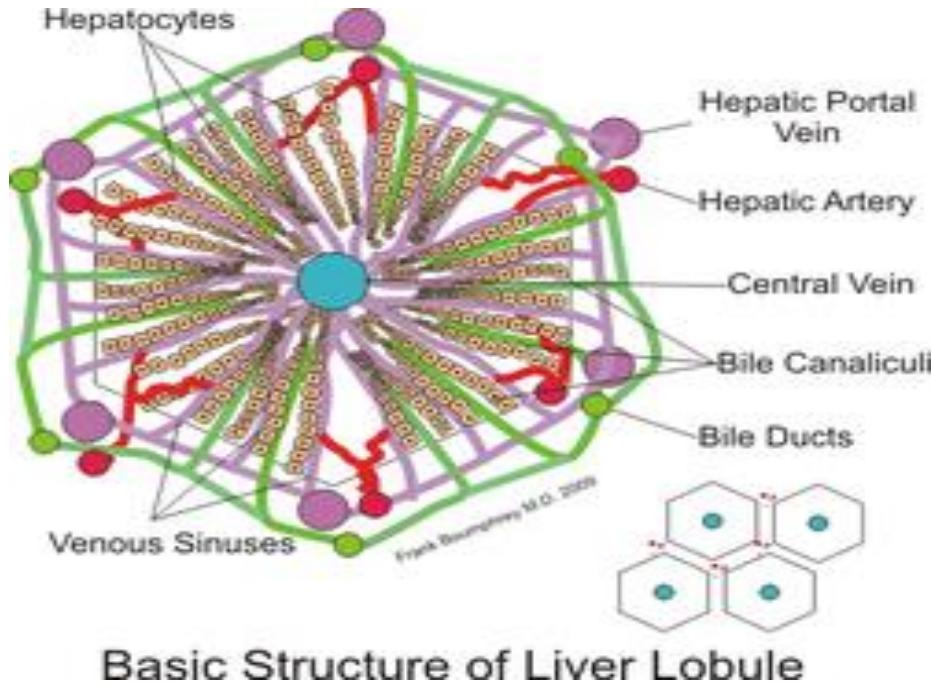


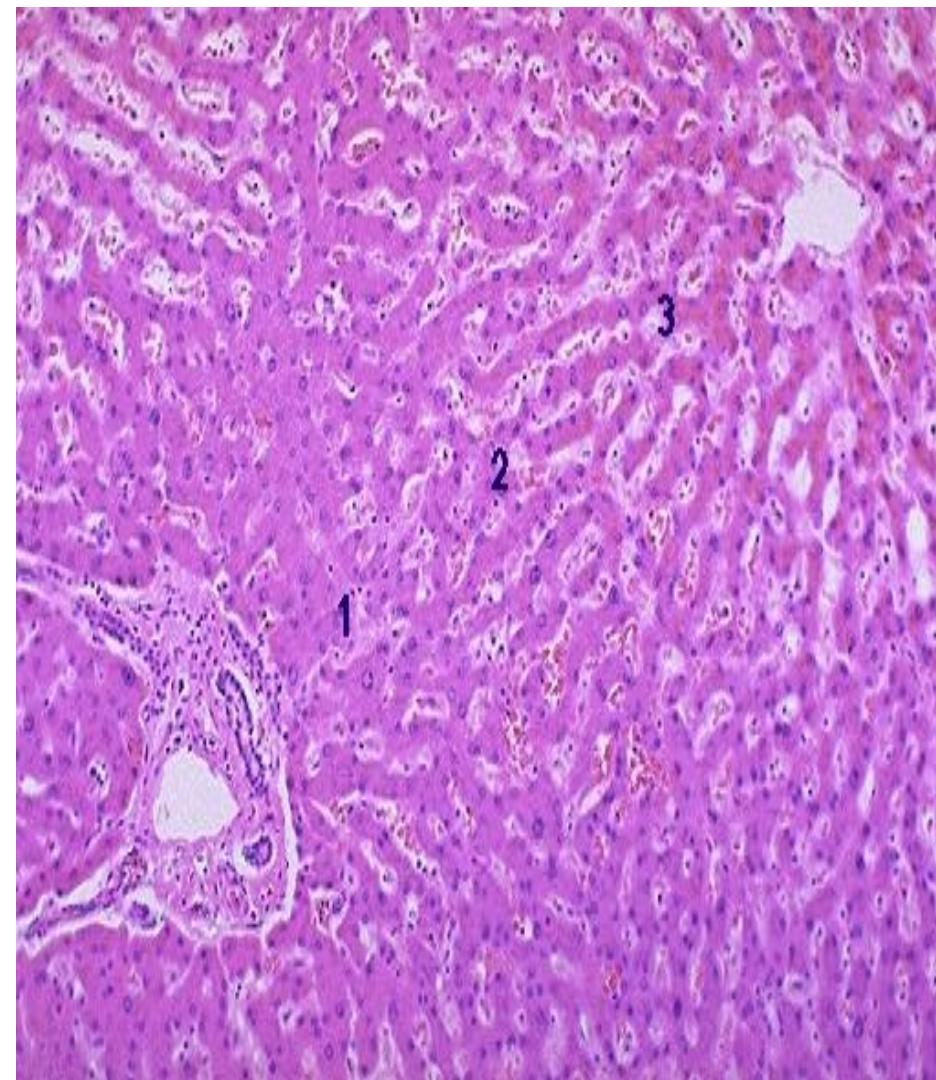
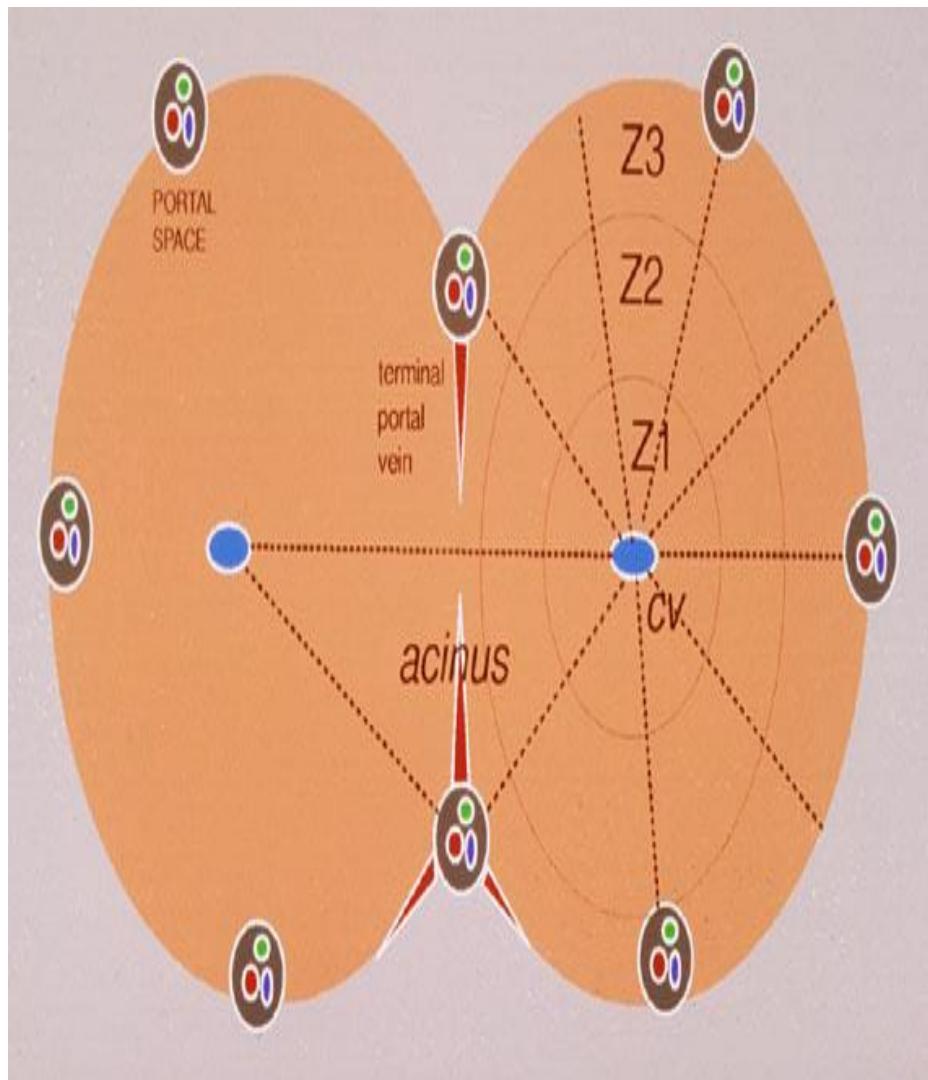


# Rappaport, 1973

- Defined hepatic lobules as functional or metabolic lobules also called as hepatic acinus
- Consists of center portal triad and pie shaped segments of adjacent anatomic lobules
- Three types of hepatocytes in metabolic lobules
- Hepatocytes surrounding the triad (Rappaport zone 1)
- Mid-zone hepatocytes (Rappaport zone 2)
- Periacinar hepatocytes (Hepatocytes near central vein)





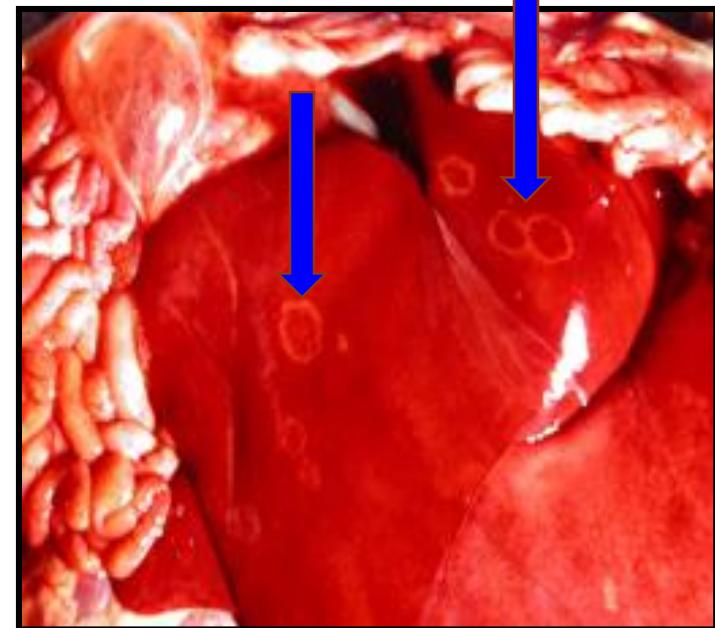


# NECROSIS

## Focal necrosis

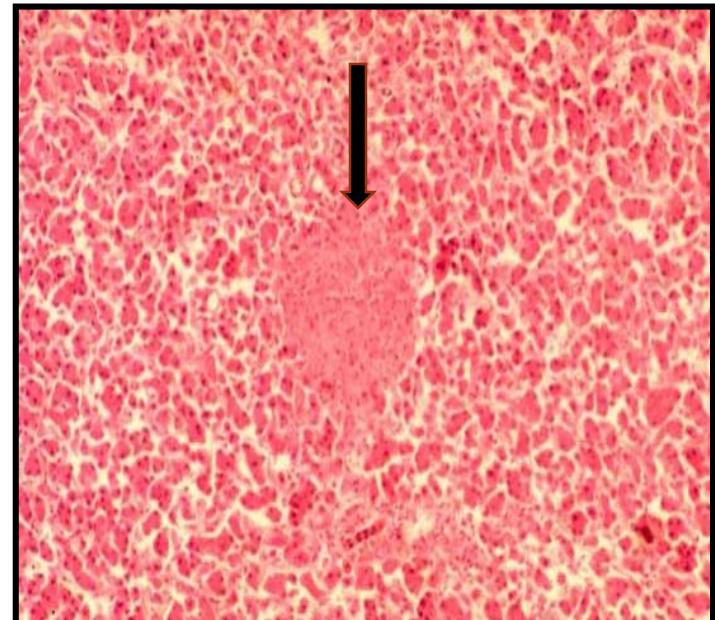
Grossly,

- ❖ Small necrotic areas or foci, of sub cellular size in any part of the lobules



