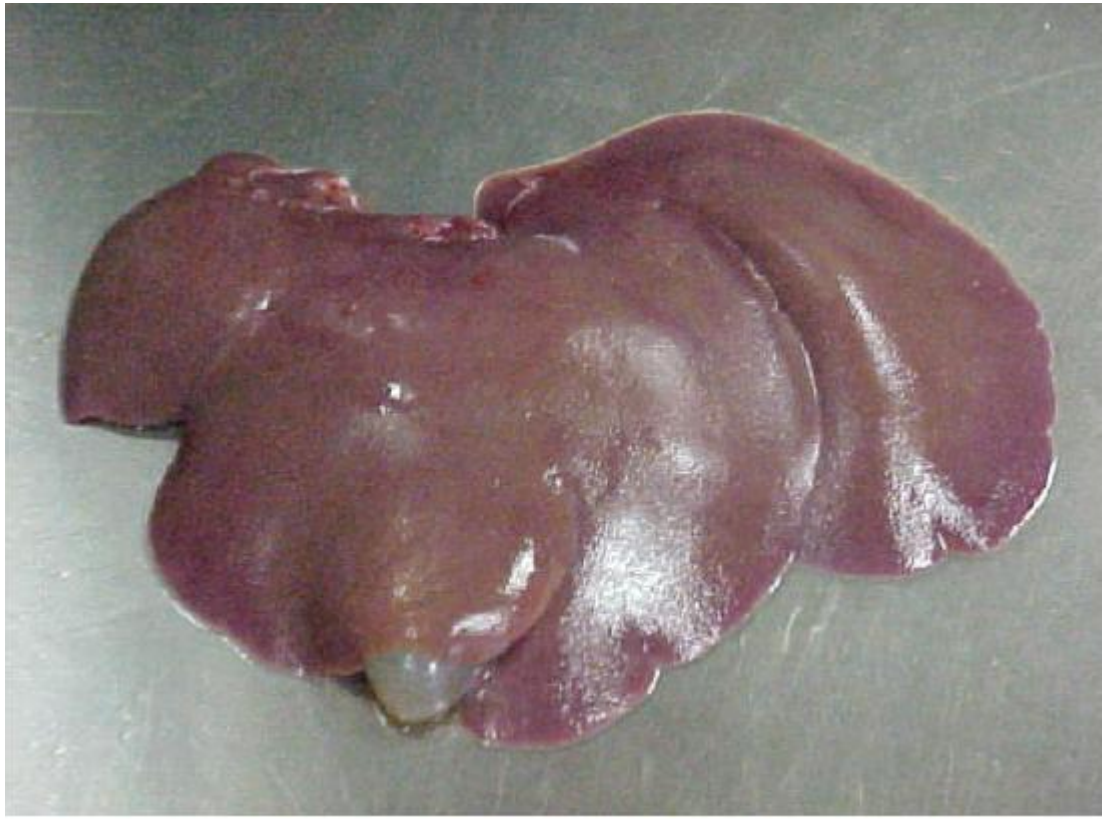
Diseases o f the liver



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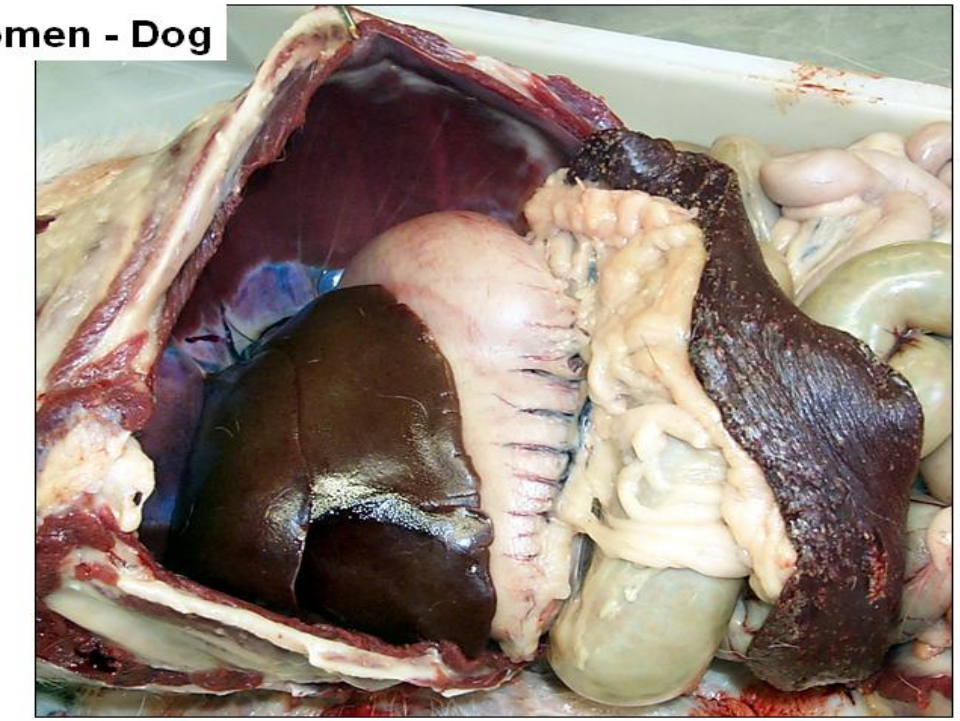
Liver

