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PATHOLOGY OF DIGESTIVE SYSTEM

- Developmental anomalies
- Pathology of Mouth cavity
- Pathology of Esophagus and crop
- Pathology of Stomach
- Pathology of Intestines
- Pathology of liver and pancreas
- Pathology of peritoneum
- Model Questions
DEVELOPMENTAL ANOMALIES

Epitheliogenesis imperfecta of tongue
Abnormal smooth surface of tongue due to small filiform papillae. It occurs as a defect in autosomal recessive gene and occurs in Holstein-Friesian cattle. This is also known as smooth tongue.

Cleft palate
This is most common congenital abnormality occurs due to failure of oral-nasal cavity to divide leaving cleft. It may also extend towards lips producing ‘hare lip’ condition.

Mega colon
There is distention of colon which abruptly terminate in rectum due to mutant gene in dogs.

Duplication of colon
In dog, the colon is duplicated from caecum to rectum and this defect is associated with malformation in the body of vertebrae T\textsubscript{4} and T\textsubscript{5}.

Atresia coli
In calf, the absence of colon occurs and the intestine terminates in blind caecum.

Atresia ani
This is absence of anal opening.

PATHOLOGY OF MOUTH CAVITY

STOMATITIS
Stomatitis in the inflammation of mucosa of oral cavity (Fig. 16.1 to 16.6). It includes:

- Gingivitis: Inflammation of gums
- Glossitis: Inflammation of tongue
- Cheilitis: Inflammation of lips
- Tonsilitis: Inflammation of tonsil
- Palatitis/Lampas: Inflammation of palates

Etiology
- Trauma due to nails, wire, or any sharp object like needle
- Physical due to hot milk, medicines etc.
- Chemical- Alkali / acids
- Microorganisms- Bacteria, virus, fungi

Macroscopic features
- Catarrhal stomatitis: Mucous exudation in oral cavity.
- Vesicular stomatitis: Vesicles in oral mucosal e.g. FMD
- Erosive stomatitis: Erosions in oral mucosa e.g. Rinderpest
- Fibrinous stomatitis: False membrane in oral mucosa.
- Ulcerative stomatitis: Presence of ulcers in oral mucosa e.g. mucosal disease.

Microscopic features
- Congestion of oral mucosa
- Presence of erosions, vesicles or ulcers
- Infiltration of neutrophils, lymphocytes and macrophages
- Presence of fibrinous exudate in the form of diphtheritic membrane.

PATHOLOGY OF ESOPHAGUS AND CROP

CHOKE
Choke is complete or partial obstruction of esophagus either due to any foreign material or pressure from adjoining areas (Fig. 16.7).

Etiology
- Beets, turnip, carrots, bone
- Abscess tumor of neck area

Macroscopic features
- Tympany
- Gangrene, sapremia and toxaemia
- Sac like dialatation “Esophageal diverticulum”
- Perforation due to sharp bone ends

Microscopic features
- Necrosis gangrene at a point of obstruction
- Congestion haemorrhage in perforated cases

ESOPHAGITIS
Esophagitis is the inflammation of esophagus caused by trauma, parasites etc. and characterized by catarrhal inflammation, ulceration or stenosis due to fibrosis.
Fig. 16.1. Photograph of mouth cavity of a bird showing stomatitis due to avian pox

Fig. 16.2. Photograph of mouth cavity of a buffalo having erosive palatitis

Fig. 16.3. Photograph of mouth of a camel showing cheilitis

Fig. 16.4. Photograph of tongue showing granulomatous lesions (ARS/USDA)

Fig. 16.5. Photograph of tongue showing ulcerative glossitis (ARS/USDA)

Fig. 16.6. Photograph of tongue showing glossitis due cysticercosis (ARS/USDA)

Fig. 16.7. Diagram of alimentary tract showing choke in esophagus due to bone

Fig. 16.8. Photograph of esophagus showing presence of cysts due to sarcosporidiosis (ARS/USDA)
**Etiology**
- Trauma due to foreign bodies
- Chemicals- Acids, alkalies
- Infection- Mucosal disease virus
- Parasite- *Spirocerca lupi*
- Nutritional- Vit. A deficiency

**Macroscopic features**
- Congestion
- Ulcer formation (Fig. 16.9).
- Red streaks of catarrhal inflammation.
- Stenosis due to fibrous nodules or inflammatory exudate.
- Enlargement of glands (Fig. 16.10).

