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Evaluation of myxomatous mitral valve disease in dogs

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ABSTRACT: This study aimed to evaluate the prevalence, clinical characteristics, diagnostic indicators, and treatment outcomes of myxomatous mitral valve disease (MMVD) in dogs in Vietnam at Procure Veterinary Hospital. From October 2024 to October 2025, a total of 3,840 dogs presenting with respiratory signs or undergoing health screening were examined. Dogs suspected of cardiac disease were further evaluated using thoracic radiography, echocardiography, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) quantification. A total of 232/3,840 dogs (6.04%) were suspected of cardiac disease, of which 84 dogs (36.21%) were confirmed to have MMVD, accounting for 2.19% of all examined dogs. The most common clinical signs were dyspnea/pulmonary edema and cough, with increasing frequency from stage B1 to C. Cardiac murmur intensity increased with disease progression, with most dogs in stages B2 and C having murmurs \geq grade III/VI. Echocardiographic variables of left-sided volume overload, including vertebral heart size (VHS), left atrium to aorta ratio (LA/Ao), left ventricular internal diameter in diastole (LVIDd), and systole (LVIDs), increased significantly across stages ($p < 0.001$), whereas fractional shortening showed no significant change ($p = 0.575$). NT-proBNP concentrations increased from 500 pg/mL (B1) to 953.8 ± 294.0 pg/mL (B2) and $2,131.8 \pm 354.4$ pg/mL (C). Treatment outcomes were favourable in stage B2 (98.18%) but markedly lower in stage C (43.48%). These findings highlight the importance of combined clinical, imaging, and biomarker evaluation for accurate staging, early detection, and improved management of MMVD in dogs.

Keywords: Myxomatous mitral valve disease; dogs; echocardiography; NT-proBNP.

Highlights

- Myxomatous mitral valve disease affected 2.19% of examined dogs; respiratory signs increased with disease stage.
- Echocardiographic variables of left-sided volume overload and NT-proBNP increased significantly from stage B1 to C.
- Early-stage treatment was highly successful, but outcomes declined markedly in stage C.

1.0 Introduction

Myxomatous mitral valve disease (MMVD) is one of the most common acquired cardiac diseases in dogs, particularly prevalent in small breeds and geriatric animals. The disease is characterized by progressive degeneration of the mitral valve leaflets, leading to incomplete valve closure, mitral regurgitation, left atrial enlargement, and chronic volume overload, which may eventually progress to congestive heart failure if not diagnosed and managed appropriately (Keene et al., 2019; Häggström et al., 2008).

In Vietnam, particularly in Ho Chi Minh City, published data on MMVD remain limited. Previous studies in small animal practice have primarily focused on clinical presentation and basic diagnostic findings, with reported prevalence ranging from approximately 1.5% to 3% among examined dog populations. However, these studies have not comprehensively evaluated the combined use of echocardiography, thoracic radiography, and cardiac biomarkers such as N-terminal pro-B-type natriuretic peptide (NT-proBNP) in disease staging and monitoring. Moreover, detailed analyses of treatment outcomes across different ACVIM stages are still lacking in the local context.

Therefore, this study aimed to evaluate the prevalence, clinical characteristics, diagnostic indicators, and treatment outcomes of MMVD in dogs at a veterinary hospital in Vietnam. By integrating clinical examination, imaging modalities, and biomarker assessment, this study seeks to provide a more comprehensive understanding of MMVD and to address existing gaps in diagnosis and disease management under practical veterinary conditions.

2.0 Materials and methods

2.1. Study Site, Animals, Housing, and Experimental Design

The study was conducted from October 2024 to October 2025 at Procure Veterinary Hospital, Phu Nhuan District, Ho Chi Minh City.

Dogs included in the study were those presenting with clinical signs such as cough, dyspnea, exercise intolerance, fatigue, or syncope, as well as dogs without clinical signs that were brought for routine annual health screening or pre-anesthetic examination.

