ASSESMENT ELECTROCARDIOGAPHIC AND ECHOCARDIOGRAPHIC PARAMETERS IN DOGS WITH DILATED CARDIOMYOPATHY

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[Received: 16.10.2022; Accepted: 20.11.2022]
{DOI 10.29005/IJCP.2022.14.2.69-75}

In the present study, a total of 262 dogs presented with clinical signs suggestive of cardiac insufficiency were screened for the DCM. Among them, DCM was diagnosed in 26 dogs (10.08 %). Echocardiographic examination revealed increased LA/AO ratio, increased left ventricular end diastolic dimensions and systolic dimensions, increased end diastolic volume and end systolic volume, increased E-point septal separation and decreased fractional shortening and ejection fraction. Mitral valve regurgitation was observed with pulsed wave Doppler, continuous wave Doppler and Colour flowDoppler echocardiography. Tissue doppler echcocardiography showed decreased systolic velocity, early diastolic velocity, late diastolic velocity and increase of E_m:A_m with normal isovolumic relaxation time and isovolumic contraction time.
Keywords: Dilated Cardiomyopathy, Dogs, Echocardiography, Electrocardiography.

Cardiology is a rapidly growing speciality in small animal medicine. Heart disease is second to cancer as a commonest disease causing premature death in dogs. Next to chronic mitral valve insufficiency (CMVI) and in some geographic regions where heartworm disease is common, dilated DCM is the second most commonly acquired cardiac disorder in the dogs (O'Grady and O'Sullivan, 2004). The DCM in dogs is characterised by chamber dilation and myocardial systolic and diastolic dysfunction of one (usually the left) or both ventricles and etiology remains to be mostly idiopathic. The rate of progression of DCM is not known. The symptomatic phase is preceded by an asymptomatic phase where several compensatory mechanisms act in concert to maintain cardiac output and prevent congestive heart failure (CHF) (Sisson *et al.*, 2000). Conventional echocardiography is a well-established technique for quantitative assessment of anatomy and function of herat allowing measurement of atrial and ventricular dimension, myocardial thickness, and segmental systolic function. Expansion of an echocardiographic examination to include spectral and colour flow dopler imaging enables clinicians to evaluate direction and velocity of blood flow direction and velocity, permitting improved assessment and understanding of cardiovascular function and disease. Tissue Doppler imaging (TDI**)** has been introduced as a new method for analyzing overall as well as segmental myocardial function in small animals (Chetboul *et al*., 2005). Cardiac ultrasound is convenient, requires no anaesthesia, and is widely available (Bonagura, 2022). Keeping in view of the above facts, the present study was designed to evaluate the echocardiographic parameters in dogs with DCM.

Materials and Methods

Among the different cases presented to the small animal medicine ward at Department of Teaching Veterinary Clinical Complex, Proddatur, a total of twenty six apparently healthy dogs of large to giant sized breeds aged above five years brought for routine health check up, vaccination and deworming were randomly selected for the study and kept under the control group. In the present study, clinical cases included the dogs presented with clinical signs suggestive of cardiacclinical signs and they were screened for the DCM using the specially designed cardiology data sheet. They were subjected to detailed clinical examination, physical examination, thoracic radiography, electrocardiography and echocardiography. Standard transthoracic echocardigraphy was

performed using Prosound α6LT Aloka US system with a 5.5–7.5 MHz phased array transducer. Two dimensional (2D), M – mode, Colour Doppler, Spectral Doppler and Tissue Doppler imaging of heart was carried out as per standard protocols. The echocardiographic parameters were interpreted as per details of stastical tool.

Results and Discussion

The 262 clinical cases suspected to be with cardiac disease were subjected to detailed examination, DCM was reported in 26 dogs (10.08 %). In the present study, the prominent clinical signs noticed on the day of presentation were: exercise intolerance, inappetence and lethargy (26/26; 100 %), Cough and weight loss (18/26: 69.23 %), abdominal distension, laboured breathing and pedal edema (14/26, 53.85 %) and syncope (6/26, 23.08 %). In the present study, lethargy in DCM dogs might be due to decreased cardiac output which was inadequate for the normal activity. Impaired skeletal muscle perfusion during exercise, related to vascular and metabolic changes that occur over time, can reduce exercise tolerance as also reported by Ware, 2009; that increased pulmonary vascular pressures and edema also lead to poor exercise tolerance. Cough might be due to congestive heart failure and cardiogenic pulmonary edema. The inadequate forward cardiac output was responsible for transient unconsciousness associated with loss of postural tone (collapse) from insufficient oxygen or glucose delivery to the brain resulting syncope as also mentioned by Ware, 2009.

