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CANINE DILATED CARDIOMYOPATHY – INSIGHTS INTO DIAGNOSIS AND MANGEMENT

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Dilated cardiomyopathy (DCM) is a syndrome characterized by impaired myocardial function (systolic +/- diastolic), ventricular dilation and frequently, tachyarrhythmias. In the dog, specific breed predispositions exist and the Doberman pinscher, Great Dane, Scottish deerhound, and Irish wolfhound appear to be over represented. This discussion will emphasize breed specific findings and newer developments in the understanding of etiologies, clinical presentation, diagnosis, screening and treatment for canine DCM as well as arrhythmogenic cardiomyopathy in the Boxer.

ETIOLOGY

The etiology of canine DCM is unknown in many cases. The development of DCM is likely to be a multifactorial process that could involve nutritional, familial and infectious agents. In some cases, there is evidence of a specific nutritional or familial link.

NUTRITIONAL

In 1991, a family of Boxers with dilated cardiomyopathy (historically known as Type III Boxer cardiomyopathy) was identified as having a myocardial L-carnitine deficiency. Affected dogs demonstrated some reversal of disease with L-carnitine supplementation. However, affected dogs eventually died from their heart disease, even while on supplementation. L-carnitine deficiency does not appear to be the cause of DCM in the majority of Boxers, or in other breeds. However, it is still considered a potential etiology for Boxers with DCM and supplementation should be considered for these rare cases.

Some American Cocker Spaniels have been reported to develop DCM associated with low taurine levels. Taurine supplementation may result in reversal of the disease and a significantly better prognosis. Although taurine does not appear to be associated with the development of DCM in other commonly affected breeds, it is still occasionally reported in unique presentations of DCM and measurement of levels may be considered in atypical breeds.

FAMILIAL

A genetic etiology has been strongly suggested in several breeds of dogs including the Doberman pinscher, Great Dane and Boxer and should be suspected in other breeds with a strong breed predisposition. The mode of inheritance has not been determined in the Doberman pinscher, but a strong male predisposition in the Great Dane suggests an X-linked mode of inheritance.

DIAGNOSIS

Clinical Presentation

This is an adult onset disease with a clinical presentation that may be as subtle as a gradual development of exercise intolerance and weight loss. However, more commonly the early signs of the disease are overlooked and the disease is not diagnosed until congestive heart failure develops and the patient presents with coughing, respiratory distress and occasionally, ascites. It would appear that the early stages of DCM are difficult to diagnose unless a clinician maintains a high level of suspicion for dogs that are of an at risk breed and annual screening is performed.

Physical Examination

A soft systolic murmur and/or gallop rhythm (S_3) may be ausculted at the left apex. In some cases, this may be the first sign of the disease. A tachyarrhythmia may be noted. Although canine DCM is predominantly a left ventricular disease, biventricular involvement and failure with jugular venous distension and ascites is frequently noted, particularly in the giant breeds.

Electrocardiography

Left atrial and ventricular enlargement and sinus tachycardia, atrial fibrillation or ventricular tachyarrhythmias are common.

RADIOGRAPHY

Left atrial and ventricular enlargement with or without pulmonary venous distension and pulmonary edema may be observed.

Echocardiography

The diagnosis of DCM in the symptomatic dog is easily determined by echocardiography. Left atrial and ventricular dilation with systolic dysfunction (decreased fractional shortening, ejection fraction and shortening area) and increased end-systolic volume are evident and often severe. Unfortunately, the diagnosis of the affected dog in the occult (asymptomatic) stage is much more difficult and will be discussed below.

Doberman pinscher dilated cardiomyopathy

In the majority of cases, DCM is diagnosed when a Doberman pinscher presents with signs of left heart failure. However, about 30% of the dogs develop ventricular tachyarrhythmias and may present for syncope or die of sudden death before ventricular dilation and systolic dysfunction have developed.

Screening for Early Diagnosis of DCM

Recent evidence that the disease is familial and that early intervention may increase survival has lead to significant interest in screening asymptomatic dogs for signs of early disease. Annual echocardiography and ambulatory electrocardiography (Holter monitoring) are believed to be the best predictors of early DCM. Criteria that are believed to be indicators of early disease include a left ventricular diastolic dimension of greater than 4.6 cm and a systolic dimension of 3.8 cm even without evidence of systolic dysfunction. These numbers are based on average sized dogs and may not be valid for very large dogs. Annual Holter monitoring has been recommended to detect Doberman pinschers that may develop ventricular arrhythmias before ventricular dilation and systolic dysfunction. Adult Doberman pinschers with greater than 50 ventricular premature complexes (VPCs) per 24 hours, or couplets or triplets are suspect for the development of DCM. Owners should be advised that since this is an adult onset disease with variability in the age of onset, screening tests should be performed annually.

PROGNOSIS

Dilated cardiomyopathy in the Doberman pinschers is a very malignant form of DCM in comparison to the disease in other breeds. Once clinical signs have developed, death usually occurs due to heart failure or sudden death within 6 months, therapy is palliative at best.

