

(F) LIVER NECROSIS AND DEGENERATION

(i) Degeneration

Liver degeneration is known as Hepatosis: It is due to fatty infiltration.

Aetiology of this condition includes:

- (i) Toxins – both endo- & exotoxins
- (ii) Nutritional and metabolic factors as seen in diabetic patients.
- (iii) Anoxia/Hypoxia as seen in anaemic patients.

Grossly, affected liver is enlarged, yellowish in colour and oily or greasy to touched when cut and put in water, fats or oil films are seen on top of the water.

Liver degeneration may be centrilobular or peripheral depending on where the lesions are observed.

When centrilobular, it is due to toxic substances coming from the lungs, when peripheral it is due to toxic substance coming from the intestine.

Sequelae – Necrosis of the hepatocytes if the cause is not removed.

- Formation of fat emboli
- Defective clotting
- Formation of fat cyst.

NECROSIS

Liver necrosis is classified based on the nature of the exudates produced and location/extent, of the area involved.

(a) Based on extent of the area involved we have;

- Focal Necrosis
- Massive/Diffuse Necrosis

(b) Based on the location of the lesion:

- Centrilobular Necrosis
- Mid zonal Necrosis
- Periportal Necrosis (Periacinal)
- Paracentral Necrosis

(b) Based on the nature of exudate produced we have:

- Coagulative Necrosis
- Caseous Necrosis
- Liquifactive Necrosis

a(i) Focal Necrosis

Here the necrotic areas are focalized or localized and restricted to a portion of hepatic lobules (sublobular size). It is seen in a Listeriosis and Leptospirosis. The liver in this case has a mottled or variegated appearance i.e. difference in coloration. The lesion can affect any part of the lobule.

(ii) Massive/Diffuse Necrosis

This involves the entire lobule of a may liver (although not the whole liver). It spreads over considerable areas without regard to lobular boundaries.

(b) (i) Paracentral Necrosis

This is an unusual form in which the necrotic area adjoins the central vein on one side but does not surround it. It has been attributed to local circulatory disorder, it is a characteristic of Rift valley fever and also seen in some anaemic diseases. The periphery of the acinus is affected

Aetiology include: vascular damage, vascular obstruction, nutritional deficiency e.g. Hapetosis dietatica.

(ii) Centrilobular Necrosis

This involves the hepatocytes central vein. It is the most common type of liver necrosis. It is found in certain cases of blood-borne toxin and anoxia due to circulatory disorder. It is usually seen in acute toxic hepatic. Causes include right sided heart failure and passive liver congestion due to stasis of blood.

(iii) Mid-zonal Necrosis

This is found in cases of yellow fever (human), Non-specific diseases of animals, Enzootic hepatitis whose appropriately involve the hepatocytes half way between the periphery and the center of the lobule. Causes include aflutoxicosis and hexachlorophene.

(iv) Periportal Necrosis

Usually seen in the peripheral zones of the lobules, it is associated with toxic injuries in the liver e.g. phosphorus, toxicity. The peripheral zone of the lobules are necrotic because the peripheral cells receive the toxic blood first and suffer most from its effect.

(b) (i) Coagulative Necrosis

Seen in diseases like Black disease and Glassers disease (lambs) Nerobasillosis (calf) of Histomoniasis (Turkey)

(ii) Caseous Necrosis

This is typical of tuberculosis parasitic infection e.g. Fascioliasis.

(iii) Liquefactive

Typically seen in corynebacterium infections

G. Metabolic disturbances in the Liver

The major ones are (i) Fatty liver and (ii) Amyloidosis.

(1) Fatty Liver/hepatic lipidosis – Defined as presence of excessive lipid within the liver due to increased accumulation of lipid and the rate of accumulation exceeds the rate of metabolism or degradation. Usually due to infiltration of fat into the liver. Grossly, the liver is enlarged, yellowish, greasy or oily to touch. The condition is not a disease entity, but a perturbation in normal lipid metabolism.

Sequelae: Fat cyst formation and this may lead to embolism and rupture.

(2) Amyloidosis

This is the extracellular deposition of amyloid (proteinaceous materials) in the liver and other parenchymatous organs. Amyloid can be demonstrated by special stain such as Congo-Red. With Lugol's iodine liver gives a reddish brown colour.

Grossly, the liver is enlarged and friable

Significance include – pressure necrosis, obstruction of sinusoids and bile canaliculi.