**Microscopic features**
- Congestion, haemorrhage
- Ulceration
- Infiltration of neutrophils, lymphocytes
- Sub-epithelial fibrosis/nodules by *Spirocerca lupi*.

**INGLUVITIS**

Ingluvitis is the inflammation of crop caused by fungi and characterized by ulcerative or diphtheritic lesions (Fig. 16.11).

**Etiology**
- Candida albicans
- Monilia albicans

**Macroscopic features**
- Turkis towel like appearance in crop mucosa.
- Round and raised ulcers.
- In moniliasis, formation of diphtheritic membrane

**Microscopic features**
- Necrotic and ulcerative lesions
- Fibrinous inflammation with infiltration of mononuclear cells

**PATHOLOGY OF STOMACH**

**TYMPANY**

Tympany is accumulation of gases in rumen due to failure of eructation as a result of obstruction or due to excessive production of gases characterized by distended rumen and dyspnoea. It is also known as *bloat* (Fig. 16.12).

**Etiology**
- Choke of esophagus
- Sudden change in animal feed with high content of legumes.
- Excessive lush green fodder

**Macroscopic features**
- Rumen is distended due to excessive accumulation of gases (CO$_2$, H$_2$S, CO)
- Distended rumen compresses diaphragm to hinder respiration.
- Tarry colour blood, pale liver and rupture of diaphragm.
- On rupture of rumen gas comes out (dry tympany).
- The gas is trapped in small bubbles in the ruminal fluid forming foams and is not easily removed. This is known as “frothy bloat”, which is produced by saponin and water soluble proteins and due to reduction in surface tension in the absence of fatty acids that favours froth formation.

**Microscopic features**
- Haemorrhage in lungs, pericardium, trachea and lymphnodes
- Atelectasis in lungs.

**RUMENITIS**

Rumenitis is the inflammation of rumen in ruminant animals caused by change in diet, chemicals or drugs and characterized by seropurulent exudate or ulcer formation with or without parakeratosis.

**Etiology**
- Change in diet, corn or alfa-alfa hay.
- Chemicals/drugs *e.g.* potassium antimony tartrate.
- *Spherophorus necrophorus* infection
Fig. 16.9. Photograph showing ulcerative esophagitis due to bovine viral diarrhoea virus.

Fig. 16.10. Photograph of esophagus showing nutritional roup

Fig. 16.11. Photograph of crop showing ingluvitis

Fig. 16.12. Diagram showing tympany in a cow

Fig. 16.13. Diagram showing penetration of needle from reticulum (Traumatic reticulitis).

Fig. 16.14. Photograph showing ulcerative abomositis

Fig. 16.15. Photograph showing proventriculitis

Fig. 16.16. Photograph of calf showing diarrhoea
Macrosopic features
- Ulcers
- Spherical white nodules of 1-2 cm diameter size.
- Sloughing of mucosa.

Microscopic features
- Seropurulent exudate
- Ulcers
- Infiltiration of lymphocytes and neutrophils
- Fibrous nodules due to hyperplasia of fibroblasts
- Parakeratosis

RETICULITIS
Reticulitis is the inflammation of reticulum in ruminant animals caused by trauma/perforation by foreign body including sharp object like needles, wires, etc. and characterized by abscess formation, adhesions, peritonitis and pericarditis (Fig. 16.13).

Etiology
- Foreign body- sharp objects like needles, wires etc.

Macroscopic features
- Perforation of reticulum by foreign body.
- Abscessation/suppuration
- Peritonitis, adhesions of reticulum with diaphragm
- Pericarditis due to foreign body (traumatic reticulo pericarditis).

Microscopic features
- Infiltiration of neutrophils, macrophages, lymphocytes
- Proliferation of fibroblasts producing adhesions.
- Liquifactive necrosis.

