

Nutritional Therapy of Heart Disease

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Nutrition can play an important role in the management of diseases, including cardiac disease. Research into specific nutrients that may be of benefit in cardiac disease and the appropriate levels of these nutrients that should be fed continues to be refined. Nutrition can modulate 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. There is great potential for nutrition as an important adjunct to medical therapy for patients with cardiac disease.

OPTIMAL WEIGHT MAINTENANCE

When veterinarians think of nutrition for patients with cardiac disease, they typically think of either excesses of nutrients such as sodium or deficiencies, such as taurine deficiency. In fact, the most common nutritional problem in cardiac disease is a deficiency or excess of a different nutrient: calories. A key goal for the optimal management of cardiac disease is to maintain optimal body weight since both obesity and weight loss can adversely affect health.

Cachexia

Cachexia, the wasting commonly seen in patients with CHF, is a common problem in dogs with cardiac disease. The distinguishing feature of cachexia is a loss of lean body mass, which has direct and deleterious effects on strength, immune function, and survival. The loss of lean body mass in cardiac cachexia is a multifactorial process caused by the adverse effects of anorexia, increased energy requirements, and increased production of the inflammatory cytokines, tumor necrosis factor and interleukin-1. Cachexia has historically been viewed as an end-stage situation manifested by an emaciated dog or cat but can be very subtle initially. It is usually not seen in patients with cardiac disease until CHF has developed. It is important to recognize the process of cachexia at an early stage to manage it effectively.

Nutritional therapy of cardiac cachexia is limited to nutritional support for anorexia and nutritional modulation of cytokine production. Another important reason for managing anorexia is that the presence of anorexia is one of the most common factors that contribute to an owner's decision to euthanize the pet. Modulation of cytokine production is another potential means of managing cardiac cachexia. One method of decreasing the production and effects of cytokines is with omega-3 polyunsaturated fatty acid (PUFA) supplementation (see below). Supplementation of fish oil, which is high in omega-3 PUFA can decrease cytokine production in dogs with CHF and improve cachexia. In some, but not all dogs with CHF-induced anorexia, fish oil supplementation can improve food intake. In addition, reduction of cytokines has been correlated with survival in dogs with CHF.

Obesity

The true prevalence of obesity is not known but is thought to occur in approximately 30–40% of dogs and cats in the United States. Therefore, it is likely that some patients with cardiac disease will be obese. It is important to rule out concurrent endocrine disease in patients that are obese, but most animals develop obesity as the result of overeating. Little research has been done on the effects of obesity in dogs and cats with cardiac disease. Nonetheless, it is likely to be deleterious since obesity has adverse effects on cardiac output, pulmonary function, neurohumoral activation, blood pressure, and heart rate in people and in experimental animal models. Owners often find that severely obese dogs and cats with cardiac disease that successfully lose weight appear less dyspneic and more active.

NUTRITIONAL MODULATION OF SPECIFIC NUTRIENTS

Nutritional deficiencies, although uncommon in dogs and cats, may also develop secondary to cardiac disease or its treatment. In addition, pharmacological uses of nutrients may provide benefits above and beyond their nutritional effects.

Protein and amino acids

Protein

As early as the 1960's, authors proposed restricted protein intake for dogs with congestive heart failure to prevent "metabolic stress" on the kidneys and liver. There is no evidence that protein restriction is necessary for dogs and cats with CHF and, in fact, it probably is deleterious in these patients that are predisposed to loss of lean body mass. Diets designed for animals with renal disease are not recommended for most cardiac patients because of the protein restriction (unless severe renal dysfunction is present).

Taurine

A few cats still develop DCM. Most current cases, however, are taurine-independent. Still, taurine deficiency should be suspected in all cases of feline DCM. A dietary history should be elicited from owners to determine whether the cat has been fed a poor quality, homemade, vegetarian, or otherwise unbalanced diets. Plasma and whole blood taurine should be analyzed, and treatment with taurine (125–250 mg PO q12h) should begin concurrent with medical therapy. If the taurine concentration is found to be normal, taurine supplementation can be discontinued.

