Proceeding of the SEVC
Southern European Veterinary Conference

Oct. 17-19, 2008 – Barcelona, Spain

http://www.sevc.info

Reprinted in the IVIS website with the permission of the SEVC
www.ivis.org
CARDIO-PULMONARY
Canine Dilated Cardiomyopathy

Dr. Y. Martínez Pereira
LdaVet CertVC MRCVS,
Senior Clinical Scholar in Cardiopulmonary Medicine,
Hospital for Small Animals, Royal (Dick) School of Veterinary Studies,
ypereira@staffmail.ed.ac.uk

Dilated Cardiomyopathy (DCM) is the most prevalent myocardial disease in dogs but much less frequent in feline patients. In general, myocardial diseases can be classified into primary (dilated, hypertrophic, restrictive, unclassified) or secondary (toxic, drug-induced, ischaemic, infiltrative, nutritional, inflammatory/infectious, genetic).

DCM is characterised by a progressive systolic and diastolic myocardial dysfunction of unknown cause, which can present as asymptomatic, congestive heart failure or sudden death. It is more prevalent in middle-aged to elderly patients and it appears to be more severe in males of some breeds.

In dogs specifically, there are important differences associated to certain breeds. Dobermanns seem to present with more severe (and often fatal) clinical signs, but Cocker Spaniels usually show a more progressive course of the disease and Portuguese Water Spaniel tend to present an early age onset. Boxers can develop cardiomyopathy in a ‘classic dilated’ form or with arrhythmias (arrhythmogenic right ventricular cardiomyopathy). In feline patients, the incidence of dilated cardiomyopathy secondary to taurine deficiency was markedly reduced after the dietary supplementation in 1990. The incidence of DCM at present is lower than in other myocardial diseases such as hypertrophic cardiomyopathy or feline restrictive cardiomyopathy.

As in humans, a genetic predisposition for DCM is suspected, although different ways for transmission have been described in different breeds (autosomal dominant trait in Irish Wolfhounds, Terranovas and Dobermanns). Other aetiologies have been suggested (nutritional anomalies, viral agents, immune-mediated diseases, arrhythmias, etc).

The histopathological changes associated with DCM are characterised by cardiomegaly (increment in the ratio cardiac weight : body weight) with dilatation of all cardiac chambers or predominance of dilatation of left cardiac chambers and decreased myocardial thickness. Some authors have described two histological patterns in myocardial degeneration: attenuated wavy (undulating) fibres and fibro-fatty infiltration.

The pathophysiology of DCM consists on the reduction of myocardial contractility, which leads to a reduced cardiac output. This causes an activation of compensatory systems, whose aim is to try to maintain an adequate cardiac output. To achieve that, the renin-angiotensin-aldosterone system and the sympathetic system will retain sodium (and water), in order to increase the circulatory volume and will cause local vasoconstriction. This tends to increase the telodiastolic volume, increasing cardiac output (Starling’s law). However, when the compensatory mechanisms are unsuccessful, it will lead to an excessive increase in venous pressure, which will lead to effusions (‘backward’ or congestive heart failure) and myocardial remodelling (fibrosis, etc), which will then exacerbate the existing myocardial dysfunction and will reduce cardiac output (‘forward’ heart failure).

During the clinical phase of the disease, the patients usually present with signs of left-sided congestive heart failure (cough, decreased exercise tolerance, dyspnoea), right-sided congestive heart failure (abdominal and/or pleural effusions) or signs of biventricular failure. Other common symptoms are weight loss, anorexia and tachycardia. Frequent findings during physical examination are weak femoral
pulses, pulse deficits, pale mucous membranes with increased capillary refill times, muscular atrophy and poor body condition, abdominal distension with fluid thrill, jugular distension/pulsation, arrhythmias and mild to moderate systolic heart murmur and gallop rhythm. In some breeds like Dobermanns or Boxers, the first clinical sign can be syncope during exercise. In cats, anorexia, lethargy and increased respiratory rate tend to be common clinical signs, although other patients can present with severe dyspnoea due to the combination of pulmonary oedema and pleural effusion.

