# Infectious Canine Hepatitis (Hepatitis Contagiosa Canis - HCC)

- Infectious canine hepatitis is a viral infection of dogs and other canids, including foxes and coyotes.
- **canine adenovirus 1.**
- is not lethal in dogs **older than two years.**
- The virus has a predilection hepatocytes, vascular endothelium, and mesothelium; fulminant disease is characterized by hepatic necrosis and widespread serosal hemorrhage that can affect a variety of organs.

# Infectious Canine Hepatitis
# (Hepatitis Contagiosa Canis - HCC)

- Exposure of susceptible dogs is most often via the oral by contact with urine from infected dogs.
- Viremia lasts for 4 to 8 days.
- Virus multiplication initially occurs in the tonsils and produces tonsillitis, which can be severe, with spread to local lymph nodes and then to the systemic circulation. Viremia is associated with leukopenia and fever. Spread of virus to the liver, endothelial cells, and mesothelial cells follows. Infection of Kupffer cells may precedehepatocellular injury
- Adenoviruses are cytolytic and cause necrosis of infected cells.

# Infectious Canine Hepatitis (Hepatitis Contagiosa Canis - HCC)

- **Clinical sings:**
- Vomiting - bloody faeces - high fever - abdominal pain,
- Petechial hemorrhages in gums,
- The mucous membranes are pale and slightly icteric,
- Nerve symptoms in some cases,
- Faringitis and tonsillitis in mild cases.

# Infectious Canine Hepatitis (Hepatitis Contagiosa Canis - HCC)

- Some dogs recovering from infectious canine hepatitis develop immune-complex uveitis (type III hypersensitivity), which produces *degeneration and necrosis of the corneal endothelium* and resultant *corneal edema* clinically known as **blue eye**.

# Infectious Canine Hepatitis (Hepatitis Contagiosa Canis - HCC)

- Grossly;
- petechiae and ecchymoses,
- accumulation of clear fluid (sometimes bloody) in the peritoneal and other serous cavities,
- Enlargement, edematous and reddening of the tonsils and Superficiall ymph nodes
- Linear bleedings in stomach serosa,
- mild icterus.

# Infectious Canine Hepatitis
# (Hepatitis Contagiosa Canis - HCC)

- Grossly;
- ***The liver*** is moderately enlarged and friable
- may contain small foci of centrilobular hepatic necrosis.
- the presence fibrin strands on the surface of the liver,
- the wall of the gallbladder is thickened by edema.
- Foci of hemorrhage in lung, brain, kidneys, and the metaphysis

of the long bones may also be evident.

# Infectious Canine Hepatitis (Hepatitis Contagiosa Canis - HCC)

- Microscopic lesions;
- ***Liver;***
- has usually widespread centrilobular necrosis.
- Sinusoids are enlarged and full of blood,
- Large deeply eosinophilic to amphophilic intranuclear inclusions are found hepatocytes, vascular endothelium, and Kupffer cells.
- Marginal hyperchromasia in inclusion nuclei.
- Inflammation tends to be mild, and *neutrophils and mononuclear cells* are the most abundant cell type.

# Wesselsbron Disease

- Wesselsbron disease of sheep is caused Wesselsbron virus, a flavivirus.
- It a zoonotic arthropod (mosquito)- transmitted viral disease that occurs in Africa.
- Zoonose?
- The virus can cause disease newborn lambs and abortion in ewes.

# Wesselsbron Disease

Clinical Signs : The typical presentation can abortions, stillbirths, mummified fetuses and weak lambs that die soon after birth. **Some aborted fetuses** may congenital CNS defects and arthrogryposis.

- The clinical signs in **neonatal lambs** can be nonspecific, with fever, anorexia, weakness, a rough hair coat and an elevated respiratory rate. Some descriptions also mention mucoid diarrhea, melena, pale mucous membranes, blood-stained nasal discharges, icterus, edema of the head or encephalitic signs. Some affected lambs can die within a few days.
- **Pregnant shee**p may also have systemic signs, with one study describing a moderate fever, mild lethargy, decreased appetite and an elevated respiratory rate after experimental inoculation. Reproductive complications, such as metritis and pregnancy toxemia, can be fatal.
- **Nonpregnant sheep** generally seem to develop a brief, mild, nonspecific febrile illness or have no clinical signs.

# Wesselsbron Disease

- Grossly;
- Affected lambs multifocal areas of generalized petechiae, and hepatomegaly a yellowish to orange-brown liver. Icterus may develop. Canalicular cholestasis is often apparent. There may also white, pinpoint necrotic foci in the liver.
- The gastrointestinal tract may contain hemorrhagic lesions, including petechiae and ecchymoses in the abomasal mucosa, and hemorrhagic lesions of varying severity in the intestines.
- Petechiae and ecchymoses can sometimes be detected on the fascia and serosal surfaces of other organs, and there may blood-stained fluid in the abdominal and thoracic cavities
- Pulmonary congestion, splenomegaly and subcutaneous edema have also been described.

