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Canine cardiac diets: efficacy unproven*

*Condensed version of article in Dutch (1)

Introduction

In veterinary practice, nutritional intervention for congestive heart failure in dogs does not distinguish between dilated cardiomyopathy (DCM) and chronic valvular disease (CVD). When a canine patient eats willingly or can be tempted to food, a commercial cardiac diet is regularly seen as an option. This type of veterinary diet is restricted in sodium and expanded with long-chain omega-3 fatty acids, L-carnitine and taurine.

Sodium restriction

For more than 50 years, low-sodium dietetic therapy is used for dogs with congestive heart disease. It is believed that sodium restriction diminishes cardiac pre- and post-load and counteracts ascites and lung edema. Cardiac diets making such efficacy claims have sodium contents ranging from 25 to 110 mg/MJ of metabolisable energy. The diets come in wet (± 0.55 MJ/100 g) and dry form (± 1.75 MJ/100 g). For a dry diet, the drinking of tap water adds about 8 mg sodium/MJ (1).

For healthy, adult dogs, the minimum and recommended sodium requirements are 18 and 48 mg/MJ (2). The inevitable sodium loss is 10 mg/MJ (3). It may well be argued that many cardiac diets are not sodium restricted and that none puts dogs into a negative sodium balance, which is required for lowering body sodium. A study in 15 dogs with congestive heart disease reports substantial negative balances at an intake of 49 mg sodium/MJ (4). This finding is impossible and must be erroneous.

Renin-angiotensin-aldosterone system

In heart disease, the renin-angiotensin-aldosterone system is activated. High aldosterone maintains inflammation and fibrosis of the heart and leads to accumulation of body sodium and water. At regular sodium intakes, healthy dogs excrete at least 90% of ingested sodium with urine (3). This is only about 2% for dogs with induced (5) or spontaneous heart failure (6).

Sodium restriction in dogs with heart disease further raises plasma aldosterone (7-9). Dogs with induced heart failure were fed diets containing 53, 275 or 1226 mg sodium/MJ (10). The items assessed were: brisk walking, appetite, respiratory rate, muscle atrophy, ascites and left ventricular ejection fraction. With increasing sodium intakes plasma aldosterone concentration dropped and the clinical profile improved.

Clinical trials

In an open, non-controlled study (11) four dogs with congestive heart disease were fed a prescription diet containing 35 mg sodium/MJ (12) as only therapy. After 4 weeks, heart and respiratory rates were lowered, clinical symptoms had disappeared and body weight was increased.

During dietary treatment for up to one year, there was remission of clinical signs. Successful recovery may relate to weight gain (13) rather than sodium supply, which was not the sole diet change.

Two trials evaluated commercial cardiac diets, but diet comparisons involved multiple differences. Dogs with CVD or DCM received medication and wet diets containing 167 or 96 mg sodium/MJ (8). The lower-sodium diet slightly reduced left ventricular internal dimension in diastole and systolic blood pressure, but clinically no improvement was noted. Dogs with asymptomatic CVD were given medication and dry foods with 361 or 148 mg sodium/MJ (9). Lower sodium intake slightly reduced left ventricular size, but did not affect systolic blood pressure.

Omega-3 fatty acids

In a placebo-controlled trial, oral administration of capsulated fish oil ethyl esters improved cachexia in dogs with heart failure, which was associated with lower plasma concentrations of interleukin-1 β and tumor necrosis factor-α (14). However, body weight was unchanged, the two treatment groups were poorly comparable and outcome reproducibility is unknown. The supplement provided 140 mg eicosapentaenoic acid (EPA)/MJ. In a retrospective study, 11 fish oil-supplemented dogs, out of 108 heart patients, tended to survive longer (13).

In canine models, long-chain omega-3 fatty acids have been shown to reduce arrhythmogenesis (15, 16). Boxer dogs with arrhythmogenic right ventricular cardiomyopathy were administered capsules containing sunflower, flax or fish oil (17). Compared with the other oils, fish oil supplementation (120 mg EPA/MJ) for six weeks diminished arrhythmia.

L-carnitine and taurine

L-carnitine is indispensible for mitochondrial β-oxidation of fatty acids. Dogs can synthesize L-carnitine from lysine, but deficiency associated with DCM has been reported for some Boxer dogs. Oral administration of L-carnitine was effective in two dogs (18), but not in another (19). Eleven Doberman Pinchers with DCM and myocardial carnitine deficiency obtained conventional therapy supplemented orally with 150 mg L-carnitine/kg body weight/day (20). Two dogs survived longer than one year.

The dog is able to convert methionine via cysteine into taurine, which has ionotropic and antiarrhythmic activity. Some dogs have DCM accompanied by low plasma taurine concentration (21). Newfoundlands with low plasma taurine appeared to have a high requirement of sulfur amino acids (22). Taurine deficiency in those dogs was reversed by methionine supplementation (23).

Heart function of three Newfoundlands with DCM improved after taurine supplementation (1 g bid) (22). Based on a retrospective study in 12 dogs with DCM and taurine deficiency, it was suggested that oral taurine administration (1-3 g/day) prolongs survival time (24). In a double-blinded, placebo-controlled trial (n= 5 or 6/group), American Cocker Spaniels with DCM and low plasma taurine were treated with 500 mg taurine and 1 g carnitine PO tid (25). Despite weaning off cardiovascular drugs, echocardiographic variables improved during supplementation.

Bottom line

There is no evidence that sodium restriction improves clinical signs in canine cardiac disease. Worse still, there are good reasons for contraindication. As cardiac patients often have arrhythmias, fish oil supplementation seems beneficial. L-carnitine and taurine alone have not been tested under controlled conditions. The above-mentioned L-carnitine doses are not met by commercial cardiac diets.

Literature

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