(ii) Hepatic Toxicosis

This refers to pathologic condition due to hepatotoxic agent/substance factors that influence hepatic toxicosis are

- (1) Dosage consumed
- (2) Time of exposure
- (3) Component of the agent
- (4) Host factors – age, sex etc.

Sequelae include

- (1) Fatty change
- (2) Hepatic necrosis
- (3) Liver Fibrosis
- (4) Liver Cirrhosis

The most important liver toxicosis is Aflatoxicosis (from mouldy feed).

Mouldy feed contains a fungal toxin called aflatoxin which produces hepatotoxicosis.

Pathology

In acute case, you will observe periportal necrosis and liver haemorrhage. In chronic form, liver is yellowish, then there is liver fibrosis and later cirrhosis other lesions include bile duct proliferation and hepatitis.

Hepatic Abscess

This is common in pigs and ruminants especially cattle. Its economic significance is partial condemnation of the affected liver. Aetiology include trauma, omphalophlebitis, corynebacterium, staphylococcus sp, streptococcus sp, as well as exo and endotoxins.

Sequelae

The abscess can rupture and this may lead to discharge vena cava leaving to pyemia, septicaemia, toxemia and peritonitis.

Nutritional Diseases of liver:

(1) Hepatosis Dietetica/Nutritional hepatic necrosis.

The condition is a syndrome of acute hepatic necrosis that occur in rapidly growing swine. It is a manifestation of a variety of disorders that are, at least in part causes by a deficiency of either vit.E or selenium. The pathogenesis is incompletely defined.

Grossly: Affected liver is distended, deep red and friable and later collapse to form dense tracts of connective tissue (post necrotic scarring).

HEPATIC TUMOURS

Liver tumour may be primary or secondary .

Primary tumour – those that originate from hepatocytes

Secondary tumour – those that metastasize to liver. Examples of such tumours are:

(a) Primary

- (1) Hepatocellular adenoma
- (2) Hepatocellular carcinoma
- (3) Cholangio cellular adenoma (from bile duct)

(b) Secondary

These are common in dogs. The most important one is lymphosarcoma caused by metastasis from the lymphoid organs like spleen to the liver.

PATHOLOGY OF PANCREAS, PERITONEUM AND GALL BLADDER.

PANCREAS

Pancreas is just like the liver is an accessory organ. It has exocrine and endocrine portions the former is responsible for enzyme secretion while the latter is responsible for hormone secretion most especially glucagons and insulin

A Pancrease Abnormalities

(i) Pancreatic Division

Here the 2 portion fails to unit to form a single organ. But individual functions are normal.

(ii) Annular Pancreas

Pancreas surrounding the duodenum instead of in loop. It can cause obstruction.

(iii) Accessory Pancreas

Also called ectopic pancreas. Here a portion of pancreatic tissue is located outside the pancreas.

(2) Acquired Abnormalities

The major one is pancreatic cyst or pancreatic bladder. Here there is blockage or obstruction of the pancreatic duct by calculi, stones, migrating ascanid, this predisposes the organ to accumulation of exocrine secretion followed by enlargement and cyst formation.

Sequelae – pressure necrosis

- Rupture
- Exudation of proteolytic enzyme
- Pancreatic necrosis
- Acute pancreatitis

B. Pancreatitis (Inflammation of Pancreatic tissue).

Aetiological agents may be enterogenous infection (through intestine) or haematogenous (via blood) characteristic lesions may include:

- Catarrhal exudates
- Haemorrhage
- Suppuration (as seen in pyogenic organism) fibrosis
- Oroliferation (as seen in Tuberculosis and Necadiazis)

C. Pancreatic Tumours

- Adenoma
- Adenocarcinoma

Note – Tumour of endocrine portion is more important because it causes excessive production of endocrine secretion e.g. insulin.

PERITONEUM

This is the serous lining of the abdominal cavity.

1. Circulatory disorder in the peritoneum

(a) Congestion/Hyperaemia

(b) Hydroperitoneum/Ascitis

(c) Haemorrhage/Haemoperitoneum

2. Inflammation is known as peritonitis, and this is always infectious, the mode of entrance of the degree varies with the causative agent(s) and relative resistance of the host. E.g. dogs seldom have serious peritonitis as against horses which are very susceptible.

Aetiology

- (1) Infectious agents – virus, bacteria e.t.c.
- (2) Trauma or rupture especially due to surgical incisions of the abdominal wall.