Some of the diagnostic techniques most commonly used are:

- Haematology and biochemistry analysis: usually unremarkable. The presence of pre-renal azotaemia is common due to neurohormonal activation.
- Troponine I: usually normal or moderate increase.
- Thyroid panel: normal levels or euthyroid sick syndrome
- Electrocardiography: the most common arrhythmias are sinus tachycardia, atrial fibrillation and ventricular ectopy (ventricular premature complexes and ventricular tachycardia), especially in Dobermanns and Boxers.
- Holter monitoring: it is commonly used to investigate the presence of arrhythmias and to evaluate the response to medical treatment. It is also used to diagnose patients during the occult phase of the disease.
- Thoracic radiography: cardiomegaly, venous congestion, presence of effusions and pulmonary oedema tend to be the most common findings in the congestive phase of the disease.
- Echocardiography: it is usually the main test to confirm the disease and rule out the presence of other cardiomyopathies. The ESVC Taskforce published in 2003 guidelines for the echocardiographic diagnosis of DCM.

The treatment of DCM consists of:

1. Reduction in afterload: arterial dilators. The most frequently used are ACE inhibitors (benazepril, enalapril, ramipril, etc), hydralazine, phosphodiesterase inhibitors (pimobendan).
2. Inotropic support: in emergency situations, the sympatoniomimetic catecholamines are often used (dobutamine), and calcium sensitizers are also used to treat chronic heart failure (pimobendan).
3. Treatment for arrhythmias: digitalis (digoxine) are frequently used to treat atrial fibrillation. Calcium channel blockers (diltiazem) are a safe and effective alternative to control ventricular rhythm in case of atrial fibrillation and can be used in combination with digoxin. Beta blockers are also a useful alternative, although contraindicated in cases of congestive heart failure due to a potent negative inotropic effect in comparison to the combination of digoxin and calcium channel blockers. To treat ventricular arrhythmias (ie ventricular tachycardia), lidocaine can be used in emergency situations and mexiletine (on its own or combined with a beta-blocker such atenolol) can be used for long term treatment. The drug of choice for ventricular arrhythmia in Boxers is sotalol.
4. Others:

   a. Beta-blockers form part of the basic treatment of DCM in humans (once the congestion signs are under control) because it seems to prolong lifespan. It is not certain if canine patients do benefit in the same way from this type of medication in DCM. Equally, it is not yet clear that medical treatment in the occult phase of the disease can actually delay the onset of clinical signs of heart failure. Carvedilol is a non-selective beta-blocker (acts on alpha and beta adrenergic receptors) with antioxidant properties and has been suggested as treatment for canine DCM.

   b. Nutraceuticals: supplementation of specific products have been recommended in
some breeds/species (taurine, L-carnitine, etc). Supplementation of ω-3 essential fatty acids (EPA, DHA) can be used in cases of cardiac cachexia.

The long term prognosis for DCM is poor, since there is no cure for the disease and it is a progressive condition. However, the short to medium term prognosis is variable and a prediction on survival time is virtually impossible. For that reason, several studies have been carried out to research indicators with prognostic value:

- In a study of 189 canine patients of different breeds (Tidholm, 1997), the three negative survival factors were:
  - Age: dogs of < 5 years presented lower survival times
  - Dyspnoea at presentation
  - Ascites

- In a more recent study with 63 canine patients of different breeds (Borgarelli, 2006), the factors associated with a poor prognosis were:
  - Severity of heart failure
  - Ascites
  - ESVI (end-systolic volume index)>140 ml/m2
  - EF< 25%
  - Pattern of restrictive transmitral flow: this was the most significant indicator of poor prognostic

The mean survival time in the first study was 27 days and 671 days in the second. The significant discrepancy can be related to the differences in the design of both studies (in the first study were included NYHA class IV heart failure patients, whereas the patients of the second study were less severely affected or even asymptomatic). As a general rule, it is useful to observe the initial response to treatment and to monitor the progression of the disease to be able to generate an accurate prognosis for each patient.

BIBLIOGRAPHY


