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Electrocardiographic changes in small-Breed Dogs with Myxomatous Mitral Valve Disease (MMVD)

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Abstract

Myxomatous mitral valve disease (MMVD) is the most common acquired heart disorder in dogs, especially in small and toy breeds. As mitral regurgitation worsens, it causes chronic overload of the left atrium and ventricle, leading to cardiac remodeling, atrial stretch, fibrosis, and changes in impulse generation and conduction. These changes increase the risk of conduction issues and arrhythmias. Electrocardiography (ECG) is a non-invasive way to detect arrhythmias and heart chamber changes related to mitral valve degeneration. In this study, ECG results and rhythm abnormalities were recorded from 18 small breed dogs with MMVD. Dogs with MMVD exhibited a statistically non-significant increase in p wave duration (0.05 ± 0.00 sec) compared to healthy controls (0.04 ± 0.00 sec). Key ECG findings included sinus tachycardia (66.67%), wide p wave or p-mitrale (55.56%), ST coving (27.77%), notched p wave (22.22%), and atrial fibrillation (5.55%). Normal sinus rhythm (4/18, 22.22%) was also recorded in MMVD dogs. These findings indicate atrial volume overload and progressive cardiac remodelling in MMVD. These results support the application of electrocardiography as a useful additional tool for early diagnosis and management of MMVD.

Keywords: MMVD, ECG, mitral valve disease, small breed dogs, arrhythmia, atrial fibrillation, p-mitrale

Introduction

Myxomatous mitral valve disease (MMVD), also known as chronic degenerative mitral valve disease, is the most common acquired cardiac disease in dogs, particularly affecting small- and toy-breed dogs. It is characterized by progressive nodular thickening and distortion of the mitral valve leaflets, resulting in mitral regurgitation. Chronic mitral regurgitation causes volume overload of the left atrium and left ventricle, leading to progressive cardiac remodeling characterized by myocardial stretch, chamber enlargement, and structural alterations. These changes may disrupt normal electrical impulse generation and conduction, predisposing affected dogs to conduction abnormalities and cardiac arrhythmias. Electrocardiographic manifestations of these alterations include P-mitrale (broad or notched P waves indicative of left atrial enlargement), tall R waves and widened QRS complexes suggestive of left ventricular enlargement, and various cardiac arrhythmias (Ogawa *et al.*, 2022) ^[7].

Echocardiography remains the gold standard for the diagnosis and staging of MMVD. However, electrocardiography (ECG) serves as a valuable complementary diagnostic tool by detecting rhythm disturbances, conduction abnormalities, and electrical changes associated with cardiac remodeling. Although many ECG abnormalities may remain subtle or absent during the early stages of MMVD, progressive disease increases the risk of clinically significant arrhythmias that may manifest as syncope or, rarely, sudden cardiac death. Therefore, ECG provides important additional information for the comprehensive evaluation and clinical management of dogs with MMVD.

The present study was undertaken to evaluate electrocardiographic parameters and rhythm abnormalities in 18 echocardiographic ally confirmed small-breed dogs with MMVD and to compare these findings with those of healthy control dogs.

Materials and Methods

Client owned dogs presented to small animal medicine ward, Veterinary clinical complex, College of Veterinary Science, Tirupati and Proddatur, with clinical signs of cough, dyspnea, exercise intolerance, syncope and abdominal distension were screened and echocardiographic ally confirmed for presence of MMVD. A total of 18 echocardiographic ally confirmed small-breed dogs with MMVD were subsequently subjected to electrocardiographic evaluation using a standard electrocardiograph (Aspen ECG-3 Plus) following the methodology described by Tilley (1992) [13]. Electrocardiograms were recorded at paper speeds of 25 and 50 mm/s with a standard calibration of 10 mm = 1 mV. The recordings were analysed for heart rate, rhythm, waveform amplitude, and interval durations in accordance with standard electrocardiographic guidelines (Willis *et al.*, 2015) [14].

Results

In the present study, Electrocardiographic parameters of 18 dogs with MMVD were compared with those of healthy control dogs. A non-significant increase in P-wave duration (0.05 ± 0.00 s) was observed in dogs with MMVD compared with healthy controls (0.04 ± 0.00 s). No significant differences ($p > 0.05$) were detected in P-wave amplitude, QRS complex duration and amplitude, T-wave duration and amplitude, PR interval, QT interval, or ST segment between the two groups (Table 1).