Routes of Infection may include:

- Haematogenous (through infection)
- Rupture/perforation of hollowed organs like uterus, stomach and intestine, e.t.c.
- Trauma due to operative incision through the abdominal wall, via an infected umbilicus in the new born and by direct extension from infected kidney. This depends on the nature of the inflammatory exudates. These are: (a) serous peritonitis (b) Sero-fibrinous peritonitis (c) suppurative peritonitis due to pyogenic organisms. (d) Granulomatous peritonitis.

Types

(3) Tumours of the peritoneum

Primary tumours are – mesothelioma and Lipoma

Secondary tumours are: - Adenocarcinoma, Lymphosarcoma and Haemangioma

GALL BLADDER

Function – stores and concentrates bile.

(A) Inflammatory Conditions.

- (i) Cholecystitis – inflammatory of the gall bladder
- (ii) Cholangitis – This is inflammation of the bile duct.
- (iii) Cholangi hepatitis – inflammation involving both bile duct and liver.

Causes

- Ascending infection from the duodenum
- Infection through haematogenous route.
- Bile retention in the bile duct retained bile cause irritation which leads to inflammation.

(B) Cholelithiasis

This is defined as presence of biliary calculi, or choleliths in the gall bladder and bile duct. Three types gall stones have been described viz: cholesterol stones, pigment stone and calcium phosphate and carbonate stones. Causes is unequivocally cholecystitis of infectious origin.

The common gall stones, however are composites of these several ingredients commonly being yellow to dark brown in colour, light in weight and somewhat fragile.

In animals, gall stone are “silent” that is asymptomatic, but in humans, they cause episodes of severe pain, dyspepsia, nausea and other gastric symptoms e.g. steatorrhea. The accompanied cholecystitis is believed to be responsible for this symptoms rather than the stones themselves.

SPECIFIC GIT DISEASES – AN OVERVIEW

These diseases are of special interest in veterinary medicine because they have major economic impact on our food industry. Based on the aetiological agent, these diseases include:

(A)

- 1) Foot and Mouth Disease (MD)
- 2) Bovine viral diarrhoea/mucosa disease
- 3) Rinderpest/peste des petits ruminants (kata)
- 4) Transmissible gastro-enteritis (Pig)
- 5) Porcine epidermic diarrhoea
- 6) Canine parvoviral enteritis
- 7) Canine distemper
- 8) Feline viral enteric disease
- 9) Malignant catarrhal fever

(B) Bacterial Diseases

- 1) Colibacillosis
- 2) Clostridial enterotoxaemia
- 3) Swine dysentery
- 4) Salmonellosis
- 5) Johne's disease (Paratuberculosis)
- 6) Tyzzer's disease
- 7) Mucoïd enteritis of rabbit

(C) Parasitic Diseases

- 1) Helminths – have been discussed earlier
- 2) Protozoa

(D) Enteric Diseases of Uncertain Aetiology

- 1) Inflammatory bowel disease
- 2) Canine lymphocytic – plasmacytic enteritis
- 3) Canine mucosa colitis
- 4) Colitis x of horses

(E) Neoplastic Diseases

- 1) Intestinal Adenocarcinoma
- 2) Intestinal adenoma (polyp)
- 3) Intestinal lymphosarcoma

A. VIRAL DISEASES

(a) Foot and Mouth Disease (FMD, Aphthous Fever)

- Caused by picorna virus which has 7 distinct antigenic types, (A, O, C, SAT-1, SAT-2, SAT-3 and Asia-1) and infection with one type confers no resistance to the other six.
- Main hosts are domestic ruminants and pigs (cloven footed animals) but virus can affect other spp including man, wild ruminants, swine and some laboratory rodents. Most feared animal disease in the world because of high morbidity even though mortality is low except in suckling.
- Virus enters through pharynx causes a viraemia, localizes in epithelial cells and replicates before vesicles etc. are formed. Entry is through fomites, saliva or ingestion of infected animal products.
- Lesions: Mucosal hyperaemia, vesicles/bullae followed by erosions/ulceration on lips, cheeks, gums, tongue, hard palate, dental pad, feet, teats. Bullae usually rupture within 24 hours leaving a raw, moist base and later scab formation. Secondary bacterial infection frequently occurs so that healing is usually by both epithelial regeneration and scarification. Affected hoofs may separate. Calves die from acute heart failure and show mottled pale areas in the ventricles of the heart (Tiger heart) due to acute myocardial degeneration and lymphocytic infiltration.

