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Review Article

Dilated Cardiomyopathy and its Management in Dog

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Dilated cardiomyopathy (DCM) is the term used to define the primary myocardial disorder characterized by reduced contractility and ventricular dilatation involving the left or both ventricles, of unknown or familial etiology. DCM is an important cause of cardiac morbidity and mortality in the dog. Next to chronic mitral valve insufficiency and in some geographic regions where heart worm disease is common, DCM is the most common acquired cardiac disorder.

Etiology and Prevalence

In dogs, the cause of the disease is generally unknown. However, some recognized causes of DCM in dogs include genetic factors, tachycardia, taurine deficiency, toxic factors and possibly carnitine deficiency. In cats, DCM is primarily due to a dietary deficiency of the amino acid, taurine. The potential for a viral or immunologic etiology also should be considered. However, Idiopathic DCM is the most common form of DCM in dogs.

Bond and Tilley (1980) stated that DCM had a strong familial predisposition in some breeds and canine DCM had long been suspected to have a genetic basis. There was clinical and experimental evidence that neonatal parvo viral infection could induce a form of idiopathic DCM in dogs under one year age (Atwell and Kelly, 1980). Higgins *et.al* (1981) showed evidence that canine distemper virus could produce severe myocardial damage in dogs.

DCM can occur due to deficiency of L- carnitine in Boxers, taurine in American Cocker Spaniels (Keene *et.al*, 1991) and protein in Dalmations (Freeman *et.al*, 1996). Panciera (1994) reported a higher prevalence of concurrent hypothyroidism and DCM, particularly in Dobermann Pinschers. A strong male disposition in Great Dane suggested an x-linked mode of transmission in DCM (Meurs *et.al.*, 2001).

Meurs (2002) stated that the etiology of DCM was unknown. The development of DCM was likely to be a multifactorial process that could involve nutritional, familial and infectious agents. An autosomal dominant transmission of DCM was reported in Irish Wolfhound, Newfoundland and Dobermann Pinschers (McEwan *et.al*, 2003).

Whatever be the initial cause, progression of the condition is as follows: Defective transport of calcium ions within the heart muscle diminishes the cells' ability to contract. The heart muscle becomes thin and flabby. Quietly, over a period of several months, the thinning worsens, the heart chambers become dilated (enlarged), electrical timing of the heart malfunctions and affected dogs begin to have visible trouble. Before long, the problems cascade into full-blown congestive heart failure and then death. Over the usual one- to two -year course of the disease, the heart deteriorates from a muscular, automated, fine-tuned pump to a bag of overstretched elastic with misfiring electronics.

According to Montoya (2002), DCM has been recognized in a number of species, including dogs, cats and human beings. DCM is typically observed in large and giant breed dogs, most commonly in Doberman Pinschers, Boxers, Great Danes, Irish Wolfhounds, Saint Bernards, Cocker Spaniels, Golden Retrievers and German Shepherd Dogs. More than 90% of cases of canine DCM are confined to these eight breeds. The disease is likely to be genetic in origin, although this has not yet been proved and the mode of inheritance is yet to be documented. Middle-aged dogs are most often affected. There is a gender bias in dogs, with male dogs more frequently affected.

Diagnosis

In dogs with DCM, the complete blood count (CBC)

findings were not different from those of normal dogs except for a moderate lymphopenia and occasionally a modest neutrophilia (Brien *et.al.*, 1993).

Tidholm and Jonsson (1997) reported hyponatremia, hyperkalemia, azotemia and modest elevation of serum liver enzymes (ALT and AST) in dogs with DCM.

The disease is usually not difficult to confirm once the patient is symptomatic. Diagnosis is much more problematic in dogs in the preclinical, occult stage of the disease. Occult DCM has been described as a specific clinical entity in the Doberman.

Clinical signs

The signs of DCM can vary widely depending on the breed of dog. Clinical findings in dogs with DCM may include cough, depression, dyspnoea, weight loss and syncope. Moreover clinical presentation of DCM commonly includes signs of CHF, pulmonary edema, pleural effusion and ascites. Clinical signs exhibited by dogs with DCM are similar in all breeds, but the observed frequency of these signs is significantly different in many of the commonly affected breeds. In some breeds of dogs with DCM, sexual differences concerning clinical characteristics have been observed.