The predominant electrocardiographic abnormalities observed in dogs with MMVD were sinus tachycardia (12/18; 66.67%), P-mitrale (broad P waves) (10/18; 55.56%), ST-segment coving (5/18; 27.78%), notched P waves (4/18; 22.22%), low-

voltage QRS complexes (3/18; 16.67%), P-pulmonale (3/18; 16.67%), and atrial fibrillation (1/18; 5.56%). Normal sinus rhythm was observed in 4 of 18 dogs (22.22%) despite echocardiographic evidence of MMVD (Table 2).

Table 1: Electrocardiographic parameters comparison between control and small breed dogs with MMVD (n=18)

S. No.	Parameter		Control	MMVD
1.	P wave	Duration (sec)	0.04 ± 0.00^a	0.05 ± 0.00^a
		Amplitude (mV)	0.43 ± 0.22^a	0.25 ± 0.05^a
2.	QRS complex	Duration (sec)	0.03 ± 0.00^a	0.04 ± 0.01^a
		Amplitude (mV)	1.23 ± 0.28^a	1.80 ± 0.40^a
3.	T wave	Duration (sec)	0.09 ± 0.01^a	0.10 ± 0.02^a
		Amplitude (mV)	0.26 ± 0.10^a	0.30 ± 0.10^a
4.	PR interval(sec)		0.11 ± 0.00^a	0.09 ± 0.01^a
5.	QT interval(sec)		0.13 ± 0.02^a	0.16 ± 0.02^a
6.	ST segment(sec)		0.09 ± 0.01^a	0.12 ± 0.01^a

Mean $\pm$ S.E followed by same superscript(s) do not differ significantly ($p > 0.05$)

Table 2: ECG findings of small breed dogs with MMVD (n=18)

S. No.	ECG Finding	Number	Percentage
1.	Sinus tachycardia	12	66.67
2.	Wide P Wave (P-Mitrale)	10	55.56
3.	ST Coving	5	27.77
4.	Notched P wave	4	22.22
5.	Normal sinus rhythm	4	22.22
6.	Low voltage QRS complexes	3	16.66
7.	P - pulmonale	3	16.66
8.	Atrial Fibrillation	1	5.55

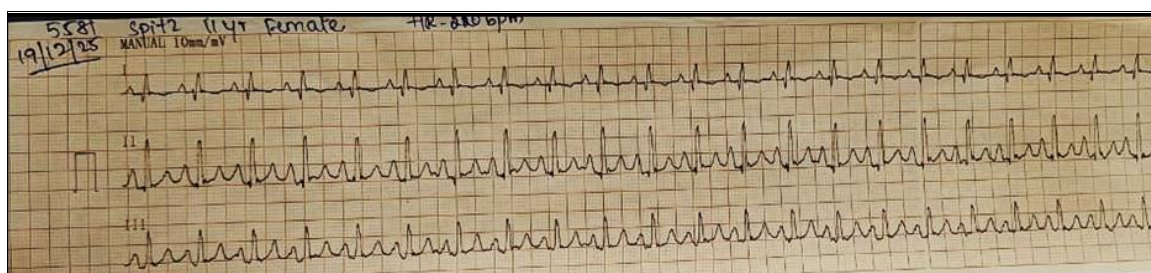


Fig 1: Sinus tachycardia recorded in an 11-year female Spitz with MMVD (220bpm)

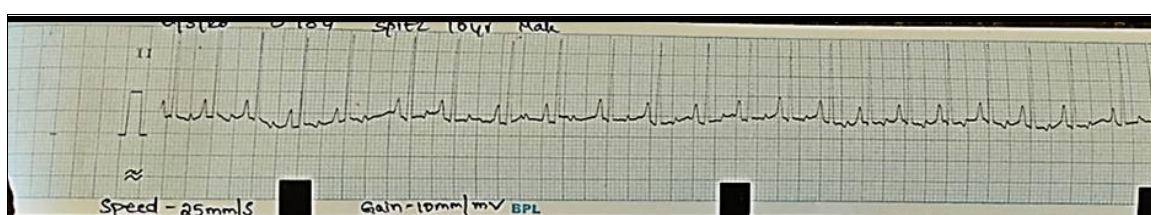


Fig 2: Sinus tachycardia with tall R-waves in a 10-year male Spitz with MMVD (175bpm)



