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Canine Dilated Cardiomyopathy: A Clinical and Diagnostic Study

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ABSTRACT

Dilated cardiomyopathy (DCM) is an acquired myocardial disorder of dogs characterized by dilation of chambers, hypokinesis and reduced contractility. Echocardiography is a standard diagnostic test for DCM however its technical expertise as well as the cost of equipment, limits its use under field conditions. This paper focuses on the clinical, hematobiochemical, radiographic and electrocardiographic changes in fifty-two dogs affected with dilated cardiomyopathy. Among the clinical signs recorded, exercise intolerance, lethargy, dyspnea, ascites, and cough were commonly seen followed by edema and syncope. Cardiomegaly is the characteristic finding seen on lateral thoracic radiograph measured as a significant increase in vertebral heart score, along with pulmonary edema and rarely pleural effusions as a sequel to congestive heart failure. Among the hematobiochemical findings, anemia and neutrophilic leukocytosis were noticed with non-significant increase of ALP, BUN, and creatinine. In the present study common arrythmias recorded were atrial fibrillation while the common morphological change noticed was ST coving indicative of myocardial hypoxia.

HIIGHLIGHTS

- Overt DCM is commonly expressed as exercise intolerance, lethargy, and dyspneainitially with gallop rhythm on cardiac auscultation.
- Increased VHS, pulmonary edema on thoracic radiography.
- Sinus tachycardia and atrial fibrillation on ECG are common.

Keywords: Canine dilated cardiomyopathy, Electrocardiography, Hematobiochemical findings, Vertebral heart score

Dilated cardiomyopathy (DCM) is an acquired cardiac disorder characterized by reduced myocardial contractility, hypokinesis and dilation of either left chambers or all the four chambers (O'Grady and O'Sullivan, 2004). Phenotypic expression of the disease varies with breed (Bonagura and Visser, 2022) as well as the stage of disease with no clinical signs seen in occult stage. Clinically the condition often mimics respiratory disease due to the presence of dyspnea and cough while ascites and edema developed with the progression of disease at later stage. Echocardiography is the gold standard for confirmatory diagnosis of DCM however the availability of equipment in field conditions is limited as well as it warrants sufficient technical expertise in handling as well as interpretation. Thorough knowledge of the clinical signs and their associated laboratory as well as diagnostic imaging findings complement the clinician in early identification of the clinical form of DCM, and to initiate appropriate treatment. This will prevent the development of congestive heart failure, early mortality along with improving the quality of pet's life. The present paper aims in describing the clinical, haemato-biochemical, and electrocardiographic changes seen in fifty-two dogs affected with dilated cardiomyopathy during the study.

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MATERIALS AND METHODS

Dogs presented to small animal outpatient unit, Madras Veterinary College between June 2021 - August 2022 with the symptoms of exercise intolerance, dyspnea, ascites were selected for the study. A total of twelve healthy dogs presented for general examination were subjected to hematobiochemical, radiographic and electrocardiographic evaluation that constituted the reference range for the study. During the study period, a total of fifty-two dogs were found to be affected with dilated cardiomyopathy which were confirmed by echocardiography (Fig. 1 and 2). Thorough clinical examination was done and all the cases were subjected to complete hematobiochemical evaluation, radiographic and electrocardiographic assessment. Phlebotomy was done aseptically at cephalic/saphenous veins and five ml of whole blood was collected of which two ml was transferred to EDTA vial for hematological studies and three milliliter to clot activator vials for serum biochemical studies. Hematological analysis was done using auto hematology analyzer (Mindray BC-2800 vet hematology analyzer) while semiautomated biochemical analyzer (A15 Biosystems Random Access Analyzer)was used for serum biochemical evaluation. Thoracic lateral radiographs were taken at inspiration on digital x-ray and were evaluated for vertebral heart score as recommended by Buchanan and Bucheler (1995). Electrocardiographic evaluation was done using a four-lead electrocardiogram (Vesta 301i electrocardiograph) in right lateral recumbency with all four limbs extended. Systolic blood pressure was examined by using a vmed doppler by placing the animal in lateral recumbency and measured over peripheral artery. Statistical analysis was done using SPSS software with t-test employed to test the significance.

