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Echocardiographic Diagnosis and Management of Myxomatous Mitral Valve Disease in a Small Breed Dog

*Shruti Dehru[1] and Dr. Raksha Devi[2]
[1]PhD Scholar, Division of Medicine, Indian Veterinary Research Institute (IVRI), Izzatnagar, Bareilly, Uttar Pradesh, India
[2]Assistant Professor, Veterinary Surgery and Radiology, Abhilashi University, Mandi, Himachal Pradesh, India
*Corresponding Author's email: shrutideryan11@gmail.com

Myxomatous mitral valve disease (MMVD) is the most prevalent acquired cardiac disorder in dogs, accounting for approximately 70–80% of canine heart diseases. The disease primarily affects small- and medium-sized breeds, particularly elderly dogs such as Cavalier King Charles Spaniels, Poodles, Dachshunds, Chihuahuas, Shih Tzus, Miniature Schnauzers, and Pomeranians. MMVD is characterized by progressive myxomatous degeneration of the mitral valve leaflets and chordae tendineae, resulting in mitral regurgitation, progressive left atrial and ventricular remodeling, pulmonary venous congestion, and eventually congestive heart failure (CHF). Echocardiography has become the gold standard imaging modality for diagnosis, disease staging, prognostication, and therapeutic monitoring of MMVD because it provides comprehensive anatomical and functional assessment of the mitral valve and cardiac chambers without invasive procedures. The integration of two-dimensional (2D), M-mode, Doppler, color flow Doppler, tissue Doppler imaging, and advanced echocardiographic techniques enables accurate evaluation of disease severity and guides evidence-based clinical management. This review discusses the pathophysiology, echocardiographic findings, disease staging according to the American College of Veterinary Internal Medicine (ACVIM) guidelines, therapeutic management, prognosis, and future developments in the diagnosis and treatment of MMVD in small breed dogs.

Introduction

Myxomatous mitral valve disease represents the most common chronic degenerative valvular disorder affecting companion dogs worldwide. The disease predominantly affects geriatric small breed dogs, although early onset has been documented in genetically predisposed breeds. The prevalence increases substantially with age, affecting nearly 30% of dogs older than ten years and more than half of dogs over thirteen years of age. Among predisposed breeds, the Cavalier King Charles Spaniel exhibits an inherited predisposition with disease onset occurring considerably earlier than in other breeds, suggesting a strong genetic component in disease pathogenesis.

MMVD is characterized by progressive structural degeneration of the mitral valve apparatus, involving thickening of valve leaflets due to accumulation of glycosaminoglycans, disruption of collagen architecture, activation of valvular interstitial cells, and elongation or rupture of chordae tendineae. These pathological alterations impair complete valve closure during systole, leading to mitral regurgitation and chronic volume overload of the left atrium and left ventricle. Over time, compensatory cardiac remodeling progresses toward decompensated heart failure, pulmonary edema, arrhythmias, pulmonary hypertension, and sudden cardiac death in advanced stages.

Early diagnosis of MMVD is critical because therapeutic intervention during asymptomatic stages has been shown to delay the onset of congestive heart failure and improve survival. Although cardiac auscultation remains an important screening tool, the intensity of the systolic murmur does not always correlate with disease severity. Thoracic radiography provides valuable information regarding cardiac enlargement and pulmonary edema but lacks sensitivity for early disease detection. Echocardiography has therefore become the diagnostic cornerstone for evaluating mitral valve morphology, quantifying regurgitation severity, assessing chamber enlargement, measuring systolic function, estimating pulmonary pressures, and monitoring disease progression.

Etiopathogenesis

The pathogenesis of MMVD involves complex molecular, cellular, and biomechanical mechanisms. Degenerative changes begin with activation of valvular interstitial cells into myofibroblast-like phenotypes under the influence of transforming growth factor-beta (TGF-β), serotonin signaling, inflammatory mediators, oxidative stress, and extracellular matrix remodeling enzymes. Progressive degradation of collagen fibers accompanied by excessive deposition of proteoglycans produces the characteristic myxomatous thickening of valve leaflets. Matrix metalloproteinases contribute to extracellular matrix degradation, while alterations in elastin architecture reduce leaflet tensile strength and elasticity. These structural abnormalities lead to leaflet prolapse and incomplete coaptation during systole. Mitral regurgitation causes chronic volume overload of the left atrium and left ventricle. Initially, compensatory eccentric hypertrophy maintains adequate cardiac output through the Frank–Starling mechanism. Continued regurgitant volume eventually overwhelms compensatory mechanisms, leading to elevated left atrial pressure, pulmonary venous hypertension, pulmonary edema, decreased forward stroke volume, neurohormonal activation, myocardial fibrosis, and congestive heart failure.

Activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system further accelerates disease progression by promoting sodium retention, ventricular remodeling, myocardial fibrosis, endothelial dysfunction, and increased afterload.

Clinical Presentation

Clinical manifestations vary considerably depending on disease stage. Dogs in early disease often remain asymptomatic despite significant structural valve abnormalities. A left apical systolic murmur detected during routine physical examination frequently represents the earliest clinical finding. As disease progresses, owners may observe exercise intolerance, reduced activity, mild coughing, tachypnea, increased respiratory effort, weakness, and episodic syncope. Development of pulmonary edema results in severe dyspnea, orthopnea, cyanosis, and respiratory distress requiring emergency intervention. Physical examination commonly reveals a left apical holosystolic murmur, hyperdynamic precordial impulse, irregular cardiac rhythm in dogs with atrial fibrillation, increased respiratory rate, pulmonary crackles, weak femoral pulses in advanced heart failure, and cachexia in terminal disease.

Role of Echocardiography

Echocardiography provides comprehensive non-invasive assessment of cardiac anatomy and function and remains the gold standard for diagnosis of MMVD. It enables visualization of mitral valve morphology, quantification of regurgitation severity, measurement of cardiac chamber dimensions, evaluation of systolic and diastolic function, identification of pulmonary hypertension, and exclusion of congenital or acquired cardiac diseases with similar clinical presentations. Serial echocardiographic examinations allow monitoring of disease progression and optimization of therapeutic strategies throughout the patient's lifetime.

Two-Dimensional Echocardiographic Findings

Two-dimensional echocardiography demonstrates characteristic morphological abnormalities involving the mitral valve apparatus. The mitral valve leaflets appear diffusely thickened,

irregular, and nodular due to myxomatous degeneration. Leaflet prolapse into the left atrium during systole is frequently observed. In advanced disease, ruptured chordae tendineae may produce a flail leaflet with severe eccentric mitral regurgitation. Left atrial enlargement develops progressively and serves as one of the most important prognostic indicators. Enlargement of the left ventricle occurs secondary to chronic volume overload, producing eccentric ventricular remodeling. Severe cases may demonstrate spontaneous echocardiographic contrast or intracardiac thrombi within markedly enlarged atria. Pulmonary artery enlargement and right ventricular remodeling may indicate secondary pulmonary hypertension.

M-Mode Echocardiography

M-mode imaging provides quantitative assessment of cardiac dimensions and systolic performance. Common findings include increased left ventricular internal diameter during diastole (LVIDd), increased left ventricular internal diameter during systole (LVIDs), increased fractional shortening during compensated disease, and reduced systolic function in late-stage myocardial failure. Hyperdynamic ventricular contraction often reflects compensatory responses to chronic volume overload rather than improved myocardial contractility.

Color Flow Doppler Evaluation

Color Doppler imaging identifies and semi-quantifies mitral regurgitation. Regurgitant jets appear as turbulent multicolored flow extending from the left ventricle into the left atrium during systole. Jet direction varies according to leaflet involvement and may be central or eccentric. Although jet area provides a rapid estimate of regurgitation severity, interpretation should consider technical factors such as gain settings, Nyquist limit, atrial size, and jet eccentricity. Multiple regurgitant jets may indicate extensive valvular degeneration.

Spectral Doppler Assessment

Continuous-wave Doppler measures peak mitral regurgitant velocity, while pulsed-wave Doppler evaluates transmitral inflow patterns and diastolic function. Elevated E-wave velocity often indicates increased left atrial pressure and worsening disease severity. Restrictive filling patterns suggest advanced diastolic dysfunction and poor prognosis. Tricuspid regurgitation velocity enables estimation of pulmonary artery pressure and identification of secondary pulmonary hypertension.

Tissue Doppler Imaging and Advanced Echocardiography

Tissue Doppler imaging evaluates myocardial velocities and provides sensitive assessment of ventricular relaxation and systolic function before conventional parameters become abnormal. Speckle-tracking echocardiography measures myocardial strain and strain rate, facilitating early detection of myocardial dysfunction associated with chronic mitral regurgitation. Three-dimensional echocardiography allows improved visualization of mitral valve anatomy and may assist surgical planning in specialized referral centers.

Echocardiographic Measurements Used in MMVD

Several standardized echocardiographic indices are routinely employed to determine disease severity. The left atrial-to-aortic root ratio (LA/Ao) remains one of the most important measurements, with values exceeding 1.6 indicating clinically significant left atrial enlargement. Normalized left ventricular internal diameter in diastole (LVIDDN) greater than 1.7 indicates substantial ventricular enlargement associated with Stage B2 disease. Fractional shortening, ejection fraction, E/A ratio, E/e′ ratio, tricuspid regurgitation velocity, pulmonary artery acceleration time, and left ventricular strain analysis provide additional information regarding ventricular function and prognosis.

