Niek J. Beijerink
DVM, PhD, DECVIM (Cardiology)

University of Sydney
Australia

niek.beijerink@sydney.edu.au

TREATMENT OF CANINE MITRAL VALVE DEGENERATION: DRUGS ON WHICH CONSENSUS IS REACHED (THE GOOD)

Mitral regurgitation (MR) caused by myxomatous mitral valve disease (MMVD) of the mitral valve leaflets and associated structures is by far the most common heart disease in dogs. Its presence is often suspected by the presence of a typical systolic heart murmur in the middle-aged to old small to medium sized dog. The initial diagnosis of MR caused by MMVD is often not complicated because the clinical (the murmur) and echocardiographic findings (MR caused by primary mitral valve lesions with or without eccentric hypertrophy of the left heart) are obvious and match. Treatment might be more controversial: At which stage is it indicated to initiate therapy? In 2009 a group of leading veterinary cardiologists published guidelines (a consensus statement) about the diagnostics and therapy of MMVD[1]. It has to be said that consensus statements by their very nature represent a compromise of positions, regardless of the subject under discussion. The ACVIM Consensus Statement on MMVD was no different. In this abstract I summarize the major points which regard to treatment of MMVD on which consensus was reached.

MMVD is a progressive disease. Because every stage of the disease requires a different type of therapy (if any therapy at all), 4 stages have been differentiated: from A through D.

Stage A:
These are dogs of predisposed breeds without any detectable abnormalities on a cardiovascular examination. An example could be a young, healthy Cavalier King Charles Spaniel without a murmur due to MR on cardiac auscultation. There is no evidence of any medication available that prevents MMVD to develop, and thus despite the high likelihood that this dog will develop the disease at an older age, any medical treatment in stage A dogs is thus not indicated.

Stage B:
These dogs have a characteristic cardiac murmur (systolic murmur with a point of maximal intensity over the point of maximum intensity of the mitral valves), but no clinical symptoms related to congestive heart failure such as dyspnea, tachypnea and coughing. All cardiologists in the panel agreed that if the heart is not enlarged (stage B1; on echocardiogram and/or chest radiographs no evidence of cardiomegaly) these dogs do not need any treatment. However, the panel did not reach a consensus about the dogs in stage B, if there is clear cardiomegaly (stage B2; cardiomegaly on chest radiographs, evidence of eccentric hypertrophy of the left heart on echocardiogram). Because there is no prove that any medication would be able to prolong the symptom-free period of dogs in this stage, many cardiologists do not recommend any medication. Others do recommend starting with lifelong use of an ACE-inhibitor. According to the strongest currently available evidence ACE-inhibitors are unlikely delaying the onset of congestive heart failure (CHF)[2], despite another paper suggesting a possible benefit of ACE-inhibitors[3]. A trial evaluation the efficacy of pimobendan in possibly reducing the progression of heart disease is under way in stage B2 dogs (and results are expected in several years).

Stage C:
These dogs are either having, or have ever had confirmed CHF (cardiogenic lung edema). All cardiologists agree that CHF can only be diagnosed with a combination of physical examination and thoracic radiographs. The most important abnormalities are: tachypnoea with or without dyspnoea, tachycardia (HR> 120 BPM; with the lack of respiratory sinus arrhythmia) as well as an enlarged left atrium in combination pulmonary venous congestion and an interstitial and/or alveolar pattern on the thoracic radiographs, typically in the hilar region. Before starting any kind of therapy (except for oxygen in cases of a severe pulmonary oedema) thoracic radiographs must be taken to evaluate the pulmonary parenchyma for the presence or absence of CHF. Many mildly to moderately symptomatic dogs may be managed on an outpatient basis. However, more severely symptomatic dogs may require hospitalization and intensive CHF therapy. These dogs are usually managed by comparably high intravenous injections of furosemide (initially 4-8 mg/kg q 2-8h; or a continuous rate infusion of 1 mg/kg/h after an initial bolus. Monitor the respiration for therapeutic effect), oxygen supplementation

and cage rest, nitroglycerin ointment and sometimes administration of a selective arterial vasodilator (such as hydralazine at 1 mg/kg q12h) ± a positive inotrope (pimobendan at 0.25-0.30 mg q 12h). Cases with severe ascites may require abdominal paracentesis as well. Once these hospitalized dogs have stabilized, they usually can be managed at home with appropriate medication. However, successful CHF therapy is always based on a good collaboration between pet owner and veterinarian. It is important that the owner is educated on the following issues: how to monitor heart rate and respiratory rate at home; importance of regular habits and medication; the possibility to modify diuretic dose within a fixed range; avoid strenuous exercise; diet; possible complications; and rechecks.
Chronic treatment of patients with CHF secondary to MMVD typically consists of diuretics on effect (i.e. furosemide (1-3 mg/kg q12-24h), pimobendan (0.25-0.30 mg/kg q12h), ACE-inhibition (mostly enalapril or benazepril; 0.25-0.50 mg/kg q24h), and sometimes additional agents. The fine-tuning of the dosages of the oral medications should be done based on careful monitoring of the respiratory rate and effort as well as the renal function.

Stage D:
These are dogs that do not respond to the highest recommended dosages of the above mentioned "CHF medications" (i.e. furosemide, pimobendan and an ACE-inhibitor). Spironolactone at a dose of 2 mg/kg q 12-24h and/or hydrochlorothiazide at a dose of 2-4 mg/kg q12h may be added in cases where the fluid retention is refractory to furosemide alone; sildenafil (1-2 mg/kg PO q12h) may be added in cases of significant pulmonary hytertension1. These latter drugs are often added stepwise as the severity of clinical signs progress.

References

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