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DIGESTIVE SYSTEM

1. **Caries**: Caries means decay of teeth in which the enamel is decalcified followed by softening and discoloration. The affected teeth usually have one or more depressed areas, which are brown or black in colour.
2. **Stomatitis**: Stomatitis means diffuse inflammation of the mucous membrane of the mouth.

A. Gingivitis: It is inflammation of the gums.
B. Glossitis: It is inflammation of the tongue.
C. Lampas: It is inflammation of the palate.
D. Cheilitis: It is inflammation of the lips.
E. Pharyngitis: It is inflammation of the pharynx.
F. Tonsillitis: It is inflammation of the tonsils.

Etiology:

A. Physical :
(i) Trauma by awns, thorns, burrs, wood pieces, glass pieces, sharp bits, irregular sharp teeth, sharp edged feeding utensils.
(ii) Hot drenches
(iii) Eating frozen foods.

B. Chemical: caustic alkalies, corrosive acids, fertilizers.
C. Deficiency of vitamins: Hypovitaminosis A especially in fowl, Niacin deficiency - Black tongue in dogs.
D. Microorganisms:
(i) Bacteria: *Actinomyces bovis*; *Actinobacillus lignieresi*; *Fusobacterium necrophorum*; *Pseudomonas aeruginosa*; *Corynebacterium pyogenes*; Streptococci and Staphylococci.
(ii) Fungi: Candida albicans and Oidium pullorum in poultry.
(iii) Viruses: Foot and Mouth disease; Rinder pest: Virus diarrhoea -mucosal disease; Infectious canine hepatitis; contagious ecthyma; vesicular exanthema; fowl pox and blue tongue.

Types of Stomatitis-

(i) **Catarrhal Stomatitis**: It is characterized by presence of excessive mucous. It forms grey patches or coating on the tongue and occludes the mucosal glands forming raised retention cysts. It undergoes bacterial decomposition producing foetid smell.
(ii) **Vesicular Stomatitis**: It is characterized by presence of small, clear, fluid filled vesicles on the mucosal surfaces in the oral cavity. In cattle these are caused by picrona virus (Foot and Mouth disease) and rhabdo virus infections. The roof of the vesicle is formed by superficial layers of the epithelium while the floor is formed by hyperemic papillary bodies which are denuated of epithelium and infiltrated with leucocytes.
(iii) **Erosive and Ulcerative Stomatitis**: It is characterized by circumscribed lose of superficial and deeper layers of epithelium. It is caused by foreign bodies and corrosive acids.

(iv) **Necrotic Stomatitis**: It is characterized by presence of yellow-grey round foci of coagulative necrosis on the border of the tongue which extend up to sub-mucosa. Necrotic area is demarcated by hypermic zone and connective tissue. It usually occurs in calves due to necrobacillar infection.

(v) **Fibrinous Stomatitis**: It is characterized by presence of dry skin like grey deposit due to epithelial necrosis and fibrin exudation.

(vi) **Sclerosing Stomatitis**: It shows broad grey white fibrous band and atrophic pale-red muscles running from dorsum to the substance. These changes are due to chronic proliferation of connective tissue.

(vii) **Papular Stomatitis**: It is characterized by presence of small (1 cm. or less) circumscribed solid elevations.

3. **Tumours of the Mouth and Pharynx:** Papillomas are the most common neoplasms in the dog and calves.

   Epulis: It is a fibroblastic tumour consisting of dense fibrous tissue with varying amounts of epithelium and a few giant cells. It usually occurs in the gums.

SALIVARY GLANDS

1. **Sialoliths**: These are salivary calculi and commonly found in horses. It appears as concentrically laminated body formed of $caco_3$. salivary calculi blocks the flow of saliva and produces stasis, distension of ducts and finally atrophy of the gland.
2. **Ranula**: It is cyst on the floor of the mouth caused by the dilatation of the salivary duct. Ranula is smooth, rounded cyst containing a clear fluid.
3. **Salivary Mucocoele:** It is a pseudocyst which is not lined by epithelium and filled with saliva.
4. **Sialoadenitis**: Inflammation of the salivary glands is known as sialoadenitis. It is caused by traumatic injury or infections.