A regurgitant systolic murmur with a low-pitched protodiastolic gallop sound is a frequent and important clinical finding. A gallop sound is evidence of severe ventricular impairment. Dobermans and Boxers generally show signs of acute left-sided heart failure or life-threatening arrhythmia. Sudden death may be the first observed sign in these dogs with DCM. Other large breed dogs have lower possibility to syncope or to die suddenly. Ascites and/or pleural effusion are even more prevalent in other giant breed dogs than Doberman or Boxers. Weight loss and muscle wasting are common in some dogs with DCM. These findings are more severe in dogs that have been symptomatic for several months.

Advanced diagnostic procedures.

Radiograph: Pulmonary edema is the most common finding on thoracic radiographs of dogs with symptomatic DCM, although signs of right-sided heart failure are reported to be common in giant breeds with DCM. Radiographic changes affecting the cardiac silhouette include left atrial enlargement alone, left atrial and left ventricular enlargement, right-sided enlargement or generalized cardiomegaly. Bright and Mears (1997) stated that generalized cardiomegaly was a typical radiographic feature.

Electrocardiography (ECG)

Electrocardiographic abnormalities are common in dogs with DCM, but the nature and prevalence of different arrhythmias varies in different breeds. Atrial fibrillation is the most commonly diagnosed electrocardiographic abnormality in Irish wolfhound, Dalmatian and Presa canario dogs with DCM, although ventricular premature depolarisations and ventricular tachycardia are reported in a majority of Dobermans. These ventricular arrhythmias may be present upto nine months prior to development of echocardiographic evidence of the disease in this breed. In Dobermans, atrial fibrillation is less common and it is a poor prognostic indicator. In Weimaraners, ventricular arrhythmias are common and may result in syncope or sudden death prior to the development of other clinical signs. Ventricular arrhythmias have also a high prevalence in Boxer, Airedale terrier and Newfoundland dogs with DCM. In Boxers, sudden death is less common than in Dobermans. Ventricular premature depolarisations in Boxers have a characteristic appearance; the configuration of the complexes is almost consistent with a right ventricular origin. In Cocker spaniels, ECG often shows tall R waves and sometimes a left bundle branch block. In Presa canario dogs with DCM, abnormalities of cardiac conduction have been diagnosed in 34 % of dogs and changes in wave morphology have been recorded in 62 % of patients. Ventricular arrhythmias are uncommon and they have a very important prognostic value in Presa canario dogs with DCM.

Most dogs with DCM showed arrhythmia, abnormal amplitude or duration of P wave or QRS complex indicating chamber enlargement or conduction abnormalities. Atrial fibrillation was the most common arrhythmia in DCM. Ventricular Premature Complexes (VPCs) and Ventricular

tachycardia were common in Boxers and Doberman (McEwan *et.al*, 2003).

Holter monitoring provides a more representative assessment of the genuine incidence of arrhythmias in patients with DCM. The role of Holter monitoring in the diagnosis of occult cardiomyopathy, particularly of Doberman pinschers and Boxers, has been recognized (Goodwin,1998). Holter recordings are particularly useful in Doberman and Boxer dogs. These breeds tend to manifest frequently occult DCM.

Echocardiogram

Echocardiography was the most sensitive method of confirming myocardial dysfunction (Meurs, 2002). Echocardiographic evaluation of left ventricular systolic performance reveals increased end-systolic and end-diastolic dimensions, dilatation of the left atrium and decreased fractional shortening, as well as changes in systolic time intervals, in dogs with DCM. Diastolic dysfunction, as evidenced by Doppler examinations derived prolonged relaxation time and left ventricular inflow pattern of increased early diastolic to atrial wave ratio have been correlated to clinical deterioration in dogs with DCM. Echocardiography has been used to screen for DCM. However, currently it is difficult to differentiate between normal and abnormal cases and to assess echocardiographic abnormalities in the absence of the classical findings of DCM, because echocardiographic parameters vary with the breed of the dog as well as with the weight of the animal.

Cardiac Catheterization

I. This diagnostic tool can be used to determine the underlying heart disease, to confirm the presence of heart failure (by measuring diastolic intra ventricular pressures and cardiac output), and to assess the severity of heart failure.

II. Since most animals with heart failure are at anaesthetic risks, it is desirable to either avoid cardiac catheterization, do the procedure with sedation and local anesthesia or do it with light anesthesia and keep the procedure as short as possible.