# Wesselsbron Disease

- Histopathologically;
- Scattered individual hepatocyte and sinusoidal lining cell necrosis accompanied pigmented macrophages and mononuclear inflammatory cells within the parenchyma are typical
- Eosinophilic inclusions can be found in hepatocytes.
- A newborn calf infected in utero, which died soon after birth, hepatic lesions resembling those seen in lambs, together congestion, edema and hemorrhages of the brain, respiratory tract, myocardium, spleen and small intestinal mucosa

❖***In Wesselsbron disease, foci of hepatic necrosis are typically less extensive than in Rift Valley fever, although cholestasis is usually more prominent.***

# Rift Valley Fever

- Rift Valley fever is an acute, arthropod (mosquito)-transmitted ***zoonotic*** viral disease that affects ruminants, causing extensive mortality among calves and lambs and abortion in ewes and cows, although adults can also be affected.
- The causative virus is a member of **family Bunyaviridae in the genus Phlebovirus.**
- The disease enzootic in southern and eastern Africa.
- In severe cases, affected animals febrile may abort, have respiratory and gastrointestinal signs, including prominent diarrhea Mortality is highest in sheep, with lambs most severely affected, but even in cattle up to one-third of calves may die.

# Rift Valley Fever

- Hepatic involvement is consistently present in fulminant cases, typically in neonates, and is hepatomegaly with a yellow-orange discoloration. Areas of congestion may be present.
- In older pale, 1- to 2-mm randomly scattered foci of hepatocellular necrosis impart a mottled appearance and sometimes an enhanced lobular pattern.

# Rift Valley Fever

- Haemorrhages and oedema wall of the gall bladder

  contain a blood coagulum or blood-tinged bile.

Most mature sheep and cattle haemorrhages and oedema in the abomasal folds, and sometimes copious amounts of free blood in the lumen of the intestines.

# Rift Valley Fever

- Microscopic lesions are characterized by the both randomly distributed foci of hepatocellular necrosis and apoptosis. Secondarily, more widespread zonal necrosis, which ranges from centrilobular to midzonal, can develop.
- Fibrin deposition within sinusoids is common, but cholestasis is not a consistent feature.

# Rift Valley Fever

- Eosinophilic intranuclear inclusion bodies may be present in degenerate hepatocytes in areas of necrosis.
- Diffuse petechiae and ecchymoses are also characteristic of the disease edema and hemorrhages of the intestinal tract and the wall of the gallbladder.

# *Bacterial Diseases*
# Liver Abscesses and Granulomas

Bacteria can reach the liver via a number of different routes and form abscesses .

Routes include the following:

- The portal vein
- The umbilical veins from umbilical infections in newborn animals
- The hepatic artery, as part of a generalized bacteremia
- Ascending infection of the biliary system
- Parasitic migration.

# Liver Abscesses and Granulomas

- Gram-positive and Gram-negative organisms can cause hepatic abscesses. Among the Actinomyces (Corynebacterium) pyogenes, Streptococci and Staphylococci are prominent.
- Hepatic abscesses are often caused by any of a variety of enteric species, as well *Francisella* spp., *Nocardia asteroides*, *Actinomyces* spp.
- Bacterial infections of the liver and subsequent formation of hepatic abscesses or foci of necrosis are especially common in neonatal foals and ruminants, in addition to feedlot cattle.
- It becomes a single or a large number and especially develops in the left lobe.
- Liver abscesses of cattle frequently are incidental lesions, but they can cause weight loss and decreased milk production.

# Liver Abscesses and Granulomas

The result is formed

- Direct implantation of foreign body in reticulum,
- direct invasion of supurative lesion at traumatic reticulitis
- vena porta embolus,
- direct omfaloflebitis

Hepatic abscesses usually occur as a sequel to toxic rumenitis because damage to the ruminal mucosa allows ruminal microflora, Fusobacterium necrophorum, to enter the portal circulation.

# Liver Abscesses and Granulomas

- Granulomas are randomly distributed (i.e., hematogenous spread) in the liver.
- They a central core of cell debris, caseation, and granulomatous inflammation surrounded a fibrous capsule.

# The Results of Liver Abscesses

- ✓ is asymptomatic and unimportant.
- ✓ It can be recovered by resorbing or around the capsule is shaped.
- ✓ nflammation with fibrin adhesions can be seen to the surrounding tissues.
- ✓ Rarely perforated.
- ✓ thrombophlebitis of the vena cava, endocarditis, lung abscesses, or lung embolism.
- ✓ In young animals omphalos abscesses are seen.
- ✓ In adults a large number of abscesses are formed and especially necrobacillary it causes death.