RESULTS AND DISCUSSION

Among the clinical signs recorded exercise intolerance and lethargy was noticed in majority of the cases (94.23%) followed by dyspnea (80.76 %), ascites (67.30 %), cough (46.15 %), inappetence (36.53 %), difficulty in sleeping (32.69 %), anorexia (30.76 %), edema (17.30 %) and syncope (1.92 %) (Fig. 3, 4 & 5).

Among the vital signs recorded, significant increase ($p<0.01$) in heart rate and respiratory rate was noticed among the DCM affected dogs with no significant difference in rectal temperature. Elevated heart rate in DCM reflects the chronotropic response induced by the sympathetic stimulation to compensate the reduced cardiac output (Santos *et al*., 2006; Borovac *et al*., 2020). Tachypnoea noticed in DCM affected dogs is a sign related to reduction in cardiac output leading to decreased tissue oxygenation and also due to the pulmonary edema developed in DCM. Pulmonary edema is a common consequence seen in DCM because of increased left atrial pressure leading to increased pulmonary hydrostatic pressure resulting in left side heart failure (Saini, 2021). Cardiac auscultation revealed presence of gallop rhythm in majority of the dogs with DCM. Gallop rhythm also called gallop ventricular sound is a third heart sound produced during early diastole at the time of ventricular filling (Englar, 2019). Significant decrease ($p<0.01$) was noticed in systolic blood pressure of diseased dogs (113.66 ± 2.27 mm Hg) in comparison with

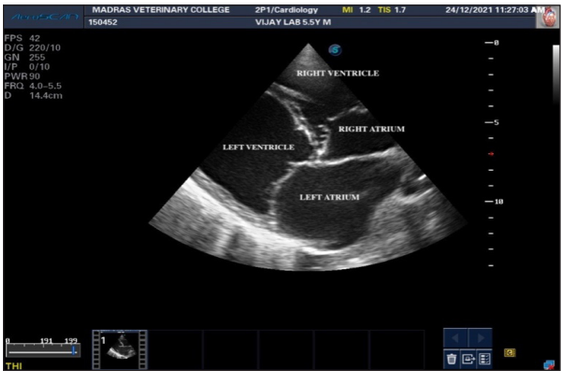

Fig. 1: Dilated cardiac chambers in canine dilated Cardiomyopathy

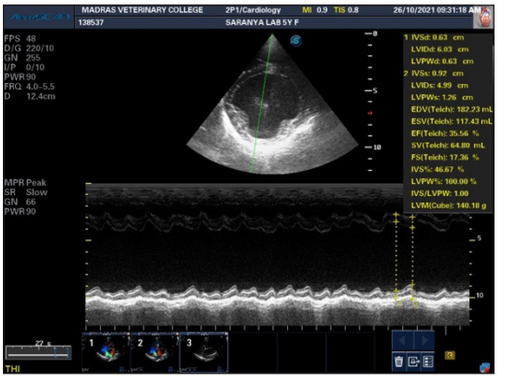

Fig. 2: M-mode evaluation of left ventricular internal diameters and systolic function