In the present study, physical examination of dogs with DCM revealed the following findings: tachycardia (69.23 %, 18/26), dyspnoea (61.53 per cent, 16/26), ascites (53.85 per cent, 14/26), pale mucous membranes (34.61 per cent, 9/26), systolic murmur (30.76 percent, 8/26), crackles (23.07 per cent, 6/26) and gallop rhythm (23.07 per cent, 06/26), it was in agreement to the report of Miller, 2010 and Jeyaraja *et al.*,2015 who narrated that in DCM dogs, dyspnoea and crackles may be due to pulmonary edema subsequent to left sided heart failure . Ascites noticed in DCM dogs was associated with right sided congestive heart failure, as reported earlier by Prosek, 2015. Pale mucous membranes in DCM dogs might be due to anaemia or peripheral vasoconstriction as also mentioned by Ware, 2009. Systolic murmur and low pitched gallop sound was an evidence of severe ventricular diastolic impairment.

In the present study, the prominent thoracic radiographic findings observed in DCM dogs (**Fig.1** and **Fig.2**) were cardiomegaly (84%), pulmonary edema (69.25%) left atrial enlargement (57.69%), pleural effusion (38.46%) and pericardial effusion (11.53%). In DCM dogs, generalised cardiomegaly is caused by volume overload or ventricular dilation, left atrial dilation may be present because of volume overload or mitral valve dysfunction from a change in shape of the mitral annulus as a result of cardiac dilation and possible pleural effusion, hepatomegaly, and/or ascites from right-sided heart failure is often seen. The radiographic appearance of heart size often fails to reflect the severity of the underlying myocardial impairment in DCM dogs as also reported earlier by Sisson *et al.,* 2000. In the present study, the important electrocardiographic findings recorded in dogs with DCM (**Fig.3**) were: presence of low voltage QRS complexes, tall R wave, atrial premature complexes, sinus tachycardia, ventricular premature complexes, normal sinus rhythm, prolonged QRS complexes, ventricular tachycardia, atrial fibrillation. Atrial fibrillation was the most common arrhythmia in dogs with DCM and this might be due to atrial stretching as also reported by Dukes-McEwan *et al*., 2003.

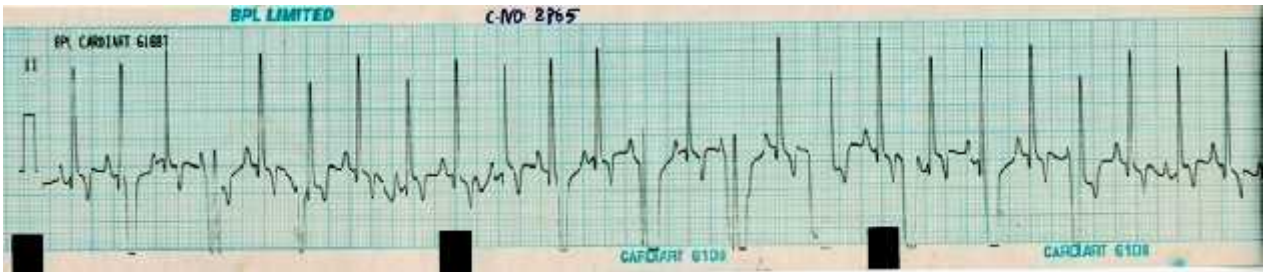
Fig.1. LATERAL THORACIC RADIOGRAPH OF DOG WITH DCM SHOWING CARDIOMEGALY AND LEFT ATRIAL ENLARGEMENT

