Review on Nutritional Management of Cardiac Disorders in Canines

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Abstract

A variety of nutritional deficiencies, dietary protein, fat, vitamins, minerals and trace elements are known to cause cardiac disease in various species. Nutritional management of dogs with cardiac problems can be handle with providing adequate calories, protein and modulating cytokine production to manage the possible cause of decreased appetite of the side effects of medications. Owners should be aware that dietary supplements are not regulated in the same way as drugs. They do not require proof of safety, efficacy, or quality control before they can be sold. Therefore, careful selection of type, dose, and brand is important to avoid toxicities or complete lack of efficacy.

Keywords: Nutrition, Management, Cardiac Disorder, Canine, Vitamin, Mineral, Fat, Protein.

Introduction

In addition to, optimal medications of dogs with cardiac disease, it also includes careful attention to the diet. Research is now beginning to show that dietary factors may be able to modulate canine cardiac disease, either by slowing the progression, minimizing the number of medications required, improving quality of life, or in rare cases, actually curing the disease.

Cardiac Cachexia

Dogs with CHF commonly demonstrate weight loss, termed cardiac cachexia. In a dog with injury or illness, including CHF, amino acids from muscle are the primary source of energy, resulting in loss of lean body mass. Loss of lean body mass is usually first noted in the epaxial, gluteal, scapular, or temporal muscles. Cardiac cachexia typically does not occur until CHF has developed.

Cardiac cachexia can occur with any underlying cause of CHF (e.g., DCM, CVD, congenital heart diseases) but most commonly occurs in dogs with DCM, particularly those with right-sided CHF.

While these factors play a role in the loss of lean body mass, a major factor in this syndrome is an increased production of inflammatory cytokines, such as tumor necrosis factor (TNF) and interleukin-1 (IL-1). These inflammatory cytokines are known to directly cause anorexia, to increase energy requirements, and to increase the catabolism of lean body mass. Of particular pertinence to cardiac disease, TNF and IL-1 also cause cardiac myocyte hypertrophy and fibrosis and have negative inotropic effects.

Nutritional management of dogs with cardiac cachexia consists primarily of providing adequate calories and protein and modulating cytokine production. One of the most important issues for managing anorexia is to maintain optimal medical control of CHF. Another possible cause of decreased appetite is the side effects of medications. Digoxin toxicity or azotemia secondary to ACE inhibitors or overzealous diuretic use can both cause anorexia.

Tips that may assist in food intake include feeding small, more frequent meals or warming the food to body temperature (or for some dogs, feeding refrigerated food increases appetite). Gradual introduction of a more palatable diet may be beneficial for some dogs (e.g., switching from a dry food to a canned food, changing to a different brand, or having a veterinary nutritionist formulate a balanced homemade diet). It also may be useful to use flavor enhancers to increase food intake (e.g., yogurt, maple syrup, or honey).

Modulation of cytokine production can also be beneficial for managing cardiac cachexia. One method of decreasing the production and effects of cytokines is with n-3 polyunsaturated fatty acid supplementation. Supplementation of fish oil, which is high in n-3 fatty acids, can decrease cytokine production in dogs with CHF and improve cachexia. A reduction of IL-1 has

been correlated with survival in dogs with CHF.

Preventing Nutrient Excesses

Veterinarians have extrapolated from the human literature since the 1960's in applying nutritional recommendations to dogs with cardiac disease.

Sodium and Chloride: A prime example is sodium restriction. Healthy dogs can easily excrete excess dietary sodium in the urine but, even before clinical signs become apparent in dogs with cardiac disease; there is activation of the renin-angiotensin-aldosterone (RAA) system and abnormal excretion of sodium.

Dogs with CHF respond differently to dietary sodium restriction. Sodium restriction is one method, along with the use of diuretics and venous vasodilators, to treat excessive increases in preload in patients with CHF. In the 1960's, when few medications were available for treating dogs with CHF, dietary sodium restriction was one of the few methods of reducing fluid accumulation. In this situation, severe sodium restriction clearly was beneficial in reducing signs of congestion.

Use low sodium foods to insert the pills before administration

• Fresh fruit (e.g., banana, orange, melon)

• Low sodium canned pet food
• Peanut butter (labeled as "no salt added")
• Home-cooked meat (without salt)

Chloride: Dietary chloride levels are often ignored but research suggests that chloride may be important in the optimal management of CHF. Chloride administration also appears to decrease plasma renin activity in salt depleted rats. The patient with heart failure has chronic activation of the RAA system, which could be significantly influenced by dietary chloride. In addition, furosemide is known to block chloride transport in the ascending loop of Henle, and hypochloremia (and hyponatremia) can develop in advanced CHF. Therefore, chloride is likely to play an important role in the CHF patient. Unfortunately, little is known about optimal dietary intake for CHF patients and additional research will be required to make specific recommendations.