III. In dogs that require invasive testing to assess the severity of cardiac disease, catheterization of the right side of the heart with a balloon-tipped thermodilution catheter is preferred. This type of catheter can be placed with the dog awake or lightly sedated, and the catheter can be maintained in place to monitor the disease or response to therapy if necessary.

Biochemical markers

Circulating markers of LV systolic dysfunction, including BNP and cardiac troponins (cTn-I) may be useful in this regard , to potentially identify the dogs in the occult stage, even before electrically or morphologically detectable abnormalities occur.

Differential diagnosis

A) Non cardiac pulmonary disease frequently mimics heart failure. Both can cause coughing and dyspnoea. It is not unusual for heart disease and pulmonary disease to be present in the same patient. One or both may be responsible for clinical signs. Thoracic radiographs are usually the best diagnostic test to differentiate the causes of cough and dyspnea, but false negative and false positive results do occur for all diseases assessed in this manner. Although measurement of left ventricular end-diastolic or pulmonary capillary wedge pressure are the ideal means of confirming pulmonary edema due to left-sided heart failure, they cannot be done routinely on a practical basis.

B) Ascites, peripheral edema, and hydrothorax can be caused by diseases other than congestive heart failure. Usually the measurement of central venous pressure will confirm whether or not right-heart failure is the cause. Evaluation of the size of the hepatic veins is a good substitute.

Management of Dilated Cardiomyopathy (DCM) Occult

Management of the occult stage of DCM is clearly of value, because this stage of DCM inevitably progresses to the overt stage and death.

- Angiotensin converting enzyme inhibitors have been demonstrated to retard the progression to overt DCM. This effect has been much more dramatic in male Dobermans compared with female Dobermans

- Currently a new beta blocker, carvedilol is found to be more effective than Digoxin and Metoprolol in the long term treatment of DCM in dogs (Sindhu, 2006), as it confers additional protection beyond that produced by angiotensin converting enzyme inhibitors.

Occult DCM with lots of PVCs:

- PVCs are a common part of DCM in both the occult stage and overt stage. It is a major sign of occult DCM as opposed to a sign of risk for sudden death. Now, Sotalol is being attempted to protect the dogs from sudden death.

Congestive Heart Failure with DCM

- Administration of angiotensin converting enzyme inhibitors and diuretics. The highest recommended dose appears to be the best dose.
- One has to search for the least dose of the diuretic that maintains ease of breathing so that it can be used for the long time treatment.
- Digoxin is associated with a lot of toxicity and unproven efficacy.

Beta blockers, especially carvedilol was proved to help people with overt DCM. This agent can initially and immediately make the dog worse. Hence, it is to be started on a very low dose and increased slowly. Also, it may best be started after pulmonary edema has been corrected.

Spironolactone is more than a diuretic. It is too weak a diuretic to be useful as such. However, it's other property as an agent that blocks the hormone aldosterone appear to be responsible for its benefits in people with heart failure.

A vital part of the treatment of this disease is the follow-up. The first recheck should occur on 3 to 5 days after first examination. The objective here is to attempt to reduce the dose of diuretic required. Perform a lateral chest radiograph and a serum assessment of kidney function and also check the heart rhythm for frequency of PVCs or atrial fibrillation. The next checkup occurs about 1 week after first examination, then at 2 weeks, 4 weeks, and then once monthly. The objective is always the same.

Atrial Fibrillation and Congestive Heart Failure with DCM

In addition to the drugs described above, dogs with atrial fibrillation need to receive drugs with the objective of reducing the heart rate. Many Dobermans with atrial fibrillation have heart rates over 200 beats per minute. The goal is to reduce the heart rate to about 160 beats per minute. Drugs to accomplish this are:

Beta blockers: Atenolol, Carvedilol, Sotalol

Calcium channel blockers: Diltiazem. Currently, this is the best agent to slow the heart rate.

Digoxin: probably it will not be effective if the heart rate is over 200 beats per minute before therapy. Hence, additional agents will likely be needed.

A vital part of the treatment of this disease is the follow-up just like above. In addition to the issues described above, the rechecks also focus on checking the heart rate with a view to determining if we have achieved the target heart rate. Some nervous dogs may be better assessed with a Holter examination, which will give us the heart rate at home and the trends in the rate throughout the day.

