



Echocardiographic Alterations of Dilated Cardiomyopathy in Dogs

Mukesh K. Srivastava ^{a++}, Anil Ahuja ^{b#}, R.D. Velhankar ^{ct},
Ashish Srivastav ^{a++}, Barkha Sharma ^{d++}
and Arpana Raikwar ^{e‡*}

^a Department of Veterinary Medicine, DUVASU, Mathura (U.P.), India.

^b Department of Veterinary Medicine, CAVS, RAJUVAS, Bikaner, Rajasthan, India.

^c Department of Veterinary Medicine, MAFSU, Maharashtra, India.

^d Department of Veterinary Epidemiology, DUVASU, Mathura (U.P.), India.

^e Department of Veterinary, Medicine College of Veterinary Science and A.H., Jabalpur (M.P.), India.

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

An extensive investigation involving 2497 dogs aimed to determine the prevalence of Dilated Cardiomyopathy (DCM). Through meticulous examination incorporating historical, clinical, electrocardiographic, and radiographic analyses, 29 cases were definitively diagnosed with DCM using echocardiography. Employing advanced echocardiographic technology, including 2-D imaging with M-mode and Doppler modalities, precise cardiac assessments were conducted. Attention to

⁺⁺ Associate Professor;

[#] Former Professor & Head;

[†] Former Professor;

[‡] Assistant Professor;

*Corresponding author: Email: arpanajbp@gmail.com;

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standard imaging planes and thoracic depilation ensured optimal transducer adherence. Comparative analysis between healthy and DCM-affected dogs revealed significant differences in left atrial dimensions, indicative of pathological cardiac remodeling. Furthermore, notable variations in ventricular dimensions and functional indices were observed, reflecting impaired myocardial contractility and ventricular compliance, particularly in cases with right-sided involvement. Doppler assessments unveiled aberrant blood flow dynamics in DCM, indicating hemodynamic disturbances. These findings underscore the diagnostic utility and pathophysiological insights provided by echocardiography in characterizing DCM pathology in dogs. In conclusion, echocardiography emerges as a valuable tool for the comprehensive evaluation and diagnosis of DCM in canine patients, facilitating early detection and effective management of this debilitating cardiac condition.

Keywords: Dilated cardiomyopathy; echocardiography; doppler; cardiac imaging.

1. INTRODUCTION

DCM is characterized by clinical, electrocardiographic, circulating biomarker, and imaging abnormalities. The diagnosis of clinical DCM with CHF is straightforward; however, identification of the preclinical stage can be challenging [1]. Echocardiography serves as a valuable tool for research into the pathophysiology, treatment modalities, and outcomes of DCM in dogs. Echocardiography is essential in veterinary medicine for diagnosing cardiac conditions in dogs. It provides real-time visualization of the heart's structure and function, aiding in the diagnosis of various diseases such as cardiomyopathies and valve defects.

2. MATERIALS AND METHODS

A comprehensive research initiative was conducted to screen 2497 dogs of various breeds, genders, and age groups to detect cases of Dilated Cardiomyopathy (DCM). This collaborative study was carried out 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.

The screening process involved meticulous historical examination, clinical assessments, electrocardiography, and radiography. Ultimately, 29 cases were definitively diagnosed with DCM using echocardiography, comprising the DCM group.

Advanced echocardiographic assessments were conducted using the MyLab40VET system equipped with a phased array Cardiac Probe

PA320 (frequency range: 3.0 to 5.5 MHz). A structured echocardiographic protocol was employed, starting with 2-D imaging and integrating M-mode, color-flow mapping, and spectral Doppler techniques. To optimize transducer contact, thoracic hair surrounding the cardiac region was carefully removed prior to examination.

During imaging sessions, dogs were positioned in right lateral recumbency on a specialized wooden table, and imaging commenced from the right parasternal position. Standard imaging planes were meticulously followed, adhering to established methodologies as described by Fox et al. [2] and Nyland and Mattoon [3]. The evaluation included precise M-mode measurements from specific anatomical landmarks to assess various cardiac dimensions and functional indices accurately.

