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Canine Cardiomyopathy: Dobermans, Boxers and More

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Arrhythmogenic Cardiomyopathy

The term "arrhythmogenic cardiomyopathy (ACM) is a useful term that refers to recurrent or persistent arrhythmia in the setting of a normal left ventricular ejection fraction. While some dogs affected with ACM clearly go on to develop classic DCM, many do not, and in some, the key to clinical management is control of the cardiac arrhythmia. ACM is particularly common in the Boxer (and some English Bulldogs) where the term arrhythmogenic right ventricular dysplasia (ARVD) is sometimes used to indicate the presumed origin of arrhythmia. The Doberman Pinscher is another breed that often manifests ventricular ectopics prior to the development of overt myocardial failure (DCM). Another common example is the Irish Wolfhound (and other giant breeds); these dogs are prone to atrial fibrillation (AF) without obvious impairment of LV contractility, a condition sometimes referred to as lone AF.

Other atrial arrhythmias may be recognized in ACM including ectopic atrial tachycardia and atrial flutter. In dogs with lone AF, the Holter data provide insight about the daily heart rate and the exercise heart rate. Average daily heart rates that exceed 90–95/min or moderate-level exercise heart rates that exceed 250/min are reasonable grounds for slowing the heart rate response to AF. This can be done with a beta-blocker such as atenolol (6.25 to 25 mg PO q12h) or metoprolol (12.5 mg PO q12h in giant breeds). The initial dose of the beta-blocker should be low to prevent lethargy, but it can be titrated up over two to four weeks to achieve an appropriate average daily rate (generally in the range of 70 to 80/min). Digoxin can be prescribed for lone AF, but cardiac glycosides are less effective for controlling excessive exercise-related rates and are not recommended by the author unless there is congestive heart failure. Diltiazem (0.5 to 1.5 mg/kg PO q8h) is very effective in controlling heart rate, but does not confer the "cardioprotection" of beta-blockers should the arrhythmia represent occult DCM.

Grading the severity of ventricular arrhythmias in terms of relative risk for sudden death is more difficult. Clearly, the presence of clinical signs (collapse, syncope) is an indication to control ventricular tachycardia if the clinician is certain that a tachyarrhythmia is the basis for the spells. If uncertain, an event monitor (client activated ECG) should be prescribed and worn by the dog. The more common problem is when there are no overt clinical signs but frequent ventricular ectopic beats. Here the clinician must attempt to judge the seriousness of the arrhythmia. If the Holter ECG shows rapid runs of ventricular tachycardia (exceeding 225/min), frequent ectopics (such as more than 7,000 per 24 hour period), or "warning" arrhythmias (such as short-coupled PVC’s, or flutter-like runs of ventricular tachycardia), antiarrhythmic therapy is recommended.

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is an idiopathic, genetic, or familial myocardial disease characterized by cardiac dilatation and reduced myocardial contractility. Histologic lesions include absence of inflammation, attenuated wavy fibers, loss of myocytes, and the presence of increased myocardial fibrosis. Coronary arteries are normal and the valves are unremarkable, except in older dogs with concurrent mitral or tricuspid valve endocardiosis. Deficiency of metabolic substrates (such as L-carnitine or taurine) is found in a minority of dogs, but the exact cause and effect relationship between these substrates and DCM is incompletely understood.

Occult DCM indicates an overtly healthy dog with echocardiographic evidence of systolic dysfunction by echocardiography. Most diagnoses are made when a breeder requests screening of an important dog or after a veterinary examination uncovers a murmur or arrhythmia. In most cases the “diagnosis” of occult DCM is based on a minor axis measure of LV systolic function (the shortening fraction). Values below 25% are considered suspicious in most laboratories, but there is no unanimity about one specific figure that indicates myocardial failure. This single linear approach can be questioned because larger dogs shorten relatively more in the apical to basilar direction and this motion is not assessed by the shortening fraction measure. Before rendering a diagnosis
of occult DCM, the clinician should request more detailed echocardiographic measures of systolic function including LV short-axis shortening area, apical-to-basilar mitral annular motion, and volumetric estimates of LV ejection fraction using the method of discs or a prolate ellipsoid model. Serial examinations also can be helpful in establishing a downward trend in LV function. Holter ECG is a useful adjunct for establishing the diagnosis in breeds prone to DCM with cardiac arrhythmias. Most would consider >50 VPC/PVC’s per day abnormal. When the diagnosis of occult DCM is certain, cardioprotection should be considered. This can be initiated with an angiotensin converting enzyme inhibitor (ACEI) such as enalapril, benazepril, ramapril (ramipril), or quinapril given once daily. If persistent arrhythmias are evident, a beta-blocker or sotalol should also be considered (see above for doses).