Microscopically-

- ❖ Focal necrotic areas scattered in the parenchyma

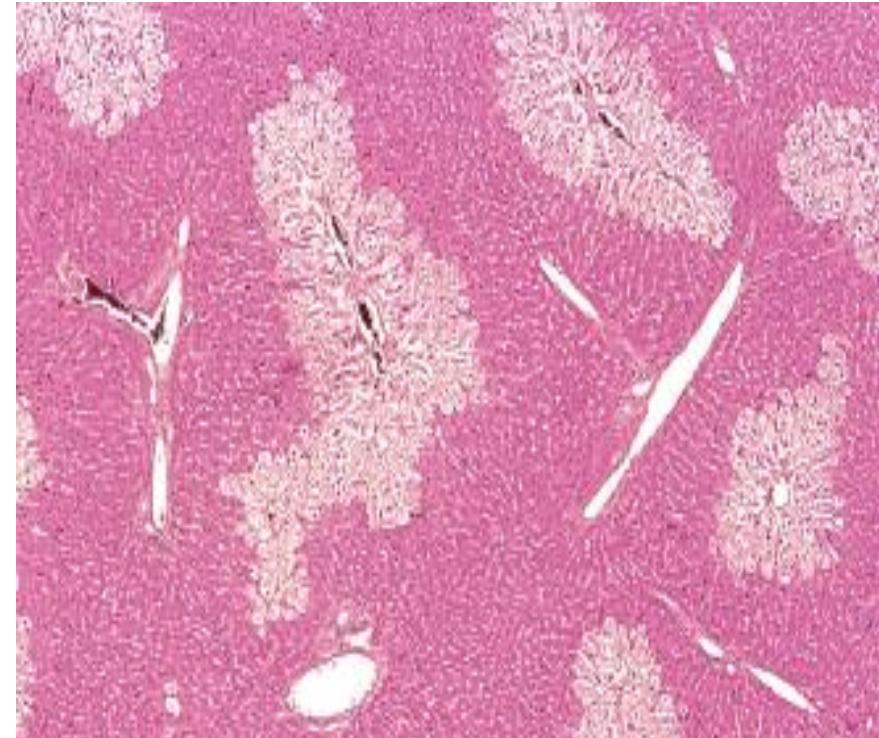
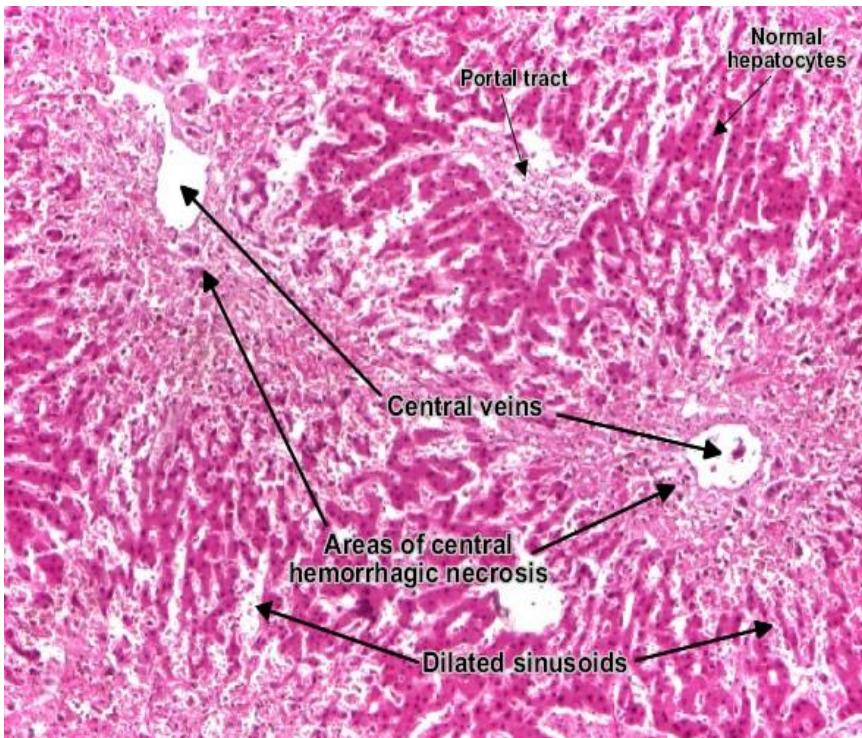


# Centri-lobular Necrosis

- Hepatocytes nearest the central vein are affected
- Receive least oxygenated blood and has highest enzymatic activity
- Most commonly seen in hypoxic condition like passive congestion and severe anemia

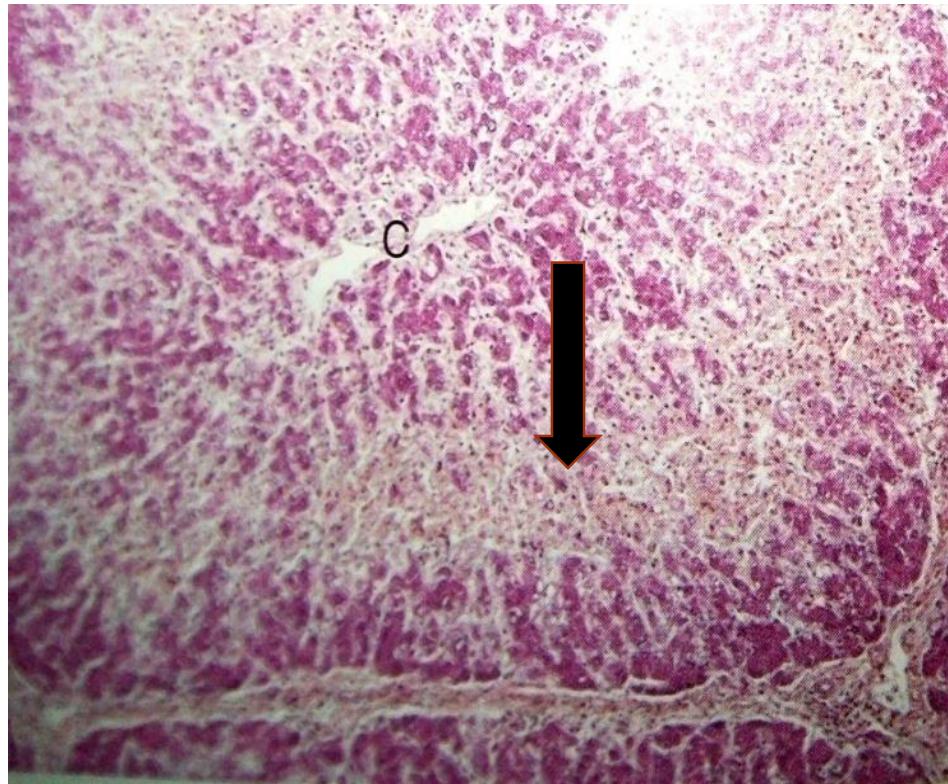
## Microscopically

- Cells near the central vein shows necrosis
- May disappear, replaced by blood
- Peripheral cells may show cloudy swelling, fatty change



## Mid-zonal necrosis

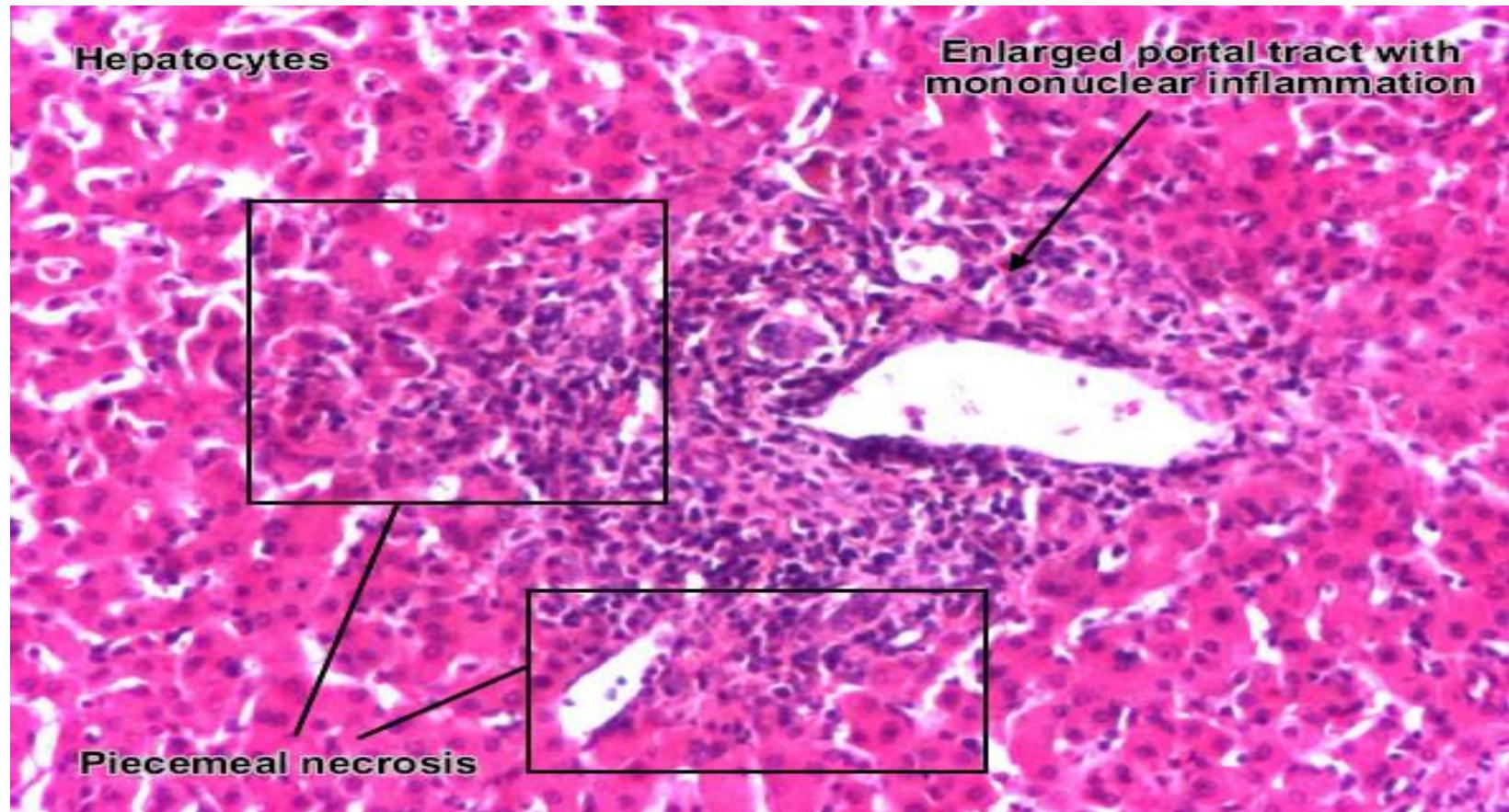
- Hepatocytes mid-way between the periphery and the central vein
- Reported in pigs and horses with aflatoxicosis



