

Diffuse diseases of the liver

Hepatitis: the word hepatitis is include all diffuse, degenerative, and inflammatory diseases that affect the liver.

There are 5 types of hepatitis as follow:

- a- Toxic hepatitis.
- b- Infectious hepatitis.
- c- Parasitic hepatitis.
- d- Nutritional hepatitis.
- e- Congestive hepatitis.

Etiology:

A) Toxic hepatitis: The common causes of toxic hepatitis in farm animals are:

- ✓ **Inorganic poisons:** copper, phosphorus, arsenic, possibly selenium
- ✓ **Organic poisons:** carbon tetrachloride, hexachloroethane.
- ✓ **Poisonous plants** Weeds, including Senecio, Crotalaria, Heliotropium.
 - ✓ Pasture and cultivated plants - Panicum eifusum, lupins, water-damaged alfalfa hay .
- ✓ Trees and shrubs -lantana (*Lantana camara*); yellow wood (*Tenninalia oblonga ta*); ngaio tree (*Myoporum laetum*);seeds of cycads (*Zamia spp.*).
- ✓ **Fungi:** *Aspergillus flavus*, *Penicillium rubrum*,. *Pithomyces chartarum*
- ✓ **Algae:** the slow death factor .
- ✓ **Insects:** ingestion of sawfly larvae (*Lophyrotoma interrupta*) .
- ✓ **Miscellaneous farm chemicals** These include dried poultry waste, cotton- seed cake, herring meal.

B)Toxemia perfusion hepatitis: Moderate degrees of hepatitis occur in many bacterial infections regardless of their location in the body, and the hepatitis is usually classified as toxic. Hepatic failure may occur in dairy cattle following mastitis or metritis , Endotoxemia. Or extensive tissue damage occurring after burns, injury and infarction.

C) Infectious hepatitis: Diffuse hepatic lesions in animals are rarely associated with infectious agents. The significant ones are

- Rift Valley fever virus
- *Bacillus piliformis*, associated with Tyzzer's disease in foals

- Equine herpesvirus-1 of viral rhinopneumonitis as a cause of abortion in horses.
- Infectious necrotic hepatitis associated with *Clostridium novyi*.
- Some of these are infectious equine anemia, salmonellosis, leptospirosis, septicaemic listeriosis.
- Systemic mycoses, e.g., histoplasmosis.

D) Parasitic hepatitis:

- ✓ Acute and chronic liver fluke infestation.
- ✓ Migration of larvae of ascaris.

E) Nutritional hepatitis:

A multiple dietary deficiency has also been suggested as the cause of a massive hepatic necrosis observed in lambs and adult sheep.

Hepatic lipidosis occurs in cattle during the transition period from late gestation to peak lactation. It is triggered by a negative energy balance occurring in the periparturient period in over conditioned dry cows.

- ✓ Deficiency of cystine and thiamine at one time.
- ✓ Methionin deficiency.

F) **Congestive hepatitis:** Due to congestive heart failure, which causes increased pressure in the sinusoids of the liver, leading to anoxia and compression of surrounding hepatic parenchyma, which causes centrilobular degeneration.

G) Idiopathic Hepatosis and Cirrhosis:

1-**Hepatic cirrhosis and hemochromatosis in horses** has been recorded. There is cirrhosis with increased iron stores in the parenchymal cells of the liver.

2- **Hepatic fatty cirrhosis (hard yellow liver)** in sheep and cattle has occurred in Texas during years of maximal rainfall. The cause is unknown, but the high incidence during periods of heavy rainfall suggests the possibility of either a mycotoxin or nutritional deficiency.

H- Portosystemic Vascular Anomaly: Portosystemic shunts in large animals have been recorded occasionally in foals and calves. There is altered blood flow through the liver and hepatic insufficiency secondary to hepatic atrophy

Pathogenesis: The causes of hepatitis depending on **type of agent**. The effects of **endotoxin** on the liver include multifocal hepatocellular necrosis, **decreased hepatic gluconeogenesis and decreased hepatic blood flow**. Endotoxin may cause the **Kupffer cells to release lysosomal enzymes, prostaglandins, and collagenase, which can damage hepatocytes**.

Hepatitis are usually the same as follow:

1. The usual lesions in toxic hepatitis is centrilobular varying from cloudy swelling up to necrosis with a terminal venoculose lesions as in some plant poisoning.
2. In infectious hepatitis the lesions varies from local cell to diffuse cell necrosis affecting all or most of hepatic parenchyma.
- 3- In parasitic type the changes depend upon the number and type of migrating or infesting parasites. In massive fluke infestations sufficient damage may occur to cause acute hepatic insufficiency, manifested particularly by submandibular edema.. In more chronic cases extension from a cholangitis may also cause chronic insufficiency.
- 4 - hepatic fibrosis develops particularly if there is massive hepatic necrosis, which destroys entire lobules. Degeneration is not possible when the necrosis zonal, and fibrous tissue replacement occurs. Fibrosis is a terminal stage of hepatitis that may have developed acutely or chronically and is manifested by the same clinical syndrome as that to hepatitis except that the signs develop more slowly.
- 5- Congestive hepatitis : Is characterized by dilatation of central veins and sinusoids with compression of the parenchymal cells. Hepatic fibrosis develops particularly if there is massive hepatic necrosis that destroys entire lobules.