Introduction; The liver (hepar) is an extremely important organ in the body of mammals and vertebrates as it provides functions essential for life.

- It is the largest internal organ and has numerous functions including production of bile and protein, fat and carbohydrate metabolism.
- During foetal development, the liver has an important haemopoetic function, producing red and white blood cells from tissue between the hepatic cells and vessel walls.

- The size of the liver varies due to its role in metabolism. In carnivores the liver weighs about 3-5% of body weight, in omnivores 2-3% and in herbivores 1.5%. the liver is much heavier in young animals than older animals as it atrophies with age.

Cranial Abdomen - Dog



Cadaver in right lateral recumbency. Abdominal wall has been removed. Ribcage & diaphragm intact. Left ribcage is retracted laterally with forceps.

- *Identify: Tendinous & muscular portions of diaphragm, left lateral & left medial lobes of liver, right medial lobe of liver, fundus & body of stomach, greater curvature of stomach & gastrosplenic ligament, spleen.*
- *Also visible: Large intestine & jejunum*

Functions of the liver

(1) Bile metabolism:

1) Conjugation and excretion of bilirubin.

2) Synthesis and excretion of bile salts.

(2) Fat metabolism:

1) Synthesis of about 75% of the circulating cholesterol, esterification and excretion of cholesterol with bile.

2) It also synthesis of **vitamin A** from carotene.

3) Binding of cholesterol with globulins to form lipoproteins

4) Incorporation of cholesterol in the synthesis of bile salts

5) It regulates phospholipid concentration in blood plasma.

(3) Protein metabolism:

1) Deamination of amino acid to form plasma protein (Synthesis of albumin, alpha 1 globulin, elimination of gamma globulins, fibrinogen, prothrombin & cholinestrase), tissue protein and stored protein.

2) Synthesis of coagulation factors : prothrombin, factors **V,** Vil, TX, X and fibrinogen.

3) Incorporation of ammonia with CO2 to form urea.

4) Conversion of uric acid to allantoic acid.

5) Conversion of the non nitrogenous residue resulting from: deamination of amino acids into glucose, ketone bodies and other materials used in metabolism.

(4) Carbohydrate metabolism:

1) Storage of glycogen and release of glucose.

2) Gluconeogenesis.

3) Formation of lipids from excess carbohydrate

4) Maintains normal blood sugar levels.

(5) Detoxification of hormones, drugs and toxic substances and excretion of many toxine.

(6) Vitamins metabolism & storage: **A, D,** E, K, thiamine, riboflavin and niacin.

(7) Erythropoesis and blood storage with spleen.

(8) Liver has a very large reserve of functions & approximately three-quarters of its parenchyma must be rendered inactive before clinical signs of hepatic

Manifestations (principles) of liver dysfunction

- Jaundice
- Nervous signs
- Diarrhoea and constipation
- Edema and emaciation
- Endocrine abnormalites:
- Blood & serum abnormalities
- Photosensitization
- Hemorrhagic diathesis
- Abdominal pain
- Alteration in size of the liver
- Hepatic coma
- Nutritional and metabolic abnormalities

Manifestations (principles) o f liver dysfunction:

[I] Jaundice:

- It is the most important clinical sign associated with liver diseases, in which bile pigments accumulates in blood (bilirubinemia) and then partly excreted by the kidney (bilirubinuria) and partly deposited in the tissue such as mucous membrane (of conjunctiva, nasal & mouth) and unpigmented part of the skin. The sweat, milk and exudates also contain bile.