# Infectious Necrotic Hepatitis/ Black disease

Infectious necrotic hepatitis, also known as **black disease**, is most in sheep and cattle but also in pigs and horses.

Infectious necrotic hepatitis is caused Clostridium novyi (type B).

Clos. novyi produces **three potent exotoxins;**

Alpha toxin is related to the clostridial cytotoxins produced by *C. difficile* and *C. sordelli.*

Beta toxin a necrotizing and hemolytic phospholipase C (lecithinase)

- Zeta toxin hemolytic properties.

The disease is actually a intoxication with exotoxins.

# Infectious Necrotic Hepatitis/ Black disease

For the disease occur;

* The animal is nonimune,

* Finding latent sports infection in tissue,

* It is necessary to shape the anaerobic environment in order to activate and proliferate the spores.

* The anaerobic environment required for the activation of spores is mostly provided in sheep with Fasciola hepatica larvae.

*Black disease is principally an acutely fatal disease of sheep in regions where the inciting helminths are endemic.* The disease most commonly initiated by migrating larvae of the common liver *Fasciola hepatica.* Sporadic cases may be related *Cysticercus tenuicollis* infection or may be idiopathic.

# Infectious Necrotic Hepatitis/ Black disease

Pathogenesis: *C. novyi* is widely distributed in soil, and the spores are continually ingested by grazing animals in areas where black disease occurs.

- In endemic areas, germination of spores is usually initiated by hepatic necrosis caused by the migration of immature liver flukes.
- Parasitic migration tracts are usually present within the affected liver.
- Ruminants take sports through the digestion. Some spores cross the mucous membranes, probably in phagocytes.
- They remain as latent infections macrophages, mainly in the liver, spleen, and bone marrow. . The duration of latency in tissue is not known, but it can be many months.

# Infectious Necrotic Hepatitis/ Black disease

Clinical signs:

- Deaths in sheep from black disease occur rapidly and usually without warning signs.
- Illness, if observed, is brief and characterized by reluctance to move, drowsiness, rapid respiration, and quiet subsidence
- Affected animals are usually in good nutritional condition. Postmortem decomposition occurs rapidly.

# Infectious Necrotic Hepatitis / Black disease

Grossly:

- *The name of the disease is derived from the appearance of flayed skins, the dark coloration being caused by an unusual degree of subcutaneous venous congestion.*
- Frequently, there edema of the sternal subcutis, and airways contain stable foam
- Serous cavities contain an abundance of fluid that clots on exposure to air. The fluid is usually straw colored, but that in the abdomen may be tinged with blood. The volume of fluid in the abdomen and thorax may vary from about 50 mL to 1.5 L.

# Infectious Necrotic Hepatitis / Black disease

Grossly:

- The pericardial sac is distended with similar fluid in amounts up to ~300 mL
- Subendocardial hemorrhages in the left ventricle are almost constant
- Patchy areas of congestion and hemorrhage may be present in the pyloric part of the abomasum and in the small intestine.

# Infectious Necrotic Hepatitis / Black disease

Grossly:

The typical and diagnostic lesions occur in the liver and are always present. They are usually clearly evident on the capsular surface, the diaphragmatic surface especially, but the organ may have to be sliced carefully to find them. The liver will be the seat of either

acute traumatic hemorrhagic lesions of acute fascioliasis the cholangiohepatitis of the chronic disease, or both.

# Infectious Necrotic Hepatitis / Black disease

Grossly:

- The lesion of black disease, and occasionally there are several, a yellow-white area of necrosis 2-3 cm in diameter, surrounded by a broad zone of intense hyperemia, roughly circular in outline, and extending hemispherically into the substance of the organ.
- There may a coagulum of fibrin on the capsular surface overlying the necrotic area. Occasionally, the essential lesions are rectilinear in shape or very irregular. The lesions appear homogeneous on the cut surface, but some contain poorly defined centers of soft or caseous material.

# Infectious Necrotic Hepatitis / Black disease

Histologically,

- The histologic evolution of the hepatic lesions begins the necrotic and hemorrhagic tracts caused by wandering immature flukes.
- These sinuous tunnels ~0.5 cm in diameter that contain blood, necrotic hepatic cells, and the leukocytes, chiefly eosinophils, attracted by the flukes.
- About the tunnels is a narrow zone of coagulative necrosis, also produced by the flukes.
- As usual, the necrotic tissue is demarcated by a thin zone of chiefly neutrophils.