(b) Bovine virus diarrhoea

- The disease first appeared in the form of epizootics (BVD in 1940 and MD in 1953) but is now sporadic and with higher mortality.
- Young animals (less than 2 years) without maternal antibodies are most susceptible.
- Virus exists as cytopathic (CP) and non-cytopathic (NCP) biotypes and an animal infected with both biotypes shows more severe disease which is also more chronic (i.e. MD).
- Transmission is through inhalation, ingestion placenta and use of live vaccines. Virus is present in a wide variety of excretions and may even be spread through artificial insemination, embryo

transfer and during rectal examination for pregnancy diagnosis. The epidemiology of the disease depends critically on the persistently infected viraemic excretor.

Forms of BVD/MD depend on the strain and virulence of the virus, the immune status of the host, whether or not the animal is pregnant and the stage of pregnancy. They include:

- i. Bovine virus diarrhoea most common form of the disease. It is usually mild or sub-clinical but may be more severe. It is seen in immuno-competent, sero-negative, non-pregnant animals.
 - ii. Persistent infection: transplacental infection of the fetus with NCP-BVD virus during the first 4 months of gestation results in the production of a persistently infected calf. The calf may be clinically normal, weak or undersized at birth, but it often becomes unthrifty. Most succumb to MD before 2 years of age. The few that survive to reproductive age always produce viraemic calves or infected semen. The epidemiology of BVD thus depends on the persistently infected host.
 - iii. Mucosal diseases: Mucosal disease occurs when a persistently infected NCP-BVD viraemic virus. It is the most severe form of the disease and is almost invariably fatal. Severe lesions are usually found in the upper and lower alimentary tract, similar to those of rinderpest.
 - iv. BVD and secondary infections: due to the immunosuppressive effects of the virus. Bacterial, mycotic and other viral infections are enhanced.
 - v. BVD-induced thrombocytopenia: seen in calves with some strains of NCP-BVD virus which cause destruction and/or sequestration of thrombocytes.
 - vi. BVD and reproductive diseases: infection of seronegative immunocompetent dams may lead to foetal infection. In early gestation this results in foetal resorption, mummification or abortion. Between 90 and 120 days of gestation, the virus has teratogenic effects including microencephaly, cerebellar hypoplasia and eye defects. Also, up to 4 months of gestation after 260 days gestation may result in the birth of normal but weak calves.
- Characteristic gross lesions are foci of necrosis, erosion and/or ulceration in the mouth, oesophagus, forestomachs, small and large intestines and fibrinonecrotic pseudomembranes over Peyer's patches (mucosal disease) similar to those of rinderpest.
 - Histo: Ulceration of squamous epithelium and cellular infiltration of the base, necrosis and inflammation of crypts, cystic dilation and crypt dropout, necrosis of Peyer's patches with herniation of crypts and ulcerative dermatitis (coronitis). Variable degree of generalized lymphoid necrosis and moderate fibrinoid necrosis of arterioles.

VESICULAR STOMATITIS

- Caused by a rhabdo virus and affects horses, cattle and pigs. Milder than FMD and occurs in Western hemisphere (tropical and subtropical).

1. PORCINE VIRAL ENTERIC DISEASES

(a) Transmissible gastro-enteritis (TGE)

Characterized by vomiting and diarrhoea in pigs under 14 days of age. High morbidity and mortality.

B. BACTERIAL DISEASES

(a) Colibacillosis

- A very common disease of newborn, especially calves and piglets caused by *Escherichia coli*. For disease to occur, the organisms must adhere to the surface of enterocytes, proliferate and produce toxins or invade the tissue.
- Predisposing factors include:
 - (i) Heavily contaminated environment
 - (ii) Failure to receive colostrums
 - (iii) Formula feeding (milk substitutes)
 - (iv) Cold stress
 - (v) Overcrowding
 - (vi) Concurrent viral or cryptosporidial infection.

Disease occurs in different forms

- (i) Enterotoxigenic E. Coli (ETEC): Newborn calves and pigs. Organisms attach to enterocytes thus producing heat-labile (LT) and heat-stable (ST) enterotoxins and cause secretory diarrhea (like cholera). They cause chloride secretion by crypt cells, sodium and water following osmotically from the mucosa. No significant lesions in intestinal mucosa.
- (ii) Entero-invasive E. Coli (EIEC): Organisms invade and damage enterocytes (like shigellosis) and may become septicemic.
- (iii) Attaching and effacing E.Coli (AEEC): Organisms attach to enterocytes and produce shigalike toxin and efface microvilli with effluent haemorrhagic enteritis and colitis. Associated with swine postweaning diarrhea.
- (iv) Septicaemic colibacillosis: Systemic infection resulting from entry per or through umbilicus or respiratory tract. Fibrinous polyserositis is characteristic if animal lives long enough.