   Macroscopically the glands are swollen and red which can cause atrophy of the gland.

ESOPHAGUS

1. **Choke**: Retention of food in esophagus due to obstruction is known as choke. Choke may be complete or incomplete type. In complete type, feed will be returned and water will come out of the nostrils following intake of water by animal. In horses, choke occurs in thoracic area where as in cattle and dogs, the pharynx is obstructed.

   Causes:

   (i) Impacted masses of feed like potato, carrot etc.

   (ii) Compression of esophagus due to enlarged lymph nodes, thyroid etc.

   (iii) Due to changes in the esophageal muscles as occur in hypertrophy of muscles, spasmand loss of elasticity in old age.

2. **Dilatation (Ectasia):** The dilatation of esophagus may be fusiform or cylindrical.

   Causes:

   (i) Accumulation of food proximal to a stenosed area.

   (ii) In ruminants accumulation of food during regurgitation on the distal side of stenosed area.

   (iii) Trauma from horns etc. rupturing the muscular coat.

   (iv) Relaxation of the esophageal muscles due to the nervous lesions.

   Sequelae:

(i) The food gets accumulated in the dilated area of esophagus leading to pressure and rupture.
(ii) The food may become decomposed and produce softening of the epithelium, inflammation, ulceration, gangrene and death.
(iii)In ruminants, bloat occurs.

3. **Esophagitis:** Inflammation of the esophageal mucosa is known as esophagitis. Grossly, mucosa is red and swollen.
4. **Neoplasms:** Neoplasms of the esophagus are not common. In the thoracic portion of the esophagus, fibrosarcomas and osteogenic sarcomas are found in connection with infection.
5. **Obstruction of Crop in Birds:**

Causes:
(i) Ingestion of large quantities of dry grain which swell in the crop and form a hard mass.
(ii) Atony or paralysis of wall leading to stasis of feed.
(iii) Foreign bodies like wire etc.

Gross pathology: The stagnated feed gets decomposed, gas accumulates and inflammation sets in.

Sequelae:
(i) Rupture of crop due to distension by feed and gas or due to penetration by the foreign body.
(ii) Death due to-
a) Heart failure due to pressure on heart.
b) Asphyxia due to compression of trachea.
c) Intoxication due to absorption of toxins from decomposed feed.
d) Starvation since, feed does not enter the proventriculus.

6. **Ingluvitis: Inflammation of crop** is called ingluvitis. It may be catarrhal or diphtheritic type.

Pathological Affections of Fore Stomach

1. **Tympanites or Bloat:** It is the distension of rumen with gas**.**

Types of Bloat-
- Based upon etiology**,** bloat may be primary due to increase production of gas or secondary due to functional disturbances as wall insufficiency and obstruction.
- Based upon course, bloat may be acute or chronic.
- Based of gas, bloat may be dry or frothy. The dry bloat is less harmful and the gases can be more easily got rid of by eructation. In the frothy bloat, the gas is trapped as small bubbles in the fluid forming a foamy mass which is not easily eructated.

Pathogenesis-Distended rumen compresses other abdominal organs and causes passive congestion. Also, 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, lungs appear oedematous. Haemorrhages observed on the pleura, pericardium, tracheal mucosa and on bronchial lymph nodes as well as in the lymph nodes of head and neck. Congestion of abdominal visceral organs

except liver and spleen occur. Liver is pale. The rumen or diaphragm may be ruptured sometimes. Within few hours of death, the ruminal epithelium peels off.

Sequelae:

If quickly relieved, acute bloat can be cured. If not, death may supervene due to asphyxia.

2. **Impaction of Rumen and Reticulum:** It is an acute atony of rumen, mainly occurs in cattle due to overfeeding with large amount of highly fermentable carbohydrate feed, lack of water and paresis of rumen.

   Pathogenesis-Impaction of rumen causes production of excessive amount of volatile fatty acids leading to fall in rumen pH. Lactobacillus bacteria also increase in number and produces excessive lactic acid. This makes the rumen pH 4-4.5 Fatty acids have osmotic effect so fluid is drawn into the rumen resulting haemoconcentration and dehydration. Rumen atony and toxaemia may be fatal to cattle.

