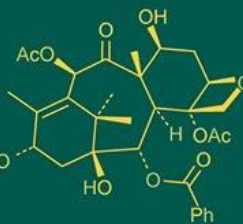
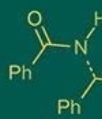
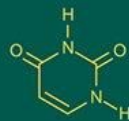
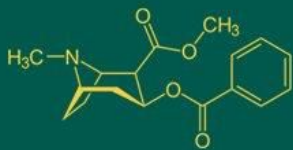


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Diagnostic use of cardiac biomarkers and echocardiography in canine dilated cardiomyopathy

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Abstract

Dilated cardiomyopathy (DCM) is a well-recognized cause of heart failure in dogs and often act silent killers. Diagnosis with use of ECG and Echocardiography is not a routine in India except at few institutes. This is the first report from Gujarat (India) on diagnostic use of cardiac biomarkers and echocardiography in canine dilated cardiomyopathy in dogs. The study included 9 healthy dogs and 56 confirmed cases of DCM out of which 18 cases were randomly selected for the detailed study. DCM affected dogs showed history and clinical signs of exercise intolerance, dyspnoea, coughing, inappetence, weight loss, abdominal enlargement, vomiting and syncope. The typical clinical features of DCM cases were tachycardia, murmur, pulmonary oedema and muffled heart sound. The heart rate, pulse rate and respiration rate of DCM affected dogs were significantly higher than healthy group with no change in rectal temperature of both groups. The average diastolic blood pressure in DCM group was significantly higher than healthy group. Important ECG findings noticed in DCM group were atrial fibrillation (38.89%), sinus tachycardia (33.33%), ventricular premature complex (16.67%), normal sinus arrhythmia (5.56%) and atrial flutter (5.56%). Significant increased LA/Ao ratio and EPSS along with low EF and FS on echocardiography confirmed the cases of DCM. Further, the biomarkers: NT-pro BNP, cTn-I and CPK-MB were found elevated in DCM group than healthy group. Results suggests that dilated cardiomyopathy being the most commonly observed acquired heart disease in middle aged dogs requires awareness in owners for regular checkup to diagnose cases in earlier so that follow-up treatment can be helpful for enhancement of lifespan of affected dogs.

Keywords: Cardiac biomarkers, canine dilated cardiomyopathy, echocardiography

Introduction

Veterinary cardiology has advanced greatly since last two decades. According to information disseminated by the American Veterinary Medical Association (AVMA), one in ten dogs suffers from heart disease. The most commonly encountered cardiac abnormalities which cause heart failure in dogs are degenerative mitral valve diseases (MVD) and dilated cardiomyopathy (DCM) whereas, other less common abnormalities include congenital heart disease, infectious endocarditis, cardiac neoplasia, heart worm disease or other conditions leading to pulmonary hypertension (DeFrancesco, 2001) ^[1]. Cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually exhibit inappropriate ventricular hypertrophy or dilation (O'Grady and Sullivan, 2004) ^[2]. Dilated cardiomyopathy is one of the most common cardiac diseases in dogs and humans especially in the dog; it primarily affects large and giant breeds (Tidolhm, 2006) ^[3]. The diagnosis of DCM is mainly based on history, clinical signs, radiography, electrocardiography (ECG) and echocardiography (Tilley *et al.*, 2008) ^[4]. Echocardiography permits non-invasive and non-ionizing visualization inside the heart including the aorta, the ventricles and atria, the auricular appendages and all of the cardiac valves. M-mode echocardiography helps to measure cardiac chamber size, measurement of wall thickness and detects cardiomegaly and hypertrophy, while, blood flow through the heart can be seen and measured with doppler ultrasound (Boon, 2011) ^[5]. The cardiac biomarkers help in the suspicion and diagnosis as well as monitoring of cardiovascular diseases and congestive heart failure. Cardiac troponin I (cTnI) has been recognized as the most sensitive and specific marker of myocardial cell necrosis in humans, demonstrating a higher specificity than the cardiac isoenzyme creatine kinase (CKMB) and better sensitivity and specificity than cardiac troponin T (cTnT) (Lavecchio *et al.*, 2009) ^[6]. N-terminal pro-B-type natriuretic peptide

(NT-proBNP) is a cardiac biomarker that increases in the blood of dogs with heart diseases (Oyama *et al.*, 2009) [7]. Still, there is paucity of information regarding use of advance diagnostic modalities for canine cardiac abnormalities in India. Looking to the need of the era, the study was undertaken and executed.