Clinical signs

The cardinal signs of hepatitis are anorexia, mental depression with excitement in some cases, muscular weakness, jaundice and in the terminal stages somnolence, recumbency and coma with intermittent convulsion. Animals that survive the early acute stages may show photosensitization, a break in the wool or hair leading to shedding of the coat.

In the horse.

- **In acute cases** the clinical findings include
- ✓ weight loss, anorexia, dullness and depression.

- ✓ Other findings include hemoglobinuria, tachycardia, intermittent fever, abdominal pain, ventral body wall edema, clotting deficiency, muscle fasciculation and diarrhea or constipation.
- ✓ **Jaundice** is a constant feature in acute hepatic necrosis. Dysphagia, photosensitization, encephalopathy and hemorrhages tend to occur terminally, particularly in horses **with cirrhosis**.
- ✓ **The nervous signs** are often pronounced and vary from ataxia and lethargy with yawning, or coma, to hyperexcitability with muscle tremor, mania, including aggressive behavior, and convulsions. **A characteristic syndrome is the (dummy syndrome)**, in which affected animals push with the head, do not respond to normal stimuli and may be blind.
- ✓ **Serum hepatitis (Thelie's disease)** is the most common cause of acute hepatic failure in the horse. Typically clinical findings become apparent several weeks after administration of tetanus antitoxin. Lactating mares appear to be at a higher risk than other horses but this may be due to the administration of the antitoxin to mares at the time of parturition.
- ❖ **In chronic liver disease** the course is several months.
- ✓ The initial anorexia is often accompanied by constipation and punctuated by attacks of diarrhea.
- ✓ The feces are lighter in color than normal and if the diet contains much fat there may be steatorrhea.
- ✓ Jaundice and edema may or may not be present.
- ✓ Photosensitization may also occur but only when the animals are on a diet containing green feed and are exposed to sunlight. A tendency to bleed more freely than usual may be observed.

In cattle hepatic disease is characterized by

- ✓ weight loss, dullness and depression.
- ✓ **Signs of hepatic encephalopathy** include blindness, head pressing, excitability, ataxia and weakness.
- ✓ The presence of fever and jaundice represents a poor prognosis.

In young animals with Portosystemic shunts the clinical findings include

stunted growth, ascites and variable neurological abnormalities resulting from hepatic encephalopathy. Calves and foals may be a few weeks to a few months of age before they are presented for examination. Apparent cortical blindness, circling and dementia are common. Persistent tenesmus is common in calves. Recurrent

episodes of unexplained neurological clinical findings in a young foal suggest the presence of a Portosystemic shunt.

Treatment:

1. Intravenous injection of glucose and electrolyte solutions especially, if the animal is off food.
2. Avoid giving proteins as far as possible because of the danger of ammonia intoxication and the given diet should be high in carbohydrate and calcium contents and consequently low in proteins and fats.
3. Give enema with soft soap and warm water in common with giving the animal purgatives.
4. Oral administration of antibiotics (broad spectrum). The results have been excellent with neomycin and chlortetracycline, the disappearance of hepatic coma coinciding with depression of blood ammonia levels.
5. Supplementation of the feed or periodic injections of the water soluble vitamins are desirable. Hepatic fibrosis is considered to be a final stage in hepatitis and treatment is not usually undertaken.

Focal diseases of the liver

- **Hepatic abscesses:** Local suppurative infections of the liver do not cause clinical signs of hepatic dysfunction unless they are particularly massive or extensively metastatic. They do cause significant losses in feedlot and grain-fed cattle because of the frequency of rumenitis in those cattle leading to hepatic abscess formation and the rejection of the affected livers at the abattoir.
- **Tumor of the liver :** Metastatic lesions of lymphomatosis in calves are the commonest neoplasms encountered in the liver of animals, although primary adenoma, adenocarcinoma and metastases of other neoplasms in the area drained by the portal tract are not uncommon, especially in ruminants. For the most part, they produce no signs of hepatic dysfunction but they may cause sufficient swelling to be palpable, and some abdominal pain by stretching of the liver capsule. Primary tumors of the gallbladder and bile ducts also occur rarely and do not generally cause clinical signs. A primary hepatic fibrosarcoma in a goat has caused loss of body weight, although appetite was maintained, anemia and jaundice.