GIANT BREED DILATED CARDIOMYOPATHY

Giant breed dilated cardiomyopathy is used to characterize DCM in the Irish Wolfhound, Great Dane, Scottish Deerhound and Newfoundland dog, among others. Dilated cardiomyopathy in these breeds is more commonly a progressive biventricular disease and may present with ascites.

A high percentage of affected dogs present with atrial fibrillation. In some cases, atrial fibrillation may develop before any other evidence of underlying myocardial disease (chamber enlargement or systolic dysfunction). These dogs should be carefully followed for the development of DCM.

SCREENING

Occasional cases of familial disease in the Great Dane, Newfoundland and Irish wolfhound have been identified. In the Great Dane, it is most likely an X-linked disease. Sons of affected females are at high risk of developing the disease; daughters of affected fathers are likely to be silent carriers. Since it is adult onset, all dogs should be screened annually with echocardiography. Dogs with atrial fibrillation without other evidence of cardiomyopathy should probably be withheld from breeding until it can be determined if they will develop DCM.

COCKER SPANIEL DILATED CARDIOMYOPATHY

Dilated cardiomyopathy is reported in both American and English cocker spaniels. The disease is not common and cocker spaniels are more likely to develop heart failure due to mitral valve endocardiosis than cardiomyopathy. However, when cardiomyopathy is present, it is likely to start as a left ventricular disease that develops into biventricular failure.

An association between the development of DCM and decreased plasma taurine levels has been reported in some American cocker spaniels with dilated cardiomyopathy. Plasma taurine levels (normal range:44-224 nmol/ml) should be evaluated to help guide therapeutic interventions and prognosis. Supplemental taurine (500 mg q 12h, PO) may be started while waiting for the results of the plasma levels and should be continued if the level is low. Additional treatment should be given as needed for heart failure, arrhythmias, etc. If taurine deficiency is documented, significant improvement may be observed with supplementation in 3- 4 months. If taurine deficiency is not identified as the etiology of the DCM, the prognosis is poorer, but progression is fairly slow and the dog may be kept comfortable on heart failure medications for some time.

DALMATION DILATED CARDIOMYOPATHY

Dilated cardiomyopathy has been reported in a group of male dalmations and was characterized as left ventricular disease with heart failure. Interestingly, the majority (8/9) of dogs had been a fed a low protein diet for all, or part of their lives. The cause and effect relationship in these cases is not known, but dalmatians that develop DCM and are being fed a low protein diet should be switched to a more balanced diet if possible.

TREATMENT OF DCM

Asymptomatic Dogs with Ventricular Dilation / Dysfunction (Occult)

Two types of therapy have been under investigation for treatment of occult DCM, ACE inhibitors and beta-blockers. Administration of ACE inhibitors (enalapril, 0.25-0.5 mg/kg q12hr) has been shown to slow the progression to heart failure in the Doberman pinscher. Since ACE inhibitors are generally well tolerated, this treatment is recommended for dogs of other breeds at this stage and provides additional support for the practice of screening at risk dogs (perhaps with a family history) to allow any intervention. Administration of beta-blockers at this stage is still being evaluated. The addition of low dose beta-blockers to the treatment of human patients with DCM and stable heart failure has demonstrated a reduction in both mortality and morbidity. However, many human patients with DCM cannot tolerate even very low doses of beta-blockers and demonstrate rapid cardiac decompensation. The use of beta-blockers for the canine patient with DCM has not yet been well studied and a consensus opinion on use of these drugs for our patients is not yet available. Beta-blockers might be considered for the patient with occult disease, but they should be very carefully monitored and should not be given once there is evidence of fluid retention and heart failure until it is very well stabilized. The optimal beta-blocker for this purpose appears to be carvedilol because of its effects on both alpha and beta-receptors. It cannot be over emphasized that the addition of beta-blockers in our DCM patients should be done very cautiously with gradual increases in dosing after a two-week period and careful monitoring of heart rate, blood pressure and symptomology.

DOGS WITH HEART FAILURE

Symptoms of heart failure should be alleviated with furosemide (1-3 mg/kg, q8-12h) and ACE inhibitors (enalapril, 0.25-0.5 mg/kg, q12h, orally). As heart failure becomes more refractory, the addition of spironolactone (1-2 mg/kg, q12h, orally) should be considered for aldosterone blocking affects. Digoxin may be added when the heart failure becomes refractory or atrial fibrillation is observed. Pimobenden can be added on a case by case basis with approval from the FDA at a dose of 0.3 mg/kg orally twice a day. Pimobenden may have significant difference for dogs with DCM and heart failure in terms of both quality of life (increased appetite, activity, etc) and survival. Dogs with DCM and chronic heart failure often develop weight loss and cardiac cachexia. Fish oil supplementation has been shown to decrease cardiac cachexia in some cases. It may be dosed at 40 mg/kg EPA and 25 mg/kg DHA . For easy dosing, most 1.0 gm capsules contain 180 mg EPA and 120 mg DHA.

PROGNOSIS

Sudden death is always possible. However, many dogs may live for years on antiarrhythmics without symptoms, some of these may develop ventricular dilation and systolic dysfunction.

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