Materials and Methods

The study was conducted during a period of 2 years (April, 2018 to March, 2020) in the Department of Veterinary Medicine in collaboration with Department of Veterinary Clinical Complex (VCC), College of Veterinary Science and Animal Husbandry, NAU, Navsari, Gujarat, India. The detail examination was performed in 18 confirmed DCM cases and 8 apparently healthy dogs presented to the hospital for health check-up and vaccination as control group. The detail study included clinical examination, blood pressure, radiography, electrocardiography, echocardiography, haemato-biochemical and cardiac biomarkers examination. The clinical examination included physical examination and special cardiovascular assessment as suggested by Tilley *et al.* (2008) [4] and Ettinger and Feldman (2010) [8]. The haemato-biochemical analysis was performed using auto-analyzer in departmental laboratory, whereas, biomarkers were got analyzed in private human laboratory using commercially available diagnostic kits. For the blood pressure measurement, the average of at least three measurements from each dog was recorded as the final blood pressure as described by Carr (2001) [9]. In thoracic radiography, Vertebral Heart Score (VHS) was calculated as suggested by Buchanan and Bucheler (1995) (Fig.5). The echocardiography was performed with phased array Cardiac Probe PA320 with frequency ranged between 5.0 to 8.0 MHz. 2-Dimensional (2-D) and Motion-Mode (M-mode) imaging was done as per standard protocols followed by Boon (2011) [5] (Fig.6).

Following measurements were recorded using 2-D and M-mode imaging

1	RVDd(cm)	Right ventricular diameter in diastole
2	IVSd (cm)	Inter ventricular septum in diastole
3	LVDd(cm)	Left ventricular diameter in diastole
4	LVPWd (cm)	Left Ventricular Posterior wall in diastole
5	RVDs (cm)	Right Ventricular diameter in systole
6	IVSs (cm)	Inter ventricular septum in systole
7	LVDs (cm)	Left ventricular diameter in systole
8	LVPWs (cm)	Left ventricular posterior wall in systole
9	EF (%)	Ejection fraction
10	FS (%)	Fractional shortening
11	LA (cm)	Left atrium
12	AO (cm)	Aorta
13	LA/AO	Left atrium / Aorta
14	EPSS (mm)	E Point to Septal Separation

From these values the contraction indices like fractional shortening and ejection fraction were calculated by the following formulas:

$$\text{Fractional shortening} = \frac{\text{LVIDd} - \text{LVIDs}}{\text{LVIDd}} \times 100 \text{ per cent (Boon, 2011) [5]}$$

$$\text{Ejection Fraction} = \frac{\text{EDV} - \text{ESV}}{\text{EDV}} \times 100 \text{ per cent (Boon, 2011) [5]}$$

Statistical analysis: Different parameters in DCM affected dogs were compared with healthy dogs using student 'T' test using IBM SPSS statistical software version 20.0.

Results

History and clinical signs: During the study period, history of DCM affected dogs showed exercise intolerance and dyspnoea (83.33%) as most frequently observed signs followed by weakness (77.78%), coughing (72.22%), inappetence (66.67%), weight loss (61.11%), abdominal enlargement (55.56%), vomiting (27.78%) and syncope (16.67%) (Fig.1-4). The mean rectal temperature of DCM affected dogs was $101.94 \pm 0.30^{\circ}\text{F}$ which could not differ from control group ($101.33 \pm 0.17^{\circ}\text{F}$). The heart rate (149.17 ± 4.59), pulse rate (145.94 ± 4.27) and respiration rate (36.39 ± 1.17) per minute in DCM affected dogs were significantly higher than control group ($p < 0.01$). The typical characteristics features of DCM observed were tachycardia (77.78%), murmur (77.78%), pulmonary oedema (38.89%) and muffled heart sound (11.11%). The average diastolic blood pressure in DCM group (127.39 ± 2.62) was significantly higher than control group (87.17 ± 2.31) ($p < 0.05$). Vertebral heart score on radiograph was significantly higher in DCM affected dogs (12.27 ± 0.25 V) than control group (8.97 ± 0.11 V) (Fig.5). Thoracic radiographs of the healthy dogs did not show any enlargement / abnormal regional dilatation or deviation of cardiac silhouette.