ACVIM Staging of MMVD

The American College of Veterinary Internal Medicine classifies MMVD into four stages.
Stage A includes dogs predisposed to MMVD but lacking structural abnormalities.

Stage B1 comprises asymptomatic dogs with mitral regurgitation but no radiographic or echocardiographic evidence of cardiac enlargement.
Stage B2 includes asymptomatic dogs with significant cardiac remodeling characterized by left atrial enlargement and ventricular dilation.
Stage C represents dogs with current or previous episodes of congestive heart failure requiring medical therapy.
Stage D includes dogs with refractory heart failure that no longer respond adequately to standard medical treatment.
Accurate echocardiographic staging directly influences therapeutic recommendations and prognosis.

Medical Management

Treatment strategies depend on disease stage and echocardiographic findings. Dogs classified as Stage B1 generally require periodic clinical and echocardiographic monitoring without pharmacological intervention. Dogs with Stage B2 disease benefit from administration of pimobendan, which has been demonstrated to delay the onset of congestive heart failure by improving myocardial contractility and reducing afterload. Stage C patients require combination therapy consisting of pimobendan, furosemide, angiotensin-converting enzyme inhibitors, and spironolactone. Supplemental oxygen, thoracocentesis for pleural effusion when indicated, dietary sodium restriction, and careful monitoring of renal function and electrolyte balance remain essential components of treatment. Stage D disease necessitates individualized therapeutic adjustments, including higher diuretic dosages, torsemide substitution, vasodilators, antiarrhythmic drugs, positive inotropes, and palliative supportive care.

Monitoring Disease Progression

Regular follow-up examinations are essential for long-term management. Serial echocardiography permits objective assessment of mitral regurgitation severity, chamber enlargement, ventricular function, pulmonary hypertension, and therapeutic response. Thoracic radiography assists evaluation of pulmonary edema and vertebral heart score.
Blood biomarkers including N-terminal pro-B-type natriuretic peptide (NT-proBNP) and cardiac troponin I provide complementary information regarding myocardial stress and injury. Owners should be instructed to monitor resting respiratory rate, appetite, exercise tolerance, coughing frequency, and body weight at home to facilitate early recognition of heart failure.

Prognosis

Prognosis depends primarily upon disease stage, degree of cardiac remodeling, pulmonary hypertension, occurrence of arrhythmias, and response to treatment. Dogs diagnosed during Stage B2 and treated appropriately often remain clinically stable for several years before developing congestive heart failure. Once CHF develops, median survival varies considerably depending upon therapeutic response, owner compliance, and presence of concurrent diseases. Early echocardiographic diagnosis combined with timely initiation of evidence-based medical therapy substantially improves both survival time and quality of life.

Future Perspectives

Recent advances in veterinary cardiology are improving understanding of MMVD pathogenesis and clinical management. Artificial intelligence-assisted echocardiographic image analysis, machine learning algorithms for automated disease staging, portable handheld ultrasound devices, and tele-echocardiography are expanding access to specialized cardiac diagnostics. Research into circulating biomarkers, genomic susceptibility loci, proteomics, metabolomics, and molecular imaging may enable earlier diagnosis before structural valve degeneration becomes clinically evident. Novel pharmacological agents targeting fibrosis, extracellular matrix remodeling, serotonin signaling, and inflammatory pathways may further delay disease progression. Minimally invasive transcatheter mitral

valve repair and advanced surgical valve reconstruction techniques continue to evolve in veterinary medicine and may become increasingly accessible for selected canine patients in specialized referral centers.

Conclusion

Myxomatous mitral valve disease remains the most common acquired cardiac disease affecting small breed dogs and represents a major cause of morbidity and mortality in geriatric canine populations. Echocardiography is indispensable for accurate diagnosis, disease staging, prognostic assessment, and therapeutic decision-making because it provides comprehensive evaluation of mitral valve morphology, regurgitation severity, cardiac remodeling, ventricular function, and pulmonary hypertension. The incorporation of conventional and advanced echocardiographic techniques has significantly enhanced the ability to detect disease during preclinical stages and to guide evidence-based management. Early diagnosis, adherence to ACVIM staging guidelines, timely initiation of appropriate pharmacological therapy, regular echocardiographic monitoring, and owner education collectively improve clinical outcomes, delay the onset of congestive heart failure, and enhance the quality and duration of life in dogs affected by MMVD.

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