OMASITIS
Omasitis is the inflammation of omasum in ruminant animals caused by Actinobacillus sp. and characterized by granulomatous inflammatory reaction.

Etiology
- Actinobacillus ligneiiresi

Macroscopic features
- Granulomatous nodules in omasum

Microscopic features
- Typical granuloma formation
- Sulfur granules of Actinobacillus in the centre of lesion.

ABOMASITIS
Abomasitis is the inflammation of abomasum in ruminants caused by chemicals/drugs, bacteria, virus or parasites and characterized by congestion, oedema and/or haemorrhagic ulcers (Fig. 16.14).

Etiology
- Chemicals/drugs
- Bacteria e.g. Clostridium septicum cause of Braxy
- Virus e.g. Hog cholera, Mucosal disease
- Parasites e.g. Theileria sp.

Macroscopic features
- Presence of ulcers (button ulcers in Hog cholera).
- Congestion, oedema of abomasal folds, haemorrhage in braxy.

Microscopic features
- Catarrhal, haemorrhagic abomasitis
- Presence of gram positive rods in case of braxy.
- Neutrophilic and lymphocytic infiltration.
- Congestion and haemorrhages.
- Ulceration with lymphocytic infiltration.

IMPACTION OF RUMEN AND RETICULUM
Impaction of rumen and reticulum is common in cattle and buffaloes caused by heavy carbohydrate diet and characterized by atony of rumen, indigestion, acidosis and haemorrhage on serous membranes.
Fig. 16.17. Photograph showing enteritis

Fig. 16.18. Photograph of catarrhal enteritis

Fig. 16.19. Photomicrograph of catarrhal enteritis

Fig. 16.20. Photomicrograph showing normal length of villi in intestine

Fig. 16.21. Photomicrograph showing reduced length of villi due to rotavirus

Fig. 16.22. Scanning electron microphotograph showing normal length of villi

Fig. 16.23. Scanning electron microphotograph showing reduced length of villi with rough surface

Fig. 16.24. Scanning electron microphotograph showing smooth surface of villi
Etiology
- Overfeeding of carbohydrate feed.
- Lack of water.
- Defective teeth or damaged tongue.
- Paralysis of rumen.

Macroscopic features
- Atony of rumen due to lactic acid production.
- Rumen is filled with hard, caked undigested food with foul odour.
- Hemoconcentration, anuria, blood becomes dark in colour.

Microscopic features
- Haemorrhage in lungs.
- Desquamation of ruminal epithelium.
- Lesions of acidosis/toxicosis.

GASTRITIS
Gastritis is the inflammation of stomach in non-ruminant animals having simple stomach caused by chemicals/drugs, bacteria, virus, parasite and characterized by congestion, oedema, haemorrhage and ulceration. Inflammation of proventriculus in poultry is termed as proventriculitis (Fig. 16.15).

Etiology
- Physical - overfeeding, trauma,
- Chemicals- Acid/alkali
- Microorganisms such as bacteria, virus, fungi,
- Parasites e.g. *Trichostrongyles* sp., *Hemonchus* sp.
- Uremia

Macroscopic features
- Congestion, oedema and haemorrhage of mucosal surface
- Thick mucous exudate in stomach
- Presence of vesicles / ulcers on gastric mucosa

Microscopic features
- Congestion and haemorrhage of gastric mucosa.
- Presence of ulcers/necrosis.
- Infiltration of mononuclear cells.
- Lymphoid hyperplasia.

PATHOLOGY OF INTESTINES
CATARRHAL ENTERITIS
Catarrhal enteritis is characterized by increased number of goblet cells, congestion and infiltration of neutrophils and mononuclear cells in mucosa of intestine (Fig. 16.16 to 16.25).

Etiology
- Physical- Foreign bodies and coarse feed
- Chemical- drugs
- Microorganisms-*E.coli, Salmonella* sp., viruses
- Parasites- *Coccidia*

Macroscopic features
- Presence of catarrhal exudate in lumen of intestine and congestion.
- Thickening of the wall of intestine.
- Diarrhoea.
- Presence of parasites in lumen of intestine.