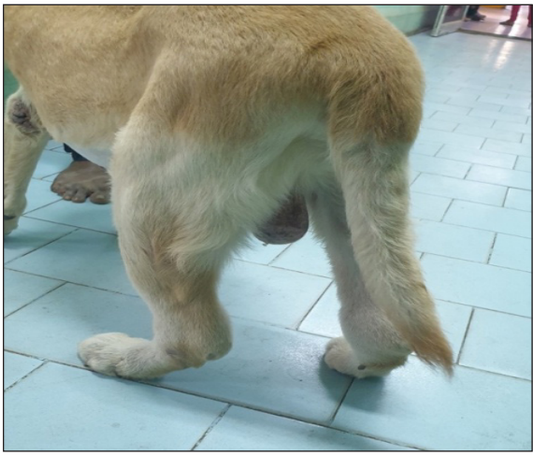

Fig. 3: Limb edema

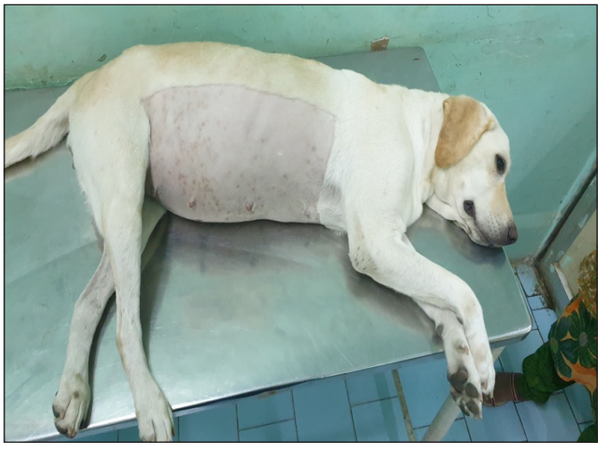

Fig. 4: Recumbent dog with ascites

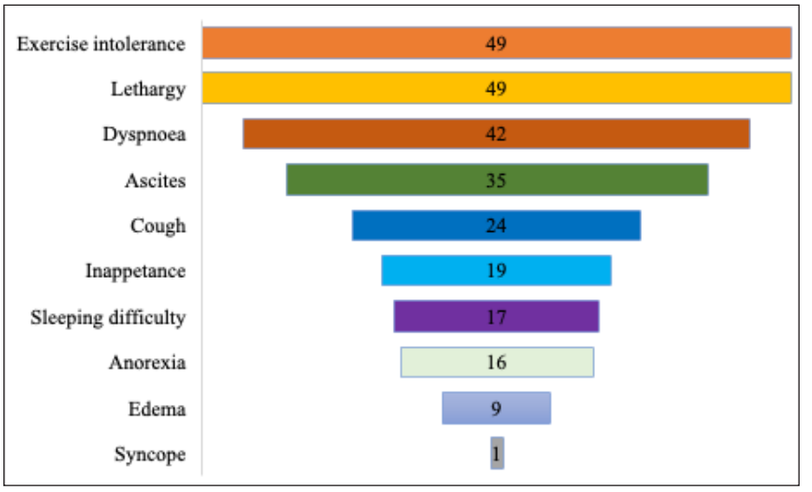

Fig. 5: Clinical signs recorded in canine dilated cardiomyopathy

healthy dogs (136.58 ± 3.19 mm of Hg) and is within the normal limit. Blood pressure is determined by the cardiac output and systemic vascular resistance and the decreased blood pressure is possibly explained due to the drop in cardiac output caused by the systolic failure in DCM. Mean ± SE of vertebral heart score in the affected cases was 12.42 ± 0.26 (Fig. 6) which was significantly increased ($p<0.01$) when compared with healthy ones (9.95 ± 0.13). DCM is characterized by dilation of chambers with resultant cardiomegaly classically represented by elevated vertebral heart score on thoracic radiograph however with some differentials like pericardial effusion, PPDH, mitral valve disease etc. Hematological evaluation revealed significant ($P<0.01$) decrease in haemoglobin, PCV, total erythrocyte count while total leukocyte count and absolute neutrophil count was significantly increased ($p<0.05$) (Table 1). Cardio renal anaemia syndrome was described in DCM affected dogs by Srivastava *et al*. (2015) and was due to decreased erythropoietin activity in these cases. Elevated neutrophils and leukocytosis in DCM cases was also reported by Vishnurahav *et al*. (2017) and Vatnikov *et al*. (2019). Neutrophilic leukocytosis in DCM is due to a proinflammatory state induced by the myocardial damage and setting up of aseptic inflammation.

