Canine Idiopathic dilated Cardiomyopathy

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Introduction

Cardiomyopathies are diseases of the myocardium associated with cardiac dysfunction and dilated cardiomyopathy is characterised by dilation and impaired contraction of the left ventricle or both ventricles. Dilated cardiomyopathy (DCM) is the most common form of cardiomyopathy in dogs and the second most common form of acquired heart disease in dogs. Diagnosis, pathophysiology and current treatment recommendations will be discussed in this article.

Signalment

DCM tends to be a disease of large breed dogs with a higher prevalence in male dogs. A genetic predisposition to the disease has been identified in Dobermans, Irish Wolfhounds, Great Danes, Newfoundlands, Spaniels and Portuguese Water Dogs. The disease in the American cocker Spaniels has been frequently characterised as a taurine deficient condition with supplementation markedly improving the outcome.

Pathophysiology

DCM is characterised by ventricular enlargement, diastolic and systolic dysfunction and resulting congestive heart failure. The symptomatic phase is preceded by an asymptomatic phase where a number of compensatory mechanisms work in concert to maintain cardiac output and prevent the development of congestive heart failure. These compensatory mechanisms include the rennin-angiotensin aldosterone-system (RAAS), catecholamine’s and other vasoactive substances. These compensatory mechanisms are beneficial in the short to medium term but have deleterious effects in the long term. Increasing blood pressure with RAAS increases the cardiac work load as does increased plasma catecholamine’s by increasing the heart rate. The end result may increase the rate of myocardial cell death. These deleterious compensatory mechanisms are balanced by vasodilator, diuretic and natriuretic factors such as the natriuretic peptides.

In symptomatic DCM plasma catecholamines have been found to be significantly increased in both humans and dogs. Increased catecholamine activity results in down regulation of the β1-receptors in the myocardium and decreased β-adrenergic responsiveness. This results in impairment of systolic and diastolic functions and exacerbation of arrhythmias.

It is widely accepted that congestive heart failure is associated with an increased RAAS activity. This results in vasoconstriction, sodium retention, water retention and an increase in thirst. A local RAAS in the myocardium is most likely the most significant area of
angiotensin II production. Increased local RAAS may lead to myocyte hypertrophy, necrosis and fibrosis.

**Clinical Findings**

Dogs with DCM can be divided into those in the pre-clinical stage where heart disease is evident but there are no outward signs of heart disease. In the clinical stage dogs will exhibit some or all of the following signs

- Breathlessness
- Dyspnoea
- Cough
- Exercise intolerance
- Anorexia
- Weight loss
- Syncope
- Abdominal distension
- Polydypsia

Clinical examination may reveal tachypnoea, crackles, tachycardia, pulse deficits, arrhythmias and in some dogs a systolic murmur, ascites, pale mucus membranes, weight loss and muscle wasting. The arrhythmia is described as irregularly irregular - and has no discernable pattern. A pulse deficit occurs when the pulse is lower than the heart rate. This occurs when the ventricles contract prematurely and the chamber is not yet filled with blood - thus there is no bolus of blood being pushed into the arterial system and thus no pulse.

Large breed dogs are also predisposed to developing pericardial effusion. These dogs however have good body condition as it is not a chronic disease. In these patients the heart rate may be rapid, but is regular and there is no pulse deficit. Auscultation reveals softer heart sounds. The right heart is compromised first as the myocardium is thinner and thus these animals develop ascites. The radiographs will also show cardiomegaly and careful evaluation is required to differentiate from DCM. Ultimately ultrasound is required to confirm a diagnosis.

**Diagnosis of DCM**

Radiographic findings in dogs with clinical DCM include cardiomegaly, venous congestion, prominent left atrium, pulmonary oedema, pleural effusion and sometimes ascites.

Echocardiography is required to appreciate increased chamber size and myocardial failure. It must also be borne in mind the DCM is not the only cause of increased chamber size and myocardial failure. Doppler echocardiography is required to exclude some acquired and congenital heart diseases that result in ventricular dilation and reduced myocardial function.
For example patent ductus arteriosus results in volume overload of the left heart with ventricular dilation. Other differentials include tachycardia induced cardiomyopathy, doxorubicin induced cardiac damage, taurine/carnitine deficiency and systemic diseases such as hypothyroidism.