Microscopic features
- Increased number of goblet cells in intestinal villi, reduced length of villi.
- Congestion.
- Infiltration of polymorphonuclear and mononuclear cells.

HAEMORRHAGIC ENTERITIS
Haemorrhagic enteritis is characterized by inflammation of the intestines along with haemorrhagic exudate (Fig. 16.26 to 16.28).

Etiology
- Bacteria- *E. coli, Bacillus anthracis, Salmonella* sp.
- Virus- Coronavirus, BVD, MD, RP
- Parasites- *Coccidia*

Macroscopic features
- Haemorrhagic exudate in intestines; blood mixed intestinal contents.
- Petechial or echymotic haemorrhage in mucosa and submucosa of intestine.
- Presence of erosions/ulcers in mucosa.
Fig. 16.25. Scanning electron microphotograph showing rough surface of villi

Fig. 16.26. Photograph of hemorrhagic enteritis

Fig. 16.27. Photomicrograph of hemorrhagic enteritis

Fig. 16.28. Photograph showing linear haemorrhage (Zebra marking) in large intestine

Fig. 16.29. Photograph showing corrugations in large intestine indicative of chronic enteritis

Fig. 16.30. Photograph showing necrotic enteritis in birds due to clostridia

Fig. 16.31. Photomicrograph showing necrotic enteritis

Fig. 16.32. Photograph showing necrotic enteritis


**Microscopic features**
- Haemorrhage in the mucosa of intestine
- Infiltration of neutrophils and mononuclear cells.
- Erosion or ulcers in intestinal mucosa
- Presence of coccidia in the mucosa

**CHRONIC ENTERITIS**
Chronic enteritis is the chronic inflammation of intestine characterized by proliferative changes like proliferation of fibrous tissue, infiltration of mononuclear cells and plasma cells in lamina propria leading to hardening of intestinal wall.

**Etiology**
- *Mycobacterium paratuberculosis* in bovines
- Intestinal helminths
- *E. coli* in poultry (Hjarre’s disease)

**Macroscopic features**
- Thickening of the wall of intestine (Corrugations in Johne’s disease) (Fig. 16.29).
- Thick mucous cover over mucosa of intestine
- Transverse corrugations in the large intestine.
- Granulomatous nodules in duodenum.
- Small, round, raised necrotic foci on serosal surface of intestine covering whole length of intestine.

**Microscopic features**
- Proliferation of fibrous tissue in lamina propria.
- Infiltration of macrophages, lymphocytes, plasma cells.
- Atrophy of intestinal glands.

**NECROTIC ENTERITIS**
Necrotic enteritis is characterized by necrosis of mucosal epithelium of intestine leading to erosions/ulcer formation and exposition of underlying tissues (Fig. 16.30 to 16.32).

**Etiology**
- Salmonella
- *Rinderpest, rotavirus, cornovirus, Hog cholera virus.*
- *Coccidia, Histoplasma*
- *Niacin deficiency*
- *Clostridium* sp. after coccidial infection in birds.

**Macropscopic features**
- Necrotic patches in intestines.
- Fibrinous deposits over necrotic patches like bran deposits
- Swelling of mesenteric lymphnodes
- Ulcers in intestine.

**Microscopic features**
- Congestion and infiltration of mononuclear cells.
- Necrosis and desquamation of intestinal villus epithelium, leading to exposed underlying tissue.
- Ulcers in mucosa.
- Proliferation of crypt epithelium, presence of abnormal epithelium over villus surface.

**PARASITIC ENTERITIS**
Parasitic enteritis is caused by parasites and characterized by catarrhal and/or haemorrhagic exudate in intestine, presence of ova/adult parasite and thickening of the wall of intestine (Fig. 16.33 & 16.34).