**Reported in pigs and horses with aflatoxicosis**

# Periportal Necrosis

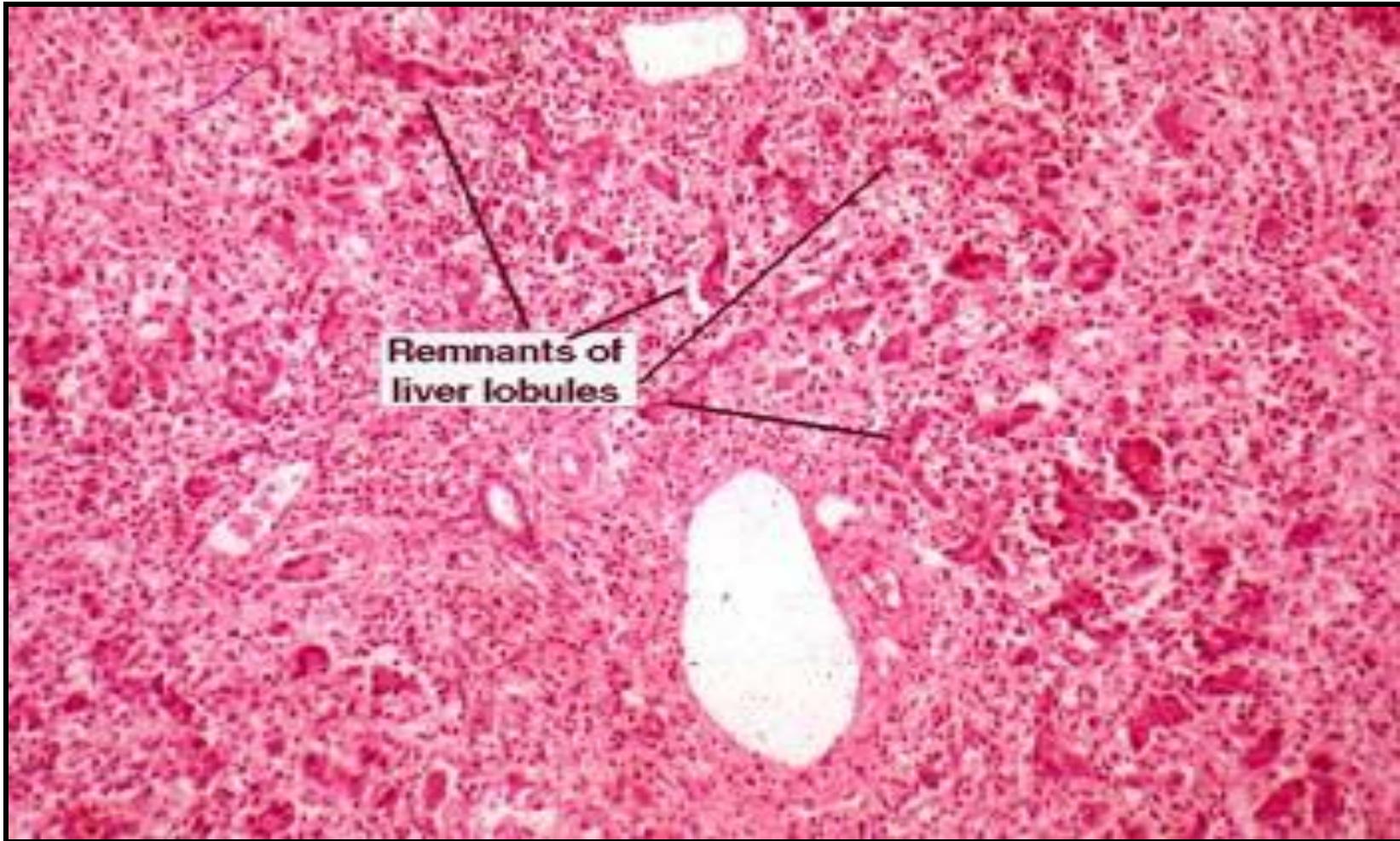
- The cells adjoining the portal triad become necrotic
- Common when the toxins have been conveyed by portal vein and do not require metabolism
- Phosphorous poisoning



**Phosphorous poisoning ,  $\alpha$ -naphthyl isothiocyanate and Allyl alcohol**

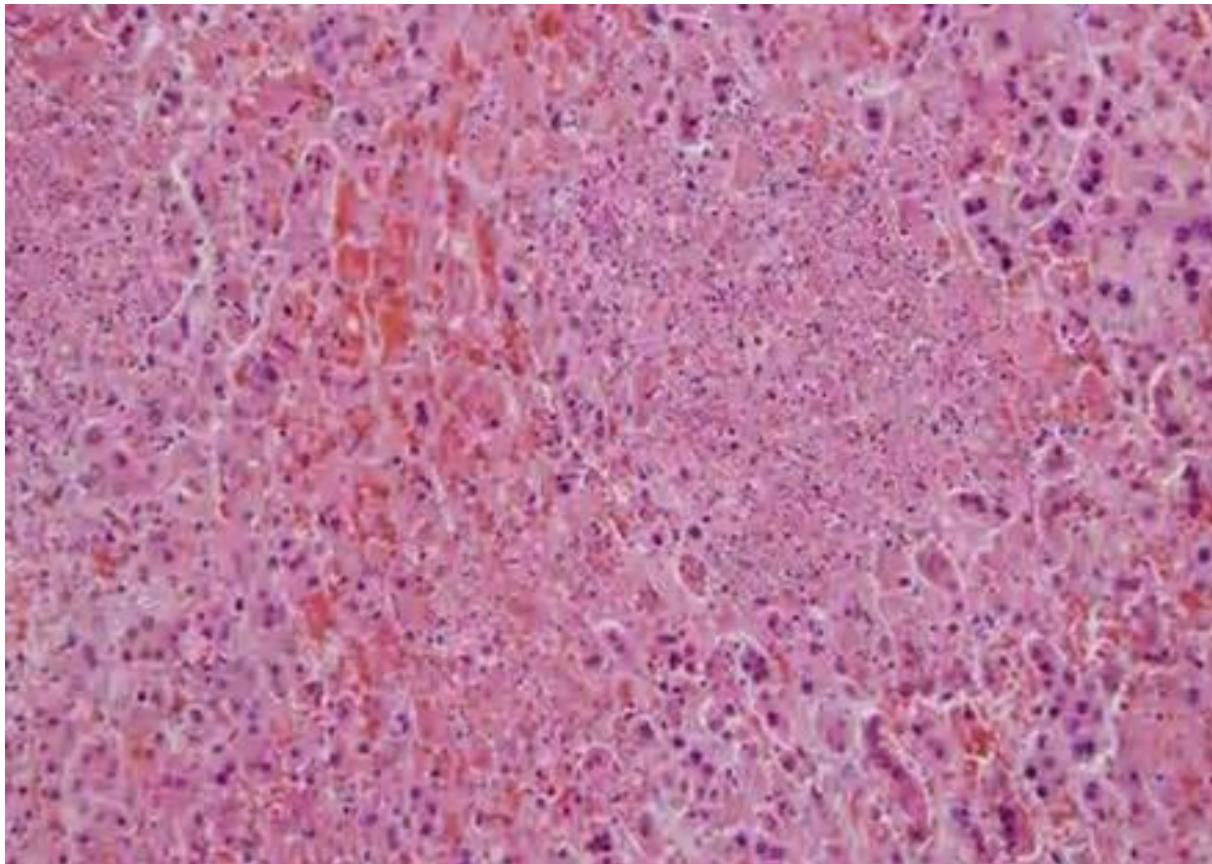
# Para-central necrosis

It is a form of coagulative necrosis, occurs when an isolated complete hepatic acinus dies and is viewed in transverse section.



# Massive Necrosis

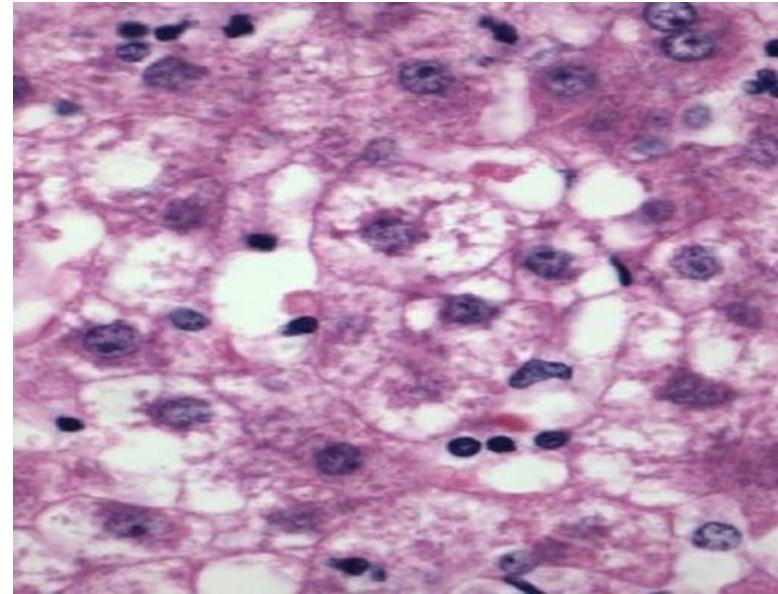
- Necrosis of entire lobules, also nearby lobules
- Outcome of extensive zonal necrosis or circulatory disease
- Also in *hepatosis dietetica* (pig)



# Response to toxic injury:

- **Acute:**

- Cellular swelling & accumulation of lipid
- necrosis, common in peri-portal areas

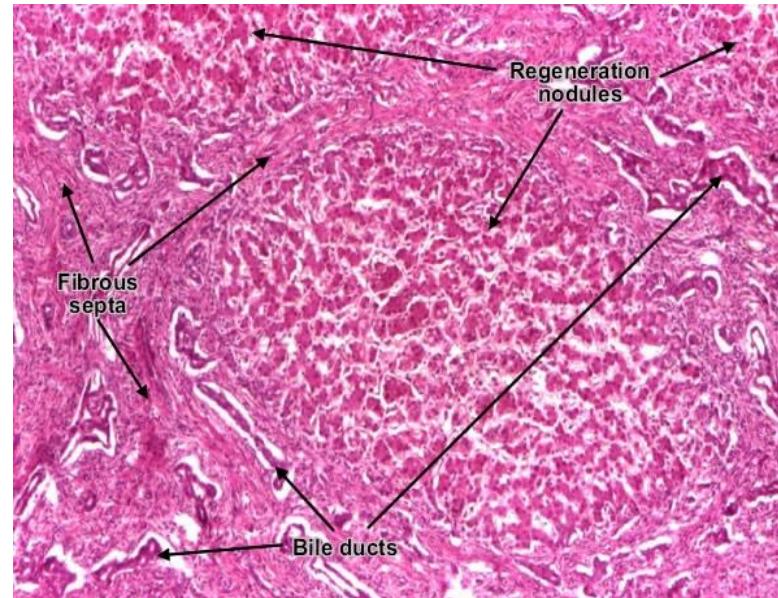


- **Chronic:**

- Fibrosis
- Biliary hyperplasia
- Parenchymal regeneration

- Toxic injury leads to cirrhosis

- Aflatoxicosis – neoplasia
- Hepatic failure



# AUTO IMMUNE HEPATITIS

- Chronic active hepatitis due to immune process directed against liver antigens
- Precipitated by exposure to environmental agent which triggers - autoimmune response
- In Dogs - *Leptospira, ICHV* Infection

## Gross Examination

- ❖ Liver is small, distorted, coarse nodular texture to an end stage Liver

## Microscopic Examination

- ❖ Periportal fibrosis which extends towards central vein – bridging fibrosis
- ❖ Accumulation of inflammatory cells lymphocytes in portal tracts and adjacent periportal areas of lobules

# EQUINE SERUM HEPATITIS

- Serum hepatitis, Equine viral hepatitis, Theilers disease
- Occurs in Horses which received an injection / therapeutic agent
- Among agents are immunizing sera against Equine infections

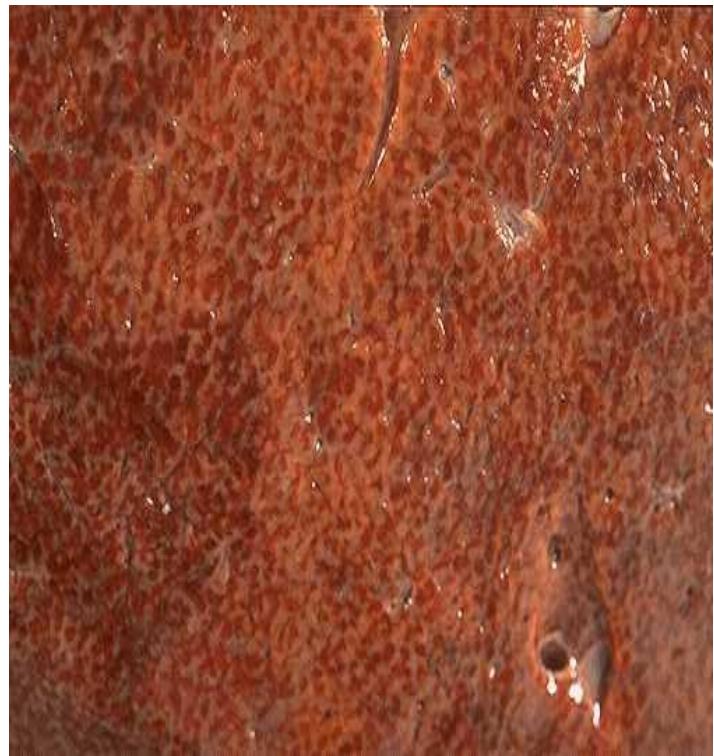
Tetanus toxin

African horse sickness

Equine encephalomyelitis

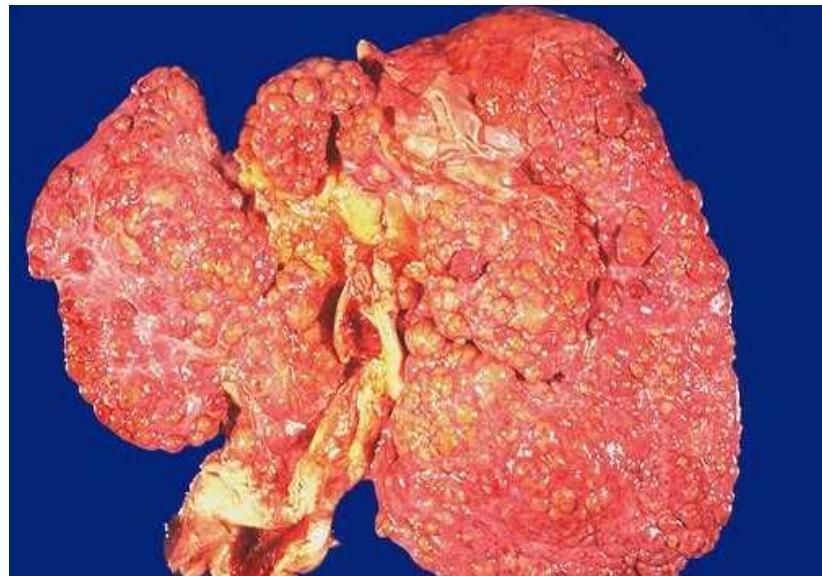
## Lesion –

- ❖ Liver is small and friable , Icterus
- ❖ Liver is swollen with nutmeg appearance
- ❖ Centrilobular necrosis



# CIRRHOSIS OF LIVER

- Kirrhos - Tawny or orange colored liver
- Best accepted definition for cirrhosis define by WHO (1977)
  - *"a diffuse process characterized by fibrosis and the conversion of the normal liver architecture into structurally abnormal lobules"*
- End-stage liver- final and irreversible
- The altered architecture is due to
  - Loss of hepatic parenchyma
  - Condensation of reticulin framework
  - Formation of tracts of fibrous tissue



- The stimulus for the fibroblastic proliferation is some irritant, chronic and severe enough to produce degeneration and necrosis of the parenchymatous cells.
- The irritant may reach the liver through (a) The portal vein (b) hepatic artery and (c) Bile ducts.