Fig.2. LATERAL THORACIC RADIOGRAPH OF DOG WITH DCM SHOWING CARDIOMEGALY, PULMONARY EDEMA AND PLEURAL EFFUSION

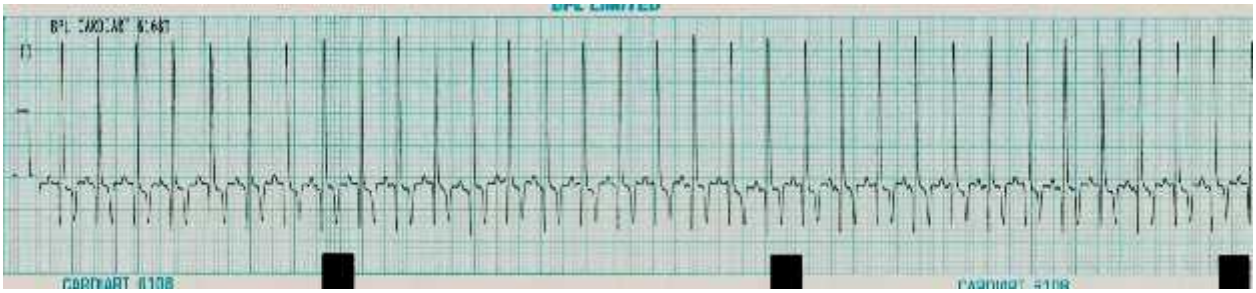
3a. LOW VOLTAGE QRS COMPLEXES IN DOG (PAPER SPEED - 25 MM/SEC)

3b.TALL R WAVE (PAPER SPEED - 25 MM/SEC

3c.VENTRICULAR PREMATURE COMPLEXES PAPER SPEED - 25 MM/SEC



3d.Sinus tachycardia in DCM dog (paper speed - 25 mm/sec



Echocardiography

It was used to assess cardiac chamber dimensions and myocardial function and differentiate pericardial effusion or chronic valvular insufficiency from DCM. Dilated cardiac chambers and poor systolic ventricular wall and septal motion are characteristic echocardiographic findings in dogs with DCM as also recorded by Ware, 2009.

The mean values of echocardiographic parameters in control and DCM dogs were given in **Table- I.** The dilated chambers, hypokinesis of the mitral valve and left ventricle are easily appreciated using 2-D echocardiography. In the present study, the mean ± S.E values of LA (mm) and LA/Ao were significantly increased (**Fig.4**) and Ao (mm) was significantly decreased in dogs with DCM when compared to the control group which was in accordance with the findings of Singh *et al.* (2014) and Jeyaraja *et al.* (2015). Decreased lumen dimensions of the aorta was an indication of a low stroke volume. In the present study, highly significant increase in left ventricular internal dimension end- diastole **(**LVIDd) and left ventricular internal dimension end- systole (LVIDs) was observed in DCM dogs when compared to the control group which was in agreement with the earlier reports of Singh *et al*.,2014 and Jeyaraja *et al.* 2015. The left ventricular end-diastolic dimensions were increased as a result of volume loading. **I**n the present study, end diastolic volume (EDV) and end systolic volume (ESV) were significantly increased in DCM dogs when compared to the control group which might be attributed to impaired myocardial systolic and diastolic function in dogs with DCM as also recorded by Sisson *et al.*, 2000. In the present study, left ventricular posterior wall end-diastole (LVPWd), left ventricular posterior wall thickness end-systole (LVPWs), inter ventricular septum thickness end-diastole (IVSd) and inter ventricular septum thickness end-systole (IVSs) were significantly reduced in DCM dogs when compared to the control group which was in agreement with the earlier observations of Singh *et al*., 2014 and Jeyaraja *et al.*,2015).

Table- I. MEAN ± SE VALUES OF ECHOCARDIOGRAPHIC PARAMETERS IN CONTROL AND DOGS WITH DCM IN

S.No.	Parameter	Control (n=26)	dogs with DCM (n=26)
1	LA(mm)	21.80±0.81[a]	39.60±0.94[b]
2	Ao(mm)	23.00±0.75[a]	18.27±0.66[b]
3	LA/Ao	0.95±0.04[a]	2.23 ±0.09[b]
4	LVIDd(mm)	34.10±2.20[a]	54.65±0.68[b]
5	LVIDs(mm)	23.30±1.03[a]	47.34±0.63[b]
6	LVPWd(mm)	9.70±0.98[a]	7.19±0.30[b]
7	LVPWs(mm)	11.50±0.91[a]	9.46±0.0
8	IVSd (mm)	10.80±0.71[a]	8.07±0.35[b]
9	IVSs(mm)	12.40±0.70[a]	8.19±0.41[b]
10	EDV (ml)	53.20±5.21[a]	149.90±3.50[b]
11	ESV (ml)	19.20±0.20[a]	104.50±0.39[b]
12	Fractional shortening (%)	34.40±0.56[a]	13.35±0.68[b]
13	Ejection fraction (%)	64.83±0.80[a]	28.10±1.30[b]