- (v) Enterotoxemic colibacillosis (gut edema): Disease of older pigs (6-14 weeks) due to a neurotoxin produced by *E. Coli* in small intestine (similar to shigella neurotoxin). Toxin is absorbed and causes widespread vascular damage and neuronal degeneration usually without diarrhea. There is marked oedema of eyelids and walls of stomach, intestine and gall bladder.

(c) Clostridial enterotoxemia

A group of disease caused by *Clostridium perfringens* and involving primarily the small intestine and characterized by severe toxemia and sudden death in well nourished animals. Synonyms include overeating disease, struck, pulpy kidney etc. Organism produces 5 types of exotoxins (A-e) and an enterotoxin. These toxins act on enterocytes by one of several mechanisms which facilitate chloride and bicarbonate transport into intestinal lumen. Water moves with the ions to result in diarrhea. Two main syndromes are of significance.

- (i) Haemorrhagic enteritis of neonatal pigs, calves and lambs due to type B, C or E.
- (ii) Pulpy kidney due to type D (epsilon toxin). Seen especially in sheep following a change in feeding or increase in carbohydrate levels. Rapid multiplication of organisms in intestine is favoured by intestinal hypomotility and presence of starch. This leads to production of exotoxin which damage the mucosa and other organs after absorption.

Gross: Haemorrhagic discoloration of intestine and serous surfaces soft and dark kidneys and pericardial effusion. Rumen is usually full of feed.

Histo: Haemorrhagic enteritis (segmental) fibrinous pericarditis and focal symmetrical encephalomalacia.

Clinical Path: Terminal glycosuria in sheep (due to rapid mobilization of hepatic glycogen stimulated by the toxin). Also detect toxin in fresh intestinal content.

(d) Salmonellosis

Disease occurs as peracute septicemia in young animals or acute/chronic enterocolitis in adults. Over 2000 serotypes but only about a dozen serotypes account for more than 75% of all isolates from man and animals. It is a serious zoonosis worldwide and many human infections are traceable to poultry foods especially eggs.

Organisms are acquired by ingestion and they invade enterocytes and macrophages. Producing enteritis and septicemia. Like other gram negative bacteria, they produce endotoxins. Infected animals may become symptomless carriers for indefinite periods.

Gross: In septicemic form, petechial haemorrhages and multifocal hepatic necrosis.

Acute forms (cattle, pig, horses): fibrino-necrotic enterocolitis and enlarged mesenteric lymph nodes.

Chronic form (pigs): ulcerative entero-colitis. Chronic ulcerative proctitis with granulation tissue formation can lead to rectal strictures.

Histo: Typical reaction to bacterial infection, including presence of "paratyphoid nodules" in liver and other organs.

(e) John's disease (Paratuberculosis)

A chronic infection of ruminants due to *Mycobacterium (avium) paratuberculosis* transmitted orally or in utero. There is an age-dependent resistance to infection after 6 months. Disease is characterized by chronic wasting and intractable diarrhea leading to mal-absorption, dehydration emaciation and death. Exacerbation of clinical disease often are associated with parturition, a low plane of nutrition, heavy milk yield and inter-current disease.

Gross: Marked thickening and enlargement of mucosal folds and enlarged mesenteric lymph nodes and lymphatics.

Histo: Massive proliferation of foamy macrophages containing numerous acid-fast organisms in lamina propria (lepromatous reaction) but could be tuberculoid in resistant animals. In a herd, many animals are infected but only 1-2% show clinical disease. Exacerbations of clinical disease often are associated with parturition, heavy milk yield and inter-current disease. The chronic wasting seen in clinical cases has been attributed to mal-absorption due to villous blunting and to the elaboration of Tumour Necrotic Factor (TNF) by activated macrophages.

C. PARASITIC DISEASES

(a) Ruminants

- (i) Haemonchosis: caused by *Haemonchus contortus* in sheep and goats and *H. placei* in cattle (barber pole worm). Heavy infestation leads to severe hypoproteinaemic oedema, emaciation, anaemia and death. Parasites are readily seen mixed with blood in abomasums. May undergo hypobiosis.

