Digestive System

PART VI

1

Abomasal Ulcers of Cattle

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ETIOLOGY

I. Primary causes

- 1. The feeding of high levels of grain in feedlot cattle. 2. Long Transportation. 3. Prolonged Surgical Procedures.

II. Secondary causes

- 1. Left and Right-side abomasal displacements. 2. Abomasal impaction. 3. Abomasal volvulus. 4. Vagus indigestion. 5. Lymphomatosis.

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- Outcomes of Abomasal Ulcers
- 1. Abomasal hemorrhage with indigestion. 2. Melena. 3. Abomasal perforation resulting in a painful acute local peritonitis.
- Classification of Abomasal Ulcers
- Type1:- Non-perforating ulcer. Type 2:- Ulcer causing severe blood loss. Type 3:- Perforating ulcer with acute local peritonitis. Type 4:- Perforating ulcer with diffuse peritonitis.

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CLINICAL SIGNS

- In the common clinical form of bleeding abomasal ulcers, there are a sudden onset of anorexia, mild abdominal pain, tachycardia, severely depressed milk production and melena.
- The important clinical findings of hemorrhagic abomasal ulcers in cattle are abdominal pain, melena and pale mucous membranes.
- Melena is the almost pathognomonic sign of acute bleeding ulcer of abomasum.
- D. D. Haematemesis Abomasal Torsion Intestinal Obstruction Acute and Chronic Diffuse Peritonitis

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Treatment

- The conservative medical approach :
- 1. Blood Transfusions and fluid therapy 2. Coagulants 3. Antacids: - The goal of antacid is to create a favorable environment to healing of ulcers and this can be done:
- Either by decreasing acid secretion of histamine type-2 receptor antagonists. These compounds increase the gastric pH through selective and competitive antagonism of histamine at the H2-receptor on basolateral membrane of parietal cells and characterized pharmacologically by their ability to inhibit gastric acid secretion and kinetically by their similarity in absorption, distribution and elimination. These drugs given orally or parenterally such as Cimetidine, Ranitidine and Omeprazole. Or, Neutralizing secreted acid by oral administration of Magnesium hydroxide, Aluminum hydroxide and Kaolin & pectin.
- 4. Surgical Excision.
- **Prevention**: - Recommendations for prevention, of abomasal ulceration in cattle, cannot be given because the etiology is so poorly understood.

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Equine Gastric Ulcer Syndrome (EGUS)

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Equine Gastric Ulcer Syndrome (EGUS)

- Equine Gastric Ulcer Syndrome (EGUS), describes a host of complex inflammatory and disruptive events that occur in the mucosa of the oesophagus, stomach and duodenum of both adult horses and foals.
- The prevalence of EGUS is estimated to range from 40-93% in performance horses and 25-51% of foals.
- The anatomy of the horse is quite unique and this may explain why horses, especially exercising horses, are prone to gastric problems. The horse is a mono-gastric, meaning it has one stomach (unlike ruminants). The equine stomach is made up of two different regions, the region on the top is called the squamous region or non-glandular and the region on the bottom is the glandular region, which is more akin to the human stomach. The stomach is continuously secreting acid to digest food however, nowadays most performance horses are not continuously grazing and therefore their stomach may not contain enough feed at all times to neutralize this acid. The bottom part of the stomach is protected from the acid by glands but the upper part is more vulnerable. The area that divides these two regions is called the margo plicatus and this is where a lot of ulceration occurs due to acid splashing up on to the non-glandular region.

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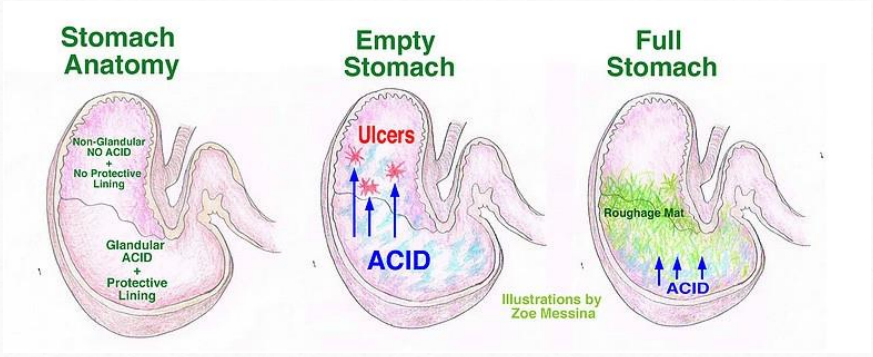
Anatomy of the horse stomach



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Risk factors

- There are multiple risk factors for developing EGUS and unfortunately most of them are due to man-made interventions where we have taken the horse from its natural habitat as a trickle-feeder with continuous access to forage. These risk factors include confinement such as stabling, reduced pasture availability, reduced hay/roughage availability and increased grain feeding.
- Physical stressors such as intensive exercise, behavioural and/or physical problems as well as NSAID (non-steroidal anti-inflammatory) use can also contribute to the high incidence of gastric ulceration.