Echocardiography

In 2-D echocardiography, the mean Left atrium (LA) diameter in DCM group was significantly higher (3.98 ± 0.13 cm) and Aortic (Ao) diameter (1.98 ± 0.07 cm) was significantly lower than the corresponding diameter of control group (Table-1) (Fig.8). The mean of LA/Ao ratio in the DCM group was significantly higher (2.05 ± 0.09) than control group ($p < 0.01$) and value recommended (LA/Ao ratio < 1.60).

In M-mode echocardiography, highly significant increased LVDd (5.30 ± 0.21 cm) and LVDs (4.57 ± 0.19 cm) was observed in DCM affected dogs than control group. The significantly higher values of RVDd and RVDs were also observed in DCM affected dogs than control group ($p < 0.05$). The mean of IVSd was significantly lower ($p < 0.01$) whereas, mean of IVSs was comparatively lower in DCM group than control group which are an indicative of reduced thickness of inter ventricular septum during dilatation of ventricles (Fig.7). The significantly decreased values of LVPWd ($p < 0.05$) and LVPWs ($p < 0.01$) in DCM group as compared to the control group was noticed. The means of EF ($26.94 \pm 2.64\%$) and FS ($13.68 \pm 1.49\%$) in DCM group were significantly lower than the control group ($p < 0.01$) (Fig.9). The EPSS in DCM group (1.71 ± 0.08 cm) was significantly higher than control group (0.40 ± 0.02) ($p < 0.01$) (Table-1).

Haemato-biochemical findings: Significant decreased means of haemoglobin, packed cell volume and RBC was observed in DCM group than control group ($p < 0.01$). Comparative increased means of monocytes and neutrophils were also observed in DCM group than control group. In DCM group, the mean of total protein was comparatively and albumin was significantly lower than control group ($p < 0.01$), whereas, AST was significantly ($p < 0.01$) and ALT was comparatively higher than control group (Table-2).

Cardiac biomarkers: The mean of cTn-I and CPK-MB were found comparatively elevated in DCM affected dogs than control group. The mean NT-proBNP in DCM group was significantly higher (319.29 ± 38.20 pg/ml or 1171.04 ± 140.23 pmol/l) than control groups ($p < 0.05$) (Table-3).

Discussion

History and clinical signs: History and various clinical signs observed in present study were in accordance with the findings of Petric *et al.* (2002) [10] who observed coughing in 79% cases followed by anorexia (71%), exercise intolerance (43%), dyspnoea (36%), vomiting (14%), syncope (14%), tachycardia (14%) and abdominal distension (7%) as the chief clinical complaints. Similarly, Tidholm (2006) [3] reported dyspnoea in all cases followed by cough (73%), exercise intolerance (60%), inappetence (42%), polydipsia (15%), weight loss (13%), syncope (11%) and abdominal enlargement (6%). Later on Martin *et al.* (2009) [11] recorded most presenting signs of DCM with their frequency. The signs were breathlessness (67%), cough (64%), exercise intolerance (48.8%), weakness (39.2%), reduced appetite (35.7%), weight loss (30.2%), collapse (26%), dullness and lethargy (20%). Two reports (Jeyaraja *et al.*, 2015; Venkateshkumar *et al.*, 2018) [12,13] had also reported similar findings in their studies on dilated cardiomyopathy in dogs. Rectal temperatures in DCM and control group did not differ. Similarly, DeFransesco (2001) [1] and Deepti *et al.* (2016) [14] did not find any difference in rectal temperature of DCM affected and healthy dogs. Whereas on contrary, McEwan *et al.* (2003) [15] reported elevation of body temperature in some cases and stated that clinical presentation could vary from case to case and according to breed.