This research underscores the importance of echocardiography in the comprehensive evaluation and diagnosis of DCM in canine populations, facilitating early detection and effective management strategies for this debilitating cardiac condition.

3. RESULTS AND DISCUSSION

Echocardiographic measurements in healthy and DCM-affected dogs revealed significant differences in LA/AO ratios (Table 1, Fig. 1). Wayne and Marc [4] proposed concurrent left atrial dilatation in left DCM-affected dogs.

The study demonstrated E point septal separation (EPSS) measurements (mm) in various canine populations: healthy (3.21 ± 0.20), overall Dilated Cardiomyopathy (DCM) (7.63 ± 0.84), left-sided DCM (8.87 ± 0.92), and bilateral DCM (10.65 ± 0.87) (Fig. 2). Significant

differences were noted between healthy and DCM-affected dogs, while comparisons within DCM groups revealed no significant variance between left-sided and bilateral DCM. EPSS findings in healthy dogs were consistent with previous studies, aligning with Nyland and Mattoon (2002), Deni et al. [5].

The study measured the mean \pm standard error (SE) of right ventricular diameter in diastole (RVDd) (mm) in different groups (Table 1). A significant difference was observed between healthy and DCM-affected dogs in relation to this parameter. Within the DCM group, analysis revealed a significantly lower value in left DCM dogs compared to those with right and bilateral DCM. The present study's observations on RVDd in DCM-affected dogs align with Lee et al. [6], indicating significantly increased chamber

diameters in Cocker Spaniels with DCM. Wide variation was observed in RVDd among both healthy and DCM-affected dogs in the current investigation.

Interventricular thickness in diastole (IVsd) measured 10.38 ± 0.38 mm in healthy dogs and 9.24 ± 0.46 mm in overall Dilated Cardiomyopathy (DCM), with similar values across left and bilateral DCM groups. These findings align with previous research by Domanjko et al. [7] and Rush et al. [8], but differ from reports by Kumar et al. [9] and Polana [10]. Septal thickness in systole (IVss) was 13.99 ± 0.43 mm in healthy dogs and 11.34 ± 0.52 mm in overall DCM, with similar trends observed across DCM subgroups. Thinning of interventricular septum and left ventricular free wall, characteristic of DCM, has been suggested by previous studies [4].

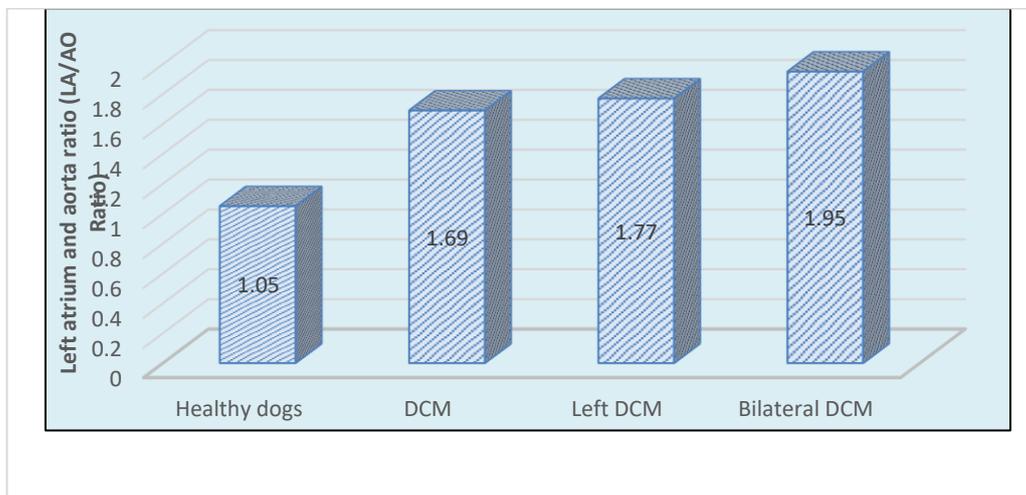


Fig. 1. M mode left atrium to aortic root diameter ratio in healthy and DCM affected dogs