Advanced cases of DCM are presented with exercise intolerance and clinical signs of CHF. There can be marked weight loss and cachexia. Clinical signs of left-sided CHF include tachypnea, respiratory distress, and coughing related to pulmonary edema. Right-sided CHF is characterized by jugular pulses and jugular venous distension, hepatomegaly, and ascites. Biventricular failure includes the above findings along with pleural effusion. Auscultation may reveal atrial and ventricular gallops, systolic murmurs, or arrhythmias. The arterial blood pressure usually is normal owing to vasoconstriction and neurohormonal activation, but will be decreased in profound DCM with cardiogenic shock.

Laboratory studies support the diagnosis in advanced cases of DCM. The EKG may demonstrate abnormalities typical of cardiomegaly (wide or tall P-waves; wide or increased amplitude QRS complexes) or myocardial disease (wide QRS, slurred R-wave descent, and ST-segment coving). One or more of the aforementioned cardiac arrhythmias may be evident, though an ambulatory EKG is needed to assess the frequency of ventricular ectopic rhythms. The signal averaged EKG may demonstrate late potentials indicating increased risk for ventricular fibrillation. Thoracic radiography reveals cardiomegaly and may demonstrate typical radiographic features of heart failure. The echocardiogram shows ventricular dilation, reduced left ventricular shortening fraction, increased E-point to septal separation, decreased wall excursion, left atrial dilation and variably right-sided cardiomegaly. Doppler evidence of mitral regurgitation and tricuspid regurgitation, pulmonary hypertension, and diastolic ventricular dysfunction are common. Routine laboratory tests are usually normal or reflect intercurrent disease, consequences of CHF, or complications of CHF therapy. Specialized blood tests for L-carnitine or taurine may be performed in selected cases.

Initial hospital therapy of CHF caused by DCM includes diuresis with furosemide (2–4 mg/kg IV, IM q6-8h), supplemental oxygen, nitroglycerin ointment (1–1.5 inches for a large breed dog q12h), and rest. Life-threatening pulmonary edema can be managed with furosemide and infusion of sodium nitroprusside (0.5–2.5 mcg/kg/min) with careful attention paid to arterial blood pressure (titrate the infusion to a systolic value of 85 to 90 mm Hg). Thoracocentesis is indicated for moderate to large pleural effusions. When there is CHF with systemic hypotension, the treatment should be furosemide, oxygen, and dobutamine (2.5 to 10 mcg/kg/min). Dobutamine can have relatively long-term benefits and is continued for at least two days, at which point the drug is tapered over a 6–12 hour period while assessing blood pressure. In the setting of hypotension, vasodilators are avoided until the pressure is stabilized by dobutamine for at least two hours after which therapy with either sodium nitroprusside or an ACEI can be initiated (see below). In dogs with atrial fibrillation, digoxin (0.005 mg/kg PO q12h) is prescribed to control the ventricular rate response.

Home therapy for CHF caused by DCM includes furosemide, an ACE-inhibitor, digoxin, and sodium-restricted diet. Fluid retention is controlled with furosemide (2–4 mg/kg PO q6-12h) and sodium restriction if possible. Digoxin therapy is initiated unless there is a contraindication (moderate renal failure, complicated ventricular ectopies). An ACEI is prescribed for once daily use (with a typical dose of 0.5 mg/kg PO for enalapril or benazepril), and the dose increased to twice daily after one or two weeks of home care. Where available, pimobendan (a phosphodiesterase inhibitor-calcium sensitizing inotropic drug with vasodilating properties) should be considered based on limited but promising clinical studies. Spironolactone (12.5 to 25 mg PO q12h in a large dog) may be added to block the cardiotoxic effects of aldosterone and impede sodium retention in the distal nephron. A beta-blocker may be considered to blunt the cardiotoxic effects of the sympathetic nervous system; however, heart failure must be well controlled first.

The beta-blocker of choice in human patients is carvedilol. This drug is both a beta-blocker and alpha-adrenergic blocker (which helps to reduce the afterload on the left ventricle). Carvedilol also had anti-oxidant properties that may benefit the myocardium. Unfortunately, the prescription drug (Coreg) is expensive. Dosing can be difficult even in large dogs that may not tolerate the negative inotropy of any beta-blocker. Thus, low initial dosages are mandatory (start with ¼ to ½ of a 3.125 mg carvedilol tablet q12h). While there are clear theoretical benefits of beta-blockers in canine DCM, one’s practical ability to initiate and maintain treatment may be very limited and
CHF can worsen (often with pleural effusion). Certainly, when AF complicates CHF, either a beta-blocker or diltiazem (0.25 to 1.0 mg/kg PO q8h) is prescribed to control ventricular rate.

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