**Etiology**
- Helminths
  - Roundworms
  - Tapeworms
- Protozoa
  - Coccidia
  - Histoplasma

**Macroscopic features**
- Presence of parasite helminths in the lumen of intestine.
- Thickening of the wall of intestine.
- Catarrhal or haemorrhagic exudate in intestine.
Fig. 16.33. Photograph showing parasitic enteritis (Coccidiosis)

Fig. 16.34. Photomicrograph showing parasitic enteritis (Coccidiosis)

Fig. 16.35. Photograph showing fibrinous enteritis

Fig. 16.36. Photograph showing granulomatous lesion in duodenum of poultry

Fig. 16.37. Photograph showing small tiny necrotic granulomatous lesion on intestine

Fig. 16.38. Photograph showing pilonidal cysts (Hair balls) recovered from stomach of calves

Fig. 16.39. Photograph showing polycystic lesions recovered from stomach of a barking deer

Fig. 16.40. Diagram (A) and photograph (B) showing intussusception in intestine
Microscopic features
- Presence of large number of goblet cells in mucosa of intestine.
- Congestion and/or haemorrhage.
- Presence of parasite/ova in the intestinal lumen
- Infiltration of eosinophils in mucosa and submucosa of the intestines.
- Coccidia can be seen on mucosal scrapings under microscope.

FIBRINOUS ENTERITIS
Fibrinous enteritis is the fibrinous inflammation of intestine characterized by presence of fibrinous exudate comprising of pseudomembrane in the mucosa of intestine (Fig. 16.35).

Etiology
- Salmonella choleraesuis
- Spherophorus necrophorus

Macroscopic features
- Presence of diphtheritic membrane over mucosa of intestine.
- Button ulcers
- Sometimes, diphtheritic membrane covers the faeces.

Microscopic features
- Congestion and haemorrhage in intestine.
- Thickening of intestinal wall due to fibrinous exudate.
- Fibrin network in mucosa.

GRANULOMATOUS ENTERITIS
Granulomatous enteritis is caused by bacteria or fungi and characterized by granuloma formation in the intestines (Fig. 16.36 & 16.37).

Etiology
- Mycobacterium tuberculosis
- Coli granuloma- E. coli in poultry (Hjarre’s disease)
- Coccidioiodomycosis / candidiasis.

Macroscopic features
- Granulomatous about cm diameter elevated/ raised areas on the serus surface of intestine.
- Thickening of the wall of intestine.
- Small, tiny, white necrotic nodules on serosa.

Microscopic features
- Granuloma formation consisting of central necrosed area covered by lymphocytes, macrophages, epithelioid cells, giant cells and fibrous connective tissue
- Extensive proliferation of fibrus tissue.
- Presence of bacteria / fungus in the lesion.

INTESTINAL OBSTRUCTION
Obstruction of intestines may occur as a result of foreign body, enterolith, piliconcretions, phytobezoars, polybezoars or due to hypermotility of intestines leading to intussusception, volvulus or torsion.

Piliconcretions
Piliconcretions are hair balls mostly found in stomach/intestines of those animals having habit of licking. This vice is more common in suckling calves and in animals with pica related to phosphorus deficiency. The hairs are accumulated in stomach which becomes in rounded shape due to movements of stomach and look like balls. Such hair balls are not degradable in gastrointestinal tract and may cause obstruction (Fig. 16.38).

Phytobezoars/Polybezoars
Concretions formed in gastrointestinal tract as a result of deposition of salts around a nidus of undigested plants or polythenes. They may cause obstruction in gastrointestinal tract (Fig. 16.39).

Foreign bodies
Foreign bodies like rubber balls, nuts, bones, stones, plastic and rubber materials, polythenes may obstruct the intestinal tract as they are not degradable in the gastrointestinal tract.
Fig. 16.41. Diagram showing volvulus in intestine

Fig. 16.42. Diagram showing torsion in intestine

Fig. 16.43. Photograph showing A. enterolith recovered from colon of a horse B. cross section of enterolith showing lamillated deposition of salts

Fig. 16.44. Photograph showing typhlitis in poultry

Fig. 16.45. Photograph of liver showing hepatitis with focal necrosis

Fig. 16.46. Photograph showing presence of fibrinous membrane on liver (Colisepticemia.)

Fig. 16.47. Photomicrograph showing focal necrosis
Hernia
Hernia is presence of intestinal loop in umbilical area, scrotum or inguinal cavity which causes passive congestion, oedema and obstruction in intestines.