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Clinicopathological Findings

- Ulcers are associated with decreased acidity and reduced blood flow to the gut mucosa. This in turn can be damaging to the gut mucosa, creating an inflammatory environment. This hostile environment can then disrupt the gut microbiome, which can also create further inflammation and damage to the mucosa.
- **Clinical symptoms** of EGUS involve reduced appetite, poor body condition, mild weight loss, poor performance, colic or more severe complications of duodenal structures. In the **foal** symptoms can include frequently lying on the back, reduced suckling, grinding of the teeth, excessive salivation and diarrhoea
- **Diagnosis**: Clinical signs, Gastroscopy

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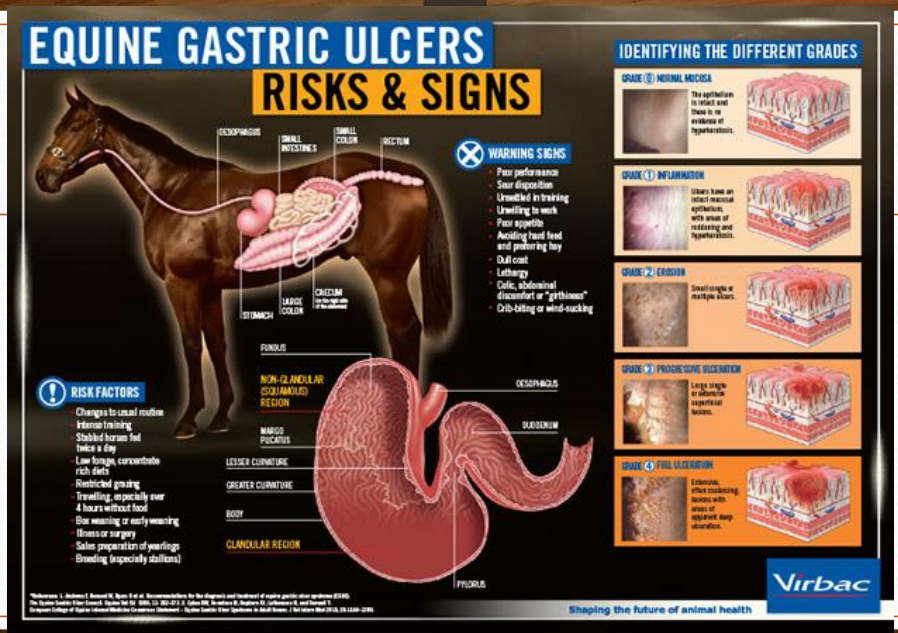

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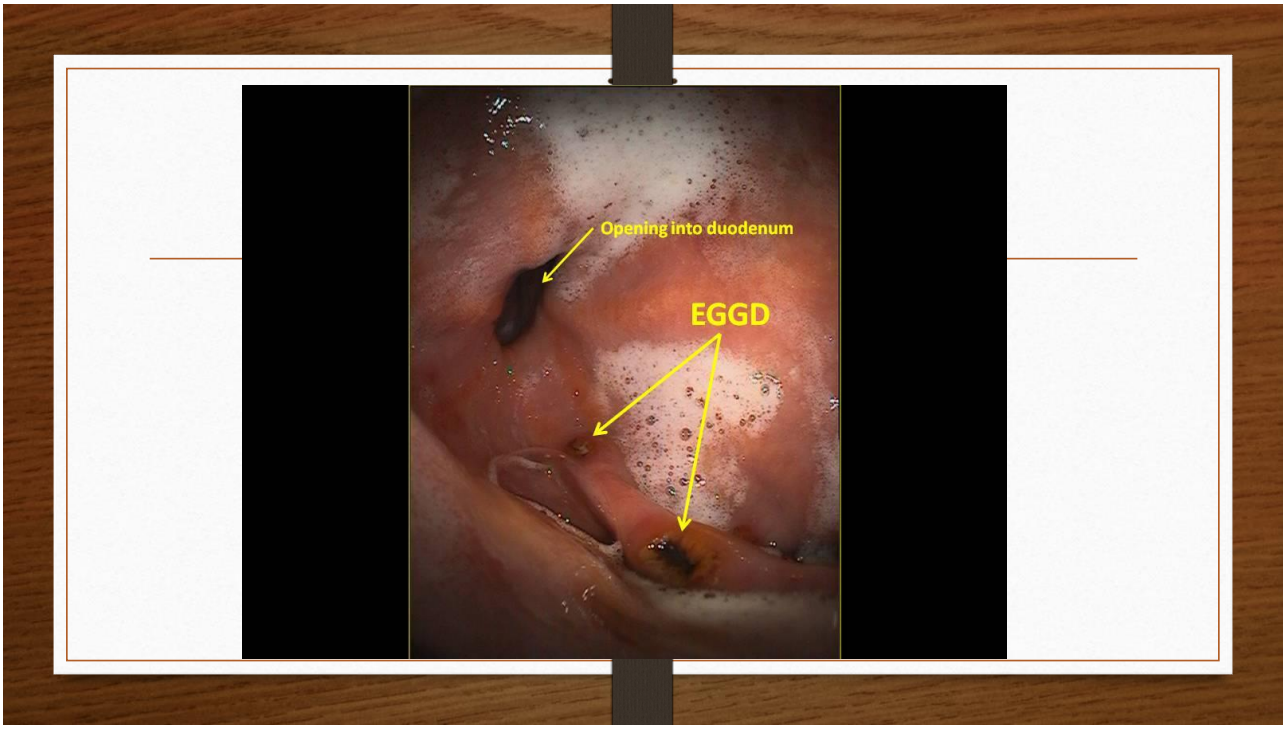

