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Cardiorespiratory

Prognostic Variables in Canine Mitral Valve Disease

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Introduction

Myxomatous mitral valve disease (MMVD) 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, and the diagnosis is usually supported by ancillary examinations such as echocardiography. Although the disease has been described and investigated ever since it was first described more than a century ago, there are surprisingly many aspects of it we still do not know, and the most important question is still to be answered. Which is (are) the primary inciting mechanism(s) that causes a dog to develop MMVD? This important question is also unknown in the human form of the disease (e.g. mitral prolapse syndrome). It is likely that the exact genetic abnormality causing MMVD will be elucidated in the future, because the disease has been shown to be inherited as an autosomal inherited polygenic threshold trait in dogs. However, if, and when, a genetic test is available, the experiences from other canine diseases, such as progressive retinal atrophy have shown that managing the disease is simply not a matter of identifying genetically abnormal individuals. There is still a need for correctly identifying the diseased phenotype, and this requires stringent diagnostic criteria and skilled examinators. Furthermore, correctly identified diseased individuals need to be managed. Because of the high prevalence of the disease and the mode of inheritance in certain affected breeds, this work is likely to be present even after the exact genetic background of MMVD has been elucidated and a genetic test is available.

Diagnosis and prognosis

The diagnosis of mitral regurgitation (MR) caused by MMVD is often not complicated because the clinical and echocardiographic findings are obvious and match. There are, however, situations where the diagnosis of MMVD may be less obvious. Especially early stages may be difficult. It may not be clinically important for managing the patient to correctly diagnose these early stages, because the effect of mild MR on the circulation is minimal, and so is the likelihood that the disease will cause clinical signs in the near future. However, it is of great importance for breeding that these dogs are correctly diagnosed as the currently employed breeding programs are founded on the principle to exclude dogs with an early onset of MMVD and to promote the use of dogs with late or no onset. Once a dog has been found to have low degree MR caused by MMVD, the question of long-term prognosis often arises. As mentioned previously, the likelihood that the disease at this stage will cause clinical signs of heart failure in the near future is low. However, some dogs do progress more rapidly to more severe forms than others. The risk factors associated with a more rapid progression for a dog with low degree MR include: breed, family, age, severity of valvular changes, and degree of MR. It is important to evaluate the presence and severity of bulging of the leaflets into the left atrium, i.e. degree of mitral valve prolapse (MVP), because dogs with MVP and MR progress more rapidly than those with MR only. The degree of leaflet displacement has also been shown to influence the long-term prognosis. Leaflet thickening has also been shown to have prognostic value, although this variable may be more difficult to evaluate depending on image quality and the lack of specific reference points to measure; leaflets may be thickened at different locations. The presence of ruptured chordae tendineae certainly also indicates a more rapid progression, although this finding is most commonly found in dogs with progressed valvular changes and significant MR, and is uncommon in dogs with less significant valve changes and mild MR. Because MMVD in small dogs is characterized by chronic progression, the owner and veterinarian often have no other alternative than to observe how the valvular lesions and MR progress slowly, often over years. At a certain point, the valve leakage can no longer be compensated for, and the forward cardiac output decreases, and the pulmonary capillary pressure exceeds the threshold for pulmonary edema. Diagnosing moderate to severe heart failure is usually not difficult as the clinical signs of congestive heart failure are usually obvious and match the findings on the radiographs, i.e. pulmonary edema and congestion. However, mild decompensated heart failure may be difficult to diagnose owing to vague clinical signs and findings and inconclusive thoracic radiographs. Furthermore, other concurrent diseases in the respiratory or the locomotor systems are common and their presence may render the diagnosing even more difficult. Thus, there is a need for clinical parameters that have prognostic and/or diagnostic value.

Diagnostic and prognostic indicators

Several physical, echocardiographical and hormonal parameters have been evaluated for these aspects. At present, the left atrial dimension (LA/AO ratio, LA/
rupture of a higher-order chordae are all associated with non-responsive pulmonary hypertension, or evidence of episodes of ventricular tachycardia, evidence of fibrillation, frequent ventricular depolarizations or Finally, the presence of a complication, such as atrial the dose, the shorter expected survival. The presence of MR. Furthermore, the required maintenance dose of furosemide is also associated with survival. The greater predicted AO etc), the vertebral heart score (VHS), and the natriuretic peptides (NP), the heart rate variability (vasovagal tonus index etc) stand out as being most reliable in predicting the presence of decompensated heart failure. The same variables stand out as having best prognostic value, as significant changes occur in all of them 0.5 to 1 year before the onset of clinical signs of decompensated heart failure. As the development of pulmonary oedema and congestion is dependant on increased pulmonary venous and capillary pressures, it is not surprising the left atrial dimension and NP have diagnostic and prognostic values. Both the atrial and ventricular dimensions contribute to the VHS score, but it appears that the left atrial size contributes significantly more to an increase in VHS score in dogs with MR. Because the increase of regurgitant volume is decisive for any change in left atrial size, NP concentration and heart rate, we can assume that an accurate estimation of regurgitant volume would be a parameter with good predictive and diagnostic value. The regurgitant volume is, today, most commonly estimated by Doppler echocardiography. Although the Doppler techniques are efficient in detecting MR and discriminating low degree MR from moderate to severe, they are less efficient in separating moderate MR from severe. This is a problem because dogs developing decompensated heart failure presumably increase their MR from moderate to severe, which means that Doppler derived estimates of MR are not parameters of high diagnostic and prognostic value. With more advanced technology, this is likely to change in the future. Once heart failure signs have developed and heart failure therapy is instituted, there is often interest for the owner and for the clinician to have access to variables that are informative of the long-term prognosis. Results from clinical trials indicate that cardiac size is prognostic for survival time. Measurements such as LA/AO, VHS and left ventricular diameter in systole are highly prognostic, which is not surprising as they are dependent on severity of MR. Type of treatment has also been shown to influence survival; drugs such angiotensin-convertin-enzyme (ACE) inhibitors, pimobendan, and spironolactone have been shown to increase survival in dogs with symptomatic MR. Furthermore, the required maintenance dose of furosemide is also associated with survival. The greater the dose, the shorter expected survival. The presence of exercise intolerance and cardiac cachexia (physical or laboratory signs) are associated with poorer prognosis. Finally, the presence of a complication, such as atrial fibrillation, frequent ventricular depolarizations or episodes of ventricular tachycardia, evidence of myocardial infarction and/or intracardiac thrombus, non-responsive pulmonary hypertension, or evidence of rupture of a higher-order chordae are all associated with a poor prognosis.

Breed differences
It should be emphasized that the bulk of the available information concerns MR in small to medium sized dogs. Presumably, there are breed-differences between different small breed dogs, but we know little of those. We also know little about MR in large dogs. Because we have assumed that dogs of various breeds and shapes have the same expression of disease and respond to it in a similar way, we have, with a few exceptions, considered significant MMVD a comparably rare condition in large dogs. Through the years, there have been a few reports focusing on primary MR in large dogs. It has been the clinical impression that some large dogs may present with massive MR and myocardial failure and arrhythmia (atrial fibrillation and ventricular ectopy) in the presence of comparably subtle changes in mitral valve morphology, and do not present the ‘classical’ thickening and prolapse of the mitral valve leaflets so common in the small dog. Why these large dogs have such prominent MR despite relatively minor changes of the mitral valve and why they seem to tolerate the MR so much poorer than the typical small dog is currently not known. Further research is warranted in this area.

Selected references

The complete list of references is available from the author.

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