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DIETARY AND THERAPEUTIC MANAGEMENT OF DOGS WITH LIVER DISEASE

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Diets for animals with liver disease are best formulated on an individual basis, with consideration given to the type and origin of the liver disease and the extent of liver dysfunction. Care must be taken to avoid overwhelming the remaining metabolic capacities of the diseased liver. The diet must be highly palatable and provide adequate energy, protein, fat, and all essential micronutrients. Impaired dietary intake,.malabsorption associated with severe cholestasis or portal hypertension, and catabolism all contribute to protein-calorie malnutrition, resulting in loss of muscle mass and hypoalbuminemia. Negative protein and energy balance promote hepatic encephalopathy (HE), reduce immune response and increase mortality.

The Aim of Dietary Management of Liver Disease Are:

(1) To supply adequate energy and nutrients to fulfil basic requirements and prevent malnutrition

(2) To limit further liver damage by preventing accumulation of copper and free radicals

(3) To support hepatocellular regeneration

(4) To prevent or minimize metabolic complications, such as hepatic encephalopathy and ascites

Energy

An adequate supply of both energy and protein is essential to prevent weight loss. The use of non-protein calories is important to prevent the use of amino acids for energy and reduce the need for gluconeogenesis. The diet should have a high energy density, since dogs with liver disease usually have reduced appetite. Normally, energy is best supplied in the form of fat since it is a highly palatable and concentrated source of energy. Dogs with liver disease can tolerate larger quantities of fat in the diet (30 - 50% of calories) than previously assumed. Fat restriction should only be considered in the few cases with severe cholestatic liver disease and suspected fat malabsorption, although adequate essential fatty acids must be provided. Boiled white rice is useful because of its high digestibility. Soluble fibers are useful in dogs with cirrhosis and a tendency to hyperglycemia, because they smoothen the postprandial glycemic response and prolong glucose delivery to the liver.

Protein

Incorrect protein restriction in dogs with liver disease causes catabolism of endogenous proteins and loss of muscle mass, both of which increase the potential for HE. In dogs, protein should represent as a minimum 10 to 14% of dietary calories, preferably at least 20%, and most dogs can tolerate higher quantities. The aim is to gradually increase the amount of protein in the diet, keeping the protein intake as close to normal as can be tolerated without precipitating signs of HE .The quality and source of the protein are important. Soy isolates, wheat gluten and dairy products are better tolerated than meat proteins in dogs. The potential benefit of vegetable proteins is attributed to their high fiber content, which causes a decrease in transit time and promotes incorporation and excretion of nitrogen in faecal bacteria, whereas the effect of dairy products is likely due to the influence of lactose on intestinal transit and pH.

Fiber

Colonic fermentation of soluble fibers such as fructo-oligosaccharides, beet pulp and gums lowers the intraluminal pH and thus reduces the production and absorption of ammonia, the effect of which is similar to that of lactulose. Colonic fermentation also favors the growth of acidophilic bacteria that produce less ammonia and promote incorporation and excretion of ammonia in faecal bacteria (e.g., Lactobacillus spp). Fiber (both soluble and insoluble) binds bile acids in the intestinal lumen and promotes their excretion. Insoluble fibers (lignin, cellulose,

hemicellulose) act by normalizing transit time, whereas they can also prevent constipation and bind toxins.

Minerals

Diets for dogs with liver disease should be potassium replete. Anorectic dogs may need supplementation by either intravenous administration of potassium chloride (10 - 40 mEq/500 ml fluids, depending on serum potassium) or oral potassium gluconate (0.5 mEq/kg once or twice daily). Potassium citrate should be avoided because of its alkanizing properties, since alkalosis can aggravate HE. Zinc benefits the urea cycle and central nervous system neurotransmission, has clear hepatoprotective effects against a variety of hepatotoxic agents, and has antioxidant functions. Zinc supplementation may reduce lipid peroxidation, has antifibrotic properties, prevents hepatic copper accumulation, and can reduce the severity of hepatic encephalopathy.

Vitamins

B Vitamins are often empirically supplemented at double maintenance dose. The diet should contain adequate levels of vitamin C in order to compensate for failing hepatic synthesis and to take advantage of the antioxidant properties of vitamin C.(lemon juice, orange juice). Mega doses of vitamin C should be avoided in dogs with copper storage hepatotoxicity, since it can function as a pro-oxidant in the presence of high concentrations of heavy metals .Vitamin E is an important endogenous free radical scavenger that protects against oxidative injury. Supplementation with vitamin E (400 - 600 IU/day) (ECARE SE, ADCELIN) is especially indicated in cholestatic and copper-associated liver disease, but is likely also important in other forms of chronic liver disease. Vitamin K deficiency is mostly relevant in cholestatic disorders, although it may also become depleted in severe chronic liver disease. Coagulopathies secondary to vitamin K deficiency should be treated with two or three doses of vitamin K1 (0.5 - 1.0 mg/kg subcutaneously every 12 hours). The same dose can be given biweekly or monthly in chronic disorders in which continued repletion of vitamin K is required.