Potassium: In the past, when digoxin and diuretics were the mainstays of therapy for people and dogs with CHF, hypokalemic was a major consideration. Now, ACE inhibitor therapy has gained widespread use in the management of dogs with CHF and this medication results in renal potassium sparing. Therefore, ACE inhibitors are known to cause

Table-1. Dietary Sodium Recommendations for Dogs Based on Stage of Disease

International Small Animal Cardiac Health Council Classification	Dietary Sodium Recommendations
1. Asymptomatic • Heart disease is detectable • Patient is not overtly affected • Does not demonstrate clinical signs of heart failure • Diagnostic findings could include a cardiac murmur, arrhythmia, or cardiac chamber enlargement that is detectable by radiography or echocardiography	• Severe sodium restriction is not required. Counsel the owner to avoid diets high in sodium (>100 mg/100 kcal) and to avoid treats and table foods that are high in sodium. • If the signs of volume and pressure compensation detected then
2. Mild to Moderate Heart Failure • Clinical signs of heart failure are evident at rest or with mild exercise - exercise intolerance, • Cough, • Tachypnea, • Mild respiratory distress (dyspnea), • Mild to moderate ascites.	• Sodium content of 50 – 80 mg/100 kcal in the main diet. Also counsel the owner to avoid treats and table foods that are high in sodium. • Sodium content of 50 – 80 mg/100 kcal in the main diet. Greater sodium restriction (<50 mg/100 kcal) is recommended if large diuretic doses are necessary to control clinical signs. • Limiting sodium intake from treats and table foods becomes more important. Counsel owner on appropriate methods for medication
3. Advanced Heart Failure • Respiratory distress (dyspnea) • Marked ascites, • Profound exercise intolerance, • Hypo perfusion at rest. • In the most severe cases, the patient is moribund and suffers from cardiogenic shock. • Death or severe debilitation is likely without therapy.	• Sodium content <50 mg/100 kcal in the main diet. Limiting sodium intake from treats and table foods is very important. Counsel owner on appropriate methods for medication administration. • Hospitalization is mandatory because cardiogenic shock, life-threatening pulmonary edema, refractory ascites, or a large pleural effusion is present. • Stabilization of acute CHF should be the goal. Diet changes should be avoided until the dog is home and stabilized on medications; a gradual change to a new diet can be instituted at that time.

increased serum potassium, with some animals developing hyperkalemia. Spironolactone, an aldosterone antagonist and a potassium-sparing diuretic is being used with greater frequency in veterinary patient after reports of improved survival in human CHF patients. This medication is even more likely than other diuretics to cause hyperkalemia. Routine monitoring of serum potassium is recommended for all patients with CHF, particularly those receiving an ACE inhibitor or spironolactone. If hyperkalemia is present, a diet with lower potassium content should be selected.

Table -1 summarizes the current recommendations, based on available literature and clinical experience. (International Small Animal Cardiac Health Council (ISACHC), 2001)

Preventing Nutritional Deficiencies versus Nutraseuticals

Historically, a variety of nutritional deficiencies have been known to cause cardiac disease in various species. These include thiamine, magnesium, vitamin E, selenium, and taurine. Although nutritional deficiencies are generally uncommon (except in owners feeding unbalanced homemade diets), they may still play a role in some cardiac diseases of dogs. Nutritional deficiencies may also develop secondary to the disease or its treatment.

Protein and Amino Acids
Protein
• Dogs with CHF should not be protein restricted, unless they have concurrent advanced renal disease. Some of the diets designed for dogs with cardiac disease are low in protein (3.6 - 4.2 g/100 kcal).
• Unless severe renal dysfunction is present (i.e., serum creatinine >3.0 mg/dL), high-quality protein should be fed to meet canine maintenance requirements (5.1 g/100 kcal).
• Azotemia occurs more frequently when ACE inhibitors are used in conjunction with diuretics although, in a small number of dogs, azotemia can develop from ACE inhibitors alone. A protein-restricted diet is not necessary in this situation unless medication changes do not correct the problem and the renal disease progresses.

Taurine: Unlike cats, dogs are thought to be able to synthesize adequate amounts of taurine endogenously and taurine is not considered to be required in canine diets. Certain breeds of dogs with DCM (e.g., Cocker Spaniels, Golden Retriever, Labrador Retriever, Saint Bernard, English Setter and Golden Retrievers) did have low taurine concentrations. Although the extent of the benefit of supplemen-tation is not yet clear, taurine supplementation is recommended until plasma and whole blood taurine concentrations from the

patient are available. The optimal dose of taurine for correcting a deficiency has not been determined but the currently recommended dose is 500-1000 mg q 8-12 hours.

Minimum taurine requirements for dogs have not been established by AAFCO, but A diet with a taurine content of 50 mg/100 kcal would provide approximately 1000 mg/day of taurine to a 40 kg dog.