Unlike cats, dogs are able to synthesize adequate amounts of taurine. Most dogs with DCM do not have taurine deficiency, but low taurine concentrations have been found in certain breeds of dogs with DCM, most notably the American Cocker Spaniel. One small study showed that Cocker Spaniels supplemented with taurine and carnitine had improvement in clinical parameters and echocardiographic measurements. Although some dogs of atypical breeds with DCM have low taurine concentrations, only some show a dramatic improvement with taurine or carnitine supplementation. Nonetheless, measurement of plasma and whole blood taurine concentrations is warranted in Cocker Spaniels and other atypical breeds with DCM. Supplementation with taurine (500 mg PO q8h–q12h) and carnitine (1 gm PO q8h–q12h) is recommended in dogs with documented taurine deficiency until additional research has been done.

Fat

In the past, the major role of fat in diets has been to provide calories and to increase the palatability of diets. The type of fat ingested, however, can significantly affect immunological, inflammatory, and hemodynamic parameters. The omega-3 PUFA, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have structures different from the more commonly ingested omega-6 PUFA, linoleic, gamma-linolenic, and arachidonic acid. A potential benefit of omega-3 PUFA supplementation is that breakdown products of the omega-3 PUFA (series 3 and 5 eicosanoids) are, in general, less potent inflammatory mediators than eicosanoids derived from omega-6 PUFA (series 2 and 4 eicosanoids). This decreases the production of cytokines and other inflammatory mediators which may reduce cachexia. Fish oil will not benefit all dogs and cats with CHF, but the author currently recommends a dosage of 40 mg/kg q24h EPA and 25 mg/kg q24h DHA in dogs with anorexia or cachexia. The amount of EPA and DHA in individual fish oil supplements varies widely so it is important to know the exact amount in brand of supplement recommended. Capsules that contain approximately 180 mg EPA and 120 mg DHA can be purchased over the counter at most human pharmacies or health food stores. Fish oil should contain vitamin E as an antioxidant, but other nutrients should not be included to avoid toxicities.

Minerals

Sodium

Although healthy animals can easily excrete excess dietary sodium in the urine, this response is blunted in animals with cardiac disease. Based on the pathogenesis, authors in the 1960's and 1970's recommended changing to a severely sodium restricted diet when a heart murmur was first detected, even before clinical signs were present. It is unnecessary to institute severe sodium restriction at this early stage, although mild sodium restriction may be useful.

As heart disease progresses and CHF ensues, however, additional sodium restriction is warranted although the use of newer and more effective medications has diminished the need for severe sodium restriction in many patients.

Potassium

Furosemide use has been implicated in the development of hypokalemia due to increased urinary loss of potassium. Hypokalemia can contribute to arrhythmias directly and by potentiating the arrhythmogenic effects of digitalis toxicosis. Hypokalemia can occur infrequently in animals with cardiac disease but the increased use of angiotensin converting enzyme (ACE) inhibitors, which decrease potassium excretion, makes hyperkalemia equally likely. Although clinically significant hyperkalemia is uncommon, potassium supplementation or feeding diets with a high potassium content to animals receiving an ACE inhibitor may increase the risk for hyperkalemia. Many commercial diets designed for animals with cardiac disease have increased concentrations of potassium so monitoring serum potassium concentrations for both increases and reductions is important.

Magnesium

Like potassium, furosemide can cause hypomagnesemia due to increased magnesium loss in the urine. Hypomagnesemia can potentiate arrhythmias, decrease myocardial contractility, and contribute to muscle weakness. Hypomagnesemia, however, is not a consistent finding in canine studies but this may be because serum magnesium concentrations are a very poor indicator of total body stores. Nonetheless, if hypomagnesemia is detected in an individual patient, especially in those patients with arrhythmias, supplementation should be instituted.