- (ii) Ostertagiosis: caused by *Ostertagia circumcincta* in sheep and goats and *O. Ostertagi* in cattle. Caused severe edema and inflammation of abomasums and nodular hyperplasia of mucous cells. Nodules are pinhead sized and may be so numerous and confluent to create a cobblestone surface or morocco leader appearance. Clinical signs follow heavy infection (type 1) or synchronous maturation and emergence of large numbers of hypobiotic larvae from mucosa (type II) Parasites are difficult to recognize grossly.
- (iii) Trichostrongylosis: caused by *Trichostrongylus axei*.
- (iv) Ascariasis: *Ascarid sp* in cattle, sheep, goat, dogs and pigs.
- (v) Cestodiasis: *Moniezia sp*
- (vi) Oesophagostomiasis (pimply gut).

(b) Horses

- (i) Gastric bots (*Gasterophilus intestinalis* in nonglandular stomach and *G. nasalis* pyloric mucosa and duodenum). Generally asymptomatic but cause erosions and ulcers. Larvae are 2cm long and have anterior pinchers.
- (ii) Habronemiasis: caused by the spiruid worms *Draschia megastoma* and *Habronema majus*. *Draschia* produces tumor-like nodules in the fundus near the margo plicatus.
- (iii) Trichostrongylosis: hyperplastic gastritis of glandular portion.
- (iv) Gastrodiscoide: very common in the caecum also called caecal fluke.

(c) Swine

- (i) Hyostrongylosis: caused by *Hyostrongylus sp* resulting in hyperplastic gastritis as in ostertagiosis of ruminants.

2. Protozoa

- (a) Coccidiosis: Produces haemorrhagic and necrotic enteritis, colitis typhitis mainly in young animals. Disease is most important in poultry and ruminants and is caused by various species of *Eimeria* and *Isospora*. Large schizonts appear grossly as pin-point white foci on the mucosa.
- (b) Cryptosporidiosis: *Cryptosporidium* is smaller coccidium and attaches to the surface of enterocytes in ileum, jejunum and caecum. It produces watery diarrhoea often in association with neonatal virus enteritis and colibacillosis. This is a zoonosis and outbreaks in humans may occur following open days on livestock farms.

D. ENTERIC DISEASE OF UNCERTAIN ETIOLOGY

1. INFLAMMATORY BOWEL DISEASES

In dogs and cats, idiopathic inflammatory bowel disease (IBD) are frequent causes of chronic vomiting and diarrhea. The diseases are characterized by (a) persistent clinical signs of several weeks duration, leading to weight loss in spite of good appetite, and (b) histological evidence of inflammation of undetermined cause in the lamina propria of the small or large intestine. Proposed pathogenetic mechanisms include a variety of immunoregulatory defects, mucosal barrier defects and environmental agents. IBD has been reported in a few horses and in a cow.

Some of the conditions include:

- (a) Canine and feline lymphocytic-plasmacytic enteritis – common, idiopathic, no gross lesions.
- (b) Diffuse eosinophilic gastroenteritis – probably allergy to some food.
- (c) Canine mucosal colitis – common, idiopathic.
- (d) Colitis X of horses – a post-stress diarrhea characterized by toxic megacolon and pseudomembranous colitis. Probably due to proliferation of colonic pathogens (*Staph. Aureus*, *Clostridium difficile*, etc) when other competitive flora are suppressed by treatment.
- (e) Equine granulomatous enteritis – probably due to strongyle larvae or *Mycobacterium spp.*

2. Intestinal lymphangiectasia and lipogranulomatous lymphangitis

E. NEOPLASTIC DISEASES

a) Intestinal Adenocarcinoma

Disease of old dogs, cats, sheep and cattle and most often involving the colon and rectum in dogs and the ileum and jejunum in other species. Tumor is usually firm and annular and may cause stenosis. Chondroid and osseous metaplasia may occur, especially in cats. There are frequent metastasis to the serosa draining lymph nodes and liver. In sheep, relatively high frequency of the tumor has been linked to bracken fern poisoning.

- b) Intestinal adenoma (polyp). Less common.
- c) Carcinoid tumor (from enterochromaffin or Kulchitsky cells) is rare.
- d) Sarcomas involving smooth muscles and fibrous tissue are not uncommon.