Nutritional Considerations for Dog and Cat Suffering from Hepato-biliary Diseases - Review

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Abstract
Diseases of liver and biliary system are common in dog and cat. As liver plays very crucial role in digestion and metabolism of nutrients, dietary modifications are required in cases of liver dysfunction in addition to therapeutic approach. Dietary protein level and its amino acid composition plays significant role in the management of liver diseases. Levels of dietary energy, water soluble vitamins, Vitamin E and Vitamin K should be increased above recommended levels. Vitamin A, Vitamin D, copper and iron should not be fed in excess amount. Zn supplementation is helpful in recovery of damaged hepatic tissue as well as to counter the copper toxicity by reducing its absorption from intestine.

Keywords: Hepato-biliary diseases, Nutrition, Dog, Cats.

1. Introduction
Liver is the largest secretary organ in the body and its functions are also matching to its size. It is responsible for metabolism of fats, carbohydrates and proteins as well as storage of vitamins, minerals, glycogen and triglycerides. It also plays an important role in maintaining homeostasis and produces factors important for the normal clotting of blood. Since the liver is an essential part of the immune system, liver disease may often lead to a serious immune deficiency syndrome. Liver also plays an important role in digesting nutrients and detoxifying chemicals and drugs taken into the body. When the liver is not functioning properly, toxins will build up, digestion will be affected, and there may be a shortage of essential nutrients, including glucose, vitamins and minerals. Since the liver has such important functions in the body, the development of liver disease may result in serious health consequences.

Liver diseases can be divided into acute and chronic, in which former being common. Causes of acute liver disease include toxins such as acetaminophen, tetracycline and sulfa drugs, hepatic lipodosis, trauma, heatstroke and infections (canine infectious hepatitis, feline cholangiohepatitis). Causes of chronic liver disease include genetics, infections (canine infectious hepatitis, leptospirosis), toxins (anticonvulsants) and idiopathic hepatitis.

2. Altered Nutrient Metabolism and Its Dietary Management in Hepato-biliary Diseases

2.1 Protein and Amino acids
Albumin, a most abundant plasma protein contributing 55 to 60% of the total plasma protein pool performs wide range of functions in body. Functions of albumin include binding and carrier protein for hormones, amino acids, steroids, vitamins, calcium and fatty acids as well as exogenous compounds, drugs, toxins, etc. and maintenance of plasma osmotic pressure. Some glycoproteins synthesized in liver are involved in hemostasis, protease inhibition, transport and ligand binding. So in liver diseases there are incidences of hypoalbuminemia and increased bleeding tendencies as a result of reduced protein synthesis in liver (Center, 1996a). Hyperammonaemia occurs because of impaired urea formation, inadequate delivery of ammonia to liver because of portosystemic vascular shunting and increased ammonia production due to amino acid deamination and gluconeogenesis (Meyer, 1998a). Liver dysfunction results in reduced level of branched chain amino acids (BCAA) and increased level of aromatic amino acids (AAA) in blood (Center, 1996a; Strombeck and Rogers, 1978) and this picture also reflects in cerebrospinal fluid with greater magnitude as both BCAAs and AAAs share common transporter to cross blood brain barrier. This -
changed blood amino acid balance may also play a role in the pathogenesis of hepatic encephalopathy (HE) (Fischer et al., 1975; Meyer, 1998b). This may be due to formation of false neurotransmitter using AAAs as substrate. HE is a disease in which CNS gets exposed to the encephalographic material (mostly ammonia) from food, digestive or metabolic processes because of inability of liver to neutralize them. In liver diseases, body protein breakdown increases to supply amino acids for various body processes. Provide usual or increased protein through diet but of high biological value to avoid load on liver.