# Bacillary Hemoglobinuria

- *Bacillary hemoglobinuria a counterpart of black disease.*
- The cause ***Clostridium haemolyticum,*** which is closely related to *C. novyi.*
- Both species produce the *beta toxin*, a necrotizing and hemolytic lecithinase (phospholipase C).
- The pathogenesis of the 2 diseases is comparable, as both depend on a focus of hepatic injury within which latent spores can germinate.
- *Bacillary hemoglobinuria as an endemic disease exists only in areas where* Fasciola hepatica *abounds, and it is probable that flukes are the primary cause of the initiating lesion.*

# Bacillary Hemoglobinuria

- Bacillary hemoglobinuria occurs *cattle and sheep.*
- It is characterized clinically *intravascular hemolysis* *anemia* *hemoglobinuria.*
- The essential lesion is hepatic and similar to that of black disease but much larger and usually single.
- **Acute,infectious&toxemic bacterial disease**
- Route of infection:

Ingestion of contaminated food.

# Bacillary Hemoglobinuria

Histopathologically

- Liver lesion was defined as a portal thrombotic infarction.
- Thrombosis is seen in the lesion areas. Thrombosis are mostly occured in v.centralis.
- There is severe anemia,
- kidneys are speckled red or brown by hemoglobin, and the urine is of port-wine color.
- Peritoneal vessels are injected, and in some cases, there severe, dry, fibrinohemorrhagic peritonitis.

# Tyzzer's Disease / Bacillus piliformis infection

- Clostridium piliforme (formerly Bacillus piliformis), a Gram-negative obligate intracellular parasite.
- in laboratory animals but occurs only sporadically in domestic animals. it has also been reported *in foals, calves, dogs, and cats.*
- Typically, only very young or immunocompromised animals are affected. The bacteria are found in the intestinal tract of rodents.
- Infection is most likely through oral route.
- *Although* the disease is probably initiated by an intestinal infection, lesions in the gut are less specific and constant than those *of focal hepatitis and necrosis.*

# Tyzzer's Disease / Bacillus piliformis infection

- After colonization of the gastrointestinal tract, organisms penetrate into the portal venous drainage and enter the liver.
- The disease is characterized enlarged, edematous, and hemorrhagic abdominal lymph nodes; hepatic enlargement; the presence of randomly distributed, pale foci of hepatocellular necrosis surrounded by a variably intense.

# Tyzzer's Disease / Bacillus piliformis infection

- **Affected foals** usually die between the ages of 1and 4 weeks; often they are found dead after a short illness
- The liver shows pale foci up to a few millimeters across; these are represented microscopically by randomly distributed foci of coagulative necrosis with moderate neutrophilic infiltrate.
- This lesion is not diagnostic in itself; its specificity depends on the presence of the causal organism in hepatocytes in the periphery of the necrotic zones.

# Leptospirosis

- Leptospirosis is caused by infection with the Gram-negative, thin, spiral, and motile bacterium of the genus *Leptospira*  *Leptospira interrogans* .
- Leptospires enter the body through the mucous membranes or through the skin if its barrier functions have been disrupted
- Contaminated water, bedding, and soil are common sources of infection because the organism is shed in urine.
- Fetuses can develop transplacental infection and are often aborted. Infection can involve red blood cells, kidney, liver, and a number of other tissues, depending on the infecting serovar.
- The liver is often involved in acute, severe leptospirosis of all domestic species because a number of serovars intravascular hemolytic anemia leading to ischemic injury to centrilobular areas.

# Leptospirosis

**Gross lesions** include icterus that produce hemolysis.

- Hepatic hemorrhage ascites can occur

**Histopathologically;**

- Acute infection can focal necrosis or centrilobular necrosis.
- dissociation of hepatocytes.
- Affected cells rounded and have eosinophilic granular cytoplasm and dark, shrunken hyperbasophilic nuclei.
- Bile casts in canaliculi are often apparent.
- Kupffer cells may contain abundant hemosiderin.

# Protozoal Diseases

- The liver can be involved in systemic infections *Toxoplasma gondii, Neospora sp.,* and other less common protozoa.
- Liver lesions are usually characterized multifocal necrosis and inflammation.
- Inflammatory cells neutrophils, macrophages, and smaller numbers of other cells
- Free tachyzoites or cysts containing bradyzoites can be found within necrotic areas or adjacent to them.