## Etiology

- ▶ Chronic toxic insult- continued ingestion of hepatotoxin (plant containing pyrrolizidine alkaloid, alcohol in human)
- ▶ Chronic cholangitis and/ obstruction- parasitic infestation like *F. hepatica*
- ▶ Chronic congestion- right sided heart failure leads to necrosis and fibrosis near central vein
- ▶ Chronic hepatitis- chronic infection with HBV in man, CHV in dog
- ▶ Inherited disorder of metal metabolism- hereditary copper accumulations, producing cirrhosis in Dalmatian, white terrier
- ▶ Idiopathic- poorly defined disease entities like ovine hepatic fatty cirrhosis

# Types of Cirrhosis

- ❖ Portal or nodular cirrhosis
- ❖ Multinodular or Atrophic or Gindrinker's or Laennec's cirrhosis
- ❖ Biliary cirrhosis (Monolobular or hypertrophic cirrhosis)
- ❖ Other forms of cirrhosis
  - Pigment cirrhosis
  - Pericellular cirrhosis
  - Glissonian cirrhosis
  - Cadiac or central or congestive or stasis cirrhosis
  - Parasitic cirrhosis

# PORTAL OR NODULAR CIRRHOSIS

## Etiology

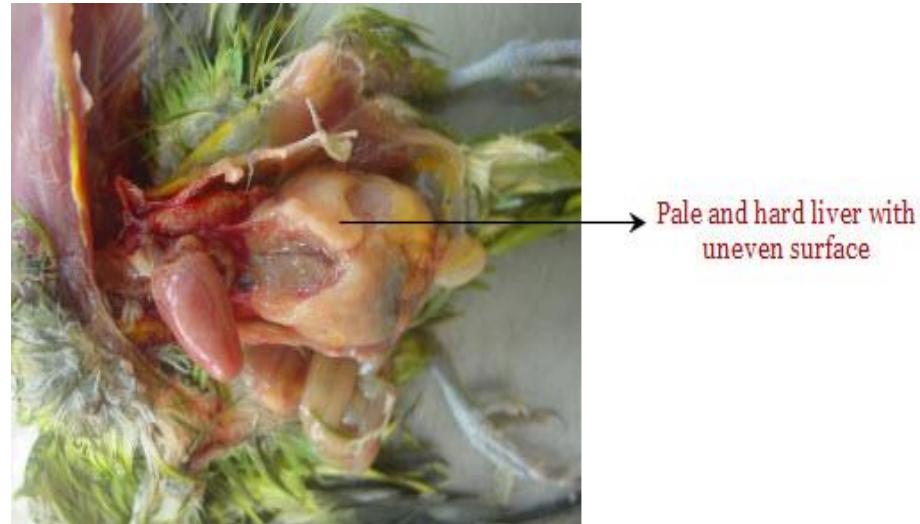
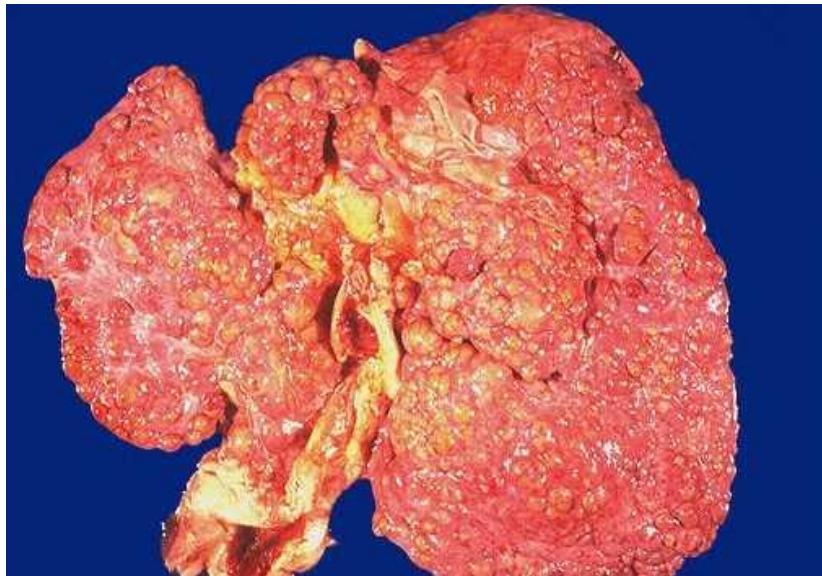
- Frequently one may not be able to ascertain the cause
- The irritant is mild and acting for a long time
- Usually, the causes are the same as described for acute focal toxic hepatitis
- Toxic plants: *Crotalaria sagittalis* in horses; plants of *Senecio* family in horses, cattle and sheep; *Atalaya intermedia* in horses; *Amsinckia intermedia* (tar weed) in horses, swine and cattle; plants containing high selenium content-in horses. (Wheat loco weed)
- Chemicals: Pitch in tar paper, repeated exposure to chloroform, carbon tetrachloride and phosphorous
- Long continued intestinal toxæmia

# Pathogenesis

- Irritant is conveyed via the portal veins, changes are first at the periphery of the lobules –area next to the portal tract
- Stimulation of the interlobular connective tissue to proliferate
- New irregular blood vessels are formed which anastomose with the network of the portal vein and hepatic artery resulting into arterio-venous shunts. Thus ischaemia of some parts of the liver occurs leading to further hepatic necrosis
- The newly formed after maturation contracts, interfering with blood circulation
- Due to resultant ischaemia, hyperplasia does not progress further
- As the fibrous tissue grows into the lobule, the hepatic cells become atrophied
- The fibrosis constricts the central vein impeding the out flow of blood, thereby rendering the irritant to stay longer in the liver
- Growth of the fibrous tissue into the lobule divides the parenchyma into small islands of hepatic cells-pseudolobulation
- In the new fibrous tissue, especially in the portal areas, new bile ducts are formed
- These are not functional, lacking an outlet and so stasis of bile occurs

## Gross pathology

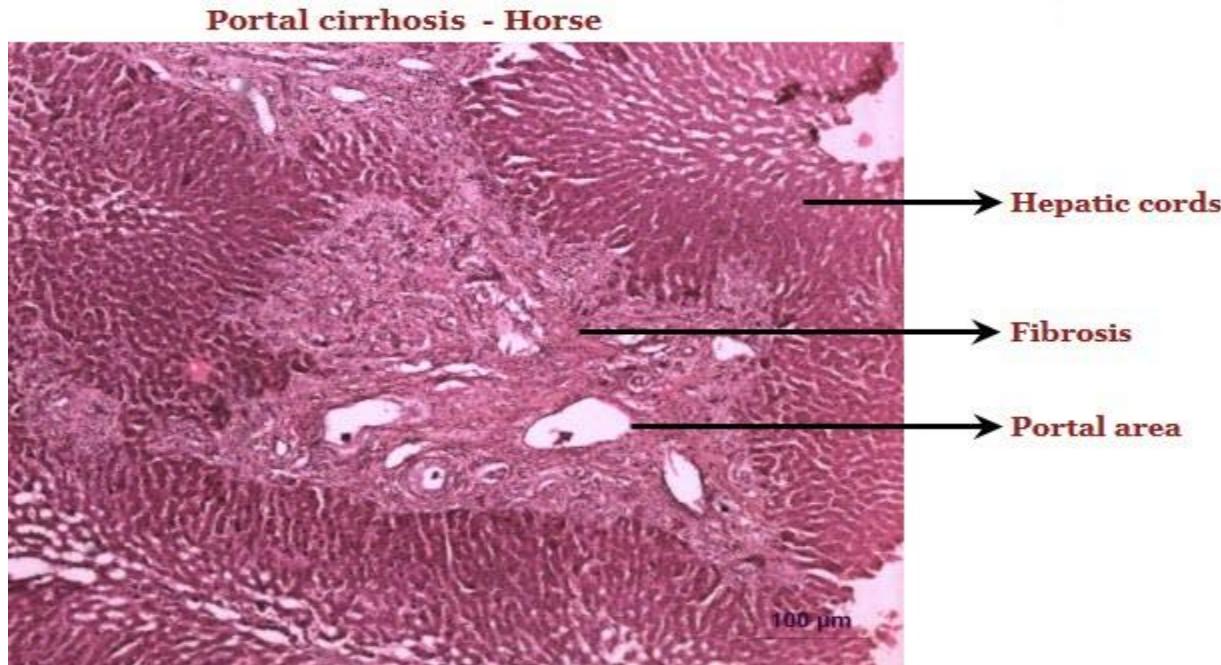
- ❖ In the early stages the liver may be large. But as the condition progresses, due to atrophy of the parenchyma, the liver is reduced in size
- ❖ Due to biliary stasis, the color of the organ **is tawny or yellowish-gray or green** and it is to this color that the name “Cirrhosis” was first applied
- ❖ The liver surface is uneven and nodular (Hobnail liver)
- ❖ The liver is hard and firm
- ❖ On section, the liver cuts with difficulty giving a **peculiar grating sound** due to the dense fibrous tissue formed



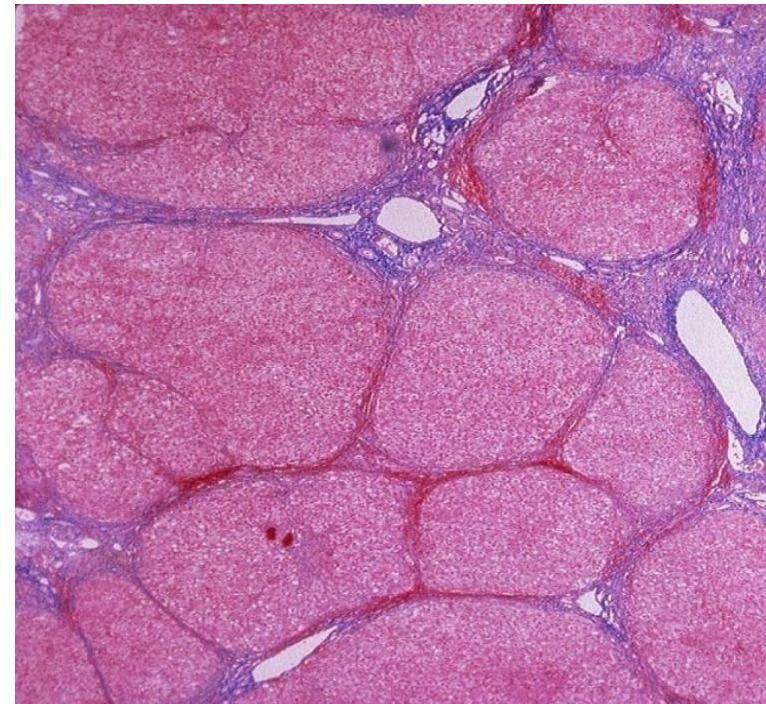
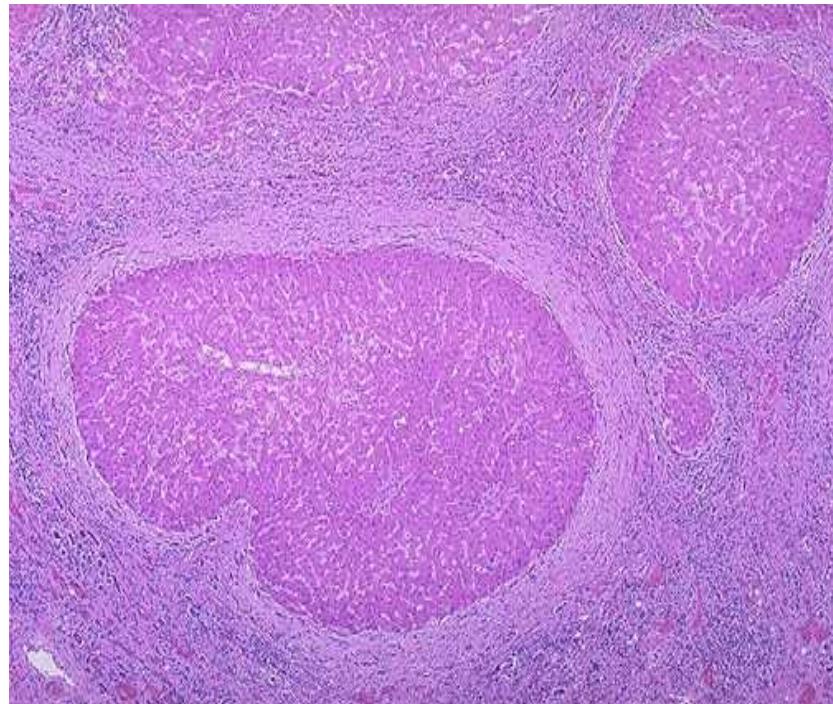
Cirrhosis of liver in a budgerigar