Intussusception
Intussusception is telescoping of intestine means a portion of intestine enters in caudal segment due to violent peristaltic movement. It causes obstruction, passive congestion and oedema (Fig. 16.40).

Volvulus
In volvulus, the loop of intestine passes through a tear in mesentry. It causes obstruction at both ends of loop (Fig. 16.41).

Torsion
Torsion is twisting of intestine upon itself causing obstruction (Fig. 16.42).

Enterolith
Concretions in intestines particularly in horses are responsible for obstruction of intestinal tract and are responsible for “colic in horse” and enterocolitis (Fig. 16.43).

TYPHILITIS
Typhilitis is the inflammation of caecum. It is particularly important in poultry caused by protozoan parasites and characterized by haemorrhage, thickening of the wall, presence of cheesy exudates and/or necrotic ulcers (Fig. 16.44).

Etiology
- *Eimeria tenella*
- *Histomonas meleagridis*

Macroscopic features
- Haemorrhage in caecum, blood mixed contents.
- Thickening of the wall, with congestion and cheesy exudates.
- Presence of necrotic ulcers in caecum in case of histomoniasis which is further supported by round, depressed, yellowish green areas of necrosis in liver.

Microscopic features
- Congestion, haemorrhage, necrosis
- Presence of protozoan parasites
- Necrotic hepatic lesions.

HEPATITIS
Hepatitis is the inflammation of liver. It may be acute or chronic. Acute hepatitis is characterized by the presence of degeneration and necrosis of hepatocytes and infiltration of neutrophils and mononuclear cells alongwith hyperemia and/or haemorrhage (Fig. 16.45 to 16.48).

Etiology
- Bacteria- Necrobacillosis, *Salmonella, E. coli*
- Virus- ICH
- Chemicals- Carbon tetrachloride
- Parasites- *Fasciola gigantica, Fasciola hepatica*

Macroscopic features
- Enlargement of liver.
- Congestion and/or haemorrhage.
- Presence of necrotic patches in liver.
- Presence of fibrinous diphtheritic membrane on liver.

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

In acute toxic hepatitis there is necrosis of hepatocytes. According to location it can be classified as under which is helpful in making diagnosis.
- **Diffuse necrosis** covers a considerable area crossing over the lobular boundaries.
- **Focal necrosis** occupying only a part of lobule e.g. EHV induced aborted foetal liver.
- **Peripheral necrosis** is characterized by necrosis at the periphery of lobule which
Fig. 16.48. Photomicrograph of liver showing diffuse necrosis

Fig. 16.49. Photograph showing cirrhosis in liver

Fig. 16.50. Photomicrograph showing cirrhosis in liver

Fig. 16.51. Photomicrograph showing cirrhosis in liver

Fig. 16.52. Photograph showing cholecystitis in birds

Fig. 16.53. Photograph showing cholangitis (ARS/USDA)

Fig. 16.54. Photograph showing pearly disease

Fig. 16.55. Photograph showing haemorrhage in mesentry due to peritonitis
occurs due to presence of strong toxins in blood.

- **Midzonal necrosis** have necrosis of cells in midway of periphery and centre of lobule.
- **Centrilobular necrosis** is characterized by necrosis of hepatocytes around the central vein occurs due to stagnation of blood with toxaemia.
- **Paracentral necrosis** is characterized by necrosis of hepatocytes at one side of central vein e.g. Rift valley fever.

**CIRRHOSIS**  
Cirrhosis is the chronic inflammation of liver characterized by extensive fibrosis, hepatic degeneration and necrosis (Fig. 16.49 to 16.51).

**Etiology**
- Bacteria- Salmonella, *Spherothorus necrophorous*
- 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.

**Macroscopic features**
- Liver becomes hard and firm.
- Surface of liver becomes uneven and nodular.
- Size of liver becomes reduced due to atrophy.
- Colour becomes yellowish, grey.

**Microscopic features**
- Increase in fibrous tissue within and around lobules.
- Infiltration of macrophages and lymphocytes.
- Central vein is either absent or placed eccentrically.
- Hepatocytes show degenerative and necrotic changes.