# Fungal Diseases

- Systemic involvement with fungi often includes the liver.
- Several genera of fungi may involve the liver, *Blastomyces, Coccidioides, Aspergillus, and Histoplasma.*

# Parasitic Diseases

- Nematodes.
- Cestodes.
- Trematodes. The majority of parasitic hepatic injury caused by trematodes is produced by members of three major families:
- *Fasciolidae, Dicrocoelidae, and Opisthorchidae.*

## Cestod Infections

- Stilesia hepatica
- Thysanosoma actinioides
- Cysticercus tenuicollis (metacestode of Taenia hydatigena)
- )

## Nematod Infections

- Capillaria hepatica

# Trematodes

- **Fasciolidae familia**
  # Fasciola hepatica
  # " gigantica

- **Dicrocoeliidae familia**
  * Dicrocoelium dentriticum
  * " hospes
  * Eurytrema pancreaticum

**Opisthorchiidae familia**
~ Opisthorchis tenuicollis (felineus)
~ " sinensis
~ Pseudoamphistomum truncatum
~ Metorchis conjunctus
~ " albidus
~ Parametorchis complexus
~ Amphimerus pseudofelineus

Diseases caused by these parasites **DISTOMIASIS.**

# Toxic Hepatic Diseases

**Acute Liver Toxication**

- Blue-Green Algae Poisoning
- Cycadales Poisoning
- Solanaceae Poisoning
- Compositae Poisoning
- Ulmaceae Poisoning
- Myoporaceae Poisoning
- Halogenated hydrocarbons Poisoning
- Phosphorus Poisoning
- Iron Poisoning
- Poisoning with Lophyrotoma

**Chronic Liver Toxication**

**Aflatoxicosis**

- Phomopsin Poisoning
- Sporidesmin Poisoning
- Poisoning with pyrrolizide alkaloids
- Lantana Poisoning
- Tribulosis
- Nitrosamine Intoxication
- Indospisin Poisoning
- Poisoning with Trifolium Hybridum

**Copper Poisoning**

- Hepatotoxicity caused by drugs.

# Aflatoxicosis

- The **aflatoxins** are a group of *bisfuranocoumarin compounds* produced as metabolites mainly *Aspergiltus flavus, A. parasiticus,* and *Penicillium puberulum.*
- The metabolites are designated by spectral qualities, and the major are B1, B2, G1, and G2.
- The *most significant and best studied of the aflatoxins* *B1* because its relative abundance and its potency as a hepatotoxin.
- Many others may be produced in minor amounts in fungal colonies or as metabolic products of the major toxins in animals.

# Aflatoxicosis

- Sheep, adult cattle, and rats are quite resistant to the toxin.
- Dogs, pigs, calves, mice, and ducklings are sensitive and may be fatally intoxicated by a dose rate of less than 1.0 mg/kg body weight.
- The most potent of these is the 8,9-epoxide metabolite of aflatoxin B1; this binds to adenine in nucleic acids in sensitive species that lack adequate glutathione S-transferase-mediated resistance.

# Aflatoxicosis

- Acute, fulminating liver necrosis is sometimes seen in dogs eat moldy bread, dog food, or garbage, which may contain very high concentrations of the toxin.
- Younger animals are much more susceptible may die within a few hours; the gross postmortem picture is dominated widespread hemorrhage and massive hepatic necrosis.

# Aflatoxicosis

Clinically; In dogs;

Acute cases: anorexia within 2-14 (mean 5) days, icterus, bile stained urine, occasional bloody faeces, vomiting sometimes bloody, epistaxis and rarely convulsions.

In chronic cases: Icterus 1-2 months later, ascites, weight loss and sometimes edema at the legs are seen.

- Similar findings are found in other animal species.

# Aflatoxicosis

- Grossly;
- Prolonged exposure moderate enlargement of the liver
- The enlargement may be partly due to hypertrophy of hepatocellular smooth endoplasmic reticulum and some degree of fatty change
- High level of aflatoxin ➔ liver;
- pallor, enlargement, bile staining, increased firmness due to fibrogenesis, and fine nodular regenerative hyperplasia.
- There may also edema of the gallbladder and bile-tinged ascites in more severe cases.

# Aflatoxicosis

**Histologically,** affected livers show increase in size of some hepatocytes and their nuclei (megalocytosis) with focal necrosis or .

- Bile ductules proliferate early, and reticulin and collagen deposition occurs throughout the acinus according to no distinct pattern
- Fatty change in affected livers is variable in extent and occurrence, bile pigments accumulate in canaliculi and hepatocytes in more severely affected livers.
- Minor degrees of megalocytosis may be seen in proximal tubular epithelium in the kidney.

# Copper poisoning

- Chronic copper poisoning occurs in sheep. In this syndrome, copper accumulates in the liver secondary to excessive intake.
- Copper toxicity also occurs in dogs. This condition differs from that of no hemolytic crisis and the main lesion is **chronic active hepatitis**.

# Neoplastic Lesions of The Liver and Bile Ducts

- Hepatocellular Tumors
  - Hepatocellular adenoma (Hepatoma)
  -
- Cholangiocellular Tumors
  - Cholangiocellular adenoma
  - Cholangiocellular
- Mesodermal Tumors
- Metastatic Tumors

# PANCREAS

- exocrine acinar lobules and endocrine cells. The pancreas is located behind the stomach in the upper left abdomen. It is surrounded by other organs including the small intestine, liver, and spleen.