   Gross pathology: The cornified epithelium is soft and peels off which produces ruminitis and ulcers.
3. **Foreign Bodies/ Abnormal Contents:** These are anything other food like sand, stone, metals, hair balls (**trichobezoars**), plant balls (**phytobezoars**) etc. which mainly lodges in rumen and reticulum of cattle.

   Ingestion of sharp objects like nails and wires penetrate the wall of reticulum causing Traumatic Reticulitis. In this condition, sharp object penetrates diaphragm and can reaches upto lungs or visceral organs. There is local fibrinous inflammation which leads to adhesion of the penetrated wall with peritoneum, diaphragm or adjacent organs. It may cause reticulitis, peritonitis and pericarditis.
4. **Trichobezoars:** These are hair balls which found mostly in the rumen.

   Gross Appearance: Trichobezoars are mostly found in animals having itching conditions. Animal may lick each other and swallows loose hair which rolled into balls during ruminal contractions. Mucus of rumen may form a smooth coat over such balls.

   Trichobezoars are found as ball in the rumen and hard in consistency.
5. **Phytobezoars:** These are plant balls which found mostly in the rumen.

Pathological Affections of Stomach/ Abomasum

1. **Acute Dilatation:** It mostly occurs in horses leading to rupture due to spasmodic closure of gastric openings in the stomach. This leads to death due to shock and absorption of toxins.
2. **Impaction:** It mostly occurs in horses due to rapid ingestion of excessive amount of heavy grain. It may lead to death due to shock and absorption of toxic products of partial digestion.
3. **Torsion:** It mostly occurs in dogs due to turning of stomach by 180 degree round the esophagus. Stomach is ballooned. The torsion may occur due to Ca:P imbalance or due to enlarged spleen. The spleen rotates with stomach and develops infarcts. Dog develops shock and cardio-respiratory interferences.
4. **Abomasal Displacement:** It usually on the left side. The condition occurs in high lactating cows which fed greater amount of succulent feed. The abomasum may over distended and twist on its mesenteric axis. Vascular supply may be interfered causing mucosal necrosis.

Symptoms: It includes anorexia, abdominal pain, weight loss, dehydration, scanty faeces, ketonuria and ruminal tympany. Animal has a tucked up appearance.

5. **Gastritis:** Inflammation of the stomach is called gastritis. Gastritis is a fairly common condition in animals. Gastritis is often associated clinically with vomiting, dehydration and metabolic acidosis.

   Etiology: Gastritis may be caused by following factors-

   (i) Physical as overfeeding, Foreign bodies, feeding of frozen feed etc.
   (ii) Chemicals
   (iii) Uremia
   (iv) Stress
   (v) Bacterial infections
   (vi) Viral infections
   (vii) Fungi
   (viii) Parasites

   Types: It may be primary or may be secondary to some other infections. Gastritis may be acute or chronic.

   Acute gastritis-

   A. Catarrhal Gastritis- There is increase mucous secretion. Macroscopically there is increased thickening and redness of gastric mucosa. Ulcers may be seen causing blood stained gastric contents.
      Microscopically, there is epithelial degeneration, desquamation and infiltration with leucocytes.
   B. Edematous Gastritis-the edematous mucosa appears gelatinous on slicing. The abomasal folds are prominent in height and thickness.
   C. Fibrinous Gastritis- It shows bran like deposits on the mucosa and occurs typically in Rinder Pest.
   D. Haemorrhagic Gastritis- It shows haemorrhage in gastric mucosa compressing mucosal glands and typically seen in Anthrax and Rani Khet Disease. 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 gastric contents.
   E. Parasitic Gastritis-Heavy infestation causing minute injuries on the mucosa and anaemia.

   Chronic gastritis-This condition is usually of a hypertrophic type with thickening of the gastric wall. It is characterized by thickening of mucosa due to over growth of interstitial connective tissue and cellular infiltration. When eosinophils are predominantly present, it is known as Eosinophilic gastritis. There is exfoliation of the epithelium.