Causes o f jaundice are classified as:

(1) Pre-hepatic (Intravenous hemolytic) jaundice:

1) Bacterial infection e.g. bacillary hemoglobinuria and leptospirosis.

2) Viruses infection (Equine infectious influenza).

3) Protozoa e.g. babesiosis, anaplasma and infectious equine anaemia.

4) Hypophosphatemia.

5) Poisoning e.g. chronic copper poisoning: Arsenic; Phosphorous; lead poisoning.

6) Isoimmune hemolytic anaemia especially in newborn.

Symptoms o f hemolytic jaundice is characterized by:

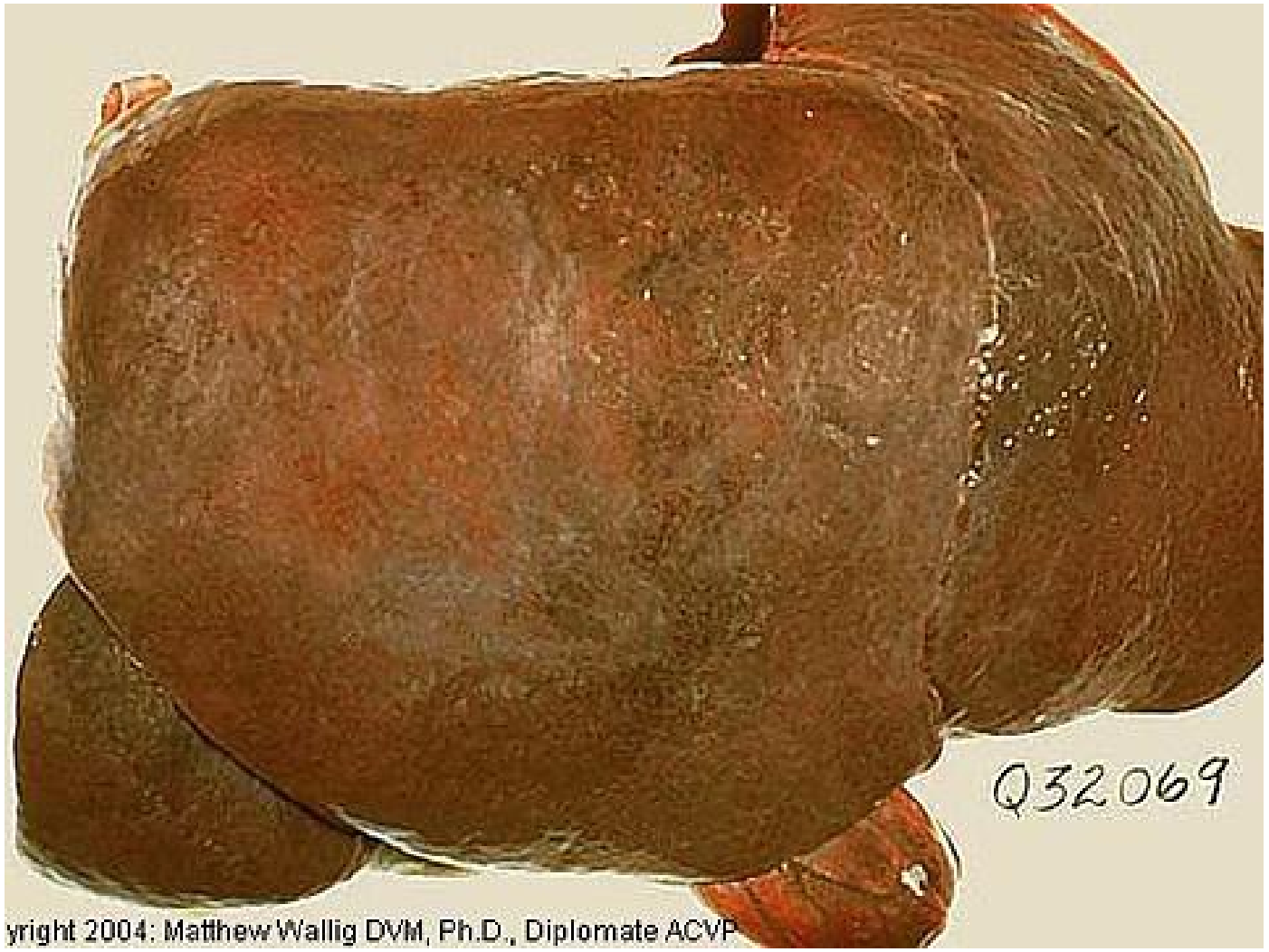
(1) Hemoglobinuria in severe cases.