# Histopathology

- Architecture of the liver is lost
- Increase in fibrous tissue within and around the lobules
- New bile ducts and inflammatory cells (lymphocytes and macrophages) are present
- Deposition of the bile pigment is present
- Central veins in some lobules are either absent or are placed eccentrically (Indication of pseudolobulation)
- Parenchymatous cells show various stages of degeneration - cloudy swelling, fatty change and even necrosis
- Hyperplasia that is present gives nodularity to the organ



- Characterized by - diffuse hepatic fibrosis , altered reconstruction of lobular parenchyma with widespread CT septa
- Circumscribed regenerative nodules of hepatocytes due to regeneration of hepatocytes between fibrous tissue
- Lack central vein (**pseudolobulation**)



## Sequelae

- Once the fibrous tissue is stimulated to proliferate, this proliferating fibrous tissue itself becomes an irritant
- So, even if the original irritant is removed or destroyed, cirrhosis progresses with more and more fibrous tissue formation until the condition terminates fatally

# BILIARY CIRRHOSIS (MONOLOBULAR OR HYPERTROPHIC CIRRHOSIS)

- Biliary cirrhosis in animals is rare because cholangitis and cholangiostasis do not occur in them
- Liver flukes that inhabit the bile ducts do not cause extensive cirrhosis but only a local fibrosis

## Etiology

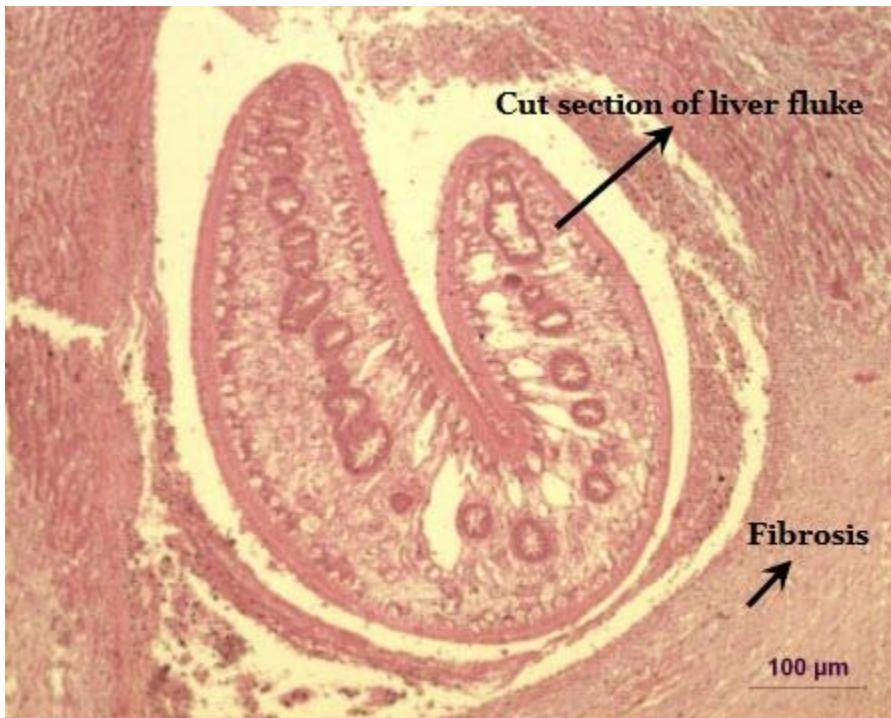
- In man this type of cirrhosis occurs consequent on obstruction and infection of the biliary tract
- The causes are
  - Cholangitis-the inflammatory exudate clogs the bile ducts
  - Stone in the common bile duct.
  - Obstruction of biliary passages by flukes (*Clonorchis sinensis*) and ascarids
  - Stricture of the bile duct
  - Extramural pressure on the bile ducts from tumour of pancreas

## Gross pathology

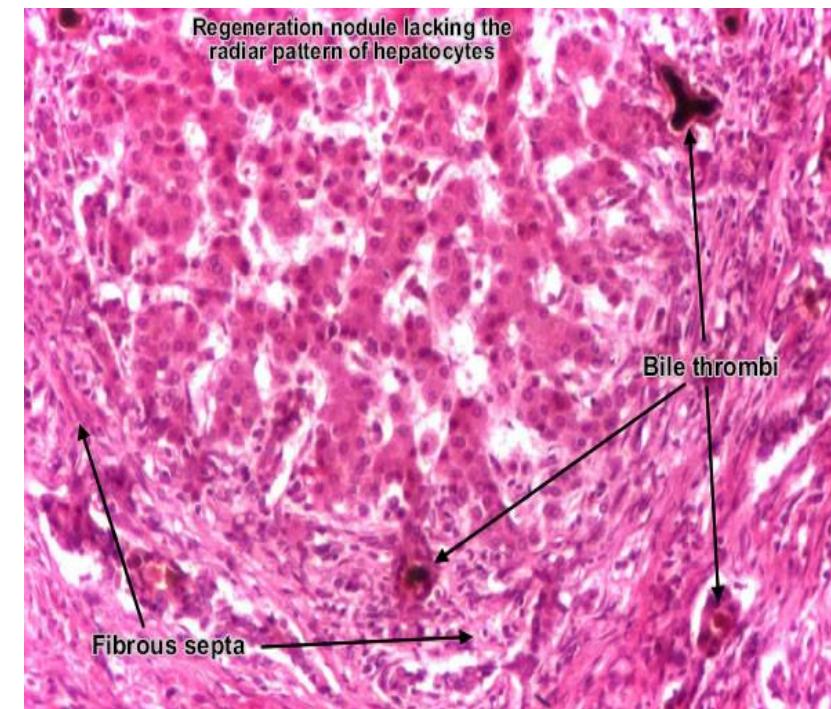
- The liver is enlarged, greenish and the surface is either smooth or finely granular

## Histopathology

- Connective tissue encircles individual lobules (hence monolobular)
- Bile ducts may be dilated and tortuous
- Infiltration of the connective tissue with chronic inflammatory cells
- Newly formed non-functional bile ducts are also found
- Hepatic cells reveal degenerative changes



Biliary cirrhosis - Liver fluke - Sheep



# MULTINODULAR OR ATROPHIC OR GINDRINKER'S OR LAENNEC'S CIRRHOSIS

## Etiology

- Deficiency of Vitamin B complex and lipotropic factors, especially in drunkards produces this condition

## Pathogenesis

- Lack of Vitamin B complex and lipotropic factors will result into a highly fatty liver and subsequently a severe fibrovascular proliferation

## Gross pathology

- The gross changes are more or less similar to portal cirrhosis

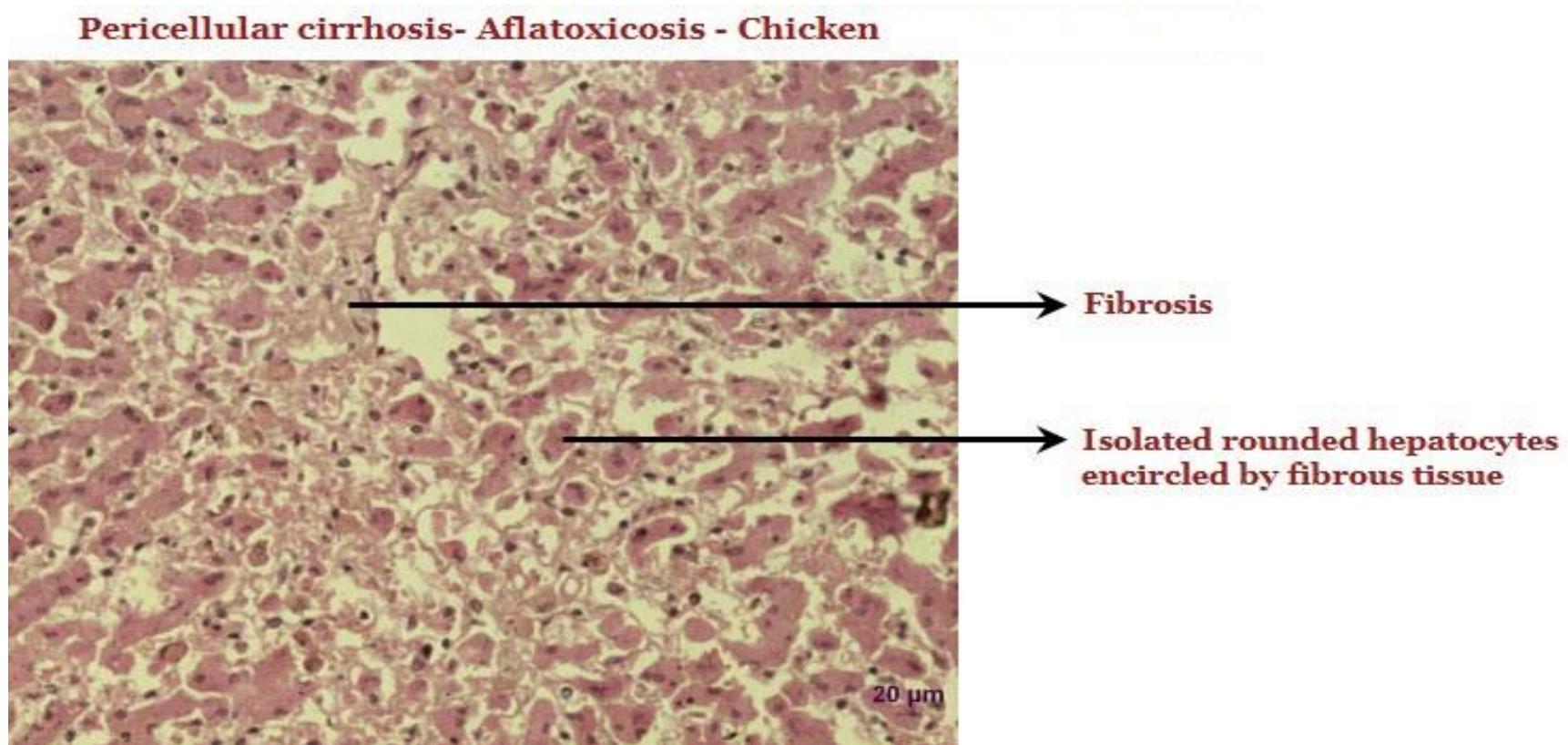
## Histopathology

- The hepatocytes show severe fatty changes
- The bulging cells, pressing on the sinusoids produce ischemia resulting in necrosis of the parenchyma
- New capillaries form and invade the lobule and connect the central vein with the portal vessels
- There is proliferation of the fibrous tissue which is infiltrated by chronic inflammatory cells
- The fibrous tissue divides the parenchyma into smaller lobules
- Some surviving cells proliferate and form nodules (Hobnail)
- Contracting fibrous tissue makes the liver smaller and hence “**Atrophic cirrhosis**” results