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Treatment

- Treating the horse with ulcers can be difficult and involves eliminating clinical signs, promoting healing and preventing reoccurrence.
- This process requires reducing fasting periods, increasing roughage availability and limiting or reducing the amount of grain fed. Taking measures to reduce the stress of the animal is also very important.
- Medical treatments use pharmaceuticals to control gastric acids, provide mucosal protectant and improve blood flow to the mucosa.
- Thus, an alternative is to adjust the nutritional status of the horse by providing less expensive, natural alternatives that could be fed with daily rations for long-term use to prevent gastric ulcers from reoccurring and to reduce discomfort in the horse.
- Several ingredients including fruit products, lecithin, sodium bicarbonate, calcium bicarbonate, omega fatty acids, natural anti-oxidants, and prebiotics or probiotics have been used in nutraceutical supplements. These nutrients have been shown to have many benefits both in the horse and other species, such as, supporting and protecting the stomach lining, supporting the normal digestive function, promoting a balanced gut microbiome, and protecting the stomach from the generation of damaging oxygen free radicals and inflammation of the mucosa.

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Prevention

- Have hay readily available at all times in the stable box
- Use a slow feeder
- Feed some chaff to the horse just prior to work so that the stomach is not empty
- Allow access to grazing every day

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Gastritis in small animals

Acute and Chronic

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Gastritis

- **Introduction**: The normal gastric epithelium serves as a barrier to the back-diffusion of acid and is quickly repaired by restitution after injury. In gastritis it is inflamed.
- **Pathophysiology**: antigens, chemical injury, ischemia, infection activate inflammatory response and increase acid secretion and disrupt the mucosal barrier. The continued interplay between ischemia and inflammation results in gastric erosion, ulceration, hypoxia, hemorrhage, edema, and necrosis
- **Etiology**: food allergy, food and drug poisoning, chemical intoxication, foreign body, infectious agents (Hleicobacter Pylori), Parasites (Physaloptera)
- **Epidemiology**: more common in dogs than cats

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Clinical signs

- Acute Gastritis:

1. Repeated Vomiting
2. Decreased appetite
3. Dehydration and electrolyte imbalance can occur in severe and consistent untreated cases

- Chronic Gastritis:

1. Intermittent vomiting

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Diagnosis

- Clinical signs
- History: drugs, chemical materials, etc….
- Radiology: chronic cases: thickening of the stomach wall
- Gastroscopy
- Histopathology
- Response to treatment/removing the cause
- Determination of the causative agent might be very difficult

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Treatment

- Removing and treating the causative agent (esp. in chronic cases)
- Withholding food in acute cases for 12-24 hours
- Using boiled and cooled water in small quantities
- Gradual returning to normal food in seven days starting by small quantities of soft food. Avoid giving oil and high protein content at the beginning.
- Administration of antiemetic agents such as metoclopramide 0.5 mg/kg or Ondansetron PO or IV
- Antiacid agents: Ranitidine, Famotidine, Cimetidin, omeprazole
- Antibacterial agents: Metronidazole, Amoxicillin, Azithromycin, …
- Fluid therapy (SC or IV according to the case severity)

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Diseases of Liver & Pancreas

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Major Hepatic Functions

- The major hepatic functions that, when disordered, are responsible for clinical signs include:

1. Maintenance of normal blood glucose levels by providing the source as glycogen.
2. Formation of some plasma proteins.
3. Formation and excretion of bile salts and excretion of bile pigments.
4. Detoxification and excretion of many toxic substances, including photodynamic agents.