6. **Gastric Ulcers**: Gastric ulcers are commonly found in calves following the early (before fourth week of age) weaning. The coarse plants induce injury to gastric mucosa producing focal gastritis and ulcer. Ulcers are commonly found in the pyloric region of the abomasum. Gastric ulcers may be superficial or deep. The ulcers may heal or perforate. When perforation occurs rapidly, diffuse peritonitis develops and when perforation occurs slowly, adhesions and local peritonitis develops.

Gross pathology-The mucosal erosions (in cattle) are of the size of millet which may later on enlarge and become deeper to form ulcers. The ulcers are usually demarcated, having raised borders with a punched-out appearance.
Histopathology-Ulcer shows mucosal degeneration, leucocytic infiltration and progressive liquefactive necrosis leads to exfoliation of mucosa.

Intestine

1. **Congenital Anomalies:**
   I. **Atresia-** It is absence of lumen in some part of intestine. It is commonly seen in case of calves and pigs in the form of absence of rectum (atresia recti) or anus (atresia ani).
   II. **Meckel's Diverticulum-** is a remnant of omphalo-mesentric duct due to failure of disappearance of omphalo-mesentric duct in the post natal life.
2. **Enteroliths:** These are mostly formed of phosphates seen mainly in horses following intestinal sluggishness.
3. **Faecoliths:** These are impacted masses of faeces usually occurs in dogs following the enlargement of prostate gland.
4. **Intussusception:** It is the invagination of one segment of intestine into another.
5. **Volvulus:** It is the twisting of the intestine on its mesenteric axis.
6. **Torsion:** It is rotation of a tubular organ along its long axis.
7. **Adynamic Ileus:** It is disorder of stomach and intestine characterized by diffuse absence of normal tone and progressive peristaltic movement.
8. **Hernia:** It is the protrusion of the abdominal viscera through a natural or artificial opening. Depending upon the location, hernia may be External or Internal. External hernia consist of a hernia sac formed as a pouch of parietal peritoneum, a covering of skin and soft tissues depending upon the location of hernia, a hernia ring and the hernia contents.Ex. - ventral hernia, umbilical hernia, scrotal hernia, perineal hernia, femoral hernia etc. In internal hernia, there is displacement of intestine through normal or pathological formina within the abdominal cavity without formation of hernia sac like diaphragamatic hernia.
   If the hernial contents can be returned in to the abdominal cavity, it is called reducible hernia.If the hernial contents cannot be returned, it is called irreducible hernia.
9. **Incarceration:** It is the trapping of intestine internally from pressure on its external surface.
10. **Enteritis:** Inflammation of the intestine is called enteritis.
    Inflammation of the colon is called colitis, caecum inflammation is typhlitis, rectum inflammation is proctitis and cloaca inflammation in poultry is cloacitis.
    Etiology: Enteritis may be caused by following factors-
    (i) Physical
    (ii) Chemicals
    (iii) Disturbed metabolic processes
    (iv)Bacterial infections
    (v) Viral infections
    (vi)Fungi
    (vii) Parasites
    Types: It may be of following types-

A. Catarrhal Enteritis- It mainly occurs in sucklings – scours in calves, lambs, foals and piglets. In acute type, grossly mucosa is reddish in color and slightly thickened, covered with a mucinous exudate. Microscopically,intestinal contents consist of mucus, fibrin and desquamated epithelial cells along with infiltration with leucocytes in the lamina propria. The tips of villi may be hyperemic and edematous.

   In chronic type, wall of the intestines is greatly thickened due to infiltration by chronic inflammatory cells. Microscopically, mucosa is thickened due to presence of numerous macrophages, plasma cells, lymphocytes and connective tissue cells in the lamina propria and even in the sub-mucosa. Theintestinal glands are atrophied.

B. Haemorrhagic Enteritis- It is characterized by presence of erythrocytes in the exudate and seen mostly in anthrax, clostridial enteritis and coccidiosis. Macroscopically, intestinal contents are blood stained. Microscopically,red blood cells may be found in the exudate of the mucosa. The villi may show necrotic changes and intestinal wall shows haemorrhages.