Protein malnutrition may lead to hepatic lipidosis because of shortage of lipoproteins involved in lipid transport and metabolism (Biourge et al., 1994). Amino acids like taurine and arginine have significant role in hepatic lipidosis of cat (Meyer et al., 2010). In case of animals exhibiting protein intolerance such as in HE, protein level can be reduced below normal requirements i.e. 15 to 20% of dry matter for dogs and 25 to 30% of dry matter for cats. Diet should not contain less than 3.3-3.5 g/kg body weight of high biologic value protein in a diet providing 70-80 kcal/Kg/day for cats and 2.0-2.2 g/kg body weight of it in a diet providing 70-110 kcal/Kg/day for dogs in case of intolerance (Centre, 1998). Protein intake must not be reduced below this level because the cat is unable to conserve nitrogen efficiently after protein restriction (Rogers et al., 1977). In animals with HE source of protein is also important. Vegetable (soya protein) and dairy protein sources are found to be effective in controlling HE instead of complete protein restriction (Thompson et al., 1986; Laflamme et al., 1993; Proia et al., 1984), but to avoid feeding meat in this case is hard in practice for anorectic cats. Vegetable and milk protein have less ammonigenic amino acids such as threonine, glycine, serine, histidine, tryptophan, and lysine. In case of soya proteins taurine supplementation is required especially for cats at a level of 1 g taurine/kg soya-based diet. This is because soya protein is deficient in methionine and prevents reabsorption of bile salts (taurocholates) by forming hydrophobic peptides that bind with it (Kim et al., 1995). Taurine appears to aid choleresis in dogs and possibly in cats; its level should be maintained as 0.10 and 0.17% of dry matter in case of dry expanded and moist foods respectively, for healthy adult cats (NRC, 2006). Higher (0.3% for cat and 0.1% of dry matter for dog) taurine levels are beneficial in liver diseases. When casein is used as protein supplement diet should be supplied with arginine which is limiting in case of it. Arginine deficiency can cause hyperammonemia and encephalopathic signs (Morris, 1985). Arginine level should be higher than normal allowance (≥0.77% of dry matter) in case of cat food containing more than 20% protein (NRC, 2006), i.e. 1.5 to 2.0% of dry matter (Meyer et al., 2010). Arginine and citrulline are amino acids required for conversion of ammonia to urea and therefore can lessen ammonia’s toxic effects on brain function. Meat and dairy proteins are also sources of L-carnitine which is essential for fat catabolism in liver to avoid lipidosis of hepatic tissue (Brehmer, 1983; Goa and Brogden, 1987). Methionine and lysine are the most limiting factors for its synthesis. L-Carnitine is helpful when given to cats in fatty liver condition at a dose of 250 mg/day along with a diet containing adequate energy and protein (Centre, 1998).

### 2.2 Carbohydrates

Hepatogenic hypoglycemia can occur in dogs with cirrhosis, congenital portosystemic vascular anomalies, fulminating hepatic failure, septicemia and extensive hepatic neoplasia (Center, 1996a). Complex carbohydrates called as fibers are important in liver diseases. Soluble fiber from pectin, gums has property of modifying intestinal fermentation and lowering enteric ammonia production by favoring acidophilic organisms (e.g. *Lactobacillus*) with lowered number of ammoniogenic organisms (Centre, 1998). Insoluble fiber causes loss of chenodeoxycholate through faeces and enhances synthesis of less toxic trihydroxy bile acids (Centre, 1998). Fiber degrading bacteria (especially pectin degraders) in this process decrease enteric ammonia production and improve fecal nitrogen elimination (Herrmann et al., 1987). Citrus pectin can be used as a source of soluble fiber and coarse bran
from bran cereals of insoluble fiber for dogs (Tweedt, 1993; Willard, 1988).

2.3 Fat
Dysfunctions of liver can lead to changes in lipoprotein and cholesterol synthesis, lecithin-cholesterol acyltransferase deficiency, defective lipolysis, abnormal recognition and uptake of lipoproteins by the liver and regurgitation of biliary lipids into plasma. Obstruction of biliary tract may lead to hypercholesterolemia and hypertriglyceridemia (Center, 1996b). No special attention is required for this nutrient unless in case of fatty liver. Cats with fatty liver can do well on diets containing up to 20 to 25% fat. Free fatty acids in blood on the other hand interfere with ammonia metabolism and augment hyperammonemia. They also contribute directly to hepatic encephalopathy by forming octanoic acid on their incomplete oxidation in liver. But dietary fat is resynthesized in enterocytes to triglycerides and incorporated into chylomicrons before leaving intestinal epithelial cells. Thus, dietary fat does not increase plasma free fatty acids but they are increased during fasting when fatty tissues are catabolized for energy (Rutgers et al., 1994).