Arginine: Based on the findings of endothelial dysfunction in patients with CHF, investigators have begun to study the effects of arginine supplementation in this group. In normal patients, L-arginine supplementation is unlikely to have an effect on nitric oxide production because L-arginine has improved endothelium-dependent vasodilation and cardiac output. These studies also have shown reduced heart rate and systemic vascular resistance, with no negative effects on cardiac contractility or other echocardiographic variables. thus, while much research is needed in this area, arginine supplementation may provide beneficial effects in patients with CHF.

Fat: Fat is a source of calories and essential fatty acids and increases the palatability of the diet.

n-3 Fatty Acids: Most human and canine diets contain primarily n-6 fatty acids. Dogs with heart failure have lower plasma concentrations of eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-6), regardless of the underlying disease Therefore, supplementation may improve an absolute or relative n-3 fatty acid "deficiency".n-3 fatty acid supplementation also reduces the more inflammatory eicosanoids. n-3 fatty acids are known to reduce the production of the more inflammatory 2- and 4-series eicosanoids (e.g., there is a shift from production of prostaglandin E2 to prostaglandin E3).

In a study of dogs with DCM, dogs supplemented with fish oil had a greater reduction in prostaglandin E2 production compared to dogs receiving the placebo. Finally, n-3 fatty acids have been shown in a number of rodents, primate, and canine models to reduce arrhythmogenesis, therefore, n-3 fatty acid supplementation may be beneficial even before CHF develops.. Recommended dosage of 40 mg/kg EPA and 25 mg/kg DHA for dogs with anorexia or cachexia.

Minerals and Vitamins

Magnesium: Magnesium plays an important role in normal cardiovascular function. It is also clear that alterations in magnesium homeostasis in people and dogs are common, and can have deleterious effects in a variety of cardiovascular conditions including hypertension, coronary artery disease, congestive heart failure, and cardiac arrhythmias. Commercial reduced sodium diets for dogs can contain between 9 - 40 mg magnesium/100 kcal (compared to an AAFCO minimum of 10 mg/100 kcal). If the dog remains

hypomagnesemic, oral magnesium supplementation will be required (e.g. magnesium oxide).

B vitamins: Little research has been conducted on the prevalence of B vitamin deficiencies in dogs with cardiac disease. Low doses of furosemide were shown to cause increased urinary loss of thiamine in healthy people and in rats. Although B vitamin status has not been reported for dogs with CHF, they may have higher dietary B vitamin requirements. Most commercial cardiac diets contain increased levels of water soluble vitamins to offset urinary losses so supplementation usually is not required.

Other Nutrients

Antioxidants: The major antioxidants include enzymatic antioxidants (e.g., super oxide dismutase, catalase, glutathione peroxidase) and oxidant quenchers (e.g., vitamin C, vitamin E, glutathione, and ß-carotene).Additional research is required to evaluate the effect, but antioxidant supplementation may hold promise in the future for the therapy of animals with cardiac disease.

L-Carnitine: L-Carnitine is a quaternary amine, whose major role is in long-chain fatty acid metabolism and energy production. Carnitine deficiency syndromes in people have been associated with primary myocardial disease and, based on this and its high concentrations in cardiac muscle, its role in canine DCM also has been of interest. L-carnitine deficiency was reported in a family of Boxers in 1991. Since that time, L-carnitine supplementation has been used in some dogs with DCM but no blinded prospective studies have been done so a causative role has not been established. The minimum or optimal dose of L-carnitine necessary to replete a dog with low myocardial carnitine concentrations is not known, but the currently recommended dose is 50 - 100 mg/kg PO q 8 hours.

Coenzyme Q10: Coenzyme Q10 is a cofactor required for energy production and has antioxidant properties. There are a number of mechanisms by which coenzyme Q10 might play a role in cardiac disease. Some investigators have proposed coenzyme Q10 deficiency as a possible cause for DCM but this has not been proven. Even in dogs with experimentally-induced CHF, serum coenzyme Q10 levels were not reduced.

The most enthusiasm for coenzyme Q10 has been as a dietary supplement in the treatment of people or dogs with DCM. Coenzyme Q10 supplementation has anecdotally been reported to be beneficial but most of the human studies of coenzyme Q10 supplementation have not been well-controlled and results are conflicting. In one study of dogs with experimentally-induced CHF, coenzyme Q10 supplementation increased serum, but not myocardial, concentrations.

The current recommended dose in canine patients is 30 mg PO b.i.d., although up to 90 mg PO b.i.d. has been recommended for large dogs. The purported benefits of supplementation include correction of a deficiency, improved myocardial metabolic efficiency, and increased antioxidant protection. Controlled prospective studies will be necessary to accurately judge the efficacy of this supplement.

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