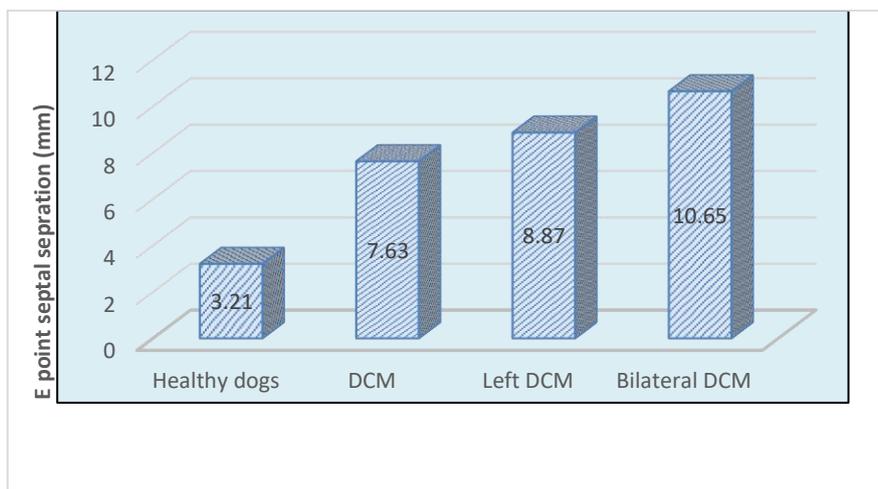


Fig. 2. E point septal separation in healthy and DCM dogs

Table 1. M mode echocardiographic parameters of healthy and DCM affected dogs

Parameters	Healthy dogs	DCM	Left DCM	Bilateral DCM
Body weight	29.62 ± 1.48 ^a	28.68 ± 1.28 ^b	29.36 ± 3.74 ^b	36.40 ± 7.24 ^b
LA/AO ratio	1.05 ± 0.02 ^b	1.69 ± 0.08 ^a	1.77 ± 0.09 ^a	1.95 ± 0.15 ^a
EPSS (mm)	3.21 ± 0.20 ^c	7.63 ± 0.84 ^b	8.87 ± 0.92 ^{ab}	10.65 ± 0.87 ^a
RVDd (mm)	9.03 ± 0.58 ^b	19.46 ± 1.92 ^a	11.93 ± 1.88 ^b	25.06 ± 2.01 ^c
IVsd (mm)	10.38 ± 0.38 ^{NS}	9.24 ± 0.46 ^{NS}	8.64 ± 0.68 ^{NS}	9.83 ± 0.93 ^{NS}
LVDd (mm)	34.81 ± 1.22 ^b	46.31 ± 3.26 ^a	57.63 ± 2.03 ^a	48.51 ± 4.30 ^a
PWd (mm)	10.37 ± 0.42 ^a	8.20 ± 0.55 ^{ab}	7.10 ± 0.76 ^b	9.40 ± 0.76 ^{ab}
IVSs(mm)	13.99 ± 0.43 ^a	11.34 ± 0.52 ^{ab}	10.85 ± 0.89 ^b	12.15 ± 0.70 ^{ab}
LVDs(mm)	23.62 ± 0.98 ^b	35.09 ± 2.87 ^a	42.08 ± 2.42 ^a	42.68 ± 2.77 ^a
PWs(mm)	13.80 ± 0.50 ^a	10.60 ± 0.68 ^{ab}	9.74 ± 0.85 ^b	11.91 ± 1.19 ^{ab}
EF (%)	63.75 ± 1.14 ^a	48.72 ± 3.95 ^b	36.36 ± 2.17 ^b	45.38 ± 6.83 ^b
FS (%)	34.08 ± 0.86 ^b	24.04 ± 2.41 ^c	18.64 ± 1.06 ^c	19.88 ± 2.20 ^c