2.4 Vitamins
As liver plays major role in absorption and storage of some vitamins, liver dysfunction may affect availability and reservoir pool of them. Subnormal Vitamin B₁₂ level was reported in cats having liver disease (Meyer et al., 2010). Provide all water soluble vitamins twice the amount actually required for normal dog. As niacin is not readily synthesized by cats, dietary niacin deficiency could impair L-carnitine synthesis in them, so its level has to be monitored in dietary niacin deficiency could impair L-carnitine synthesis in them, so its level has to be monitored diet. In case of copper or iron storage disease, extra Vitamin C can worsen injury caused by reactive oxygen species ROS (Sokol and Hoffenberg, 1996). Absorption of all fat soluble vitamins hampered in liver diseases affecting enterohepatic circulation, but more severe in case of Vitamin E and Vitamin K (Centre, 1998). Vitamin E is a free radical scavenger so its need is higher in liver injury. Food should contain minimum 1.63 and 1.0 mg/kg dry matter of Vitamin K (Menadione) for dog and cat respectively (NRC, 2006). Vitamin E should be supplemented at 20 to 100 IU/day orally to cats suffering from liver diseases (Centre, 1998). Excess of Vitamin A and Vitamin D should be avoided.

2.5 Minerals
Liver acts as storage organ for some trace minerals such as copper, manganese and selenium. Some dog breeds have tendency to accumulate high copper in liver and this condition is more severe than copper toxicosis observed in cholestatic diseases (Spee et al., 2006). Free copper can directly damage hepatocyte mitochondria resulting in electron leak with free radical formation leading to lipid membrane peroxidation (Sokol et al., 1989). Iron also accumulates in liver in some diseases as chronic hepatitis/cirrhosis and congenital portosystemic shunting (Schultheiss et al., 2002; Simpson et al., 1997). Iron damages Kupffer cells and subsequently releases inflammatory cytokines which can harm hepatic tissue. Also as a transition metal like copper it can cause free radical injury to hepatocytes (Britton, 1996; Sokol and Hoffenberg, 1996). Zinc (Zn) is a component of glutamate dehydrogenase in muscle and ornithine transcarbamylase in the urea cycle both of which are important in nitrogen metabolism. Zn is also involved in tissue healing as an essential factor for DNA synthesis (Centre, 1998). Zn as a part of superoxide dismutase (SOD) also involved in free radical scavenging and cell membrane stabilization of the hepatocytes. So supplementation of Zn over normal required level i.e. 60 and 74 mg/kg dry matter for dogs and cats respectively (NRC, 2006) has beneficial effect in liver diseases. Foods for canine patients with copper-associated hepatotoxicosis should also contain more than 200 mg/kg dry matter zinc as it blocks copper absorption (Marks et al., 1994). Cu rich feeds such as shellfish, organ meats to the dog or cat with lives disease should be avoided. Hypokalemia and hypophosphatemia are found in cats with fatty liver and required to be corrected. If there is edema sodium intake should be restricted.

2.6 Energy
Provision of adequate dietary energy is one of the important aspects for dietary management of hepatic lipodisosis (Biourge et al., 1994). Generally energy requirements are increased in liver diseases due to increased protein turnover and thereby increased maintenance needs. In acute inflammatory condition energy need of patient is more than in case of chronic condition. Extent of increase in energy needs also depends on stress, level of physical activity, presence of anorexia and preexisting malnourishment etc. (Centre, 1998). Energy density required for cat suffering from hepatic lipodisosis and cholangitis is 18.4 KJ ME/g on dry matter basis. To achieve this energy density minimum 25% dietary fat is required. Dogs and cats suffering from chronic hepatitis, portal hypertension and portosystemic shunt and dogs with copper-associated hepatotoxicosis require 16.7 and 17.6 KJ ME/g on dry matter basis for dog and cat respectively (Meyer et al., 2010).
3. Conclusion

It is understood that altered dietary level of certain nutrients (increased or decreased protein, increased Vitamin E, Vitamin K, water soluble vitamins, decreased copper and iron) and energy level (increased) may be helpful in recovery of dog or cat suffering from hepato-biliary diseases. Feed should be given in small amounts and increased frequency in order to avoid load on liver, increase its assimilation efficiency and to mitigate HE problems. Feeding management is only supportive to therapeutic management which cannot be compromised.

References