# Acute Pancreatic Necrosis

- Acute pancreatic necrosis  a common disease of dogs.
- A comparable condition occurs in humans and is occasionally seen in cats and rarely in horses and swine.
- The condition may manifest  an acute and potentially life-threatening syndrome or as a chronic relapsing syndrome that may culminate in exocrine pancreatic insufficiency and diabetes mellitus.
- Important morphological changes are not formed in the parenchyma. Lesions are seen  interstitial tissue and adipose tissue around the organ.

# Acute Pancreatic Necrosis

- The disease develops two important ways in dogs and humans
- Acute pancreatic necrosis (dog)➔

It is located perilobular area. The inflammation settles around the lobules of the pancreas and spreads to mesenchymal tissues with surrounding fat tissues. The middle parts of the ducts and lobules remain intact.

** Acute hemorrhagic pancreatitis (human)➔

The lesions begin the middle of the ducts and lobules, then expand and cover a large portion of the pancreas. It can be complicated common bleedings and sepsis.

# Acute Pancreatic Necrosis

- Pathogenesis is unknown.
- But; It can develop ➔

  after the surgical manipulation of the pancreas,

  after long-term corticosteroid treatment,

*** fed on a rich in fat /poor in protein.

- It is mostly seen in <u>overweight female dogs.</u>

# Acute Pancreatic Necrosis

- Clinically;
- Initially it may not cause any symptoms.
- It may be characterized by abdominal pain and cardiovascular collapse.
- Death is formed within 2-3 days.
- High levels of lipase and amylase in serum and abdominal fluids **diagnostic.**
- In addition, there oil droplets in the abdominal fluid (which are released from the destroyed fat tissue).
- It is common cause of Diabetes mellitus. Because, the asinus tissue and the islets of Langerhans are also affected.

# Acute Pancreatic Necrosis

- Grossly;
- Serosanguineous fluid containing free lipid droplets is usually present
- Petechial and ecchymotic hemorrhages may be present in the pancreas, omentum and
- Numerous small white chalky areas of fat necrosis, each with an intensely hyperemic border, are present adjacent to the pancreas and in the mesentery.
- The adhesions between the pancreas and the visceral aspect of the omentum and liver are characterized by yellowish or hemorrhagic fibrin adhesions.
- The pancreas edematous, swollen and soft and in extreme fat appearance.
- The cut surface of the pancreas has a variegated appearance due to merging of white areas of fat necrosis and gray-yellow areas ofparenchymal necrosis.

# Acute Pancreatic Necrosis

- Histopathologically;
- There necrosis of peripancreatic adipose tissue and pancreatic parenchyma, edematous separation of the interstitium, mural necrosis, and thrombosis of blood vessels and reactive inflammation.
- Infiltrating leukocytes, chiefly neutrophils and macrophages, congregate at the boundary of necrotic and viable tissue.
- The necrotic fat saponifies and may undergo mild dystrophic mineralization.
- Phlebothrombosis may be apparen
- Much fibrin may be precipitated in the edematous interstitium. Capillaries at the margins of by fibrin thrombi.

# Diabetes mellitus

- Diabetes mellitus is a common of dogs and cats, but is rarely seen in cattle, small ruminants, pigs, or horses
- The insulin deficiency be absolute or relative, and a consequence of inadequate synthesis and release by sensitivity of target organs to insulin, antagonism of insulin by other hormones or drugs, or a combination of these mechanisms.

# Diabetes mellitus

- In the human classification;

✓Type 1 diabetes mellitus (*insulin-dependent)*

✓Type 2 diabetes mellitus *(non-insulin-dependent)*

✓Seconder diabetes mellitus

# Diabetes mellitus

✓Type 1 diabetes mellitus (*insulin-dependent);*

*is generally consistent juvenile-onset and insulin-dependent diabetes mellitus.*

- Patients with type 1 diabetes have a *severe absolute deficiency of insulin* due to autoimmune destruction of beta cells
- No pathogenesis caused by diabetes in domestic animals!

# Diabetes mellitus

✓Type 2 diabetes mellitus *(non-insulin-dependent);*

is generally consistent with adult-onset and non-insulin-dependent diabetes mellitus.

- Genetic effects, obesity, exercise and diet are important in the formation of the disease.

both impaired insulin action (insulin resistance)

inadequate insulin secretion to meet metabolic demands. The capacity of beta cells to secrete insulin and the number of beta cells decline as the disease advances.

Depending on the stage of the disease and the functional capacity and number of beta cells present.

# Diabetes mellitus

✓Type 2 diabetes mellitus *(non-insulin-dependent);*

- Obesity significantly increases the risk of developing glucose intolerance and clinical disease in cats.
- Obese cats are insulin-resistant and chronic hypersecretion of insulin to combat this may ultimately exhaust the beta cells.