- The primary diseases of liver are seldom in occurrence in farm animals except as a result of poisoning.
- The secondary diseases of liver are arising as part of generalized disease or by spreading from another.
- In primary hepatic disease, the clinical manifestations are caused solely by lesions in liver.
- In secondary hepatic disease, the syndrome may include clinical signs unrelated to hepatic lesions.

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Major Hepatic Dysfunctions

1. Diffuse and Focal Hepatic diseases :- The diffuse diseases of liver are commonly accompanied by signs of insufficiency than focal diseases that produce their effects either by toxins formed in the lesions or by pressure on other organs such as the biliary system. The diffuse diseases can be classified as hepatitis and hepatosis. Clinically the differences between these two diseases are not marked, although some assistance can be obtained from clinicopathological examination
2. Hepatic Dysfunction: - The liver has several important functions, so any diffuse disease for it will interfere with most or all functions to the same degree and the variations occur in the acuteness and severity of the damage but the effects are the same and the clinical manifestations vary in degree only.

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Manifestations of Liver & Biliary Disease

Jaundice Photosensitization Abdominal Pain Hemorrhagic Diathesis Hepatic Encephalopathy Diarrhea & Constipation Edema & Emaciation Alteration in size of Liver Liver Displacement Liver Rupture Black Livers in Sheep.

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Jaundice

- Jaundice is a clinical sign that often arises in diseases of liver and biliary system but, also, in some diseases that don't have lesions in these organs. It does not always occur & may prominently be absent in acute hepatitis.
- The staining is much more pronounced with conjugated (Direct) bilirubin than unconjugated (Indirect) bilirubin. Thus, the jaundice is more sever in cases of obstructive and hepatocellular jaundice than in hemolytic jaundice.
- It's best detected clinically in sclera & may be detectable easily at necropsy.
- Some indications of the type of jaundice can be derived from clinical examination. Thus, jaundice is usually much more severe when impairment of flow occurs and when bile pigments are absent from feces.
- The only accurate basis for differentiation between jaundice with impaired bile flow and jaundice without impaired flow is the examination of urine for presence of bilirubin and urobilinogen and determination of the relative amounts of conjugated and unconjugated bilirubin present in serum.
- Unconjugated (indirect) bilirubin that has not passed through hepatic cells is not excreted by kidney.

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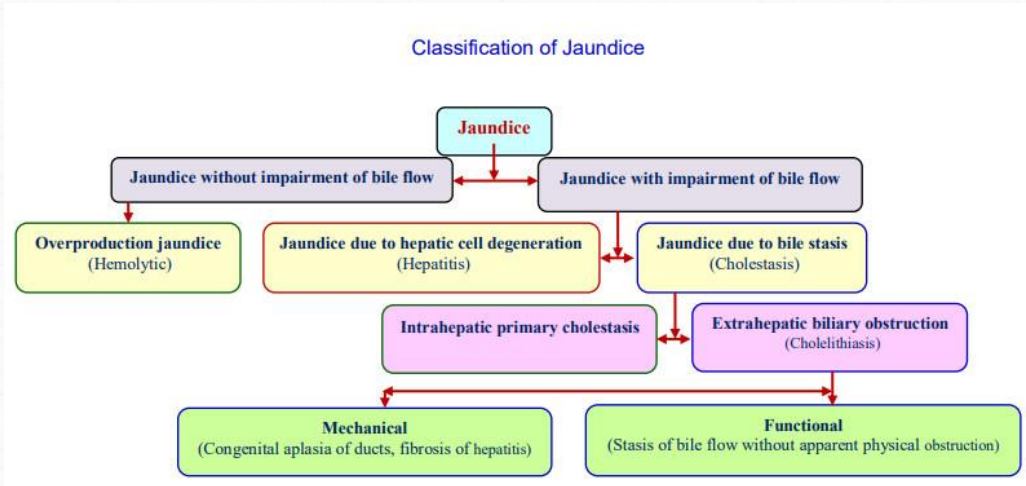

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Hemolytic Jaundice

- It's common in animals & may associate with bacterial toxins, invasion of erythrocytes by protozoa, rickettsia or viruses, inorganic and organic poisons and immunological reactions:
- Bacterial toxins that cause intravascular hemolysis include bacillary hemoglobinuria and leptospirosis.
- The common protozoan & viral diseases include babesiosis, anaplasmosis, and equine infectious anemia.
- Other common causes are chronic copper poisoning, selenium poisoning in sheep, phenothiazine poisoning in horses, pasturing on rape & other cruciferous plants and bites by some snakes.
- Postparturient hemoglobinuria has uncertain etiology but is usually attributed to a deficiency of phosphorus in diet and feeding of cruciferous plants.