2.2. Data Collection

2.2.1. Clinical Examination and Classification of Dogs Suspected of MMVD

After collecting a complete history from the owner, dogs underwent a general clinical examination with particular focus on the cardiovascular system. Cardiac auscultation was performed using a stethoscope to detect systolic murmurs, a characteristic sign of MMVD, most clearly heard at the 4th–6th intercostal spaces (Turgut et al., 2019). Murmur intensity was graded from I to VI according to severity (Pedersen et al., 1999).

Heart rate was recorded, with normal physiological values ranging from 60 to 160 beats per minute (Ettinger et al., 2017). Peripheral pulse was assessed to identify eventual weak or irregular pulses that may be associated with cardiac dysfunction.

Resting respiratory rate was measured, with normal values below 30 breaths per minute, and values above 40 breaths per minute often suggesting congestive heart failure (Goutal et al., 2010)

2.2.2. Procedures for Assessing Paraclinical Parameters in MMVD

Paraclinical evaluation followed a standardized protocol including thoracic radiography, echocardiography, and NT-proBNP testing.

Thoracic Radiography:

Thoracic radiography was performed in dogs presenting respiratory signs or when cardiac disease was suspected based on clinical examination. Dogs were positioned in right lateral and ventrodorsal recumbency with gentle restraint. Images were obtained using a digital X-ray system and analyzed for cardiac size and shape, left atrial and ventricular

enlargement, and signs of pulmonary edema or pleural effusion. The vertebral heart size (VHS) was calculated according to Buchanan and Bücheler, with normal values ranging from 8.7 to 10.5 vertebrae (Buchanan & Bücheler, 1995).

Echocardiographic Evaluation:

Echocardiography was performed in all dogs suspected of MMVD based on clinical or radiographic findings. Dogs were positioned in lateral recumbency, and transthoracic echocardiography was conducted using two-dimensional (2D), M-mode, and color Doppler imaging. Two-dimensional imaging was used to evaluate mitral valve morphology.

M-mode was used to measure left ventricular internal diameter in diastole (LVIDd) and left ventricular internal diameter in systole (LVIDs). These values were normalized to body weight to obtain LVIDd normalized (LVIDdN) and LVIDs normalized (LVIDsN) according to Cornell et al. (2004). Fractional shortening (FS) was also calculated.

The left atrium to aorta ratio (LA/Ao) was measured to assess left atrial enlargement, with values > 1.6 indicating significant enlargement (Keene et al., 2019). Color Doppler imaging was used to detect and assess mitral regurgitation. Additional parameters included ejection fraction (EF) and E/A ratio for evaluation of systolic and diastolic function (Oyama et al., 2009).

NT-proBNP Testing:

NT-proBNP testing was performed in a subset of dogs with suspected MMVD to support diagnosis and assess disease severity. Blood samples were collected from peripheral veins, processed according to the manufacturer's instructions, and analyzed using a fluorescence analyzer.

NT-proBNP concentrations < 900 pmol/L were considered within normal limits, whereas values > 1500–1800 pmol/L indicated increased risk of heart failure, and concentrations > 2000 pmol/L were commonly associated with overt congestive heart failure (Oyama et al., 2009).

2.2.3. Classification of Dogs with MMVD

Based on clinical findings and results from diagnostic imaging and biomarker analysis, dogs diagnosed with MMVD were classified into stages B1, B2, C, and D according to the criteria established by the American College of Veterinary Internal Medicine (ACVIM) guidelines (Keene et al., 2019).

2.3. Statistical Analysis

Data were collected on an individual basis and analyzed using Excel 2010 and Minitab 17. Quantitative variables were expressed as mean \pm standard deviation (SD) for normally distributed data, or as median (minimum–maximum) for non-normally distributed data. Comparisons of means were performed using one-way analysis of variance (ANOVA, F-test) followed by Tukey's post-hoc test for normally distributed variables. For non-normally distributed data, appropriate non-parametric tests were applied. Percentages were compared using the Chi-square (χ^2) test. Differences were considered statistically significant when $p \leq 0.05$.