- **Biliary cirrhosis** is characterized by proliferation of fibrous tissue around the bile ducts encircling them e.g. *Fasciola gigantica*.
- **Glissonian cirrhosis** is mostly confined to areas at a short distance beneath the capsule.
- **Pigment cirrhosis** is associated with yellow discoloration.
- **Central or cardiac cirrhosis** is increase in fibrous tissue around the central vein as a result of chronic passive congestion.
- **Parasitic cirrhosis** occurs due to damage caused by migration of parasites e.g. *Ascaris lumbricoides, Schistosoma* sp.

**CHOLECYSTITIS**  
Cholecystitis is the inflammation of gall bladder characterized by congestion, thickening of wall and infiltration of mononuclear cells. Cholangitis is the inflammation of bile duct (Fig. 16.51 & 16.53).

**Etiology**
- Parasites- *Fasciola* sp.
- Foreign body- Stones
- Bacteria- *E. coli*.

**Macroscopic 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.

**Microscopic features**
- Congestion
- Proliferation of fibrous tissue in the wall of gall bladder
- Infiltration of mononuclear cells
- Increased number of mucus secreting cells.

**PANCREATITIS**  
Pancreatitis is the inflammation of pancreas characterized by necrosis of pancreatic tissue, infiltration of neutrophils and mononuclear cells and fibrous tissue proliferation.
Etiology
- Bacteria
- Virus- Reovirus in poultry
- Parasites

Macroscopic features
- Pancreas becomes pale, swollen, oedematous.
- In chronic cases, atrophy of pancreas
- Pancreas becomes hard, firm, and fibrous.

Microscopic features
- Necrosis of pancreatic cells.
- Oedema, 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 features
- Serofibrinous, fibrinous, suppurative or granulomatous lesions
- Accumulation of clear fluid is known as Hydroperitoneum or Ascites.
- Presence of nodules in tuberculosis is also termed as “Pearly disease”.

Microscopic features
- Serofibrinous, suppurative or granulomatous lesions.
- Thickening of peritoneum, adhesions due to fibrosis.

MODEL QUESTIONS

Q.1. Fill in the blanks with suitable word(s).
1. In esophagus sub-epithelial fibrous nodules are produced by .................
2. Esophageal choke may lead to ............... in ruminants characterized by ................. rumen.
3. Omasitis is the inflammation of.............caused by.............and characterized by...........nODULEs.
4. Clostridium septicum may cause ............. in sheep characterized by ................., ................. and ................. of abomasal folds.
5. Haemorrhagic enteritis is the inflammation of ............... along with ............... exudates caused by ................., ................. and ................. bacteria and characterized by ................. or ................. haemorrhage in the intestinal wall.
6. Chronic enteritis is the.............inflammation of intestine characterized by.............changes like ................., ................. and ................. in lamina propria leading to ................. of intestinal wall.
7. In poultry necrotic enteritis is caused by .............after the primary damage caused by .............
8. Coligranuloma is also known as ................. in poultry and is caused by .................
9. ............. is the cause of ingluvitis in poultry which produce ................. like lesions.
10. In acute toxic hepatitis, necrosis occupying a considerable area in lobule is known as .................

Q.2. Write true or false, correct the false statements.
1. ..........Ulcerative stomatitis is a feature of mucosal disease in cattle.
2. ..........Impaction of rumen may lead to alkalosis.
4. ..........Actinobacillosis in omasum is characterized by haemorrhagic lesions.
5. ........Focal necrosis of liver covers a considerable area of lobules.
6. ........Cirrhosis is the extensive fibrosis of liver.
7. ........Once cirrhosis starts it can’t be checked inspite of removal of causative agent.
8. ........Parasitic cirrhosis is caused by *Fasciola gigantica*.
9. ........Cholangitis is the inflammation of gall bladder.
10. ........Mid zonal necrosis occurs in rift valley fever.