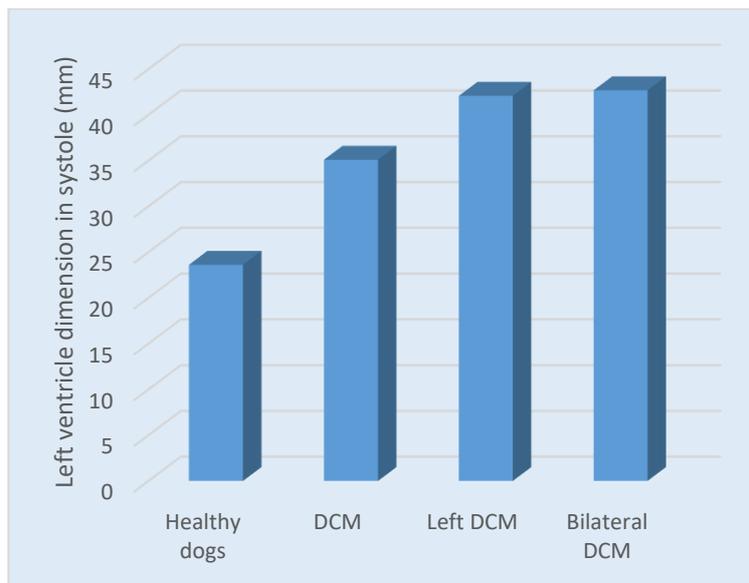


Fig. 3. Left ventricular diameter in systole in healthy and DCM dogs

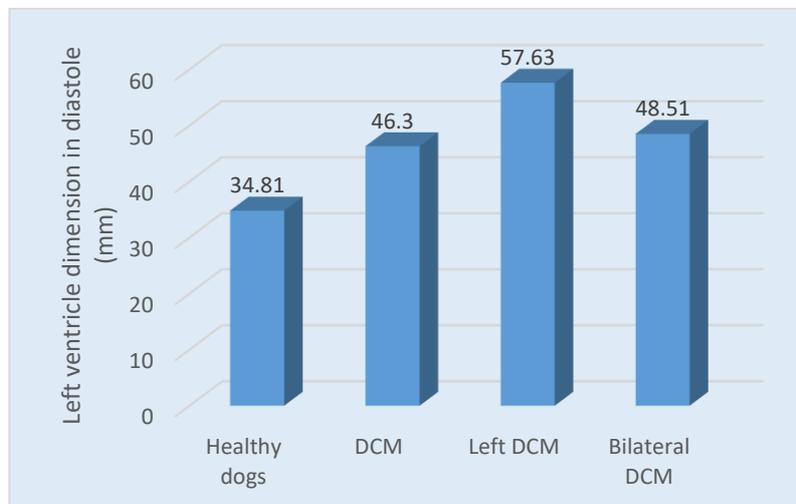


Fig. 4. Left ventricular diameter in diastole in healthy and DCM dogs

Left ventricular diameter in diastole and systole (LVdD and LVdS) in Healthy and different groups of DCM (Table 1, Figs. 3 & 4). Significant differences were observed between healthy and DCM-affected dogs in terms of LVdD, with right DCM showing a significant decrease compared to left, bilateral, and healthy dogs. These findings are consistent with previous studies.

Posterior wall thicknesses in diastole and systole (PWd and PWs in mm) were 10.37 ± 0.42 and 8.20 ± 0.55 in healthy dogs and 13.80 ± 0.50 and 10.60 ± 0.68 in DCM-affected dogs, respectively. In left DCM, PWd and PWs measured 7.10 ± 0.76 and 9.74 ± 0.85 mm, in right DCM they were 9.03 ± 1.23 and 10.83 ± 1.70 mm, and in bilaterally DCM they were 9.40 ± 0.76 and 11.91 ± 1.19 mm. Changes in thickness were not statistically significant within the DCM groups. These findings align with previous research by

Rush et al. [8] and Rosangela et al. [11], which reported similar patterns of posterior wall thickness in DCM-affected dogs. Thinning of the posterior wall in DCM may be attributed to progressive left ventricular enlargement and remodeling of the heart.