# Diabetes mellitus

✓Seconder diabetes mellitus;

- There is severe weakness and dehydration.
- yellow and fatty.
- The pancreas may appear normal, or the lesions associated with postpartum degeneration and pancreatitis are observed
- Lipemia can be seen.
- Blood serum may be milk-like in appearance.

# Diabetes mellitus

Histopathologically;

Pancreas➔

- There may be vacuolation of islet cells and of epithelial cells lining the smaller ducts.
- There is usually severe diffuse macrovesicular hepatic lipidosis and often biliary epithelial vacuolation due to glycogen accumulation.
- In dogs, glycogen may also accumulate within the nuclei of hepatocytes

Liver➔

- Severe lipidosis.

# Diabetes mellitus

Histopathologically;

Kidney➔

Glycogen nephrosis is a specific lesion of diabetes.

- The glycogen is chiefly deposited in tubular epithelium of the loops of Henle and the distal convoluted tubules and mainly in the nephrons of the inner cortex.
- There is fatty degeneration of the epithelium of the proximal renal tubules
- Lipid emboli are occasionally detectable in glomerular capillaries.
- In chronic *diffuse or nodular glomerulosclerosis* may develop.

# Diabetes mellitus

Histopathologically;

Eye ➔

- Cataract formation is an early and common complication of diabetes in the dog. Dogs with poorly controlled diabetes and erratic fluctuations in blood glucose concentration appear to be particularly at risk of developing cataracts.
- Cataracts can develop rapidly and are irreversible. Cataracts are observed infrequently in diabetic cats suggesting that their lens metabolism differs from that of dogs or that the lens capsule is less permeable to glucose.

# Diabetes mellitus

Histopathologically;

Nervous system ➔

- *Peripheral demyelinating neuropathy* is a well-recognized complication of diabetes in cats but occurs very infrequently in dogs.
- The neuropathy is reversible if glycemic control is achieved soon after it manifests, but not in protracted cases.

# Neoplasia of The Islets

- Insulinoma
- Gastrinoma
- Glucagonoma

# Peritonitis

- Damage to the serosa of the peritoneal lining associated with inflammation, or **peritonitis,** is very common in the large domestic animals, and less common in dogs and cats.
- *Peritonitis may be classified;*

*a primary or secondary acute or chronic local or diffuse septic or nonseptic; and on the basis of the type of exudate, which may be serofibrinous, fibrinopurulent, purulent, hemorrhagic, or granulomatous.*

# Peritonitis

Chemical peritonitis may be induced by a variety of agents;

Intraperitoneal drug applications for treatment,

- Experimental antibiotic lavages,
- Surgical glove powder (Talk-Starch),
- Chemical substances that accidentally spilled into the abdominal cavity (barium sulfate, etc.),
- Causes of bile and pancreatic enzymes.

# Peritonitis

Bacterial Peritonitis,

Perforations of the gastrointestinal and urinary system organs or abdominal wall,

Opening of the abdominal cavity to external environment through genital system in female animals,

Uterine ruptures and vaginal laceration,

Cystitis and contaminated bladder rupture,

Propagation of the inflamation in the abdominal organs,

* Extensive expanding of gangrenose inflamation on the intestine or uterus wall is caused.

# Results of Peritonitis

- Within a few hours the movements of intestine are increased, the paralytic ileus is formed in a short time.
- Negative effects on cardiovascular functions, circular hemostasis and acid-base balance occur.
- However, all cases of generalized peritonitis do not result in death in a short time.
- According to the structure of the exudate, diffuse adhesions may be formed or may develop chronic active or adhesive peritonitis in localized areas.
- If the fibrin masses deposited on the collapsed serosal surfaces cannot be melted, they may undergo organization with connective tissue and adhesions may develop.
- It may lead to adherence of adjacent organs or stenosis in the intestines.
- Diffuse peritonitis is mostly acute and results in death.

# Feline Infectious Peritonitis - FIP

- The group 1 species in genus Coronavirus, family Coronaviridae, Feline coronavirus (FCoV), and its two biological types Feline infectious peritonitis virus (FIPV) and Feline enteric coronavirus (FECV), as well as Transmissible gastroenteritis virus of swine and Canine coronavirus
- It occurs in 6 months to 3 years old cats.
- Sporadic.
- Mortality approaches 100%
- *Most cats do not develop disease when exposed to FIPV, and those cats that do develop the disease are usually under 2 years of age.*

# Feline Infectious Peritonitis - FIP

- **Clinically;**
- **Cats with noneffusive FIP** have a chronic disease of insidious onset and frequently develop signs specific to organs severely affected by vascular lesions.
- These ocular disease central nervous disorders such as ataxia, paraparesis, head tilt; specific nerve palsies, nystagmus, _____; renal failure hepatic or pancreatic insufficiency.
- An uncommon intestinal manifestation may be seen as a protracted period of vomiting and diarrhea with a palpable mass usually at or about the ileocecocolic junction.