3.0 Results

3.1. Survey of Dogs Suspected of and Diagnosed with MMVD

From October 2024 to October 2025, a total of 3,840 dogs were presented for health screening or examination due to respiratory signs at Procure Veterinary Hospital. Dogs showing clinical signs suggestive of cardiac disease were selected for further evaluation using diagnostic imaging and supportive laboratory tests; the results are presented in Figure 1.

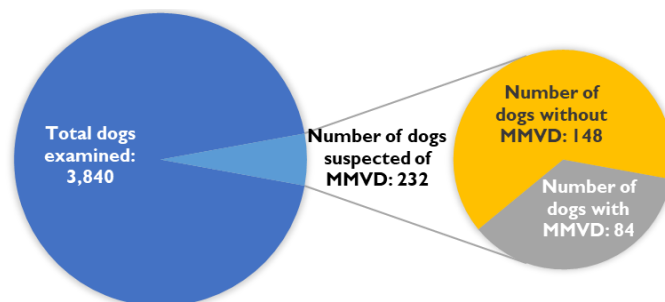


Fig. 1. Number of Dogs Suspected of MMVD and Dogs Diagnosed with MMVD at Procure Veterinary Hospital

According to Figure 1, 232/3,840 dogs (6.04%) were suspected of cardiac disease based on clinical signs such as heart murmur, coughing, and reduced exercise tolerance. Echocardiography confirmed MMVD in 84/232 dogs (36.21%), corresponding to 2.19% of all examined dogs.

3.2. Frequency of Clinical Signs in Dogs with MMVD

The frequency of clinical signs in dogs with MMVD is presented in Table 1.

Table 1. Frequency of Clinical Signs in Dogs with MMVD (n = 84)

Clinical sign	Stage B1		Stage B2		Stage C		Total		P-value
	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	
Tachypnea/ Dyspnea/ Pulmonary oedema	0	0.00	55	36.67	23	44.23	78	37.50	0.000
Cough	0	0.00	53	35.33	13	25.00	66	31.73	
Asymptomatic / Decreased appetite	6	100	22	14.67	1	1.92	29	13.94	
Reduced exercise tolerance	0	0.00	19	12.67	2	3.85	21	10.10	
Syncope / Transient loss of consciousness	0	0.00	1	0.66	13	25.00	14	6.73	

The most common clinical signs were dyspnea/pulmonary oedema (37.50%) and cough/respiratory inflammation (31.73%).

In stage B1, clinical signs were minimal, with most dogs presenting only cardiac murmurs. In stage B2, dyspnea/pulmonary oedema (36.67%), cough (35.33%), and decreased appetite (14.67%) were observed.

In stage C, dyspnea/pulmonary oedema (44.23%), syncope (25.00%), and reduced exercise tolerance were recorded.

3.3. Classification of Cardiac Murmur Intensity by Disease Stage According to ACVIM

Cardiac murmur intensity increased across disease stages (Table 2).

Table 2. Cardiac Murmur Intensity by Disease Stage According to ACVIM

Heart murmur grade	Stage B1		Stage B2		Stage C		Total		P-value
	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	Number of dogs	Percentage (%)	
0/VI	2	33.33	0	0.00	0	0.00	2	2.38	0.000
I/VI	3	50.00	2	3.64	0	0.00	5	5.95	
II/VI	1	16.67	12	21.82	0	0.00	13	15.48	
III/VI	0	0.00	31	56.35	6	26.09	37	44.05	
IV/VI	0	0.00	8	14.55	12	52.17	20	23.81	
V-VI/VI	0	0.00	2	3.64	5	21.74	7	8.33	
Total	6	100	55	100	23	100	84	100	

In stage B1, murmurs were predominantly low grade. In stage B2, grade III/VI murmurs were most frequent (56.35%).

In stage C, murmurs were mainly grade IV/VI (52.17%) and grades V–VI/VI (21.74%), although grade III/VI murmurs were also observed (26.09%).

3.4. Radiographic and Echocardiographic Indices in Dogs at Different ACVIM Stages

Radiographic and echocardiographic parameters are presented in Table 3 and Figures 2–3.