# OTHER FORMS OF CIRRHOSIS

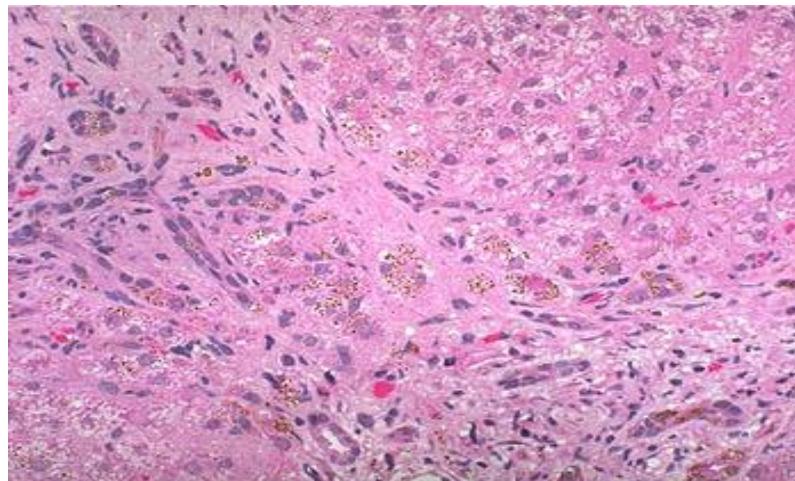
## Pericellular cirrhosis

- In this condition the fibrous tissue invades the parenchyma and encircles individual cells
- This picture may be seen in the far advanced stages of the multi and monolobular cirrhosis
- In well developed aflatoxicosis pericellular cirrhosis is often found



## Pigment cirrhosis

- ❖ This is the fibrotic condition of the liver that is found in hemochromatosis (bronzed diabetes of man)
- ❖ The large amounts of hemosiderin deposited in the hepatic cells irritate the organ causing cirrhosis
- ❖ The macroscopical and microscopical appearances are similar to mild portal cirrhosis with nodulation

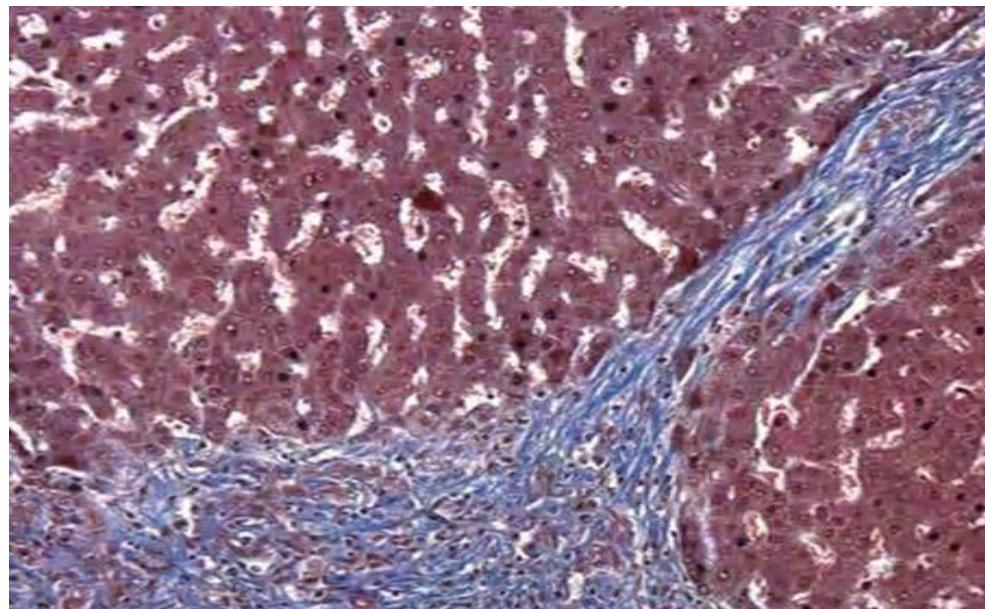


## Glissonian cirrhosis

- ❖ This is not true cirrhosis since the liver as a whole is not affected
- ❖ Inflammation and thickening of the Glisson's capsule (the result of regional peritonitis) extends to the adjacent liver parenchyma
- ❖ Though macroscopically resembling portal cirrhosis, microscopically the fibrosis is seen to extend from the capsule to a short distance beneath it

## Cadiac or central or congestive or stasis cirrhosis

- In chronic venous congestion resulting from cardiac lesions, the cells round about the central veins suffer degeneration and necrosis due to pressure and hypoxia
- **Hepatic fibrosis around the central vein resulting from CHF**
- As the hepatic cells disappear, a relative increase in the fibrous tissue is evident
- Later, diffuse fibrosis and alteration in the architecture occurs in some cases
- This may give rise to atrophy and granular appearance of the organ



# Parasitic Cirrhosis

- The cause is usually a chronic obstruction of the bile ducts by flukes or other parasites
- In **swine mature ascarids** invading the bile ducts cause biliary obstruction
- The changes are localized and are usually restricted in animals to fibrosis of parenchyma for a short distance around the biliary passages
- Cirrhosis may spread out due to the diffusion of bile into the parenchyma that surrounds the bile ducts
- The gross and microscopic picture is more or less similar to biliary cirrhosis

# SEQUELAE / EFFECTS OF CIRRHOSIS

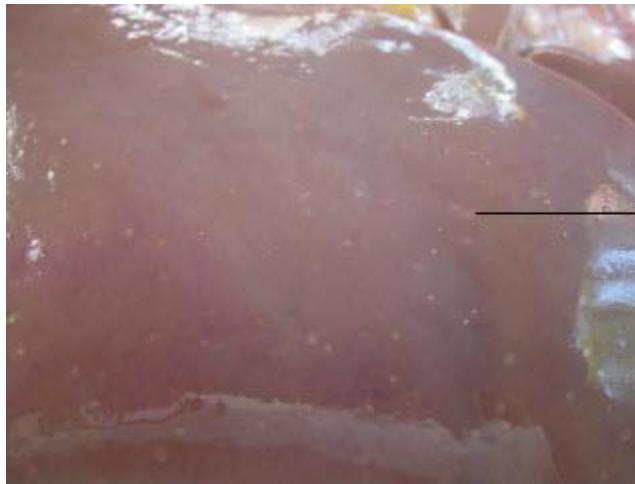
- Due to disturbance in portal circulation
  - Ascites due to
    - Increased hydrostatic pressure in portal veins due to compression of veins and distortion of the portal and hepatic veins as well as sinusoids
    - Decreased colloid osmotic pressure due to decreased production of plasma proteins, particularly albumin
    - Hormones are not inactivated by a damaged liver. Hence mineralocorticoids and the anti-diuretic hormone leads to retention of sodium chloride and generalised oedema
  - Varicosity and rupture of oesophageal veins may lead to haematemesis
  - Splenomegaly
  - Chronic gastroenteritis as a result of chronic venous congestion of abdominal viscera

- Loss of Inactivation of hormones and detoxification
  - Estrogens normally are inactivated in the liver in the male. In hepatic cirrhosis this does not occur and so **gynecomastia and testicular atrophy** occur.
  - Toxins – exogenous or endogenous are normally detoxified by the liver. If this is not done, the toxins affect the brain, producing degenerative changes resulting in “**Walking disease**” in horses.
- Jaundice: Pressure on bile canaliculi by the compressed cord cells (by fibrous tissue) - **Obstructive jaundice**
- Bleeding due to deficiency in production of prothrombin
- Anaemia: Iron and erythrocyte maturation factor cannot be stored
- Vitamin A deficiency

# Other Pathological Conditions of Liver

## SAW DUST LIVER

- In well-fed young cattle, at post-mortem, focal necrosis of the liver is commonly seen
- Irritant is borne by the portal veins from the gut and so the lesions are found nearer the portal areas
- These spots are evidently scars resulting from inflammatory reaction
- Hepatic cells undergone coagulative necrosis
- Infiltration by lymphocytes and neutrophils

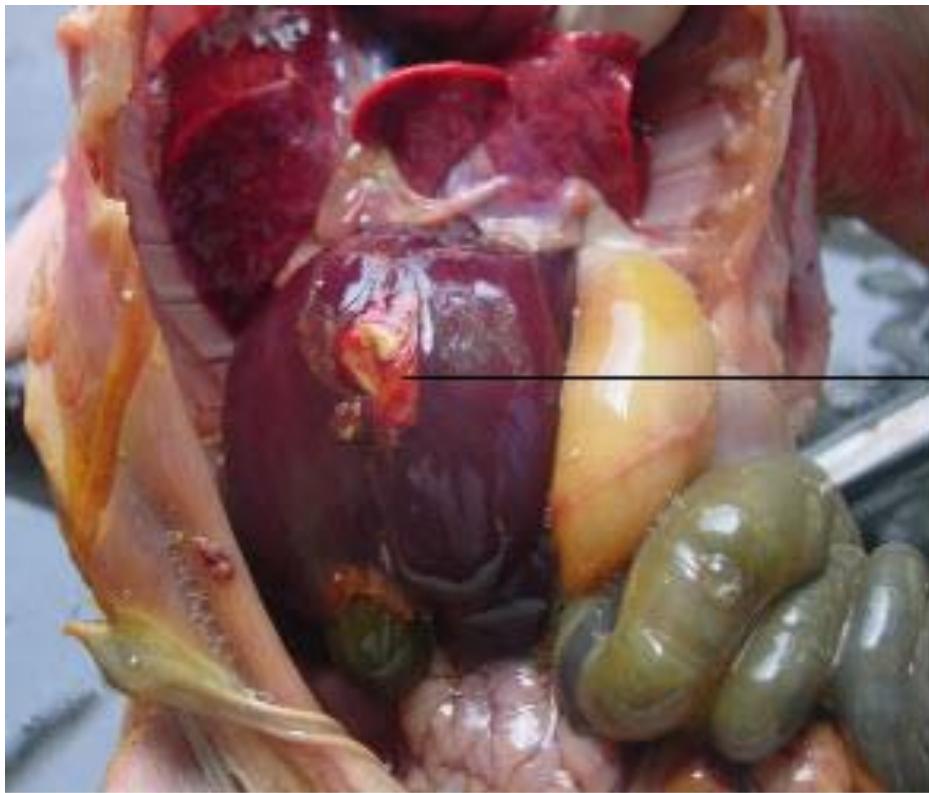


→ Multifocal necrotic spots  
on the liver surface

Sawdust liver in a cow

## TRAUMATIC INJURY TO LIVER

- Traumatic injury to liver may occur due to automobile accidents, crushing by dam in case of young ones etc.
- At the site of rupture of liver blood clots and fibrin may be present



Rupture of liver in a piglet - Crushing injury

# NEOPLASMS OF LIVER

- Tumours of the liver may be primary (arising from the liver parenchyma and bile ducts) or secondary metastases from elsewhere

## Primary neoplasms

- The most common primary tumours are hepatomas, hepatocellular carcinomas and cholangiocellular carcinoma
- In dogs, hemangiomas are common
- Primary fibroma may also be found in the liver
- In chicken, lymphoid leucosis and Marek's disease primarily affect liver

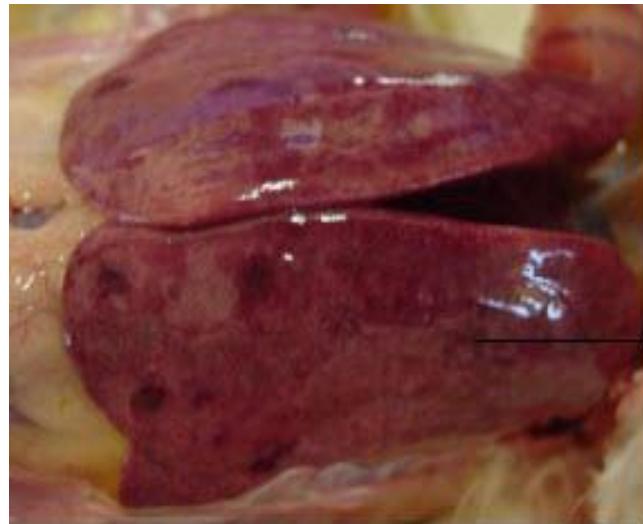
## Secondary

- Metastases of any malignant tumor may be found in the liver
- Metastases of lymphocytoma and pancreatic carcinoma are mostly seen
- In the cow, metastases of uterine carcinoma are common
- Mammary gland carcinoma in the dog metastasizes in the liver