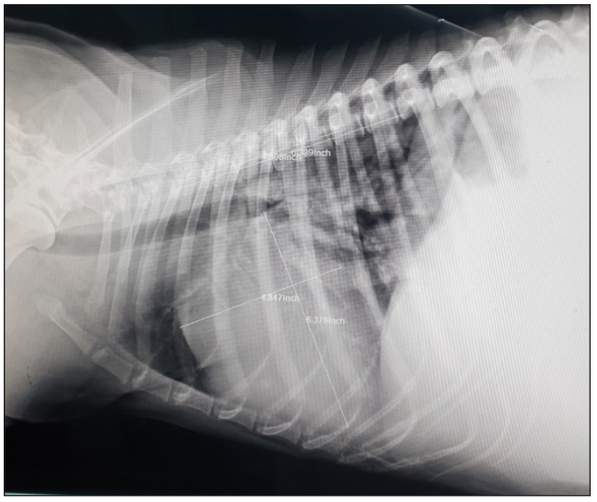

Fig. 6: Vertebral heart score measured in a dog with DCM (VHS=12.5), Pulmonary edema is visible in caudal lung lobes

No significant difference was seen in serum biochemical parameters (Table 2) with non-significant increase noticed in BUN, creatinine, alkaline phosphate with non-significant decrease in total protein and albumin. The findings were in accordance with the reports of Adin *et al*. (2019) and Haritha *et al*. (2020). Congestive hepatopathy developed in DCM could have resulted in elevated levels of alkaline phosphate. Elevated renal markers i.e., BUN and creatinine was due to the reduced cardiac output leading to reduced renal perfusion, venous congestion and reduced glomerular filtration rate.

Electrocardiographic examination revealed significant increase in P wave duration ($p<0.05$), R amplitude ($p<0.05$), QRS duration ($p<0.01$) and a significant decrease ($p<0.01$) of PR interval compared to healthy dogs however the respective values were within the normal limits. Increased chamber diameters i.e., left atrial dilation leads to

Table 1: Hematological changes in DCM affected dogs

Sl. No.	Parameter	DCM dogs (n=52) (Mean ± SE)	Healthy dogs (n=12) (Mean ± SE)
1	Haemoglobin (g/dl)	11.11^{a}± 0.35	14.75^{b}± 0.29
2	Packed Cell volume (%)	31.53 ^{a}± 1.04	41.63^{b}± 1.41
3	Total erythrocyte count (10^6/cmm)	5.32^{a}± 0.69	6.43^{b}± 0.17
4	Total leukocyte count (/cmm)	19129^{a}± 1891	11025 ^{b}± 748
5	Platelets (/cmm)	207541^{a}± 19922	247750^{a}± 18951
6	Neutrophil (%)	79.29^{a}± 0.48	73.69^{b}± 0.52
7	Lymphocyte (%)	14.61^{a}± 1.52	20.58^{b}± 0.43
8	Monocyte (%)	4.75^{a} ± 0.51	4.78^{a}± 0.31
9	Eosinophil (%)	0.50^{a} ± 0.23	0.67^{a} ± 0.28

Table 2: Serumbiochemical changes in DCM affected dogs

Sl. No.	Parameter	DCM dogs (n=52) (Mean ± SE)	Healthy dogs (n=12) (Mean ± SE)
1	Glucose (mg/dl)	92.17^{a} ± 4.21	80.67 ^{a} ± 1.72
2	ALT (U/L)	54.00^{a} ± 6.69	50.75 ^{a} ± 7.01
3	ALP (U/L)	109.83^{a} ± 27.93	69.17 ^{a} ± 19.86
4	Total protein (g/dl)	6.60^{a} ± 0.29	7.26 ^{a} ± 0.09
5	Albumin (g/dl)	2.53^{a}± 0.14	3.00 ^{a}± 0.10
6	Globulin (g/dl)	4.26^{a} ± 0.13	4.26 ^{a} ±0.13
7	BUN (mg/dl)	23.42^{a} ± 6.33	11.32 ^{a} ± 1.30
8	Creatinine (mg/dl)	1.25^{a} ± 0.12	1.09 ^{a} ± 0.09
9	Calcium (mg/dl)	9.91^{a} ± 0.34	10.69 ^{a} ± 0.25
10	Phosphorus (mg/dl)	5.84^{a}± 0.42	4.78 ^{a}± 0.18
11	Sodium	141.58^{a} ± 0.72	140.16 ^{a} ± 0.81
12	Potassium	4.57^{a} ± 0.23	4.26 ^{a} ± 0.07
13	Chloride	106.77^{a} ± 1.28	110.35 ^{a}± 0.66