Fig 3: Notched P waves recorded in a 10 year male MMVD dog

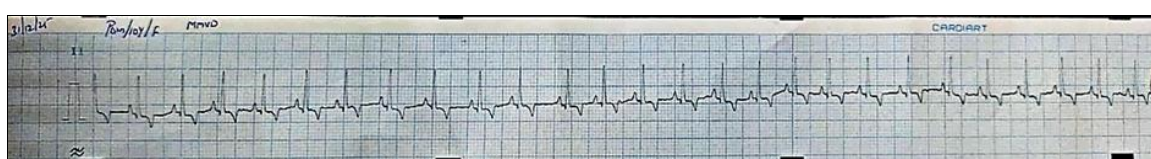


Fig 4: Normal sinus rhythm recorded in a 10 year male MMVD dog

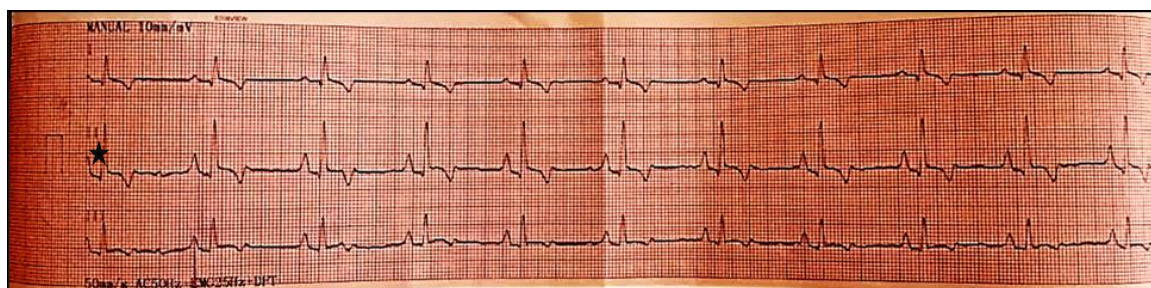


Fig 5: P-pulmonale (0.5mv) recorded on ECG in a 13 year Spitz with MMVD.

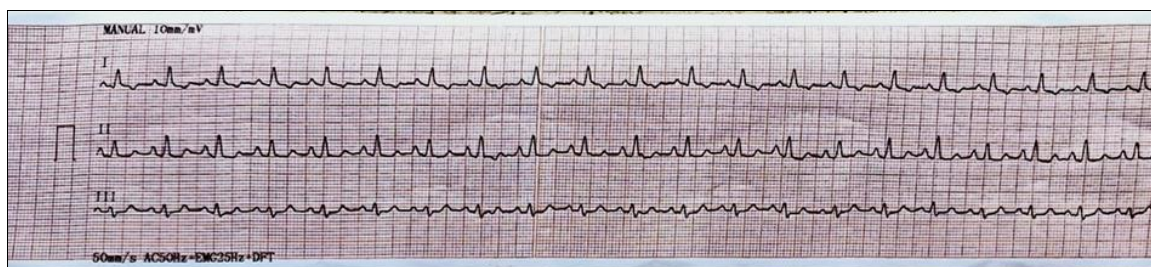


Fig 6: Low voltage complexes in a Spitz with MMVD

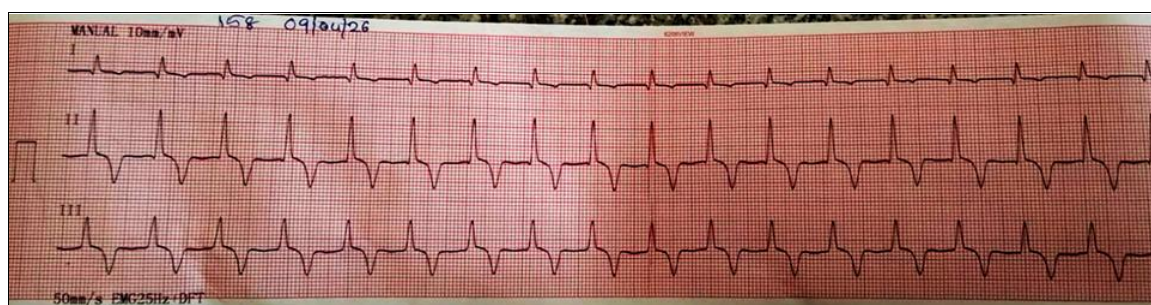
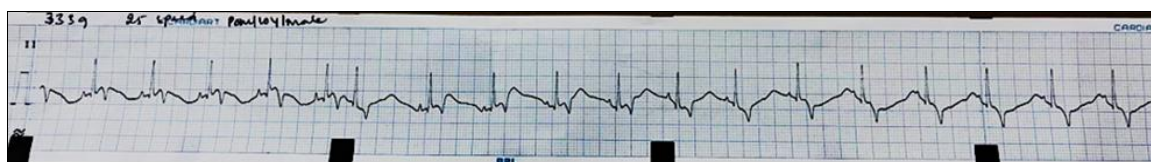


Fig 7: Atrial fibrillation recorded in canine MMVD



Pre-treatment appearance of notched P waves on ECG



Absence of notched P-waves on 30th day of therapy

Fig 8: Resolution of notched P-waves during therapy

Discussion

The electrocardiographic parameters of dogs affected with MMVD were within normal reference limits, except for a mild increase in P-wave duration (0.05 ± 0.00 s), which is suggestive of delayed intra-atrial conduction secondary to left atrial enlargement (P-mitrale). Although the increase was not statistically significant, Ogawa *et al.* (2022) [7] identified P-wave duration as a useful electrocardiographic indicator of left atrial remodelling in dogs with MMVD, supporting the trend observed in the present study.

The predominant electrocardiographic abnormalities observed were sinus tachycardia, P-mitrale, ST-segment coving, notched P waves, low-voltage QRS complexes, P-pulmonale, and atrial fibrillation. Similar ECG findings have been

reported in dogs with MMVD by Lopez-Alvarez *et al.* (2014), Petric (2015), Kim *et al.* (2017), Jan *et al.* (2018), Rosa *et al.* (2019), Greeshma *et al.* (2023), and Rajan *et al.* (2025) [2, 4, 5, 6, 8, 9, 10]. Normal sinus rhythm was also observed in a proportion of affected dogs, indicating that clinically important structural cardiac changes may occur despite minimal electrocardiographic abnormalities.

Sinus tachycardia most likely reflected a compensatory response to reduced forward cardiac output and increased sympathetic activity rather than a primary arrhythmia (Tilley, 1992) [13]. P-mitrale and notched P waves were indicative of left atrial enlargement and altered atrial conduction associated with chronic volume overload (Santilli *et al.*, 2018; Ogawa *et al.*, 2022) [7]. P-pulmonale may indicate right atrial

enlargement secondary to pulmonary hypertension in advanced MMVD. Low-voltage QRS complexes were probably associated with pleural or pericardial fluid accumulation accompanying congestive heart failure (Greeshma *et al.*, 2023) ^[2]. ST-segment coving may reflect myocardial hypoxia, nonspecific electrolyte disturbances, or ventricular hypertrophy (Cote, 2010). The occurrence of atrial fibrillation was consistent with previous reports demonstrating that marked left atrial enlargement predisposes dogs to atrial fibrillation through atrial electrical remodelling and ectopic activity originating from the pulmonary veins (Chen *et al.*, 2000; Rush, 2002; Guglielmini *et al.*, 2020) ^[1, 3, 11].

During the 90-day treatment period, no significant changes were observed in routine ECG parameters within or between treatment groups. Nevertheless, dogs exhibiting sinus tachycardia, P-mitrale, notched P waves, and P-pulmonale showed progressive improvement in electrocardiographic findings following therapy. The dog diagnosed with atrial fibrillation received adjunctive antiarrhythmic therapy with oral diltiazem and digoxin in addition to standard cardiac treatment, resulting in restoration of normal sinus rhythm by the 90th day. These observations indicate that although routine ECG measurements remained largely unchanged, rhythm disturbances and conduction abnormalities improved with appropriate medical management.

Conclusion

This study demonstrates that electrocardiographic evaluation in small breed dogs with MMVD reveals a range of rhythm and morphological abnormalities that reflect the hemodynamic consequences of chronic mitral regurgitation. Sinus tachycardia and P-mitrale were the most frequent findings, reflecting sympathetic activation and left atrial enlargement, respectively. Although routine ECG parameters showed minimal change during therapy, electrocardiography proved valuable for detecting rhythm disturbances and monitoring their response to treatment. These findings support the use of ECG as a complementary diagnostic and monitoring tool alongside echocardiography in the clinical management of dogs with MMVD.

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