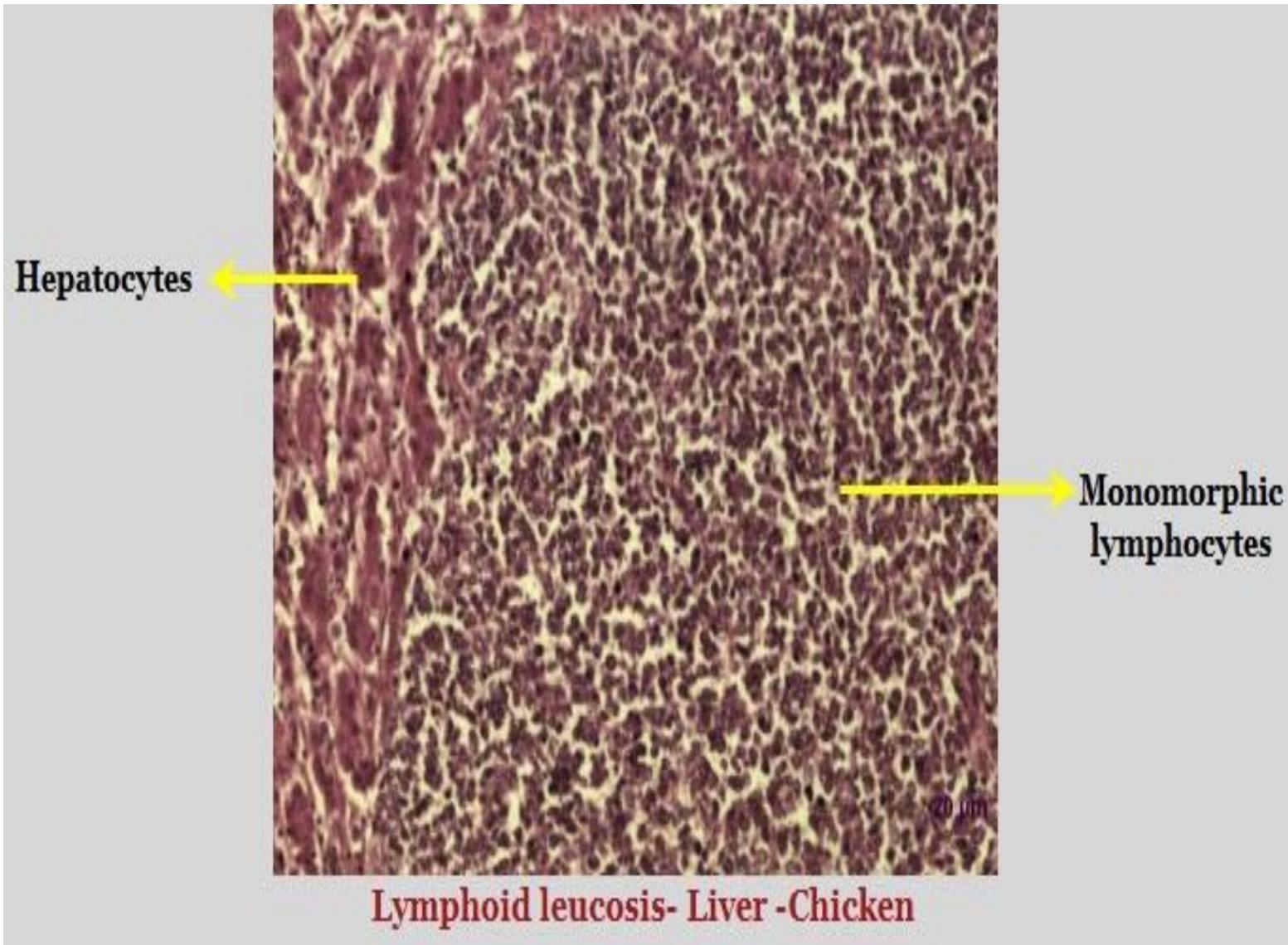
Hepatocellular carcinoma in a duck

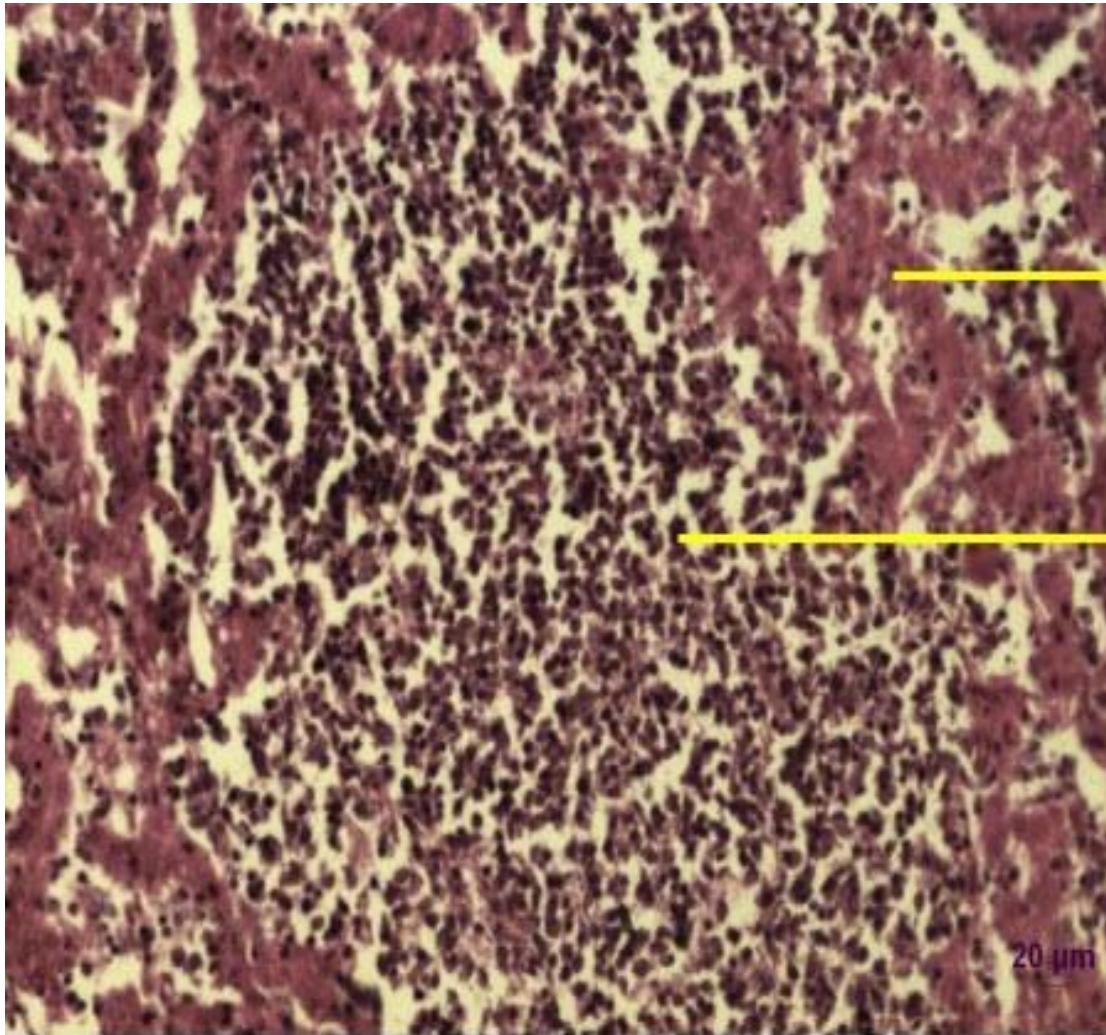
→ Nodular bile stained hepatic parenchyma



Lymphoid leucosis in a chicken

→ Big liver occupying most of the abdominal cavity





Hepatocytes

Pleomorphic  
lymphoid cell population

20 μm

Marek's disease - Chicken

# CHOLANGITIS

Definition: Inflammation of bile duct is called cholangitis

## Etiology

- Mostly liver fluke infection
- Hepatic Coccidiosis by *Eimeria stiedae* in rabbits

## Pathogenesis

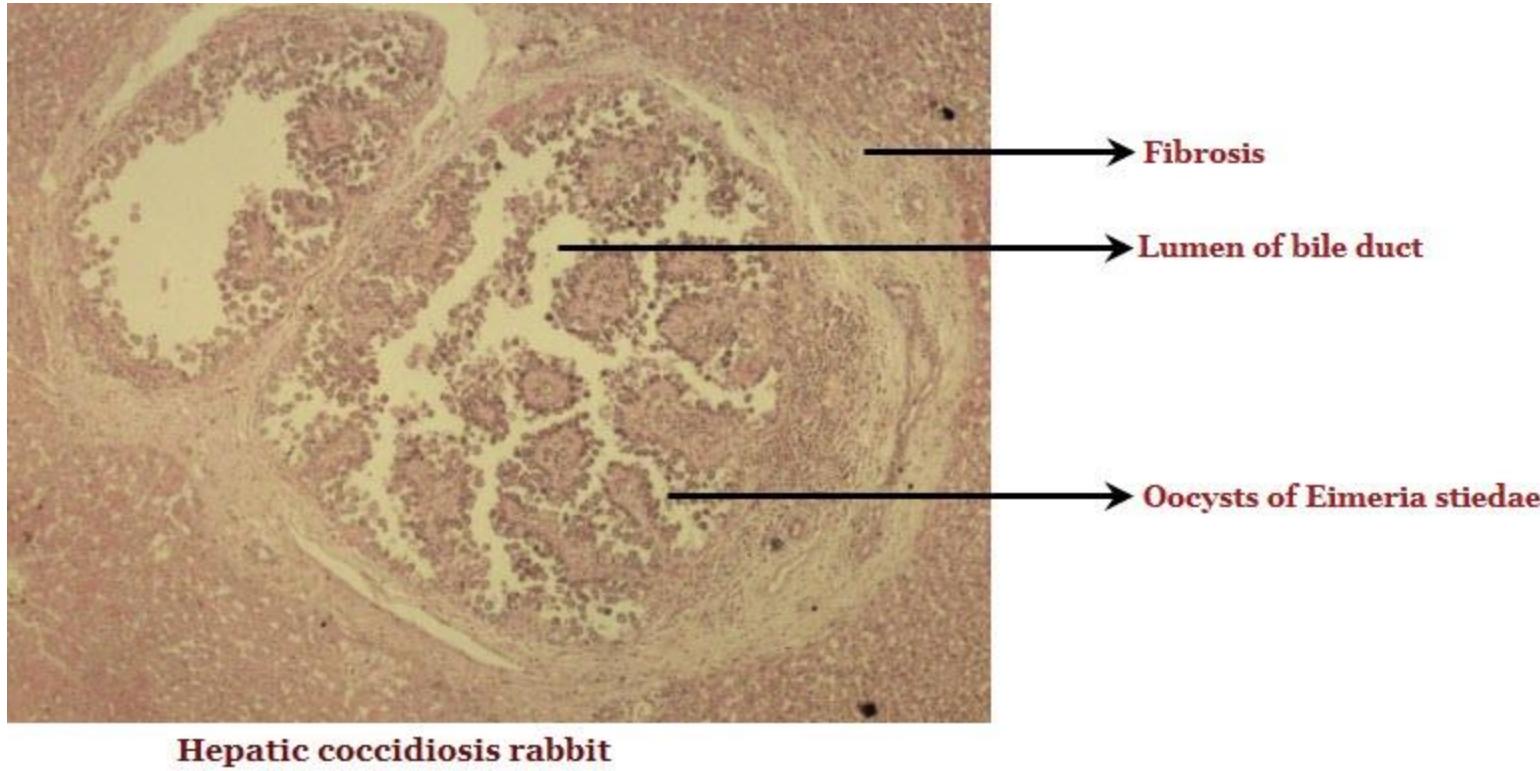
- Cholangitis is caused by the irritation of the spines on the cuticle of the parasites as well as the toxins

## Gross pathology

- Lumen of the bile duct is dilated and its wall is thickened
- The ducts stand out as thick cords
- In some cases calcification makes them hard (**Clay pipe appearance**)

## Histopathology

- The bile duct mucosa is hyperplastic with papillary projections
- The bile duct wall is infiltrated by macrophages and lymphocytes
- The fibrous tissue proliferates around the walls of the bile ducts and may extend to a short distance into the parenchyma of the liver



# CHOLECYSTITIS

- Inflammation of gall bladder is called **cholecystitis**
- It is caused when stasis of bile occurs by
  - the presence of foreign bodies
  - parasites
  - concretions
  - Extramural pressure on bile duct
- *E.Coli* and *Salmonella* are frequently associated with cholecystitis
- Infection is usually ascending from the duodenum
- The retained bile itself acts as an irritant
- Usually the catarrhal variety is noticed with congested mucosa and increased secretion of mucus by the glands