14	EPSS (mm)	2.71±0.34[a]	15.01±1.20[b]
Pulsed wave Tissue Doppler Echocardiographic findings			
1	Systolic velocity (Sm in cm/s)	12.60±0 .88[a]	8.17 ±0 .85[b]
2	Early Diastolic velocity (Em in cm/s)	12.76±1.47[a]	9.92±0.96[b]
3	Late Diastolic velocity (Am in cm/s)	7.59±1.08[a]	4.74±0.23[b]
4	Em/ Am	1.59±0.17[a]	2.17±0.12[b]
5	Isovolumic relaxation time (IVRT in ms)	72.00±10.73[a]	72.62±3.65[a]
6	Isovolumic contraction time (IVCT in ms)	57.60±13.49[a]	50.75±4.24[a]

Mean ± S.E followed by different superscript(s) differ significantly (P<0.01)

Fig.4 Dilated Left atrium; Increased LA/AO (2.01)

Fig.5 Elevated EPSS (23 mm)

In the present study, highly significant decrease in FS (**Fig.6** and **Fig.7**) and ejection fraction (EF) values were observed in dogs with DCM. The present findings were in agreement with earlier studies of Thomason *et al*., 2014 and Jeyaraja *et al.*, 2015. Systolic dysfunction causes the ventricle to be hypodynamic, which results in a decreased fractional shortening. The mean ± SE values of EPSS were significantly increased (**Fig.5**) in DCM dogs when compared to the control group. In the normal heart, the mitral valve opens in diastole, and its anterior leaflet almost contacts the interventricular septum. In dilated hearts with decreased contractility in DCM, the mitral valve does not reach the septum as also reported by Belanger, 2005, causing increase in EPSS in the present study.

Spectral Doppler and Color flow Doppler imaging techniques are used to evaluate blood flow velocity and direction in the heart and great vessels as also mentioned by Belanger, 2005. In the present study, pulsed wave Doppler (**Fig.8**), continuous wave Doppler (**Fig.9**) and color Doppler (**Fig.10**) findings revealed DCM with mitral valve regurgitation in ten dogs (57.69%). The regurgitation may be attributed to abnormal dilation of the chambers leading to altered geometry and consequent leak in the AV valves as also recorded by Jeyaraja *et al.*, 2015. Tissue Doppler Imaging (TDI) is more sensitive than conventional echocardiography for the diagnosis of myocardial dysfunction and for differentiating physiological from pathological hypertrophy as also stated by Chetboul, 2002. In the present study, the mean values of systolic velocity (S_m), early diastolic velocity (E_m) and late diastolic velocity (A_m) were significantly decreased in DCM dogs (**Fig.11**) when compared to that of control group. The present findings were in agreement with the earlier observations of Pereira *et al*., 2016. While, the mean values of E_m: A_m were significantly increased in DCM dogs when compared to the control group which was in accordance with the

findings of Pereira *et al.* 2016. There were no significant changes in IVRT and IVCT in DCM dogs when compared to the control group.

Fig.6. Right parasternal long axis view showing FS:11.0 percent

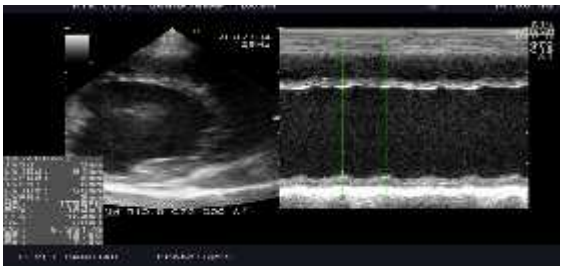


Fig.7 Right parasternal short axis view showing FS: 7.6 percent

Fig.8 Pulsed wave Doppler showing mitral valve regurgitation

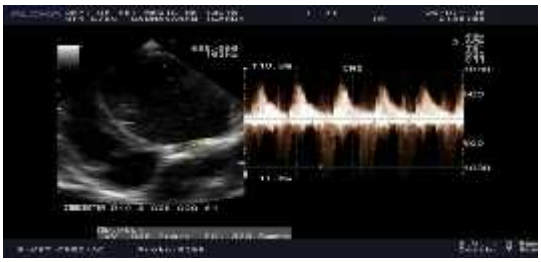


Fig.9 Continuous wave Doppler showing mitral valve regurgitation

Fig.10 Colour flow Doppler showing mitral valve regurgitation

Fig.11 Tissue Doppler image in DCM dog showing diastolic dysfunction

Conclusions

The findings of the present study, it was concluded that detailed collection of patient history, results of physical examination, electrocardiography and thoracic radiography provides a practical means of diagnosing DCM in dogs. The echocardiography is useful for confirmatory diagnosis and quantifying the left ventricular dysfunction in dogs with DCM.

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