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Hemolytic Jaundice

- Isoimmunization hemolytic anemia of newborn is caused by an immunological reaction between the sensitized cells of newborn and antibodies in colostrum of dam. The occurrence of acute hemolytic anemia & jaundice in calves that drink large quantities of cold water may be of the nature of an immunological response.
- The neonatal jaundice is, relatively, common in babies and is regarded as a benign condition. Although it is generally stated that the jaundice is hemolytic and results from destruction of excess erythrocytes when postnatal life begins, it appears more probable that it is due to retention of bile pigments because of immaturity of hepatic excretion mechanism.

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Hemolytic Jaundice

- Hemolytic jaundice is characterized
- Clinically by:- 1. A moderate degree of yellowing of mucosa. 2. Presence of hemoglobinuria in severe cases.
- Clinicopathologically by:- 1. Anemia. 2. Increase in urobilinogen. 3. Absence of bilirubin in urine and a preponderance of indirect bilirubin in serum.

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Extrahepatic Biliary Obstruction

- The obstruction of bile ducts or common bile duct may occur by:
- 1. Biliary calculi.
- 2. Compression by tumor masses that rarely in occurrence in farm animals.
- 3. Inflammation of bile ducts by extension from enteritis.
- 4. Infestation with Nematodes or Trematodes.

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Hepatitis

- **I. Toxic Hepatitis**
- 1. Inorganic poisons (copper, phosphorus, arsenic, possibly selenium). 2. Organic poisons (carbon tetrachloride, hexachloroethane, Gossypol, creosols & coal tar pitch, chloroform & copper diethylamine quinoline sulfonate. 3. Poisonous plants. 4. Miscellaneous farm chemicals.
- **II. Infectious Hepatitis**
- 1. Rift valley fever. 2. Bacillus piliformis, associated with Tyzzer's disease in foals. 3. Viral rhinopneumonitis as a cause of abortion in horses. 4. Postvaccinal hepatitis of horses such as tetanus antitoxin.

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Hepatitis

- **III. Parasitic Hepatitis**
- 1. Acute and chronic liver fluke infestation. 2. Migrating larvae of Ascaris sp. 3. Fibrosing granulomas of liver in horses with chronic shistosomiasis. 4. Hepatic sarcocystosis in a horse.
- **IV. Nutritional Hepatitis** (Trophopathic Hepatitis) 1. White liver disease in sheep.

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- **Focal Diseases of Liver**
- 1. Hepatic abscess. 2. Telangiectasis of bovine liver (Sawdust liver). 3. Tumor.
- **Diseases of Pancreas** 1. Diabetes mellitus (characterized by polyuria, polyphagia, and polydipsia in dogs and less frequently cats, uncommon in farm animals). 2. Pancreatic adenocarcinoma. 3. Pancreatic adenoma. 4. Pancreatitis (severe vomiting unresponsive to treatment).

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Special Examinations of liver

- Palpation & percussion
- Biopsy
- Medical imaging of liver (ultrasonography & radiography)
- Laboratory tests for hepatic disease and function (Albumin, total protein, bilirubin, prothrombin)
- Liver enzymes: ALT, AST, ALP

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Principles of treatment of liver diseases

- 1) In diffuse diseases, no convincing treatment. The main aim should be to remove the source of damaging agent.
- 2) In acute hepatitis, the level of blood glucose must be maintained by oral or intravenous injections of glucose. Death may occur during this stage due to hypoglycemia.
- 3) There is some doubt as to whether protein intake should be maintained because of the high level of protein during liver dysfunctions may metabolized, incompletely, and this resulting in toxic effects particularly in kidney.
- 4) Diets that are high in carbohydrate, calcium and protein are of high biological value and a number of specific substances are known to have a protective effect against hepatotoxic agents.
- 5) In chronic diffuse hepatic disease, the high-protein diet causes stimulation of metabolic activity of liver and increase the deposition of fat that may cause further retarding in hepatic function.
- 6) Local diseases of liver may require a surgical or medical treatment depending upon the causes & specific treatments.

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