Fig. 2. Measurement of VHS in Dogs with MMVD

The vertebral heart size (VHS) increased significantly from stage B1 to B2 and C ($p < 0.001$), with mean values of 10.00 ± 0.19 , 10.85 ± 0.18 , and 11.66 ± 0.32 , respectively.

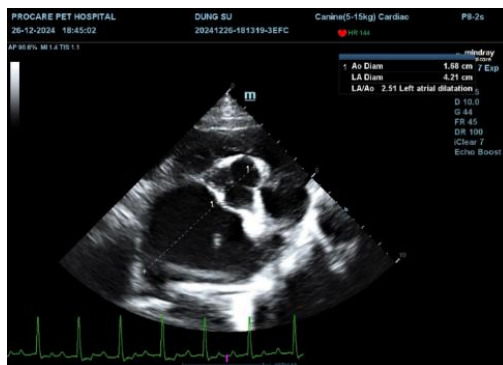


Fig. 3. Measurement of the LA/Ao Ratio in Dogs with MMVD

The left atrium to aorta ratio (LA/Ao) increased significantly ($p < 0.001$), from 1.38 ± 0.10 (B1) to 1.76 ± 0.08 (B2) and 2.18 ± 0.17 (C).

Table 3. Radiographic and Echocardiographic Parameters in Dogs with MMVD at Different ACVIM Stages

Stage	Statistical parameters	Indices				
		VHS	LA/Ao	FS	LVII Dd	LVIDs
B1	n	6	6	6	6	6
	Mean	10.00 ^c	1.38 ^c	59.82 ^c	1.47 ^c	0.67 ^c
	SD	0.19	0.10	9.35	0.03	0.12
	Min	9.80	1.26	40.51	1.42	0.54
	Max	10.30	1.51	60.72	1.50	0.85
B2	n	55	55	55	55	55
	Mean	10.85 ^b	1.76 ^b	52.03 ^a	1.75 ^b	0.81 ^b
	SD	0.18	0.08	9.47	0.03	0.10
	Min	10.60	1.62	37.26	1.70	0.65
	Max	11.20	1.91	80.35	1.84	0.99
C	n	23	23	23	23	23
	Mean	11.66 ^a	2.18 ^a	49.70 ^a	2.03 ^a	0.99 ^a
	SD	0.32	0.17	9.90	0.06	0.09
	Min	11.00	1.96	29.64	1.70	0.79
	Max	12.00	2.50	81.49	1.84	1.10
P – value		0.000	0.000	0.575	0.000	0.000

Fractional shortening (FS) showed no significant difference among stages ($p = 0.575$).

Both LVIDd and LVIDs increased significantly across stages ($p < 0.001$).

3.5. NT-proBNP Quantification Across Disease Stages

NT-proBNP concentrations increased significantly with disease progression ($p < 0.001$) (Table 4).

Table 4. NT-proBNP Concentrations in Different Disease Stages

Stage	Statistical parameters	Indices NT-proBNP
B1	n	6
	Mean	500 ^c
	SD	0.00
	Min	500
	Max	500
B2	n	55
	Mean	953.8 ^b
	SD	294.0
	Min	500
	Max	1356.2
C	n	23
	Mean	2131.8 ^a
	SD	354.4
	Min	1625.5
	Max	2935.2
P – value		0.000

Mean NT-proBNP values were 500 pg/mL (B1), 953.8 ± 294.0 pg/mL (B2), and $2,131.8 \pm 354.4$ pg/mL (C).

3.6. Treatment Outcomes of MMVD According to Disease Stage

Treatment outcomes are summarized in Table 5.

Table 5. Treatment outcomes of MMVD by disease stage

Stage	B1	B2	C	Total
Number of cases with effective treatment	6	54	10	70
Treatment success rate (%)	100	98.18	43.48	83.33
Number of cases with ineffective treatment	0	1	13	14
Treatment failure rate (%)	0	1.82	56.52	16.67

In stage B2, the treatment success rate was 98.18%, with 1 case (1.82%) showing no response. In stage C, treatment success was 43.48%, whereas 56.52% of cases were non-responsive. At stage B1, no specific cardiac treatment was administered; therefore, treatment outcomes were not evaluated for this stage.