The echocardiogram should be carried out in lateral recumbency according to the standard views. 2D, M-mode and Doppler evaluation should be performed to evaluate the heart correctly. A full description of the echocardiographic evaluations is beyond the scope of this article.

The following criteria are used to make a diagnosis of DCM

- Left ventricular dilation (especially in systole). There are breed and weight tables for normal values.
- Depressed systolic function. Measurement of fractional shortening and ejection fractions. Fractional shortening of less than 20-25%. Fractional shortening does vary slightly between different breeds and there are tables available listing the expected fractional shortening for different breeds. Echocardiography of healthy Dobermans recently found that the average fractional shortening was 26% using a short axis view, and 22.5% using a long axis view. In other breeds a fractional shortening of 25% or less in the short axis view is considered abnormal. This either indicates that a large percentage of healthy Dobermans have occult DCM or that the Doberman heart at baseline is not comparable to that of most breeds.
- Left ventricular sphericity. A ratio of less than 1.65
- Left or bi-atrial enlargement.
- Increased E point to septal separation (EPSS) >6.5mm.
- Arrhythmias such as atrial fibrillation and ventricular arrhythmias. Atrial fibrillation more common in Irish Wolf Hounds and ventricular arrhythmias more common in Dobermans and Boxers.

Holter monitors can be used to look of sub-clinical myocardial disease which may present with episodic arrhythmias.

**Treatment of DCM**

When treating a patient with DCM we need to address the underlying myocardial dysfunction, the harmful counter regulatory mechanisms, pulmonary oedema, arrhythmias, ascites and possible pleural effusion. Supplementation with taurine, carnitine and free fatty acids can also be considered.

Before starting a patient on treatment it must be borne in mind that the median survival time from diagnosis is about 19 weeks. The best single variable for assessing prognosis is the left ventricular diameter at end systole. Other variables negatively associated with survival include the following
• Presence of pulmonary oedema
• Presence of ventricular premature complexes
• Higher plasma creatinine levels
• Lower plasma protein
• Great Dane breed.

Standard therapy includes an Angiotensin converting enzyme inhibitors (ACE inhibitor), Pimobendan and furosemide.

Pimobendan requires a special mention as it has demonstrated a marked increase in survival time compared to placebo treated groups. A study by Luis Fuentes and colleagues looking at Dobermans and Cocker spaniels demonstrated a median survival of 329 days in the Pimobendan groups compared to 50 days in the placebo group. A similar study by O’Grady and colleagues demonstrated a survival time of 130.5 days in the Pimobendan group compared to 14 days in the placebo group.

The Bench study looked at the use of Pimobendan to prevent the onset of congestive heart failure in Dobermans diagnosed with pre-clinical DCM. The Study found that Pimobendan significantly prolonged (by 9 months) the median time to onset of heart failure or sudden death in Dobermans with pre-clinical DCM. This drug has now been registered for this use in Dobermans.

Pimobendan acts by inhibiting phosphodiesterase III and by increasing the calcium sensitivity of the cardiac myofibrils to calcium. The effect is increased contractility without any increase in myocardial oxygen consumption. Pimobendan results in a reduction of pulmonary capillary wedge pressure, an increase in cardiac output and an increase in stroke volume.

Beyond standard therapy other therapies addressing atrial fibrillation such as digoxin can be considered. Digoxin also has a weak positive inotropic effect to cause increased cardiac output however its main benefit is from its central effect of promoting vagal tone and thus slowing SA discharge and AV conduction rates. This will decrease the heart rate thus reducing myocardial oxygen consumption. It is vital to initiate therapy at the appropriate dosage and digitalise the patient. Digoxin should be dosed on lean body mass at 0.22mg/m², po, bid, and the dose reduced in animals with ascites. Due to the variable half-life in dogs accurate dosing is difficult and serum levels should be checked 5-7 days after initiating treatment. Trough concentrations (8-12 hrs post-pilling) of 0.8 – 1.2ng/ml (1 – 1.5 nmol/L being optimal. Beta-blockers can also be considered as they may have a positive long term effect by reducing the sympathetic drive and thus will protect the myocardium in the long run. Beta blockers should be used with care and introduced very slowly and never used in a patient with decompensated patient with signs of pulmonary oedema or hypotension.
References:


