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Electrocardiographic Alterations of Dilated Cardiomyopathy in Dogs

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

The study encompassed 2497 dogs, identifying 29 cases of Dilated Cardiomyopathy (DCM). Electrocardiographic (ECG) evaluations revealed significant differences in key parameters between DCM-affected and healthy dogs. Notably, dogs with DCM exhibited alterations in P wave amplitude and duration, QRS duration, and R wave amplitude, indicative of cardiac dysfunction. Proportional analysis further delineated distinct ECG abnormalities specific to left and bilateral DCM presentations. In left DCM, increased QRS and P wave duration, and elevated R wave amplitude were predominant, while bilateral DCM displayed similar anomalies alongside low voltage QRS and non-measurable P wave. Increased P wave duration was prevalent in both forms of DCM, with atrial fibrillation rendering P waves non-measurable in a notable proportion. Deep Q wave presence substantiated DCM diagnosis. Furthermore, dogs with left and bilateral DCM exhibited prolonged QRS duration, indicative of cardiac enlargement and global myocardial degeneration. Low voltage QRS complexes indicated potential pericardial effusion or ascites. However, PR and QT intervals did not significantly differentiate between healthy and DCM-affected canines. Rhythm disturbances were prevalent in DCM, with atrial fibrillation most common, followed by ventricular premature complexes, sinus arrhythmia, and other variants. Left bundle branch block and diverse degrees of AV block and electrical alternans were also detected. Proportional analysis delineated varied prevalence rates of rhythm abnormalities between left and bilateral DCM groups, reflecting nuanced pathological distinctions. This comprehensive investigation underscores the pivotal role of ECG assessments in diagnosing and monitoring DCM in canine populations, elucidating specific electrocardiographic aberrations associated with distinct DCM presentations.

Keywords: Atrial fibrillation; cardiomyopathy; DCM; electrocardiography.

1. INTRODUCTION

Canine cardiac diseases are potentially lethal, highly devastating and slowly progressive in nature, therefore considered as silent killer. Dilated cardiomyopathy (DCM) is characterized by decreased systolic function and dilation of one or both ventricles, often leading to heart failure or sudden death [1]. DCM is a frequent cause of cardiac disability, congestive heart failure (CHF), and arrhythmic death in dogs. World Health Organization's World Heart Federation classified cardiomyopathy in five groups, *i.e.* dilated (DCM), hypertrophic (HCM), restrictive (RCM), arrhythmogenic right ventricular cardiomyopathy (ARVCM) and non-classified cardiomyopathy. In dogs, DCM and ARVCM are the most commonly reported inherited forms of myocardial disease, out of which, DCM accounts for approximately 60-87% of total cardiomyopathy cases in dogs.

ECG is a valuable tool in the identification and characterization of cardiac arrhythmias, analysis of an irregular rhythm that do not meet the criteria for a sinus arrhythmia, syncope or collapse [2], determination of the etiology of bradycardia or tachycardia, information regarding cardiac chamber enlargement identification of dogs with risk of developing ventricular arrhythmias or dilated cardiomyopathy, individualization of therapy of heart diseases and conduction disturbances. Deep Q wave (more than 0.5 mV) was seen in 13.79 % dogs, which confirmed for either left or bilateral DCM.

2. MATERIALS AND METHODS

The present study was conducted collaboratively at the Department of Veterinary Clinical Medicine, College of Veterinary and Animal Science, RAJUVAS, Bikaner, and the Chandrika Chimanlal Doshi Cardiovascular Unit for Animals, Department of Veterinary Clinical Medicine, Ethics, and Jurisprudence, Bombay Veterinary College, Parel, Mumbai-12. A comprehensive investigation involving 2497 canines of diverse breeds, ages, and genders was conducted to ascertain occurrences of Dilated Cardiomyopathy (DCM). Through meticulous examination, which encompassed historical analysis, clinical evaluation, electrocardiography, and radiography, researchers definitively diagnosed 29 cases of DCM utilizing electrocardiography. Readings were recorded with a standard electrocardiograph single channel BPL 108 DIGI-T Model recording of ECG, subsequent measuring of complexes and intervals, their calculations and interpretation, guidelines laid down by Tilley (1992) were followed

3. RESULTS AND DISCUSSION

Results of the major electrocardiographic waveform, complexes and interval in healthy and

DCM dogs are summarized in Table 1 and Fig. 1. The values of the all the ECG parameters of control group were within normal canine range [3,2]. The rhythm of heart in all the dogs was sinus arrhythmia (slight variation in R-R interval), which is a normal feature of canine heart (Tilley, 1992). Electrocardiographic measurements of healthy and DCM dogs are similar to the findings of Noszczyk (2012). Another study of Gnieszka et al. [4] reported 0.055±0.01 sec. duration of the P wave, 0.55±0.1 mV amplitude of the P waves, 0.1 sec. duration of the P wave, 0.08±0.012 sec. duration of the QRS complex, 2.84±0.3 mV amplitude of the R waves and 0.23±0.02 sec. duration of the ST-T segment in dogs with DCM. Dharmasuriya et al. [5] also reported wide QRS interval of 0.1 sec, wide QT interval of 0.027 sec. and R wave height of 4-5mV in a 4-year-old female Dobermann Pinscher with DCM. Similar to present findings increased height of the R wave (>2.6 mV) has been reported in a nondescript dog with DCM by Rajesh et al. [6]. Statistical analysis showed significant differences in P wave amplitude and duration, QRS duration and R wave amplitude, while PR interval, QT interval and mean electrical axis did not differ significantly between healthy and DCM affected dogs.

Results of same waveform, complexes and intervals were analyzed to interpretate these parameters in different groups of DCM (Table1, Fig. 1). Proportional group analysis showed no significant difference between left and bilateral DCM group in respect to P wave duration. However, left DCM group found to differ significantly from bilateral DCM group. QRS duration did not differ significantly between left and bilateral DCM groups. In respect to PR interval, QT interval and mean electrical axis no significant difference was seen between two groups of DCM and healthy diogs. All the findings in group wise analysis supplements the views of Martin [3], Tilley et al. [2], Ware [7] and Mark et al. [8].

Table 1. Electrocardiographic waveform, complexes and interval in healthy and DCM dogs

Measurements (lead II)	Healthy	DCM	Left DCM	Bilateral DCM
P wave duration (sec)	0.042 ± 0.007 b	0.056 ± 0.001 a	0.061 ± 0.002a	0.059 ± 0.003a
P wave amplitude (Mv)	0.193 ± 0.030 c	0.307 ± 0.017 b	0.236 ± 0.013c	0.363 ± 0.026 ab
PR interval (sec)	0.133 ± 0.021 ns	0.143 ± 0.023 ns	0.144 ± 0.007ns	0.148 ± 0.007 ns
QRS complex duration	0.041 ± 0.01 b	0.064 ± 0.01 a	0.066 ± 0.002 a	0.064 ± 0.003 [a]
R wave amplitude	1.295 ± 0.205 b	1.991 + 0.315 a	2.207 ± 0.089 a	1.963 ± 0.107 c
QT interval (sec)	0.190 ± 0.030 ns	0.187 ± 0.030 ns	0.180 ± 0.007 ns	0.195 ± 0.013 ns
Mean electrical axis (degree)	78.750 ± 12.46 ns	77.58 ± 12.27 ns	75.000 ± 4.235 ns	78.75 ± 8.45 ns

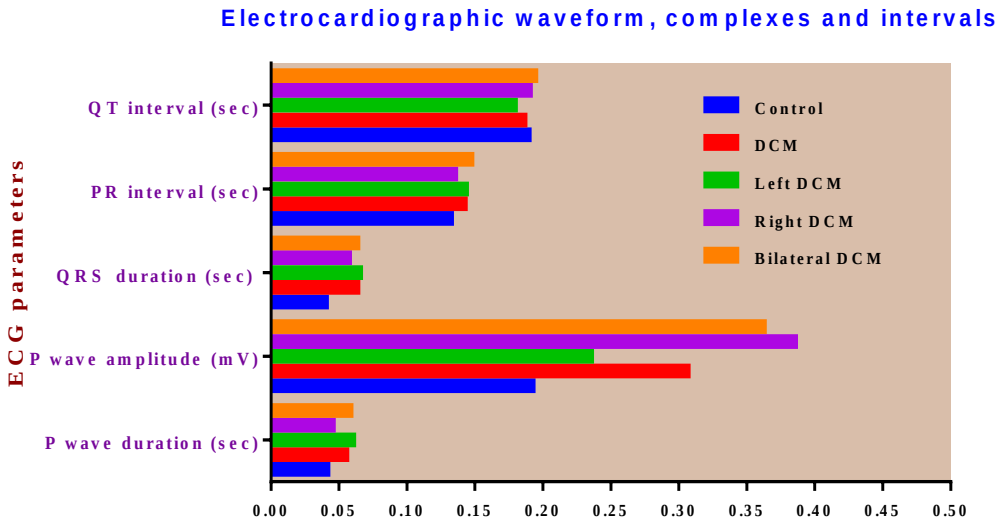


Fig. 1. Electrocardiographic waveform, complexes and interval in healthy and DCM dogs

Electrocardiographic abnormalities like more than 0.06 sec QRS duration, low voltage QRS, more than 0.04 sec P wave duration, more than 0.4 mV amplitude of P wave, non-measurable P wave, more than 3 mV amplitude of R, more than 0.5 mV depth of Q wave and deep S wave are 62.06, 37.93, 44.82, 20.68, 48.27, 55.17, 13.79 and 17.24 % dogs affected with DCM (Table 2, Fig. 2). These findings are partially similar to Polana [9] in dogs with DCM. Proportional analysis of these abnormalities between groups showed increased QRS and P wave duration in 85.71 % and increased R wave amplitude in 78.57 %, non-measurable P wave in 64.28 %, deep Q wave in 14.29 % dogs with left DCM, while dogs with bilateral DCM showed increased QRS and low voltage QRS in 75 %, increased P wave duration, amplitude and non-measurable P wave in 25 % and increased R wave amplitude in 62.5 %, deep S wave in 37.5 % and deep Q wave was seen in 25 % dogs.

Table 2. Frequency of alteration in electrocardiographic waveform, complexes and interval in DCM affected dogs

Measurements	DCM (No. of dogs)	Percent	Left DCM (No. of dog)	Percent	Bilateral DCM (No. of dog)	Percent
QRS > 0.06 sec	18	62.06	12	85.71	6	75
Low voltage QRS	11	37.93	0	0	6	75
P wave >0.04 sec	13	44.82	12	85.71	2	25
P wave >0.4 mV	6	20.68	0.00	0.00	2	25
Non-measurable P wave	14	48.27	9	64.28	2	25
R wave >3 mV	16	55.17	11	78.57	5	62.5
Deep Q > 0.5 mV	4	13.79	2	14.29	2	25
Deep S wave> 0.35 mV	5	17.24	0.00	0.00	3	37.5

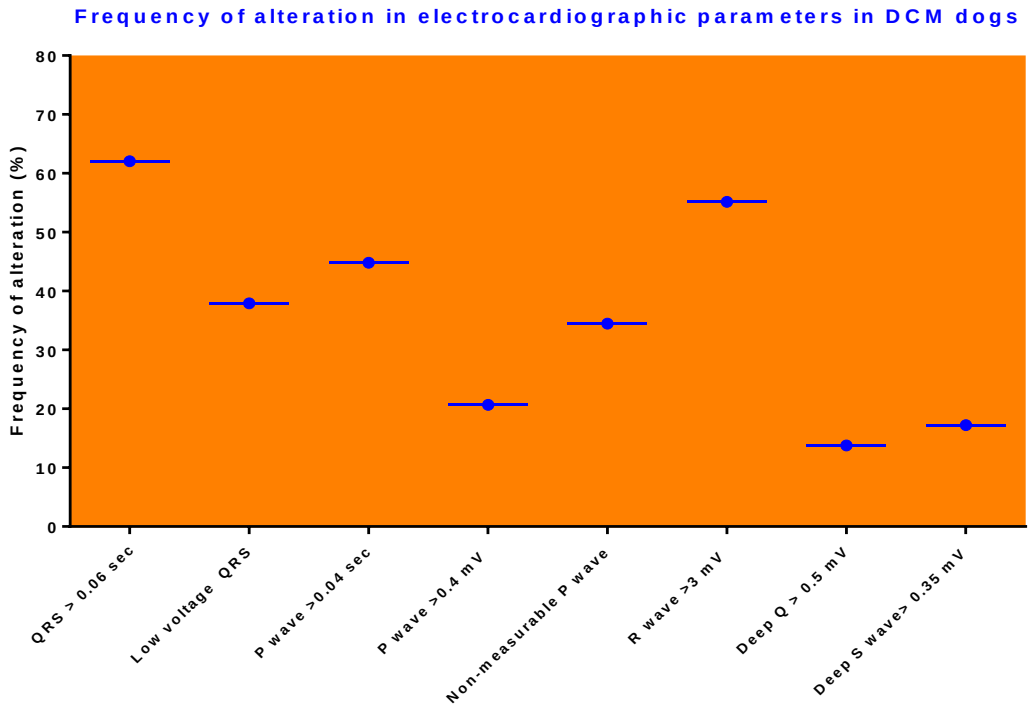


Fig. 2. Frequency of alteration in electrocardiographic waveform, complexes and interval in DCM affected dogs

Study revealed increased P wave duration in 44.82 % DCM affected dogs; all of them confirmed for either left or bilateral DCM, but increased P wave amplitude was seen in 20.68 % dogs with bilateral DCM (Fig. 2). In 34.48 % dogs P wave was non-measurable due to atrial fibrillation. These findings supplements the view of Richard and Elisa [10], Tilley et al. [2] and Ware [11] suggested that P wave longer than 0.04 ms is diagnostic of left atrial enlargement, while amplitude of more than 0.4 mV is diagnostic for right atrial enlargement. First half of P wave represents sinus impulse activated the right atrium and reaches to AV node, while second half indicates activation of left atrium and AV node. The long or abnormally shaped P wave occurs because of delay in electrical activation of the enlarged left atrium, as electricity moves leftward from the SA node, therefore increased P-wave duration signify either slow conduction or an enlarged atrium [12].

Deep Q wave (more than 0.5 mV) was seen in 13.79 % dogs, which confirmed for either left or bilateral DCM (Table 2). These findings supplement the views of Nicole [13] as he described deep Q waves as common abnormalities in biventricular and right ventricular enlargement, Kumar et al. [14] also reported deep Q wave ECG abnormalities of DCM. The Q wave represents the electrical transmission through interventricular septum and depth of the Q wave has been reported to be connected with the activation processes of cardiac ventricles.

More than 0.06 ms QRS complex was recorded in 62.06 % dogs affected with left and bilateral DCM (Table 2), which was indicative for enlarged cardiac size and global myocardial degenerationin the end stages of DCM. Simiz et al. [15] described widened QRS complex with a sloppy R descent as a classical electrocardiographic pattern of DCM in dogs with good correlation with survival time [16]. QRS duration has been reported to have significant prognostic impact as reported to be correlated with the dimension [17] and function of the left ventricle along with increased filling pressure. Additionally linear relationship has been reported between increased QRS duration and decreased ejection fraction [18].

Low voltage QRS complexes were seen in 37.93% dogs with DCM, which were due to bilateral DCM group and suggestive of either pericardial effusion or ascites [19] in relation to cardiovascular dysfunction. Criteria for low voltage QRS is less than 1.0 mV [20]. Additionally, 17.24 % DCM affected dogs showed electrical alternans, which was another electrocardiographic indication for pericardial effusion. Although many noncardiac reasons have been suggested behind these changes but percentage of low voltage QRS and electrical alternans in present study correlate well with clinical signs, radiography and echocardiography, as all the dogs showing low voltage were found to be affected with ascites or pericardial effusion or both [3].

Present investigation did not find significant difference in PR and QT interval of healthy and DCM affected dogs. PR interval represent amount of time required for both atrial depolarization and the delay in the A-V node, while QT interval is a dynamic physiological variable that can be affected by the velocities of both the ventricular conduction and repolarization [21]. In canine electrocardiography, the QT interval alone is not helpful in the diagnosis, but a useful rule is that the QT interval should be less than half of the preceding RR interval, which is not observed in present study.

More than 3 mV amplitude of R wave was seen in 55.17 % of dogs with DCM. The R wave is good indicator of left ventricular compliance and contractility and higher amplitude signify enlargement of left side [22]. Enlarged ventricle with an increased surface area and thickened walls produces greater potential. This finding is in harmony with Dharmasuriya et al. [5] and Rajesh et al. [6] reported R wave height of 4-5 mV in a Dobermann Pinscher and R wave of more than 2.6 mV in a nondescript dog with DCM, respectively.

Present investigation revealed deep S wave in 17.24 % dogs with DCM, which was confirmed for either right or bilateral DCM. S wave produce during third phase of ventricular depolarization signify apicobasilar activation of muscle fibers [3]. Similar to observations of present study regarding S wave, Tilley et al. [2] suggested more than 0.35 mV depth of S wave in lead II as an indication for right ventricular enlargement. Ware [7] also suggested deep S wave in lead II, III and a VF and its presence in lead I represent right ventricular enlargement.

Table 3. Abnormalities of rhythm and cardiac conduction in DCM affected dogs

Abnormalities	Recorded in no. of dogs with DCM	Percent	Left DCM n=14	Percent	Bilateral DCM n=8	Percent
Type of rhythm (Normal- 31.03%, abnormal 68.96 %)						
Normal sinus rhythm (sinus arrthymia)	9	31.03	2	14.29	3	37.5
Sinus tachycardia	1	3.45	1	7.14		0
Atrial fibrillation	15	51.72	11	78.57	2	25
Ventricular premature complexes	12	41.38	9	64.29	2	25
Ventricular tachycardia	4	13.79	2	14.29	1	12.5
Conduction abnormalities (24 out of 29= 82.75)						
Left bundle branch block	2	6.90	1	7.14	1	12.5
ST slurring	4	13.79	1	7.14	3	37.5
First degree AV block	7	24.14	4	28.57	3	37.5
Second degree AV block	11	37.93	6	42.86	2	25
Electrical alternans	5	17.24		3		

Mean electrical axis (MEA) did not differ in healthy and DCM affected dogs of present investigation. It describes the average direction of the ventricular depolarization process in the frontal plane and represents the summation of the various instantaneous vectors that occur from the beginning until the end of ventricular muscle activation [7].

Present investigation recorded abnormalities in both cardiac conduction and rhythm in DCM affected dogs (Table 3), which is in agreement with Kumar et al. [14]. Yamaki et al. (2007) reported normal sinus rhythm (sinus arrhythmia) in 45% dogs with DCM. Study of Martin et al. [23] showed arrhythmia in 89% of dogs and 11% of DCM affected dogs reported without an arrhythmia, but all had either QRS enlargement pattern consistent with cardiomegaly or ST depression. Morales et al. (2001) reported 9% dogs without any electrocardiographic abnormalities.

Study revealed rhythm disturbances in 68.96% of dogs, while 31.03 % failed to demonstrate any rhythm disturbances. Among various rhythm abnormalities, DCM affected dogs showed highest occurrence of atrial fibrillation (51.72%) (Figs. 5A to F) followed by ventricular premature complexes (41.38%) (Figs. 6, 7), sinus arrhythmia (31.03%), ST slurring (Fig. 8) and ventricular tachycardia (13.79%) (Fig. 9), and sinus tachycardia (3.45%), left bundle branch block (6.90%). First degree, second degree AV block (Fig. 10) and electrical alternans were detected in 24.14%, 37.93% and 17.24% DCM dogs, respectively. Proportional analysis between groups showed 14.29, 57.14 and 37.50% sinus arrhythmia, 78.57%, 28.57 and 25.00% atrial fibrillation, 64.29, 14.29 and 25.00% ventricular premature complexes, 14.29 and 12.50% ventricular tachycardia in left and DCM groups. Sinus tachycardia was seen only in 7.14% dogs with left DCM. Left bundle branch block was observed in 7.14 and 37.50 % dogs of left and bilateral DCM affected dogs. Similarly, ST slurring was seen in 7.14 and 12.50% of left and bilateral DCM affected dogs.

Abnormalities of rhythm and cardiac conduction in DCM affected dogs in Fig 3.

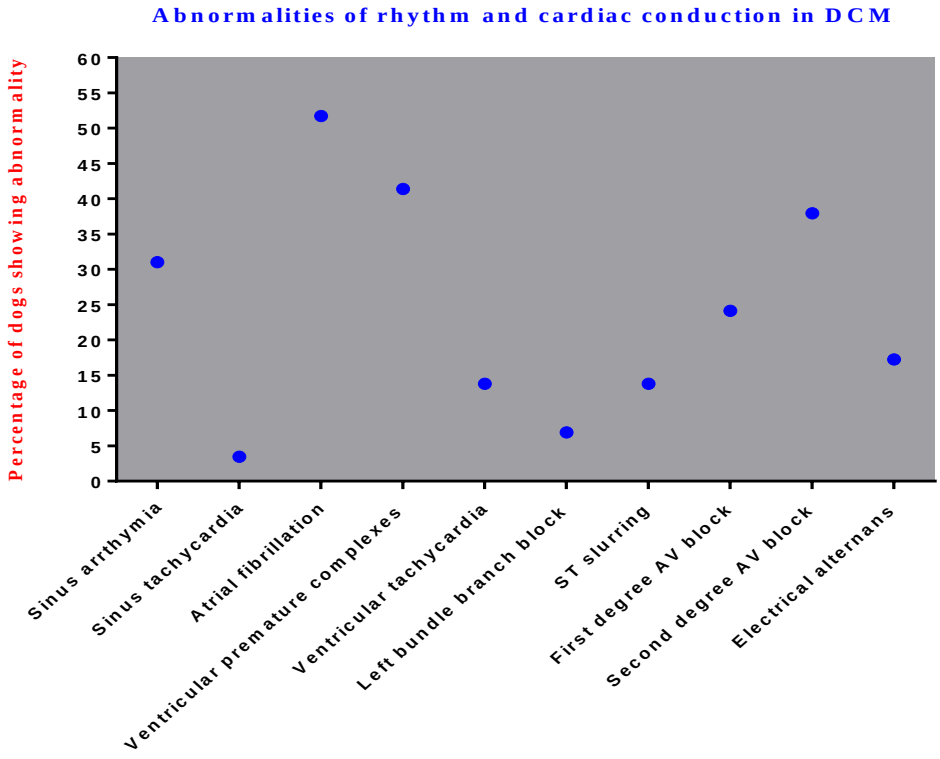


Fig. 3. Abnormalities of rhythm and cardiac conduction in DCM affected dogs

ECG ABNORMALITIES IN DCM AFFECTED DOGS

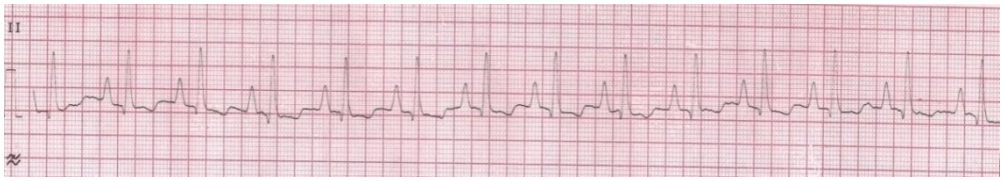
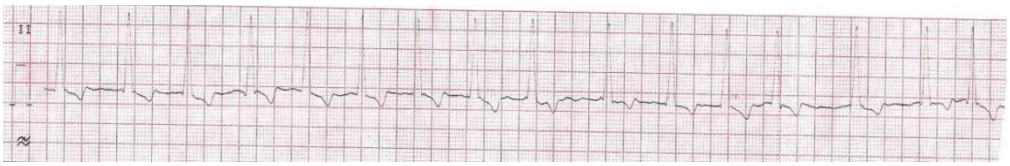
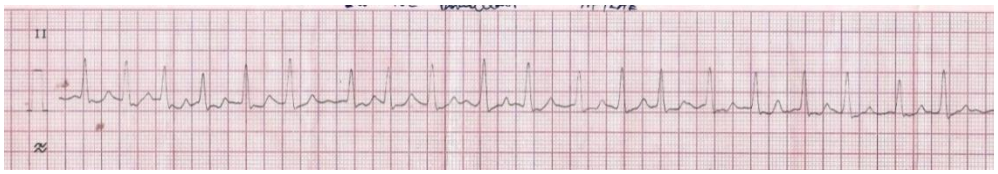


Fig. 4. Long amplitude and duration of P wave in bilateral DCM



(A)



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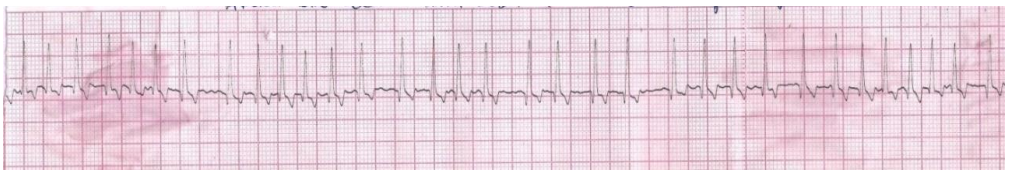
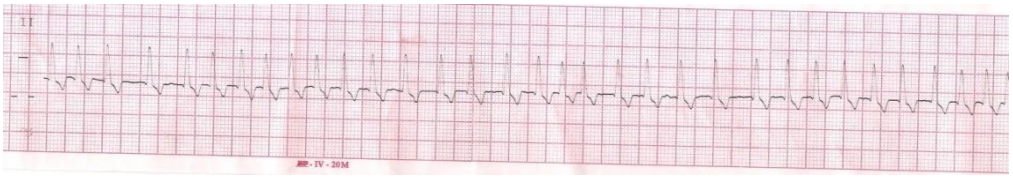
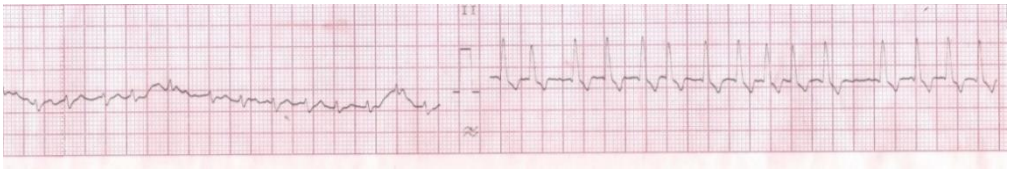


(C)



(D)



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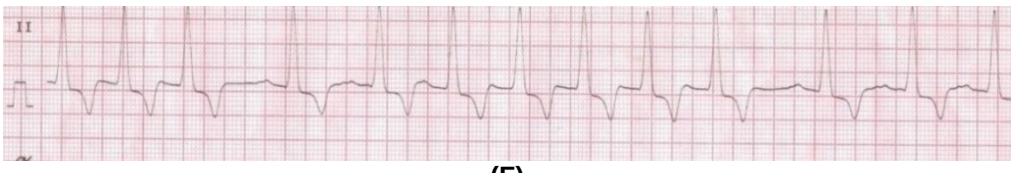
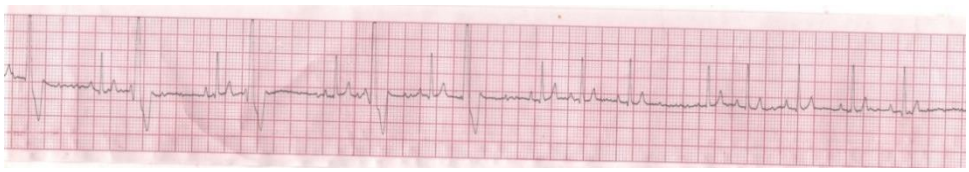


(F)

Fig. 5. A to F Atrial fibrillation



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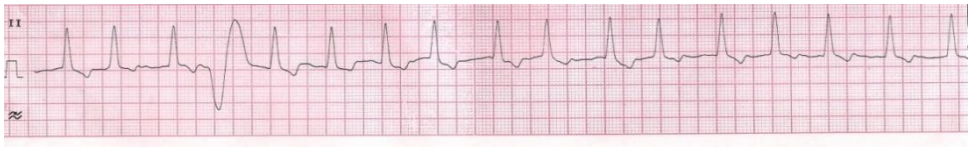

(7)

Fig. 6. and Fig. 7. Ventricular premature complexes

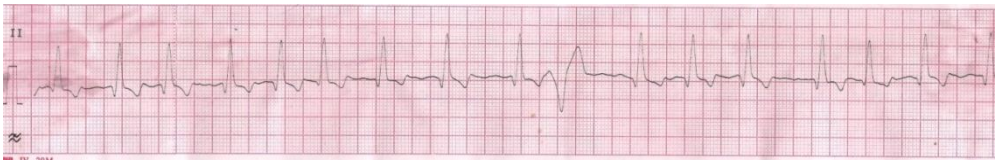

Fig. 8. Atrial fibrillation with ventricular premature complex

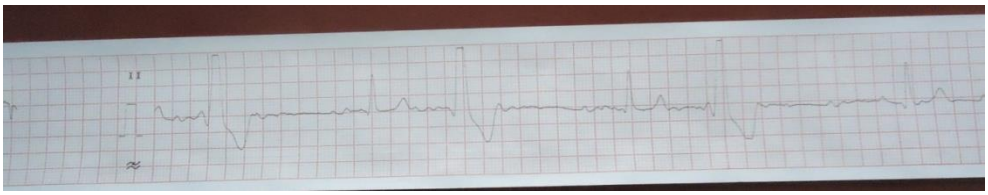

Fig 9. Ventricular bigeminy

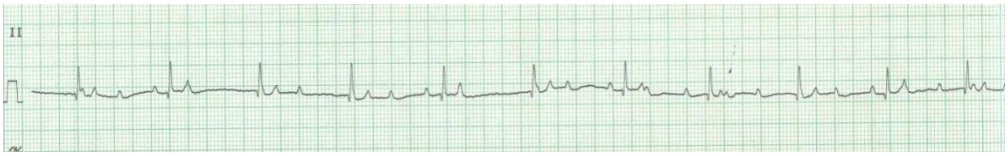

Fig. 10. Second degree heart block

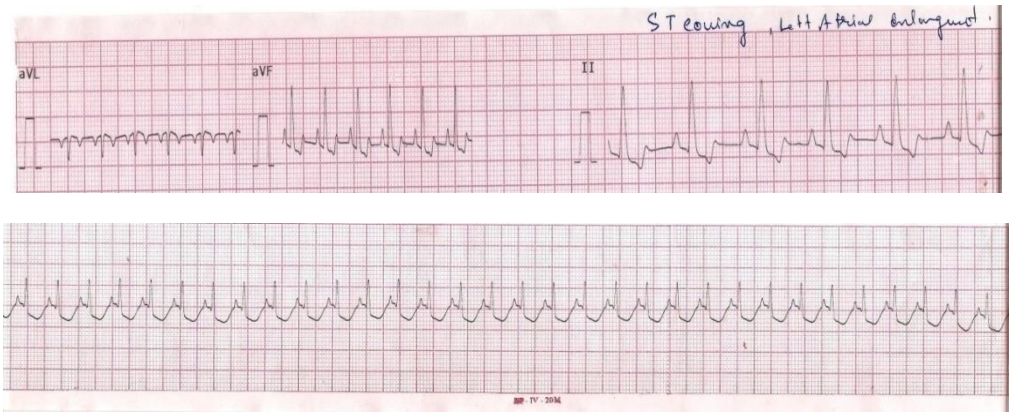

Fig. 11. ST segment slurring/ coving

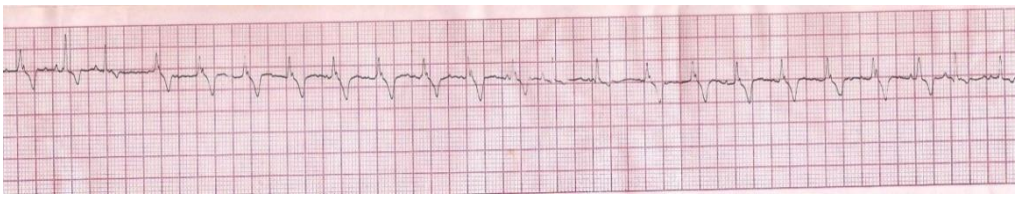

Fig. 12. Ventricular tachycardia

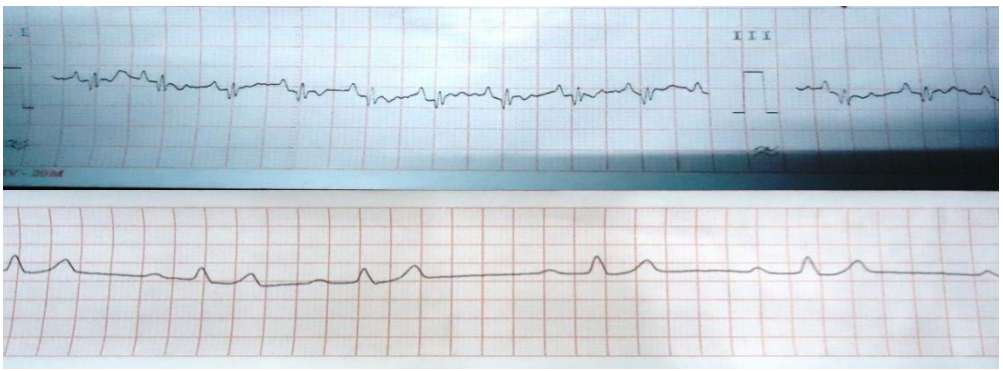

Fig. 13. Low voltage QRS complexes

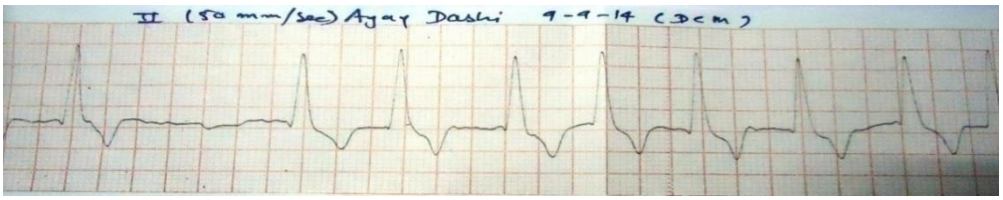

Fig. 14. Wide QRS complexes

Among various rhythm disturbances recorded in present study, atrial fibrillation predominated, which supplements the view of Borgarelli et al. (2001) Present study comprises of 65.51 % large sized dogs and 34.49 % medium to small sized dogs, which correlated well with previous reports where atrial fibrillation has been reported more frequently in giant breeds and ventricular premature complexes and the ventricular tachycardia are more frequent in Dobermanns. In contrast, some giant-breed dogs exhibit atrial fibrillation with no evidence of underlying cardiac disease, which is known as lone atrial fibrillation. Kumar et al. [14] suggested that these arrhythmias may be present up to nine months prior to the development of echocardiographic evidence of DCM in certain breeds like Doberman.

Present investigation showed ventricular premature complexes (VPCs) (41.38 %) (Figs. 6, 7) followed by ventricular tachycardia (13.79%) as most common ventricular arrhythmias. Severity of ventricular tachycardia is often associated with the degree of dilation and dysfunction of the left ventricle [24]. Some studies have found that most Doberman Pinschers in the occult phase have evidence of both abnormalities. Other studies reported that VPCs are often the first evidence for cardiomyopathy in the occult phase.

Cardiac conduction abnormalities were seen in 82.75% dogs with DCM, which was more than previous report of Kumar et al. [14]. Although, these rhythmic and conduction disturbances are not specific to this disease, but their clinical, prognostic and therapeutic evaluation aspects exalt the importance of consideration in all dogs affected by DCM. Branch block pattern and other interventricular conduction disturbance seen in present investigation supplements the view of Ware [11], but these changes are not specific and rarely contribute to the diagnosis and are considered as benign alterations [25,26].

4. CONCLUSION

This study revealed significant electrocardiographic differences between healthy

and DCM-affected dogs, with notable abnormalities in P wave amplitude and duration, QRS duration, and R wave amplitude among DCM dogs. Specific patterns of electrical disturbances were identified in left and bilateral DCM groups, highlighting the distinct manifestations of the disease. Increased P wave duration was a marker of left atrial enlargement, while increased amplitude indicated right atrial enlargement. Prolonged QRS complexes and deep Q waves were associated with ventricular enlargement and myocardial degeneration. Low voltage QRS complexes and electrical alternans suggested pericardial effusion or ascites. Rhythm disturbances, especially atrial fibrillation, were common in DCM-affected dogs, underscoring the need for comprehensive electrocardiographic evaluation in diagnosing and managing DCM. These findings enhance understanding of DCM's electrocardiographic characteristics, aiding in the early detection and treatment of this condition in dogs.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Bannasch DL, Oertle DT, Vo J, Batcher KL, Stern JA, Kaplan JL, Li RHL, Madden IE, Christen M, Leeb T, Joshi N. Naturally occurring canine laminopathy leading to a dilated and fibrosing cardiomyopathy in the nova scotia duck tolling retriever. Sci Rep. Nov 4, 2023;13(1):19077.
2. Tilley LP, Smith (Jr) FWK, Oyama MA, Sleeper MM. Electrocardiography Manual of Canine and Feline Cardiology. 4th Edition, Saunders (Elsevier) Saint Louis, Missouri. USA. 2008;49-77, 90,91, 115, 139 – 149, 180, 277, 296 -297.
3. Martin M. Small Animal ECGs: An Introductory Guide. edn. Wiley. 2007;1-76.
4. Gnieszka, Noszczyk-Nowak, Marcin Nowak, Józef, Nicpoń Janusz, Madej A. Evaluation of desmin expression by immunohistochemistry in myocardial cells of dogs with dilated cardiomyopathy. Bull. Vet. Inst. Pulawy, 2009 53: 509-513.
5. Dharmasuriya JYC, Akila NV, Kumara WRB, Rajapaksha E. Diagnosis and treatment of dilated cardiomyopathy and third degree atrioventricular block in dogs health and hygiene. In: Proceedings of the Peradeniya Univ. International Research Sessions, Sri Lanka. 2014;18:Abstract No: 806 (Poster)
6. Rajesh K, Suresh K, Lakshmi Kavitha K, Syaama SN. therapeutic profile of dilated cardiomyopathy in a dog. Anim. Sci. Reporter. 2010;4(1):37-40.
7. Ware WA. The cardiovascular examination. In: Cardiovascular disease in small animal medicine. 3rd Edn. Manson Publishing. The Veterinary Press. Iowa State University Ames, Iowa, USA. 2011;26-33.
8. Mark A. Oyama, Marc S, Kraus Anna, Gelzer R. Evaluation of electrocardiogram. In: Rapid review of ECG interpretation in small animal practice. CRP press. Taylor Francis Group USA. 2014;17-27.
9. Polana S. Dilated cardiomyopathy in the Dobermann dog: survival, causes of death and a pedigree review in a related line. Molecular genetics of dilated cardiomyopathy. In: The dobermann dog. Faculty of Veterinary Medicine, Universiteit Utrecht, The Netherlands; 2005.
10. Richard BF, Elisa M. Interpretation of electrocardiogram. In: Kirk & Bistner's Handbook of veterinary procedures and emergency treatment. 9th Edn. Elsevier Health Sciences. 2011;189-210.
11. Ware WA. Myocardial diseases of the dog. In Nelson, R.W. & Couto, C.G. (Eds). Small Animal Internal Medicine. 3ª ed. Rio de Janeiro, Brasil: Elsevier Editora Ltda. 2006;103-112.
12. Hideki Hayashi, Minoru Horie. Prognostic value of p wave for developing atrial fibrillation. in: Atrial Fibrillation - Basic Research and Clinical Applications. Edt. Jong-Il Choi. 2012;189-198.
13. Nicole Van Israel. Canine cardiomyopathies: aetiology and breed characteristics UK VET. 2003;8(7):1-7.
14. Kumar Satish K, Tirumala Rao DS, Nagaraj P. Dilated Cardiomyopathy in Dogs: A Study for 3 Years (2006 To 2009). Vet Scan. 2011;62(93):1-10.
15. Simiz F, Falca C, Moł T, Ciulan V, Morar D, Cristina P. Echocardiographical

aspects in canine cardiopathies. Lucrări Tiinłifice Medicină Veterinara. 2010;43(1): 334-337.
16. Brigite M Pedro, Joana V Alves, Peter J Cripps, Mike J. Stafford Johnson, Martin MWS. Association of QRS duration and survival in dogs with dilated cardiomyopathy: A retrospective study of 266 clinical cases. Journal of Veterinary Cardiology. 2011;243-249.
17. Huh J, Noh CI, Yun YS. The usefulness of surface electrocardiogram as a prognostic predictor in children with idiopathic dilated cardiomyopathy. J. Kor. Med. Sci. 2004;19:652-655.
18. Adesanya CO, Yousuf KA, Co C, Gaur S, Ahmed S, Pothoulakis A, Suryaprasad A, Gupta S. Is wider worse? QRS duration predicts cardiac mortality in patients with right bundle branch block. Annals of Nonivasive Electrocardiography. 2008;165-170.
19. Pandian JS. Clinical and ultrasonographic investigation of ascites in dogs. M.V.Sc. Thesis, Kerala Agriculture University, Thrissur, India; 2005.
20. Jinks J. Pericardial effusion in a mixed breed dog. Can. Vet. J. 2001;42:877-879.
21. De Ponti F, Poluzzi E, Cavalli A, Recanatini M, Montanaro N. Safety of non-antiarrhythmic drugs that prolong the QT interval or induce torsade de pointes. An overview. Drug Safety. 2002;25: 263-286.
22. Ettinger S, Feldman E. Textbook of Veterinary Internal Medicine. Disease of Dog and Cat. 6th ed. Elsevier. W.B. Saunders, St. Louis, Missouri, Philadelphia, USA. 2005;137-145, 914-1082.
23. Martin MWS, Johnson MJ, Stafford, Celona B. Canine dilated cardiomyopathy: A retrospective study of signalment, presentation and clinical findings in 369 cases J. Small Anim. Pract. 2009;50:23–29.
24. Calvert CA, Wall M. Results of ambulatory electrocardiography in overtly healthy Doberman pinschers with equivocal echocardiographic evidence of dilated cardiomyopathy. Journal of American Veterinary Medical Association. 2001; 219(6):782-784.
25. Guglielmini C, Civitella C. ECG of the month. J. Am. Vet. Med. Assoc. 2004;224(7):1076-1078.
26. Guglielmini C, Civitella C, Diana A, Di Tommaso M, Cipone M, Luciani A. Serum Cardiac troponin I concentration in dogs with precapillary and post capillary pulmonary hypertension. J. Vet. Intern. Med. 2010;24:145-52.

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