4.0 Discussion

This study evaluated the prevalence, clinical characteristics, selected diagnostic indicators, and treatment outcomes of MMVD in dogs presented to a veterinary hospital in Vietnam. Among 3,840 examined dogs, 232 (6.04%) were suspected of cardiac disease based on clinical findings, and 84 dogs were definitively diagnosed with MMVD by echocardiography, representing 36.21% of suspected cases and 2.19% of all examined dogs. These findings support the importance of echocardiography in confirming MMVD among dogs initially suspected on the basis of nonspecific clinical signs. In routine practice, heart murmur, coughing, and exercise intolerance may raise suspicion of MMVD, but these findings alone are insufficient to establish a definitive diagnosis because they may overlap with respiratory and other systemic disorders.

The clinical manifestations observed in this study were generally consistent with the expected progression of MMVD across ACVIM stages. Dogs in stage B1 showed minimal clinical abnormalities, whereas dogs in stages B2 and C showed increasingly frequent respiratory and systemic signs. Dyspnea/pulmonary oedema and cough/respiratory inflammation were the most common signs overall, and their frequency increased with disease severity. This pattern is pathophysiologically plausible, as chronic mitral regurgitation leads to progressive left atrial enlargement and elevated pulmonary venous pressure, which may result in pulmonary congestion and oedema. The marked increase in syncope frequency in stage C also reflects the hemodynamic consequences of advanced disease, including reduced forward cardiac output and impaired tissue perfusion. However, clinical signs should always be interpreted cautiously, because they are not specific for MMVD and may be influenced by concurrent respiratory disease, especially in small-breed and older dogs.

Cardiac murmur intensity also increased with disease stage in the present study. Mild murmurs predominated in stage B1, whereas grade III/VI murmurs were most common in stage B2 and more severe murmurs were more frequently observed in stage C. This finding is consistent with the clinical course of MMVD, in which progressive valvular degeneration and worsening mitral regurgitation generate a more prominent systolic murmur. Previous studies have similarly shown that murmur intensity is associated with the severity of mitral regurgitation and may help identify dogs requiring further diagnostic evaluation. Nevertheless, murmur grade should not be considered a surrogate for disease stage in all cases. Some dogs in stage C still had grade III/VI murmurs, which may be explained by factors such as thoracic conformation, pulmonary crackles, reduced cardiac output, or variation in the audibility of turbulent regurgitant flow. Therefore, although auscultation remains a valuable screening tool, staging of MMVD should rely on integrated clinical and imaging findings rather than murmur intensity alone.

Radiographic and echocardiographic findings in this study clearly reflected disease progression. Vertebral heart size, LA/Ao ratio, LVIDd, and LVIDs all increased significantly from stage B1 to stages B2 and C, indicating progressive chamber enlargement and cardiac remodelling associated with chronic volume overload. In particular, the increase in LA/Ao from 1.38 in stage B1 to 1.76 in stage B2 and 2.18 in stage C is clinically relevant, because left atrial enlargement is one of the most important indicators of disease progression and risk of developing congestive heart failure. Likewise, the increase in VHS and left ventricular internal dimensions supports the value of both thoracic radiography and echocardiography in the evaluation of MMVD severity.

The present findings are in agreement with previous reports showing that indices of left-sided volume overload are highly informative in dogs with MMVD. Boswood et al. (2016) used left atrial enlargement and increased ventricular dimensions as key criteria to identify stage B2 dogs likely to benefit from early intervention, while other studies have shown that progressive increases in VHS and chamber size are associated with worsening clinical status and poorer outcome. In contrast, fractional shortening did not differ significantly among disease stages in this study. This observation is not unexpected, because MMVD is primarily a disease of volume overload rather than primary systolic myocardial failure. In such patients, systolic indices such as FS may remain within normal or even apparently increased ranges despite substantial structural remodelling. Therefore, the lack of a significant difference in FS does not imply absence of disease progression; rather, it underscores the greater clinical value of structural measurements such as LA/Ao, LVIDd, LVIDs, and VHS in monitoring MMVD.