The heart rate, pulse rate and respiration rate of DCM affected dogs were significantly higher than control group. Increased heart rate, pulse rate and respiration rate might be due to congestion or pulmonary edema with resultant hypoxemia and hypercapnia (Tilley *et al.*, 2008) [4]. Tachycardia as a chief clinical complaint of DCM was also reported by Petric *et al.* (2002) [10]. Further, variation in heart rate and rhythm due DCM were also observed by various authors. Further, Tidholm (2006) [3] found soft systolic murmur (grades III/VI) in 56% cases of DCM. Martin *et al.* (2009) [16] also noticed murmur and gallop sound in 33 and 10% DCM affected dogs. In an Indian study, Jeyaraja *et al.* (2015) [12] also recorded gallop rhythm and systolic murmur in 87 and 73% DCM cases as major clinical features.

In our study, an average diastolic blood pressure in DCM group was significantly higher than control group. But, reports of Velhankar (2013) [17] and Thirunavukkarasu (2019) [18] showed non-significant differences in systolic and diastolic blood pressures of DCM affected and healthy dogs. The vertebral heart score on radiography of DCM affected dogs was significantly higher than control group. It might be suggestive of the enlargement of cardiac silhouette due to presence of structural heart disease and due to dilatation of cardiac chambers in DCM. Similarly, increased VHS in DCM group was also recorded previously by Venkatesakumar *et al.* (2018) [13] and Bhargavi *et al.* (2019) [19].

Echocardiography: The mean of LA/Ao ratio in the DCM group was significantly higher than control group and value recommended (LA/Ao ratio < 1.60) by Tilley *et al.* (2008) [4]. It is suggestive of left atrial enlargement due to increased diameter of left atrium (LA) and decreased diameter of aorta

(Ao). Such findings are in agreement with Deepti *et al.* (2016) [14] who reported increased diameter of left atrium (5.3 cm) during end systole and decreased aortic diameter (2.15 cm) during end diastole in DCM affected dogs. It might be due to the elevated ventricular end systolic pressure which creates low pressure gradient from the atrium into the ventricle resulting in less atrial emptying leading to high left atrial pressures and ultimately atrial dilatation in DCM affected dogs (Calvert and Brown, 1986) [20].

In M-mode echocardiography, increased LVDD and LVDs in DCM group are in agreement with Meurs *et al.* (2001) [21] and Calvert & Wall (2001) [22] who observed increased left ventricular internal dimension in DCM affected Doberman Pinchers. In above context, McEwan *et al.* (2003) [15] stated that increased left ventricular lumen (particularly during systole) can be an important M-mode echocardiography criteria for the diagnosis of DCM. Further, Boon (2011) [5] stated that reduced thickness of inter ventricular septum and wall results into decreased function. The finding of decreased thickness of posterior wall in DCM is also in agreement with reports of McEwan *et al.* (2003) [15] and Boon (2011) [5]. Dilated cardiomyopathy alters the thickness of left ventricular posterior wall owing to eccentric hypertrophy and its severity is dependent on breed, its genetic predisposition and stage of disease at the time of examination (Tilley *et al.*, 2008) [4]. The above changes resulted into reduction of EF and FS values which is clearly suggestive of left ventricular systolic dysfunction with significant reduction in cardiac output (EF) and left ventricular myocardial contractility (FS) in DCM affected dogs. The finding of significant reduction of EF is in agreement with previous reports in which they stated that low fractional shortening ($< 25\%$) is the best M-mode echocardiography criteria for the diagnosis of DCM (Boon, 2011; Meurs *et al.*, 2001; McEwan *et al.*, 2003) [5, 21, 15]. Whereas, for reduced FS, O'Grady and Sullivan (2004) [2] stated that fractional shortening (FS) is the major indicator of systolic function in Veterinary echocardiography and values of FS less than 15% are suggestive of strong evidence of DCM. The finding of increased EPSS in DCM affected dogs is in accordance with reports of Jeyaraja *et al.* (2015) [12] and Wess *et al.* (2017) [23] who reported EPSS > 6.5 mm (0.65 cm) in DCM cases. Previously, McEwan *et al.* (2003) [15] also opined that the increased mitral valve EPSS can be considered as one of the important criteria for the diagnosis of DCM. Boon (2011) [5] also stated that E-point to septal separation (EPSS) is one of the consistent and popular mitral valve measurements for diagnosis of DCM.

