The ABCDs of MMVD: Diagnosis and Staging of MMVD in Real-World Patients

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Introduction

Myxomatous mitral valve disease (MMVD) makes up 75% of canine cardiac disease and has a prevalence of 10-15% in the general dog population[1]. It is the most common acquired cardiac disease in dogs and warrants an understanding by the general practitioner. This discussion will review the pathophysiology of MMVD, outline techniques to diagnose MMVD, and review the stages of MMVD.

Pathophysiology of MMVD

The mitral valve apparatus consists of the mitral valve annulus, anterior and posterior mitral valve leaflets, the chordae tendineae, and the papillary muscles in the left ventricle. MMVD is defined as a myxomatous change to the mitral valve leaflets. Myxomatous change is a non-inflammatory, non-infectious pathology caused by a proliferation of the inner layers of the valve leaflets. This change tends to affect the anterior leaflet more than the posterior leaflet which results in an eccentric jet of mitral regurgitation. The chordae tendineae can also thicken and weaken/tear which results in valve leaflet prolapse and more significant regurgitation. The mitral regurgitation (MR) is what is heard as a left apical systolic murmur on physical exam. MMVD is most common in small and medium size breed dogs of mature age. There is a slight male predisposition.

Over time, this regurgitation results in a volume overload to the left atrium and left ventricle causing eccentric hypertrophy (dilation). The rate of this progression is variable and largely based on the degree of regurgitation (MR). The end stage of this disease is left sided congestive heart failure. Fortunately, there are numerous tools at the disposal of general practitioners to diagnose, stage, and treat MMVD.

Diagnosis of MMVD

There are a variety of tools used to diagnose MMVD, many of which are available in a private practice setting. One of the most important is the cardiovascular physical examination. Dogs with MMVD of hemodynamic significance will have a left apical systolic heart murmur. This is a defining feature of the disease process. The intensity of the murmur does not *always* correlate to the severity of the disease, but in general, the greater the intensity the more severe the MR.

A blood pressure is recommended in any patient > six years of age with a murmur on physical exam. The intention of this diagnostic in even an asymptomatic patient is to screen for systemic hypertension (systolic blood pressure > 160 mmHg)[2]. Systemic hypertension results in an increased afterload on the left ventricle. In the presence of MR, this will lead to worsened heart disease.

Thoracic radiographs are another pillar of diagnosing and staging MMVD. Orthogonal views are essential to allow thorough interpretation. Thoracic radiographs allow for evaluation of the cardiac silhouette, pulmonary vasculature, pulmonary parenchyma, mediastinum, trachea, pleural space, and other extra thoracic structures. Thoracic radiographs can be used to stage MMVD; this will be discussed later.

An electrocardiogram (ECG or EKG) also has some utility in MMVD. A diagnostic ECG can be used to assess for chamber enlargement patterns (eg wide P waves or tall R waves consistent with left atrial and left ventricular enlargement, respectively). A rhythm ECG will assess for arrhythmias. Supraventricular arrhythmias such as atrial premature complexes (APCs) and/or atrial fibrillation (AF) tend to be more common than ventricular arrhythmias in MMVD but both are possible and distinguishable with a rhythm/single lead II ECG.

Cardiac biomarkers such as a troponin (cTnI) and/or natriuretic peptides (NT-proBNP) are available through some laboratories. Their use may be more valuable in cases of feline cardiac disease and canine dilated cardiomyopathy given cardiomyopathy may not have an audible murmur to alert the clinician to the presence of disease. There is breed variation in the NT-proBNP among canines[3] which can make interpretation challenging. However, it may be useful for monitoring a single patient or to know when a referral to a cardiologist is more urgent[4]. If a cardiologist is not available, then additional diagnostics can be done to determine the best treatment recommendations. Ultimately, this cardiologist recommends thoracic radiographs rather the NT-proBNP or cTnI in dogs where MMVD is the top differential.

Ultimately, an echocardiogram is the gold standard for diagnosing the etiology of cardiac disease. An echocardiogram should be performed by a cardiologist or trained sonographer with access to a cardiologist to review. An echocardiogram will allow for a definitive diagnosis as well as the secondary changes to the heart such as degree of valve thickening, degree of MR, degree of left heart enlargement, estimation of left atrial pressure, and heart function.

Staging of MMVD

The ACVIM Consensus Statement from 2019[1] has an updated staging schematic for dogs with MMVD. The stages and abbreviated treatment recommendations are as follows:

Stage A

- Patient is AT RISK for disease
- No diagnostics needed for diagnosis
- Client education and preparation is key
- Recommend monitoring plan
- Do not breed until older (>6 years)
- No medications/therapy indicated

Stage B1

- Patient with myxomatous valve changes
- Usually identified based on presence of murmur
- No to mild cardiac remodeling
- Unlikely to develop clinical signs in < 12 months
- No treatment/medications proven beneficial at this stage

Stage B2

- Patient with myxomatous valve changes
- Tend to have a louder murmur
- **May or may not have clinical signs associated with cardiac disease**
- At least moderate cardiac remodeling
    - VLAS ≥2.5, VHS ≥11
- No evidence of *congestive* heart failure
- *These patients are believed to benefit from pimobendan therapy*
- Diet change can be considered

Stage C

- Patient with myxomatous valve changes
- Clinical signs and/or diagnostic findings consistent with congestive heart failure (CHF)
- Require quadruple therapy
    - Loop diuretic
    - Pimbobendan
    - ACE-inhibitor
    - Aldosterone antagonist
- Diet change is recommended

Stage D

- Patient with myxomatous valve changes
- Patient with persistent clinical signs despite traditional therapy
- > 8 mg/kg/day furosemide or equivalent
- Higher doses or additional medications are required to support quality of life
- Afterload reducers, anti-arrhythmics, cough suppressants, sildenafil, etc
- Ideally on a calorie-dense, high protein, low sodium diet with omega-3 FA

References:

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