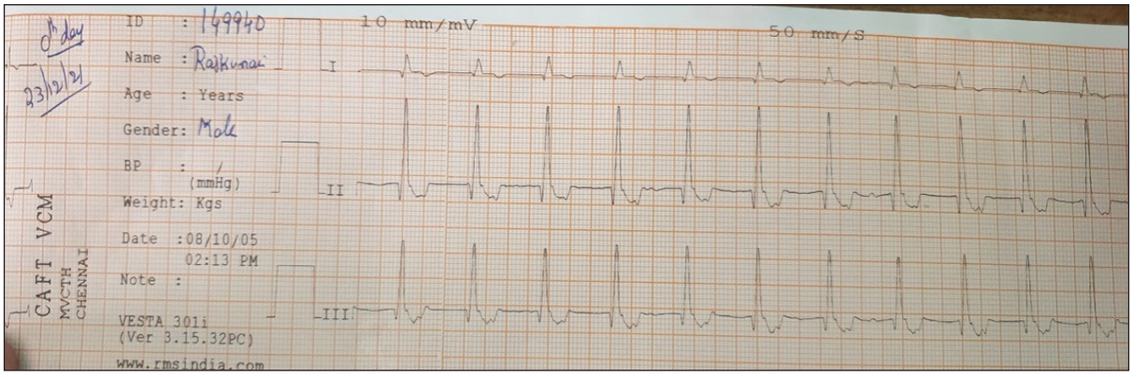

Fig. 7: Atrial fibrillation (absence of p waves) in a dog with DCM

Table 3: Electrocardiographic parameters in DCM dogs vs Healthy dogs

Parameter	P wave amplitude (mv)	P wave duration (sec)	P-R interval (sec)	R wave amplitude (mv)	QRS duration (sec)	T wave amplitude (mv)
Dilated cardiomyopathy (Mean ±SE) (n=52)	$0.16^{a} \pm 0.01$	$0.047^{a} \pm 0.003$	$0.08^{a} \pm 0.06$	1.67 ± 0.10^{a}	0.042 ± 0.001^{a}	0.228 ± 0.02^{a}
Healthy dogs (Mean ±SE) (n=12)	$0.14^{b} \pm 0.01$	$0.040^{b} \pm 0.00$	$0.12 \pm 0.01^{b**}$	1.2 ± 0.08^{b}	0.03 ± 0.00^{b}	0.21 ± 0.03^{a}

increased P wave duration while increased left ventricular diameter reflects as increased R wave amplitude and QRS duration (Willis *et al*., 2018). Among the arrythmias noticed atrial fibrillation was found to be the most common one seen in dilated cardiomyopathy (Fig. 7) followed by ventricular premature complex. Among the morphological abnormalities ST coving is another prominent finding of DCM and is indicative of myocardial hypoxia.

CONCLUSION

In the present study hematobiochemical evaluation is not found to have significant diagnostic importance in canine dilated cardiomyopathy however is useful in evaluating the severity of disease and monitoring the therapeutic response. Presence of prerenal azotemia in DCM is often mistaken as renal failure and need to be ruled out with other diagnostic parameters for the confirmation of renal involvement and cardiorenal syndrome. Regular blood pressure monitoring is advised to prevent hypotension as a side effect of treatment. Thoracic radiographs were found useful to assess the degree of cardiomegaly, severity of pulmonary edema and subsequent response to therapeutic management. Electrocardiographic evaluation was useful to assess arrythmias and myocardial hypoxia as their treatment is vital in determining the course of the disease. Thus, a thorough physical examination, meticulous radiographic interpretation of thorax and timely electrocardiographic study will almost diagnose the congestive heart failure and treatment may be initiated early for better prognosis. Later echocardiography may be employed for the confirmation of the primary condition.

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