Acute Liver Diseases

Acute liver disease is most commonly caused by toxic injury, and less frequently by infection (e.g., infectious canine hepatitis, sepsis), trauma, heat stroke, or vascular compromise. The spectrum of disease can range widely, and signs vary from mild to fulminant hepatic failure. Vomiting and diarrhoea are common, whereas HE, melena, hematochezia, and disseminated intravascular coagulation (DIC) may occur in acute liver failure.

1) Fluid therapy with a balanced electrolyte solution is necessary for initial stabilization. Potassium and glucose should be supplemented as appropriate, and correction may reduce the severity of HE.
2) Vomiting may be controlled by anti-emetics (metoclopramide, 0.2 - 0.5 mg/kg q 6 - 8h IV, IM, PO).
3) Gastroprotectants (ranitidine 2 mg/kg q 8 - 12h IV, PO) are indicated in dogs with bloody vomiting and/or diarrhoea.
4) Treatment of HE is needed using lactulose and oral antibiotics.

Dogs with acute liver disease are typically hypercatabolic and need prompt nutritional intervention in order to prevent debilitating malnutrition. The liver has tremendous regenerative capacities, but this is dependent on the availability of sufficient nutrients. Enteral nutrition via tube feeding of frequent meals (3 - 6 hrs) should be instituted when the dog remains anorexic after 48 to 72 hours, provided there is no intractable vomiting. Tube feeding is usually first started using a naso-esophageal tube; esophagostomy or gastrostomy tubes may have to be used at a later stage when the dog remains anorexic.

Oral feeding should be started gradually with small frequent meals in order not to overload the liver's metabolic capacity. Half of the daily requirements should be fed initially, and this should be increased by 10% every day dependent upon the dog's response. The diet should contain normal amounts of protein (20%) if at all possible, since a positive nitrogen balance is essential for hepatic regeneration. Moderate protein restriction may be

necessary in patients with persistent HE. However, in acute liver disease it is especially important not to over-restrict dietary protein, since this could result in endogenous ammonia production from protein catabolism as well as a reduction in the availability of protein for hepatocellular repair. The diet should also include free radical scavengers and antioxidants, such as vitamin E and vitamin C. Ursodesoxycholic acid (10 - 15 mg/kg PO q24h) can be given as a hepatoprotectant in the sub acute stage when serum bile acids remain high.

Chronic Liver Disease

Chronic hepatitis includes a diverse group of disorders characterized by mixed inflammatory cell infiltrates, in which lymphocytes and plasma cells predominate. The etiology is often never determined. Documented causes include abnormal hepatic copper accumulation and drug- or toxin-induced hepatic injury (anticonvulsants).

Every effort should be made to get the dog to eat voluntarily. Food should be palatable, at room temperature and be fed in small portions 3 to 6 times daily. Dogs that refuse to eat or consume insufficient amounts to meet minimum requirements may require tube feeding, usually initially via a nasogastric tube, in order to halt the vicious cycle of excessive muscle catabolism and worsening signs of liver dysfunction. If the dog remains anorexic, esophagostomy or gastrostomy tubes may have to be inserted in order to ensure continuing nutritional adequacy.

Dietary protein should ideally represent 17 - 20% of metabolic energy, be highly digestible and of high biological value. Protein restriction should only be instituted when there are signs of HE. Protein utilisation can be increased by administering lactulose (0.5 ml/kg orally three times daily), which may be combined with oral antibiotics (metronidazole 7.5 mg/kg q 12hr, or ampicillin 20 mg/kg q 8hr). Increasing dietary levels of vegetable, soy or dairy protein may also help to reduce the likelihood of HE. Assessment of the protein-calorie adequacy of the diet is generally based upon weekly monitoring of body weight and serum albumin concentrations. Progressive hypoalbuminemia (in absence of proteinuria) is indicative of protein malnutrition and/ or progressive liver disease. The diet should contain both soluble fiber, in order to promote an acidic colonic pH and decrease NH4+ absorption, and insoluble fiber, which helps to normalize transit time, prevent constipation and bind toxins. It is essential that the diet contains increased zinc levels and a mixture of antioxidants including vitamins E and C. Additional oral zinc supplementation (zinc acetate 2 mg/kg daily) may be helpful because it is an antioxidant that also has antifibrotic properties and can reduce the severity of HE.

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