**Q.3. Define the followings.**

1. Necrotic enteritis
2. Atresia ani
3. Plicocconcretions
4. Glossitis
5. Cleft palate
6. Intussusception
7. Phytobezoars
8. Cardiac cirrhosis
9. Cholangitis
10. Pearly disease

**Q.4. Write short notes on.**

1. Frothy blot
2. Hjarre’s disease
3. Enteroliths
4. Cholecystitis
5. Traumatic Reticulitis
6. Fibrinous enteritis
7. Impaction

**Q.5. Select appropriate word(s) from the four options given with each statement.**

1. Turkish towel like lesions are observed in .................
   (a) Candidiasis  (b) Histomoniasis  (c) Moniliasis  (d) Coccidiosis
2. Vesicular stomatitis is seen in cases of .................
   (a) Rinderpest  (b) Mucosal disease  (c) Hog cholera  (d) FMD
3. Choked esophagus may cause ................. in ruminants.
   (a) Impaction  (b) Vomition  (c) Tympany  (d) Gastritis
4. Rumen is distended due to accumulation of ................. in bloat.
   (a) H₂S  (b) CO₂  (c) CO  (d) All of the above
5. Traumatic reticulitis may lead to .................
   (a) Pericarditis  (b) Peritonitis  (c) Pleurisy  (d) All of the above
6. Increase in ................. cells is observed in catarrhal enteritis.
   (a) Mast cells  (b) Eosinophils  (c) Goblet  (d) Neutrophils
7. Punched out ulcers are produced by .................
   (a) Theileria  (b) Babesia  (c) Hog cholera  (d) *Clostridium* sp.
8. Granulomatous lesions in intestine of poultry are observed in .................
   (a) Coli granuloma  (b) *E. coli* infection  (c) Hjarre’s disease  (d) All of the above
9. Telescoping of intestine is also known as .................
   (a) Torsion  (b) Volvulus  (c) Intussusception  (d) None
10. *Eimeria tenella* causes ................. in intestines.
    (a) Typhlitis  (b) Enteritis  (c) Colitis  (d) Proctitis
11. Necrosis of hepatocytes at one side of central vein in liver is known as ................. necrosis.
    (a) Centrilobular  (b) Midzonal  (c) Paracentral  (d) Focal
12. Parasitic cirrhosis is caused by .................
    (a) *Hemonchus* sp.  (b) *Ascaris lumbricoides*  (c) *Fasciola* sp.  (d) Amphistomes
13. Cholecystitis is the inflammation of .................
    (a) Urinary bladder  (b) Bile duct  (c) Gall bladder  (d) Pancreas
    (a) Hypertrophy  (b) Atrophy  (c) Hyperplasia  (d) Hypoplasia
15. ‘Pearly disease’ is caused by .................
    (a) Streptococci  (b) Staphylococci  (c) *Mycobacterium* sp.  (d) None
16. Erosive stomatitis is seen in .................
    (a) Rinderpest  (b) Mucosal disease  (c) Pox  (d) FMD
17. Ingluvitis is the inflammation of .................
    (a) Colon  (b) Rectum  (c) Jejunum  (d) Crop
18. Sub-epithelial fibrous nodules are produced in ................. esophagitis.
    (a) Traumatic  (b) Bacterial  (c) Viral  (d) Parasitic
19. Sudden change in feed with lush green fodder is the cause of .................
    (a) Impaction  (b) Tympany  (c) Reticulitis  (d) None
20. Acute abomasitis characterized by oedema, congestion and haemorrhage of abomasal folds is feature of .................
    (a) Enterotoxaemia  (b) Black disease  (c) Braxy  (d) Blue tongue
21. Corrugations in large intestines are observed in .................
    (a) Tuberculosis  (b) Paratuberculosis  (c) Pseudotuberculosis  (d) All of the above
22. Pica may lead to formation of .................
    (a) Piliconcretions  (b) Polybezoars  (c) Both a & b  (d) None
23. Enterolith may cause ................. in horses.
    (a) Enterotoxaemia  (b) Colic  (c) Lameness  (d) Diarrhoea
24. Frothy bloat occurs in buffaloes due to .................
    (a) Saponin  (b) Fatty acids  (c) Carbohydrate  (d) None
25. Button ulcers are produced in abomasum due to .................
    (a) *Salmonella* sp.  (b) Staphylococci  (c) *E. coli*  (d) FMD