Bright and Mears (1997) opined that important components of therapy included increasing contractility, optimizing HR and rhythm and decreasing preload and afterload. Other therapies included low-sodium diet, exercise restriction and supplementation of taurine, carnitine, fish oil, magnesium, co-enzyme Q 10 and Vitamin E.

Effect of medical therapy on survival (Montoya, 2002)

- Medical therapy for DCM is generally unsatisfactory once overt CHF develops.
- Neither diuretics nor nitrates have been shown to exert a favourable influence on survival in terms of altering disease progression in dogs with DCM.
- Digoxin exerts neither a favourable nor an unfavourable influence.
- Increased survival times were detected in a group of Dobermans with DCM treated with

conventional therapy and pimobendan. However, the same protocol failed to show any effect on survival in a group of Cocker Spaniel dogs with DCM.

- ACE inhibitors have been shown to exert a favourable influence on disease progression.
- Beta-blocking drugs is known to influence survival in dogs and humans with CHF, but it has been only evaluated in few of studies.
- Antiarrhythmic therapy can prolong survival in dogs with DCM. In that, short-term (from 2 to 7 months) efficacy is generally good.

Prognosis

With dilated cardiomyopathy, the prognosis is usually fairly grim, and sufferers usually succumb soon after developing heart failure. But this isn't always true; the disease may be reversed in some Boxers using carnitine, a dietary supplement that helps the muscle use fatty acids for energy. For Dobermans, a positive diagnosis means fewer than six months to live, and for Irish Wolfhounds it can mean a few years more.

Prediction of survival times and identification of factors influencing mortality are of interest for canine patients (Montoya, 2002). Predictive factors of survival rate include clinical class of CHF, presence of ECG changes, increased left ventricular filling pressure, increased systemic vascular resistance, increased pulmonary capillary wedge pressure, left intraventricular conduction delay, increased hypertrophy-dilatation index, Doppler indices, and Doppler-derived assessment of pulmonary hypertension.

In dogs with occult DCM, survival times depends, in part, on the degree of myocardial failure. Sudden death is common in Boxer and Doberman dogs and can occur even with normal heart size and contractility. Sudden death is less common in other breeds. In general, dogs with a younger age of onset of echocardiographic abnormalities or clinical evidence of DCM have a more rapidly progressive course.

Survival times are generally short after the onset of overt CHF. Many dogs die without ever being stabilized from their first episode of failure. Dobermans have the worst prognosis.

After the advent of clinical signs of CHF, poor prognostic indicators include the presence of pleural effusion, pulmonary oedema and tachydysrhythmias.

In dogs with overt CHF, echocardiographic data and/or radiographic heart size does not predict relative survival time. In Dobermans with overt CHF, both atrial fibrillation and bilateral heart failure exert a negative influence on survival.

New Developments in DCM

Understanding the diet fed to our pets has become more important than ever. Taurine is an essential amino acid naturally produced by the dog. Just like a recipe, taurine is made up of multiple "ingredients." The sulfur amino acids methionine and cystine are critical to the production of taurine. Dogs can only produce adequate amounts of taurine when the correct levels of methionine and cystine are available in their diet.

Taurine deficiencies in cats and foxes have been associated with low plasma taurine concentrations and subsequent heart problems, specifically DCM. Sadly, similar signs have been observed in dogs fed commercial diets low in methionine and cystine, thus inhibiting the body's ability to produce taurine.

Alternative Medicine

Dwivedi and Udupa (1989) reported that T. arjuna bark contains a triterpene saponin which was responsible for diuretic property in the treatment of CHF. Bharani *et.al* (1995) observed marked improvement in the signs of heart failure and echo parameters –Decrease in left ventricular end diastolic and end systolic volume indices, increase in left ventricular stroke volume index and left ventricular ejection fraction in CHF patients using T. arjuna bark extract 500mg 8-hourly for two weeks.

Various Ayurvedic preparations are used including Neocardio, Biocardio and Cardana marketed by Thorne research laboratory, USA for therapeutic management of congestive heart failure in human beings and dogs that contain bark extract of T.arjuna as one of the active ingredient. The

saponin glycosides might be responsible for possible inotrophic and diuretic effects of barks of T.arjuna in treatment of a group of dogs with DCM. The affected dogs were treated orally (Vykuntarao, 2004) with bark extract of T. arjuna 250 mg/dog twice daily before meals, enalarpil 9.25 mg/kg b.wt once daily and furosemide 1 mg/kg.b.wt. twice daily.

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