C. Fibrinous Enteritis- It is of diptheric type and characterized by presence of fibrinous exudation into the mucosa and coagulative necrosis of the mucosa.The mesenteric lymph nodes are swollen, haemorrhagic and juicy. Microscopically, the exudate consists of strands of fibrin along with infiltration of neutrophils and exfoliated epithelial cells.Coagulative necrosis of the epithelium occurs in some places.

D. Suppurative Enteritis-It may result due to infection by pyogenic organisms.Macroscopically, the exudate contains pus. Microscopically, exudate contains mucus, exfoliated cells, neutrophils and bacteria.

E. Necrotic Enteritis- There is necrosis of the intestinal epithelium and underlying tissues. Macroscopically, patchy necrotic areas are seen and mesenteric lymphatic nodes are swollen and juicy. In hog cholera, the characteristic lesion is the “button ulcer”. Microscopically, necrosis of the epithelium of the mucosa is seen along with hyperemia, exudate and cellular infiltration.

F. Proliferative Enteritis- It is typically seen in John’s disease. It produces chronic hyperplastic enteritis in cattle, sheep, goat, camel and horses. Generally ileum and jejunum are involved showing 2-20 times thickening of mucosa. Histopathologically, the mucosa mainly shows epitheloid cell infiltration.

G. Granulomatous Enteritis- It is seen in intestinal T.B. and Coli granuloma. In tuberculosis, lymphoid follicles show tubercles which project about the mucosa and microscopically show tuberculous granulation tissue formed by conglomeration of numerous tubercles. The coli granuloma appears as miliary to pea sized nodules at the entrance of caecum and in the liver.

Pathological Conditions of Liver

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. Hepatitis is classified as alternative inflammation because the inflammatory process is caused by the same etiological agents that also produce degeneration and so alteration in the parenchymatous cells is produced.

Hepatitis is of two types:

1. Infectious Hepatitis
2. Non-Infectious Hepatitis or Toxic Hepatitis-
    A. Acute Hepatitis
    B. Chronic Hepatitis

1. Infectious Hepatitis- It occurs in certain infectious diseases like infectious canine hepatitis in which centrilobular degeneration and necrosis of hepatocytes present. On the periphery, endothelial cells, kuffer cells and hepatocytes show intra nuclear inclusion bodies.

Routes of infection to liver: These are as follows-

- Portal Vein
- Hepatic Artery
- Bile Ducts
- Umbilical Vein if the new born animals
- Direct Extension from neighboring organs

2. Non-Infectious Hepatitis or Toxic Hepatitis-

A. Acute Toxic Hepatitis- Acute hepatitis is characterized by the presence of degeneration and necrosis of hepatocytes and infiltration of neutrophils and mononuclear cells along with hyperemia with or without haemorrhage.

According to location of necrosis, it can be classified as under –

(i) Focal necrosis- Numerous small necrotic areas are seen scattered in the liver and may be found in any part of lobule.

(ii) Diffuse necrosis- It covers a considerable area of necrosis in a lobule which crossing over the lobular boundaries. It causes acute yellow atrophy of liver.

(iii) Periportal necrosis- It is characterized by necrosis of hepatocytes adjoining the portal tract at the periphery of lobule which occurs due to presence of strong toxins in blood.

(iv) Mid zonal necrosis- It is characterized by necrosis of hepatocytes in midway of periphery and centre of lobule.

(v) Para central necrosis- It is characterized by presence of peculiar type of wedge-shaped necrosis which occurs only on one side of the central vein extending up to the periphery.

(vi) Centri lobular or Periacinar necrosis- It is characterized by necrosis of hepatocytes around the central vein occurs due to stagnation of blood with toxaemia.

Macroscopic Findings-

- The liver is enlarged and paler than normal. In severe cases the organ may be dark red due to venous stasis.

- The lobular markings are exaggerated. This is due to the difference in color at the center and periphery. When congestion of the central part is present, the periphery is paler due to degenerative changes in the cells.
- If necrosis of the cells in the center occurs, then the center will be pale while the periphery is darker.
- Presence of necrotic patches in liver.

Microscopic Findings-

- Necrosis of hepatic parenchyma.
- Infiltration of neutrophils, macrophages and lymphocytes.
- Cloudy swelling and/or fatty changes in liver.
- Congestion in blood vessels and in sinusoidal area.