# CHOLELITHIASIS

## Etiology

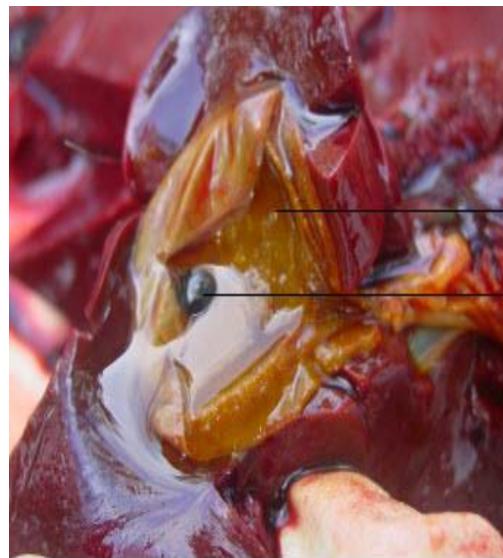
- Almost always gall stones occur as a result of cholecystitis

## Pathogenesis

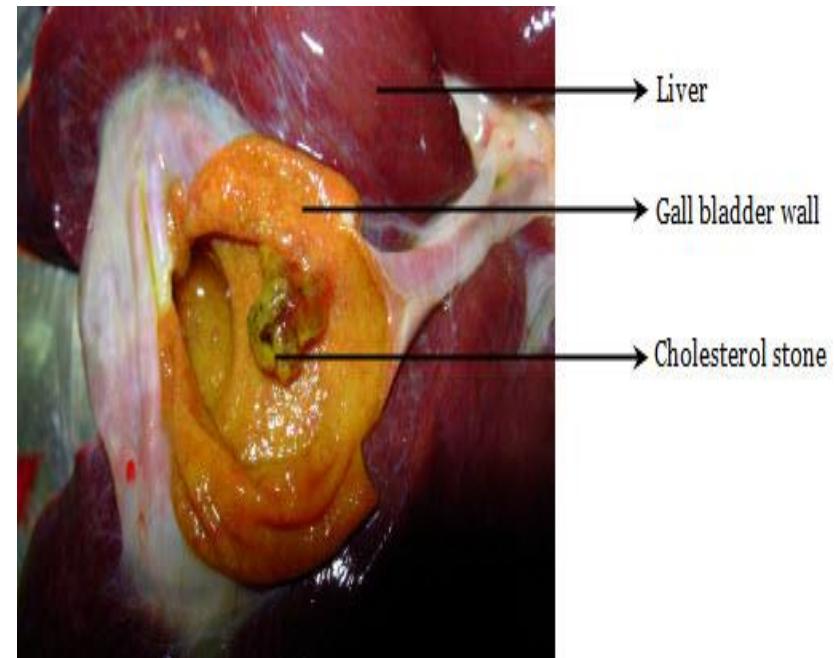
- The dead cells, bacteria or mucus may form the nuclei for development of cholelithiasis
- Sand particles and food materials that may reach the gall bladder through the bile duct from the duodenum during violent peristalsis may also form nuclei of the choleliths
- Cholesterol is normally held in solution by loose combination with bile salts.
- In cholecystitis, the bile salts are rapidly absorbed leaving the cholesterol which is precipitated
- Gall stones are composed of cholesterol, bilirubin, bile salts, calcium and organic matrix

## Gross pathology

- Gall stones may be found in the gall bladder or bile ducts but unlike in man, bile ducts are more often affected because of frequency of parasitic involvement
- Numerous small stones or a few large ones in the gall bladder
- The larger ones may be faceted due to rubbing against one another
- Dark brown or yellowish-green in color
- Light and friable



Cholelith - Pigment type in a bitch



# **EXOCRINE DISORDERS OF PANCREAS**

## **Acute pancreatic necrosis**

- It may sometimes be met with in dogs, cat, swine and horses
- Ruminants are usually not affected

## **Etiology**

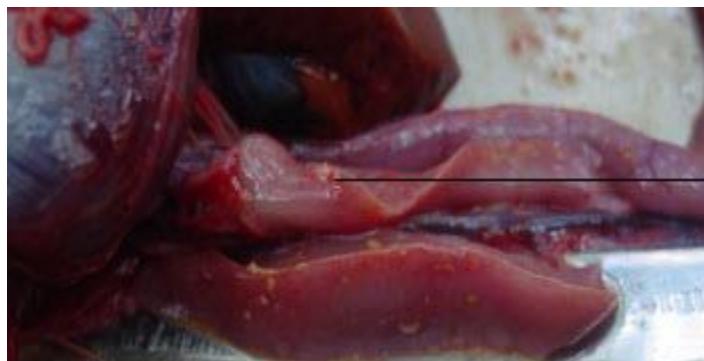
- Occlusion of pancreatic duct (by parasites) or injuries or circulatory disturbances or regurgitation of bile or bacterial infection (via blood or ascending infection from the intestines)

## **Pathogenesis**

- The essential lesion is necrosis of pancreas by its own enzymes especially the proteolytic enzymes
- The enzymes escaping out of the pancreatic tissue digest the surrounding peripancreatic fat (fat necrosis) first and the pancreatic parenchyma subsequently
- Entering lymph channels, the lipase may produce fat-necrosis in distant organs, even as far away as anterior mediastinal region

## Gross pathology

- In fatal cases, there is a small quantity of fluid in the abdominal cavity
- Haemorrhages may be present in the omentum
- In the mesentery and around the pancreas whitish areas or nodules of fat necrosis with an inflammatory zone surrounding them are found
- The pancreas is swollen and soft, yellowish or slightly hemorrhagic



Necrotic spots on the  
pancreatic surface

## Histopathology

- Pancreas may show the following changes
- Peripancreatic fat reveal necrosis, granulomatous reaction and calcification
- Necrosis of the parenchymatous cells
- Edema, haemorrhage and infiltration by a few leucocytes
- Thrombosis of vessels

Pancreatic necrosis in a duck

# Ascites or Hydroperitoneum

## Definition

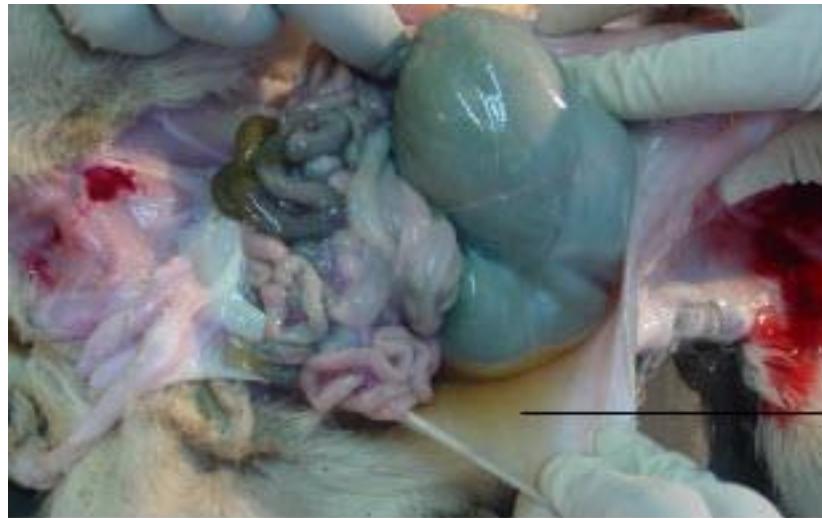
- This is edema of the peritoneum

## Occurrence

- This is common in dogs and cats but may also be encountered in sheep and cattle

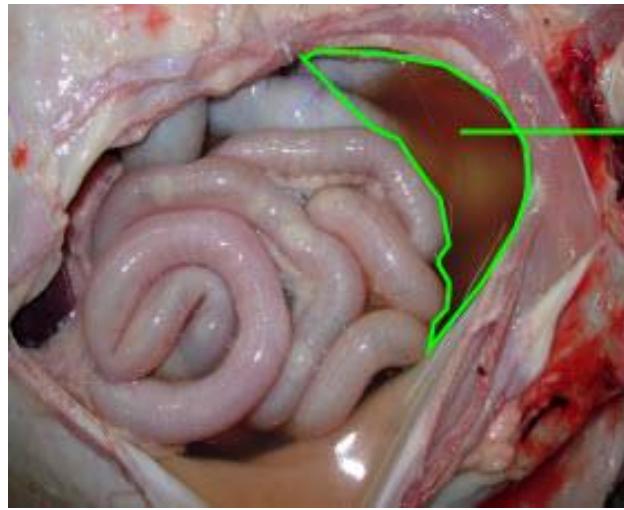
## Etiology

- Hypoproteinemia – gastrointestinal trichostrongylosis and Johne's disease
- Cachectic diseases: anaemia and starvation
- General chronic venous congestion – cardiac or pulmonary lesions
- Portal obstruction due to hepatic lesions
- Urinary obstruction in male cattle and sheep with or without rupture of bladder
- Increased capillary permeability
- Lymphatic obstruction in peritoneal cavity
- Carcinomas : primary (malignant mesotheliomas) or secondary (carcinomas)



Hydroperitoneum in a goat

→ Excessive fluid in the abdominal cavity



→ Serosanguineous fluid in the abdominal cavity

# PERITONITIS

## Occurrence

- Peritonitis is a very common condition in most of the domestic animals

## Types

- It may be localized or generalized

## Etiology

- Bacteria: *E.Coli*, *Streptococci*, *Staphylococci*, *Corynebactreium*, *Clostridia*, *Pasteurella* group and Anthrax in pigs
- Viruses of bovine encephalitis
- Helminths
- Chemicals – introduced for medication
- Endogenous – Bile and pancreatic juice
- Visceral gout of birds
- Glasser's disease of swine

## Routes of entry

- Externally through surgical wounds or from trauma
- By rupture of an abdominal organ
- Extension through the walls of stomach, intestine or uterus when their mucosa is inflamed
- From an infected umbilicus
- Direct extension from an infected kidney
- By blood stream as in bovine viral encephalitis
- By way of lymphatics from scrotal infection and infection of abdominal wall

## Pathogenesis

- The irritant first produces a serous inflammation which later becomes fibrinous or fibrinopurulent
- The fibrin is helpful in localizing the inflammation by forming adhesions
- Being a very large absorptive surface of the body, toxins are speedily absorbed from the peritoneum damaging other parenchymatous organs
- In Glasser's disease of swine, diffuse serofibrinous peritonitis is seen
- Tuberculosis of the peritoneum is very frequent in cattle, less frequent in dogs and rarely met with in other animals
- In **visceral gout** of birds, ***uric acid peritonitis*** occurs and is characterized by the deposition of urates on the serous membrane which consequently shows inflammatory changes

# DIABETES MELLITUS

## Definition

- ❖ Diabetes mellitus is derangement of the carbohydrate metabolism due to insulin deficiency and characterized by hyperglycemia and glycosuria

## Etiology

- Insulin deficiency

It may arise when

- Insulin may not be adequately synthesized due to necrosis of pancreas
  - Insulin may not be liberated into the circulation though synthesized by the Beta cells
- Diminished production of insulin due to “work-exhaustion”

## Animals affected

- Diabetes mellitus may be found in dogs and cats.
- In dogs it is a disease of older animals, especially in females, due to chronic pancreatitis
- For some unknown reason, such dogs develop cataract in the eye
- In lambs, diabetes mellitus is seen in those that are overfed on carbohydrates
- **Glycosuria** is met with in **enterotoxaemia in sheep**

## Clinical Pathology

- Lipemia is evident with the serum appearing white

## Gross Pathology

- The animal is emaciated and dehydrated
- Highly fatty Liver
- Pancreas may either be normal or show pancreatitis and necrosis with fibrosis

## Histopathology

- **Pancreas**
  - Beta cells: Vacuolation, necrosis and hyalinization of Beta cells have been noticed
  - The epithelium of ducts show vacuolation and is due to glycogen infiltration
- **Kidney**
  - Glycogen infiltration of the epithelial cells of the Henle's loops and the distal convoluted tubules is noticed
- **Liver**
  - Hepatocytes are loaded with fat
- **Other organs**
  - The retinal and vascular lesions of man are not met with in animals

# NEOPLASMS

- ❖ The primary tumors, **mesotheliomas** (malignant) arise from the serosa and are common in the young and newborn animals
- ❖ The secondary tumors are metastases from the liver or uterus
- ❖ Transcoelomic implantation of ovarian tumour found in women is not common in animals

Thank You