(2) Anemia.

(3) Yellow mucosa (moderate degree).

(4) Increase urobilinogen & absence of bilirubin in urine.

(2) Hepatic causes of diffuse hepatitis (toxic, infective and obstructive).



(3) Post-hepatic (obstructive):

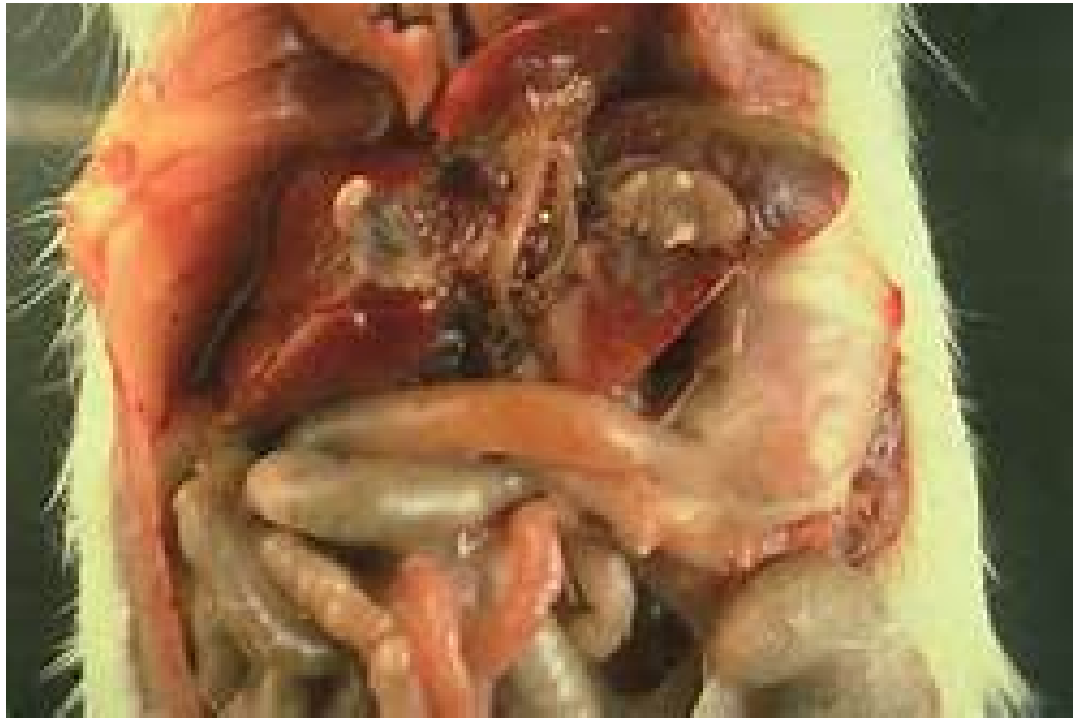
Extra-hepatic biliary obstruction

occurs by:

1) Infestation with trematodes (Fascioliasis) & nematodes.

2) Inflammation of the bile ducts by extension from enteritis.

3) Calculi or compression by tumor masses.



Symptoms o f jaundice:

(1) Indigestion, latter on the MM, unpigmented skin, sclera, tongue & gum are discolored from lemon yellow to orange yellow or greenish yellow or intense yellow.

(2) The urine, sweat, milk and other exudates stained with bile pigment. Hemoglobinuria may be occurred in haemolytic jaundice.

(3) Constipation, feces have a fetid odor and pale in color, contain fats. Feces become soft & dark in haemolytic jaundice.

(4) The animal is dull, depressed, loss its body weight & emaciated.

Treatment:

1) Complete rest, Treat the real cause, Give easily digested diet free from fat & salt, rich in protein.

(2) In constipation, direct cholagogues & laxative are used, for horse give: Mag. sulphate 60, sod. bicarbonate 30 & sod.citrate 10 gm dissolved in sufficient quantity of water given as drench for 5 days.

(3) Oral and IV injection of glucose, calcium, polyvitamines daily.

[2] Nervous signs:

- Hyperexcitability, convulsions, terminal coma, muscle tremor & weakness may be occur due to hypoglycemia & or failure of hepatic detoxication which resulted in accumulation of excess amino acids and ammonia.
- Inability to work, drowsiness & yawning occurred with more slowly liver damage & persistent hypoglycemic encephalopathy (decrease of brain glucose).