Saw Dust Liver: This condition can observed in liver of the cattle at the time of post-mortem examination. The foci of necrosis may be few or many and appear to the naked eye as through saw dust is sprinkled on the liver.

B. **Chronic Hepatitis/ CIRRHOSIS**:

Cirrhosis of the liver is chronic hepatitis characterized by-

- Degeneration and necrosis of hepatic cells
- Hyperplasia of hepatic cells
- Extensive fibrosis

The stimulus for the fibroblastic proliferation is some irritant, chronic and severe enough to produce degeneration and necrosis of the parenchymatous cells.

Etiology-

- Bacteria~ Salmonella, Spherophorusnecrophorous
- Virus- Infectious canine hepatitis
- Chemicals- Carbon tetrachloride
- Parasites- Fasciola hepatica, F. giantica
- Poisons/ toxins- Aflatoxins
- Once cirrhosis of liver starts, it is not checked even after removal of the cause as the newly formed fibrous tissue itself acts as an irritant to cause further proliferation of fibroblasts.

Pathogenesis-

- Loss of liver tissue i.e. degeneration of the hepatic parenchyma by irritants
- Stimulation of proliferation of connective tissue by which fibroblasts start to form new Fibrous connective tissue
- Infiltration by lymphocytes and macrophages as it is chronic inflammation
- New irregular blood vessels are formed which anastomose with the network of the portal vein and hepatic artery
- Hyperplasia of the surviving hepatocytes takes place which replaces the destroyed cells
- The newly formed fibrous tissue after maturation contracts, interfering with blood circulation so hyperplasia does not progress further due to decreased blood supply
- 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** which is characteristic feature of cirrhosis
- 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.

Macroscopic Findings-

- Liver becomes hard and firm.
- Colour becomes yellowish or greenish due to fatty or icteric changes respectively.
- Surface of liver becomes uneven and nodular.
- In the early stages the liver may be large but as the condition progresses, size of liver becomes reduced due to atrophy.
- Increase in fibrous tissue within and around lobules.
- On section, the liver cuts with difficulty giving a peculiar grating sound due to the dense fibrous tissue formed.
- Cut surface shows altered lobular markings in the form of irregular shaped and variable sized lobules with no or eccentrically central vein.

Microscopic Findings-

- Architectural details of the liver are lost. Parenchymatous cells show various stages of degeneration - cloudy swelling, fatty change and even necrosis.
- The characteristic picture is pseudolobulation due to the increase in fibrous tissue within and around the lobules.
- Central veins in some lobules are either absent or are placed eccentrically.
- Infiltration of macrophages and lymphocytes**.**
- Proliferated as well as small new bile ducts are present.

Types of Cirrhosis-

(i) **Portal Cirrhosis-**It occurs in the portal area extends into the surrounding tissue and usually caused by plant toxins.

(ii) **Biliary Cirrhosis-** It is characterized by proliferation of fibrous tissue around the bile ducts encircling them and usually seen in parasitic conditions such as liver fluke infestation. It shows newly formed non-functional bile ducts.
Grossly, liver is greenish in colour due to stasis of bile and enlarged.
Microscopically, dilated, tortuous and also newly formed non-functional bile ducts are found along with infiltration of the connective tissue with chronic inflammatory cells.

(iii)**Central or Cardiac Cirrhosis**- There is increase in fibrous tissue around the central vein as a result of chronic passive congestion.

(iv) **Pigment Cirrhosis-**It is characterized by yellow discoloration of liver associated with haemochromatosis.

(v) **Parasitic Cirrhosis-**It is usually caused due to the damage by migration of parasites and a chronic obstruction of the bile ducts by flukes or other parasites. The gross and microscopic picture is more or less similar to biliary cirrhosis and characterized by fibrosis of parenchyma for a short distance around the biliary passages.

(vi)**Glissonian Cirrhosis-**This is not true cirrhosis since the liver as a whole is not affected. Inflammation and thickening of the Glisson's capsule extends to the adjacent liver parenchyma.

(vii) **Pericellular Cirrhosis**-In this condition the fibrous tissue invades the parenchyma and encircles individual cells. Ex- Aflatoxicosis.