Other nutrients

Carnitine

L-Carnitine is concentrated in the skeletal and cardiac muscle and is critical for fatty acid metabolism and energy production. Carnitine deficiency is associated with primary myocardial disease in a number of species, including a family of boxer dogs. Although anecdotal reports exist regarding the efficacy of carnitine in canine DCM, no blinded prospective studies have been done so a causative role has not been established. In fact, the myocardial carnitine deficiency seen in some dogs with DCM may be merely a secondary event. There are few side effects of carnitine supplementation but high cost is a deterrent for some owners. We currently offer the option of carnitine supplementation (50–100 mg/kg PO q8h) to owners of dogs with DCM, but do not consider it obligatory.

Coenzyme Q10

Coenzyme Q10 is a cofactor in a number of reactions required for energy production and is an antioxidant. Coenzyme Q10 deficiency has been proposed as a possible cause for DCM but this association has not been proven. Although most human studies have not been well controlled, some encouraging results have been found. Anecdotal reports of success in canine DCM have been made but controlled prospective studies will be necessary to accurately judge the efficacy of this product. The commonly recommended dosage is 30 mg PO q12h, although up to 90 mg PO q12h has been recommended for large dogs. It is unclear whether the benefits of supplementation are due to the correction of a deficiency or to pharmacological effects.

Antioxidants

A great deal of media attention has been given to antioxidants for their potential role in the prevention and treatment of human cardiac disease. An imbalance between oxidant production and antioxidant protection may arise in certain situations. Most of the research on antioxidants in human cardiology has been done in coronary artery disease but there is recent evidence that dogs with DCM have increased oxidative stress compared to normal dogs, and that vitamin E concentrations decrease as the disease progresses. Therefore, while much additional research is required, antioxidant supplementation may hold promise for the therapy of dogs and cats with cardiac disease.

CONCLUSION

- There is usually not a single “best” diet for any patient. Select several diets that would be appropriate for an individual patient based on the patient, diet, and feeding practice and offer them as choices for the owner and for the pet.
- For animals with CHF, wait until the patient is stabilized to gradually change to a new diet.
- Consider the owner’s expectations. Their pet’s quality of life is of tremendous importance so provide diets that are readily eaten by the pet and that are within the owner’s financial limitations.
- Since most owners give treats or table food, be sure to specifically discuss treats with the owner. Also, consider the foods being used to administer medications. Most owners are unaware of treats that would be contraindicated (e.g., high salt treats or table food). The author typically provides a list of foods that are appropriate and foods to avoid as treats to assist the owner in wise selection:
 - Acceptable treats or foods to increase palatability*
 - Pasta
 - Rice (plain white or brown rice, not flavored rice)
 - Honey
 - Maple syrup
 - Low-salt cheese
 - Lean meats cooked without any salt (chicken, turkey, beef, or fish)
 - Eggs cooked without salt
 - Homemade soup without salt
 - Low-salt breakfast cereal (the label should read, “this is a low-sodium food”)
 - Fresh vegetables/fruit
 - Dog treats specifically labeled as “low sodium”
 - *All foods in this list should be prepared without salt
 - Foods to avoid
 - Fatty foods (meat trimmings, cream, ice cream)
 - Baby food
 - Pickled foods
 - Bread
 - Pizza
 - Condiments (ketchup, soy sauce, barbeque sauce, etc)
 - Sandwich meats/cold cuts (ham, corned beef, salami, sausages, bacon, hot dogs)
 - Most cheeses (unless specifically labeled as “low sodium”)
 - Processed foods (e.g., potato mixes, rice mixes, macaroni and cheese)
 - Canned vegetables (unless “no salt added”)
 - Potato chips, packaged popcorn, crackers, and other snack foods
 - Soups (unless homemade without salt)
 - Most dog biscuits and other dog treats

REFERENCES

References are available upon request.