[3] Diarrhoea and constipation:

- In hepatitis and hepatic fibrosis, **the partial or complete absence of bile salts from the alimentary tract deprives bile salts from their laxative and mild disinfectant effect resulting** in :

- anorexia & vomiting, in some species
- and constipation punctuated by diarrhoea with pale faeces.

[4] Edema and emaciation:

- Failure of the liver to **anabolic amino acids and protein during hepatic insufficiency is manifested by tissue wasting and fall in the plasma protein,** which lower the osmotic pressure of the plasma lead to edema as Bottle Jaw.
- Edema is much more severe & is limited to the abdominal cavity in cases of obstruction of the portal circulation

[5] Photosensitization:

Most photosensitizing substances including **phylloerythrin** (the normal breakdown product of the chlorophyll in the alimentary tract) are excreted in the bile.

In hepatic or biliary insufficiency, excretion of these substances is retarted and photosensitization occurs.

[6] Hemorrhagic diathesis:

(1) In severe diffuse diseases of the liver, there is **a deficiency in prothrombin** formation, which **prolonged the clotting** time of the blood.

(2) **Absence of bile salts** from the **intestine retards** the absorption of the fat & fat soluble vitamins especially vitamin K formation which is essential for prothrombin, fibrinogen & thromboplastin formation.

[7] Abdominal pain:

It is caused by:

(1) Distension of liver with increased tension of the capsule (due to liver engorgement with blood in acute inflammation or CHF).

(2) The lesion of the capsule, beneath the capsule or in parenchyma, causes local irritation to its pain end organs. Pain may be included arched back, disinclined

to move, tenseness of abdomen, even pain on deep hepatic palpation.

[8] Alteration in size of the liver:

- It is seen in advanced congestion of the liver due to CHF and when multiple neoplastic metastasis occurs.
- In acute hepatitis the swelling is not sufficiently large to be detected clinically.
- If fibrosis occurs, the liver becomes smaller.

[9] Hepatic coma:

It is usually seen in chronic than acute hepatic failure due to hypoglycemia and ammonia toxicity which is caused by breakdown of protein & urea by intestinal bacteria which increase endogenous urea formation.

It causes metabolic encephalopathy & hepatic coma.

[10] Endocrine abnormalites:

Due to the role of liver in endocrine metabolism.

[11] Nutritional and metabolic abnormalities:

Principles o f treatment in diseases o f liver:

(1) Rest, try to treat the real cause.

(2) Diet free from fat, rich in carbohydrate, protein of high biological value, calcium & vitamins.

(3) Easily digested food, with mild laxative.

(4) Oral & or injected glucose 5% (hepatic wash), calcium guanidate (to reduce intoxication, easily excreted), vitamins A, C, K & B complex, diuretics, liver extract or oral liver preparation.

(5) Specific antimicrobial drugs.

(6) In chronic diffuse hepatitis, fibrous tissue replacement causes compression of the sinusoids, which is irreversible except in the very early stages, where removal of fat from the liver by administration of lypotrophic factors including choline associated with diet low in fat and protein.

Treatment of edema ***and ascites by:***

(1) Salt restriction in diet.

(2) Increase dietary glucose and carbohydrate.

(3) Dietary proteins lgm/kg BW to correct hypoalbuminemia, but should be cut short if hepatic encephalopathy appears.

(4) Oral plenty of vitamins B.

(5) Diuretic (lasix, 1 ampule /50kg)

(6) Plasma transfusion & dextran 6% IV to increase plasma osmotic pressure.

(7) Abdominal paraceutesis to relieve dysnea & pressure symptoms.

Treatment o f hepatic encephalopathy by:

(1) Restriction of protein in diet to control blood ammonia.

(2) Excess carbohydrate in diet to prevent breakdown of proteins and increase liver glycogen.

(3) Potassium chloride in diet for hypokalemia .

(4) Vitamins in diet specially B complex .

(5) Antibiotics to sterile the colon and inhibit ammonia production organisms (Neomycin 4 gm daily orally for 70 kg BW).

(6) Enema or purgative daily to remove ammonia from the colon.

(7) Sedative as chloral hydrate (excreted in urine, paraldehyde (excreted in breath), chloropromazine but morphine is contraindicated.

Symptomatic treatment o f viral hepatitis by:

(1) Diet rich in protein and carbohydrate, low in fat.

(2) Vitamin K & E.

(3) Corticosteroids in acute hepatic necrosis.