NT-proBNP concentrations increased markedly with disease progression, from 500 pg/mL in stage B1 to 953.8 ± 294.0 pg/mL in stage B2 and $2,131.8 \pm 354.4$ pg/mL in stage C. These findings support the utility of NT-proBNP as a biomarker of myocardial wall stress and progressive cardiac remodelling in dogs with MMVD. The increase observed in stage B2 is especially important, because these dogs may not yet show overt signs of congestive heart failure despite already having significant cardiac enlargement. In this context, NT-proBNP may provide additional support for risk stratification, particularly when interpreted together with echocardiographic findings. The markedly higher values observed in stage C are also consistent with overt decompensation and increased hemodynamic burden. Previous studies have similarly demonstrated that NT-proBNP is useful for distinguishing dogs with cardiac from non-cardiac causes of respiratory signs and for assessing disease severity in MMVD. Thus, the present results reinforce the role of NT-proBNP as a complementary tool, rather than a standalone test, in the diagnosis and monitoring of this disease.

Treatment outcomes differed substantially between disease stages. Dogs in stage B2 had a very high treatment success rate, whereas the response rate in stage C was much lower. This result is clinically meaningful and is consistent with the concept that earlier detection and intervention are associated with better disease control. Stage B2 dogs generally still retain effective compensatory mechanisms despite structural cardiac remodelling and therefore may respond favourably to appropriate treatment and monitoring. In contrast, dogs in stage C have already developed clinical heart failure, and treatment becomes more challenging because of greater hemodynamic instability, pulmonary oedema, and

possible concurrent complications. These findings emphasize the importance of recognizing MMVD before overt decompensation occurs. They also support the current clinical approach of close monitoring and timely therapeutic intervention in dogs with evidence of cardiac enlargement even in the absence of advanced heart failure.

From a practical perspective, this study highlights the importance of combining physical examination, thoracic radiography, echocardiography, and NT-proBNP testing when evaluating dogs suspected of MMVD. No single parameter alone was sufficient to characterize disease severity across all stages. Clinical signs and murmur intensity were useful for initial screening, but imaging and biomarker evaluation provided the strongest information for staging and monitoring. In a clinical setting such as Vietnam, where access to advanced diagnostic tools may vary among facilities, these findings may help support a more structured diagnostic approach and encourage earlier identification of dogs at risk of progression.

Limitations

This study has several limitations. First, the number of dogs definitively diagnosed with MMVD was relatively limited, which may reduce the generalizability of the findings. Second, the number of dogs in the more advanced stages was relatively small, and no dogs in stage D were included, limiting assessment of the full spectrum of disease severity. Third, only a limited number of echocardiographic variables were evaluated. Additional parameters with known pathophysiological and prognostic importance, such as left atrial volume and peak E-wave velocity, were not assessed. Fourth, survival analysis was not performed, and therefore the natural history of MMVD and the long-term effects of different variables and treatment protocols could not be explored. Fifth, electrocardiographic findings were not included, and no data were available regarding arrhythmias or the use of antiarrhythmic drugs, despite the clinical relevance of rhythm disturbances in dogs with MMVD. Sixth, other cardiovascular complications, such as pulmonary hypertension and left atrial rupture, were not evaluated. Finally, information on comorbidities, particularly renal disease and tracheobronchial collapse, was not systematically collected, although such conditions may substantially influence clinical presentation, treatment response, and survival. Future studies with larger sample sizes, broader cardiovascular assessment, and long-term follow-up are needed to better define the clinical course and prognostic factors of MMVD in dogs under local practice conditions.

5.0 Conclusion

In this study, MMVD was confirmed in 2.19% of examined dogs, with prevalence increasing among those presenting with respiratory signs. Clinical signs and cardiac murmur intensity increased with disease progression, while echocardiographic variables of left-sided volume overload and NT-proBNP concentrations showed significant changes across stages.

These findings highlight the importance of combining clinical examination, diagnostic imaging, and biomarker assessment for accurate staging and monitoring of MMVD, thereby supporting earlier detection and more effective disease management in dogs.

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