# Feline Infectious Peritonitis - FIP

- **Clinically;**
- Cats **the effusive form of FIP** may develop abdominal distension.
- Ascites!
- Pleural effusion is present in "-25% of cases and may cause dyspnea.
- Cardiac tamponade due to pericardial effusion rare.
- Ocular and central nervous signs rare.
- The hypergammaglobulinemic and leukocytosis and neutrophilia.
- Most go on to die; very few recover after passing through a phase of noneffusive disease.

# Feline Infectious Peritonitis - FIP

- **Grossly;**
- Cats **the effusive form of FIP**
- Up to 1 liter of abdominal exudate may be present in cats with effusive FIP.
- The viscous, clear, and pale to deep yellow, although it may be flocculent and contain strands of fibrin.
- The serosal surfaces may be covered with fibrin.Fibrin is frequently prominent over the visceral peritoneum, and fragile adhesions may be present.

# Feline Infectious Peritonitis - FIP

- **Grossly;**
- Cats **the effusive form of FIP**
- There white foci of necrosis or raised plaques or nodular cellular infiltrations on the serosa and extending into the organs or wall of the intestine. These vary in size from a few millimeters to a centimeter in diameter
- The omentum may be contracted into a mass in the cranial abdomen, and adherent to itself and other abdominal surfaces
- Mesenteries may be thickened and opaque.

# Feline Infectious Peritonitis - FIP

- **Grossly;**
- Cats **the effusive form of FIP**
- The kidneys may be enlarged and nodular, with few or many, small to large, white, firm nodules protruding from the cortex
- Hepatitis and pancreatitis of variable degree may also be present, characterized by small, white foci of inflammation.
- Fibrin is usually less prominent in the thorax, but firm white foci may be present under the pleura,
- Hydropericardium and epicarditis occur less frequently.
- Abdominal and thoracic lymph nodes may be enlarged and have a lobulated pattern.

# Feline Infectious Peritonitis - FIP

- **Grossly;**
- **Cats with the noneffusive**
- In cats with noneffusive FIP, there may inflammatory loci in the abdominal or thoracic organs, or lesions may be restricted the eyes and nervous system
- Diffuse uveitis or chorioretinitis may progress to panophthalmitis fibrin is often present in the anterior chamber.
- Lesions in the central nervous system can involve leptomeninges, spinal cord, or brain, but usually are visible grossly only in the leptomeninges as thickening or white streaks. hydrocephalus or syringomyelia may result from ependymitis.

# Feline Infectious Peritonitis - FIP

- **Histopathologically;**
- *The characteristic microscopic lesion is generalized vasculitis and perivasculitis,* especially of venules, with a focal mixed inflammatory reaction. This lesion occurs in the serous membranes, in the connective tissue of the parenchymatous organs, the eye, and the meninges.
- Neutrophils, lymphocytes, plasma cells, and macrophages accumulate in and around affected vessels.
- The endothelium swells, and medial necrosis
  thrombosis may occur.
- The vascular lesion results in the serofibrinous and cellular exudate on the serosal surfaces, and the nodules visible on the surfaces and deeper in solid organs.

# Feline Infectious Peritonitis - FIP

- **Histopathologically;**
- The microscopic changes on the omentum, mesentery, and serosal tissues vary in severity
- Mild changes are proliferation of mesothelial cells, slight fibrin exudate with fibroblast proliferation, and scattered neutrophils and mononuclear cells.
- Severe changes result in a thick layer of fibrin adherent to the serosa, with necrosis and/or hypertrophy of mesothelium. Large numbers of neutrophils, mononuclear cells, and necrotic debris may be embedded in the fibrin.

# Feline Infectious Peritonitis - FIP

- **Histopathologically;**
- Subcapsular infiltrates occur particularly in the liver, lung, and pancreas, perivasculitis can develop deep in the parenchyma, especially in the kidney.
- There diffuse interstitial pneumonia close to the visceral pleura.
- Severe focal or lymphoplasmacytic interstitial nephritis may develop
- Cellular infiltrations in the spinal or cerebral meninges, the choroid plexuses, ependyma, and perivascular spaces.
- Degenerative and necrotic lesions in the parenchyma of the central nervous system.

# Feline Infectious Peritonitis - FIP

- *Effusive FIP _____bacterial peritonitis in particular.*

- *Noneffusive forms _____lymphosarcoma, steatitis, mycotic infections, and toxoplasmosis.*