Haematology: The haematological observations of present study are in accordance with previous reports in which significant increased means of white blood cell count and absolute neutrophil count in DCM affected dogs was noticed (Sesh *et al.*, 2013; Singh *et al.*, 2014; Jeyaraja *et al.*, 2015) [24, 25, 12]. Increased level of leucogram and neutrophils in the DCM group could be due to other primary or concurrent diseases.

Biochemical studies: The mild elevation in liver enzyme could be due to liver damage and decreases in total protein and albumin have been noticed due to impaired liver function in DCM affected group. Previously, Ettinger and Feldman (2010) [8] observed non-significant increase in ALT and AP levels due to degree of hepatic damage associated with its engorgement. Similarly, previous reports mentioned also

elevation of ALT along with low total protein and albumin level in DCM affected dogs (McEwan *et al.*, 2003; Martin *et al.*, 2009; Haritha *et al.*, 2019) [15, 16, 26].

Cardiac biomarkers: The finding of elevated level of cTn-I in DCM group is in agreement with observation of Spratt *et al.* (2005) [27] who reported significant elevated cTn-I level in dogs with acquired mitral valve disease, dilated cardiomyopathy and pericardial effusion. Later, Lavecchio *et al.* (2009) [6] explained that cardiac troponin-I (cTn-I) is a polypeptide found specifically in cardiac muscle tissue that has been used as a diagnostic and prognostic indicator of cardiomyopathy in dogs and cats. Along with elevated cTn-I,

UshaShree (2013) [28] also found elevated CK-MB (35.04±4.93 U/L) in DCM dogs as compared to control dogs (13.79±2.83 U/L). In context to significant elevated NT-proBNP, Kimmenade and Januzzi, (2009) [29] stated that BNP is rapidly produced by cardiomyocytes after stimuli like myocardial stretch or hypoxia and revealed that a concentration of less than 900 pmol/l of NT-proBNP is not compatible with myocardial increased stretch and stress. Whereas, on contrary Oyama *et al.* (2009) [7] stated that serum NT-proBNP cut-off concentration >1,158 pmol/ L discriminated between dogs with congestive heart failure and primary respiratory tract diseases.

Table 1: Mean values of 2D and M-mode echocardiography parameters in control and DCM group

Sr. No.	Parameters	Control group (n=8)	DCM group (n=18)	P values
1	LA (cm)	2.40±0.06	3.98±0.13	0.000**
2	Ao (cm)	2.20±0.03	1.98±0.07	0.006**
3	LA/Ao	1.08±0.01	2.05±0.09	0.000**
4	LVIDd (cm)	3.48±0.26	5.30±0.21	0.000**
5	LVIDs (cm)	2.38±0.21	4.57±0.19	0.000**
6	RVIDd (cm)	0.77±0.09	1.21±0.15	0.019*
7	RVIDs (cm)	0.59±0.10	1.11±0.16	0.031*
8	IVSd (cm)	0.91±0.04	0.73±0.05	0.009**
9	IVSs (cm)	1.23±0.07	0.97±0.11	0.055
10	LVPWd (cm)	0.77±0.08	0.54±0.04	0.020*
11	LVPWs (cm)	1.16±0.06	0.74±0.04	0.000**
12	FS%	32.05±1.20	13.68±1.49	0.000**
13	EPSS (cm)	0.40±0.02	1.71±0.08	0.000**
14	EF (%)	60.63±1.16	26.94±2.64	0.000**

* Indicates significant at $p < 0.05$; ** indicates highly significant at $p < 0.01$

Table 2: Mean values of various hemato-biochemical parameters in control and DCM group