(viii) Multinodular or Atrophic or Gindrinker's or Laennec's Cirrhosis-This is portal cirrhosis of man. The gross changes are more or less similar to portal cirrhosis in which contracting fibrous tissue makes the liver smaller hence, named as Atrophic Cirrhosis. Microscopically, there is proliferation of the fibrous tissue which is infiltrated by chronic inflammatory cells. The fibrous tissue divides the parenchyma into smaller lobules and nodules formation due to proliferation of cells.

Effects of Cirrhosis-

(i) Ascites.

(ii) Varicosity and rupture of esophageal veins may lead to hematemesis

(iii) Splenomegaly
(iv) Chronic gastroenteritis
(v) Caput medusae in man – This is dilatation of the cutaneous veins around the navel
(vi) Loss of inactivation of hormones and detoxification.

Foamy Liver-Postmortem decomposition of liver occurs rapidly since gas-forming organisms (*Cl. welchii*) invade from the intestines which are close by. Liver, particularly rich in nutrients, is a good medium for the growth of these bacteria. Gas bubbles form in the blood vessels. The parenchyma and the blood vessels adjacent to the bowel are stained by hemoglobin, bluish-black. The presence of gas gives the liver a foamy appearance named Foamy Liver.

NEOPLASMS OF LIVER:
Hepatoma: Benign tumour of liver
Hepato-cellular carcinoma-Malignant tumour of liver
Cholangioma- Benign tumour of bile ducts
Cholangio-cellular carcinoma-Malignant tumour of bile ducts

CHOLANGITIS: Inflammation of bile duct is called cholangitis. It most commonly occurs in liver fluke infections. Grossly, the lumen of the bile duct is dilated and its wall is thickened due to fibrous tissue proliferation around it. Clay pipe appearance of bile ducts may be seen due to calcification. Microscopically, bile duct mucosa is hyperplastic with papillary projections. The bile duct wall is infiltrated by macrophages and lymphocytes. The lumen may contain parasites, cell debris and mucus.

CHOLECYSTITIS: It is inflammation of the gall bladder which is characterized by congestion, thickening of wall and infiltration of mononuclear cells.
Etiology-
• Parasites- Fasciola sp.
• Foreign body- Stones
• Bacteria- E. coli.
Macroscopic and microscopic features-
• Thickening of the wall of gall bladder.
• On opening of gall bladder, there may be parasites/ stones/foreign body.
• Contents of gall bladder may be watery or thick oily.
• Congestion.
• Proliferation of fibrous tissue in the wall of gall bladder.
• Infiltration of mononuclear cells.
• Increased number of mucus secreting cells.

CHOLELITHIASIS: Gall stones may be numerous small stones or a few large ones in the gall bladder .Gall stones are composed of cholesterol, bilirubin, bile salts, calcium and organic matrix. These may be dark brown or yellowish-green in color.

PANCREATITIS: Pancreatitis is the inflammation of pancreas. It is characterized by necrosis of pancreatic tissue, infiltration of neutrophils and mononuclear cells and fibrous tissue proliferation.
Etiology
• Bacteria
• Virus- Reovirus in poultry
• Parasites
Macroscopic and microscopic features
• Pancreas becomes pale, swollen, oedematous.

• In chronic cases, atrophy of pancreas
• Pancreas becomes hard, firm and fibrous.
• Necrosis of pancreatic cells.
• Edema, infiltration of leucocytes, haemorrhage.
• Fibrosis characterized by proliferation of fibroblasts.

PATHOLOGY OF PERITONIUM:

Peritonitis is the inflammation of peritoneum characterized by suppurative, serofibrinous or nodular lesions.

Etiology-

• Bacteria- Staphylococci, *Mycobacterium* sp.
• Virus
• Neoplasia
• Parasites

Macroscopic and microscopic features-

• Serofibrinous, fibrinous, suppurative or granulomatous lesions.
• Accumulation of clear fluid in the peritoneum which is known as Hydroperitoneum or Ascites.
• Serofibrinous, suppurative or granulomatous lesions.
• Thickening of peritoneum, adhesions due to fibrosis.