Sr. No.	Parameters	Control group (n=8)	DCM group (n=18)	P values
1	HB (g/dl)	15.98±0.67	10.89±0.62	0.000**
2	PCV (%)	46.54±1.68	31.41±1.60	0.000**
3	RBC (m/cmm)	7.30±0.36	5.35±0.18	0.000**
4	WBC (10 ³ /cmm)	11.96±0.73	16.58± 2.16	0.055
5	Neutrophil (%)	69.13±2.86	73.33±2.30	0.170
6	Lymphocyte (%)	26.88±2.77	21.44±1.98	0.068
7	Eosinophil (%)	1.38±0.18	0.83±0.22	0.080
8	Monocyte (%)	2.63±0.42	4.33±0.79	0.045*
9	MCV (fl)	56.56±1.17	57.71±1.16	0.691
10	MCH (pg)	17.51±2.38	19.76±0.64	0.396
11	MCHC (%)	34.40±0.44	34.21±0.85	0.977
12	BUN (mg/dl)	10.08±0.83	25.22±3.58	0.000**
13	Creatinine (mg/dl)	1.21±0.02	1.34±0.12	0.151
14	Total protein (gm/dl)	6.58±0.14	5.57±0.34	0.074
15	Albumin (gm/dl)	3.20±0.08	2.35±0.12	0.000**
16	ALT (IU/L)	37.49±2.45	55.47±12.41	0.201
17	AST(IU/L)	22.67±1.96	49.63±9.53	0.009**

* Indicates significant at $p < 0.05$; ** indicates highly significant at $p < 0.01$

Table 3: Mean values of cardiac biomarkers in control and DCM group

Sr. No.	Parameters	Control group (n=8)	DCM group (n=18)	P values
1	Cardiac troponin-I (ng/ml)	0.01±0.00	16.45± 12.74	0.218
2	CPK-MB (u/l)	6.29±1.60	49.73±27.71	0.138
3	NT-proBNP (pg/ml)	<15*	319.29±38.20	0.007**

* Values not detected in machine as below 15 pg/ml; ** indicates highly significant at $p < 0.01$



Fig.1: Ascites in DCM affected dog



Fig.2: Emaciated body condition and severe weakness in DCM affected dog



Fig.3: Jugular engorgement in DCM affected dog



Fig.4: Lead placement for ECG measurement on right lateral recumbency



Fig.5: VHS measurement in lateral thoracic radiograph of DCM affected dogs



Fig.6: Echocardiographic examination using cut out wooden table in RPLAX position

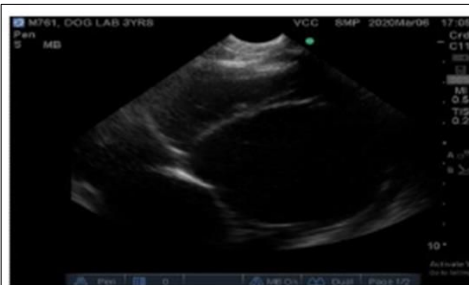


Fig.7: Dilated four chambers in DCM affected dog (RPLAX view)

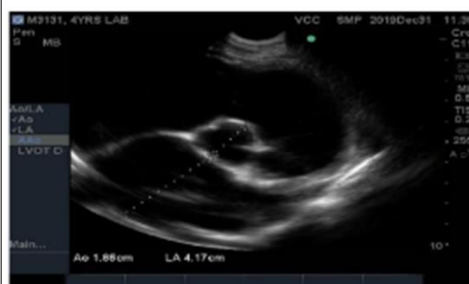


Fig.8: 2-D measurement showing left atrial enlargement in DCM affected dog

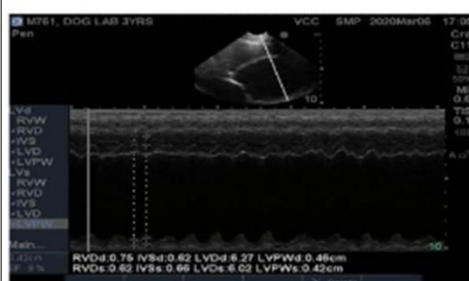


Fig.9: Reduced ejection fraction and left ventricular enlargement with hypo kinetic inter-ventricular septum and left ventricular posterior wall in DCM affected dog on M-mode echocardiography (RPLAX view)

Conclusions

Dilated cardiomyopathy in dogs is characterized by significant clinical, radiographic, echocardiographic, haematological and biochemical alterations. Echocardiography and cardiac biomarkers, particularly NT-proBNP and cTn-I, are valuable diagnostic tools for confirming systolic dysfunction. Early and comprehensive evaluation aids accurate diagnosis, disease monitoring and improved therapeutic management of affected dogs.

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