In healthy dogs, EF and FS were $63.75 \pm 1.14\%$ and $34.08 \pm 0.86\%$ respectively, contrasting with DCM-affected dogs where EF and FS were $48.72 \pm 3.95\%$ and $24.04 \pm 2.41\%$ respectively (Figs. 5 & 6). Specifically, left DCM dogs showed EF and FS of $36.36 \pm 2.17\%$ and $18.64 \pm 1.06\%$, while right DCM dogs displayed EF and FS of $77.29 \pm 3.76\%$ and $44.57 \pm 3.70\%$. Bilaterally DCM-affected dogs had EF and FS of $45.38 \pm 6.83\%$ and $19.88 \pm 2.20\%$ respectively. These findings underscore significant disparities, with notably higher values detected in right DCM cases, indicating a distinct cardiac impairment.

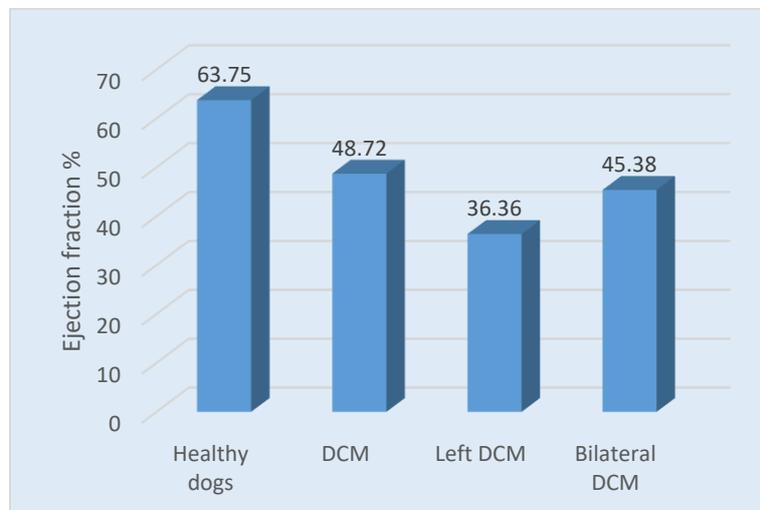


Fig. 5. Ejection fraction in systole in healthy and DCM dogs

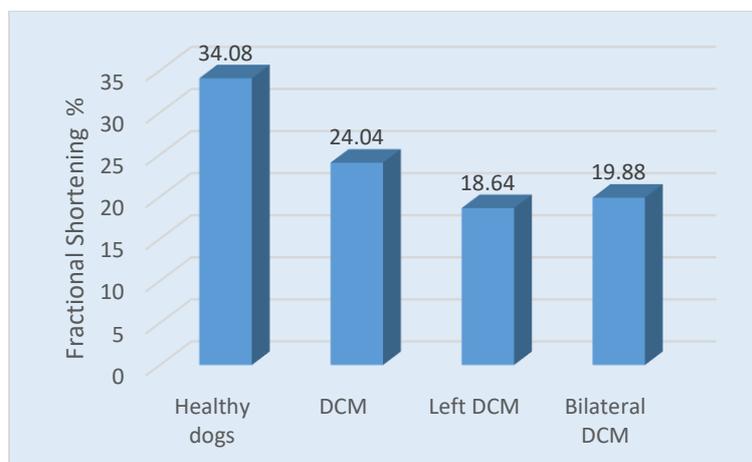


Fig. 6. Fractional shortening in systole in healthy and DCM dogs

Table 2. Doppler echocardiographic parameters of healthy and DCM affected dogs

Parameters	Healthy dogs	DCM	Left	Bilateral
Mean peak mitral velocity (m/sec)	0.84 ± 0.03 ^c	1.33 ± 0.08 ^{ab}	1.36 ± 0.12 ^a	1.54 ± 0.09 ^a
Tricuspid velocity (m/sec)	0.76 ± 0.02 ^b	1.01 ± 0.05 ^a	1.00 ± 0.07 ^a	1.00 ± 0.13 ^a
MVrg (m/sec)	NAD	3.40 ± 0.26 ^a	3.72 ± 0.36 ^a	3.77 ± 0.27 ^a
TVrg (m/sec)	NAD	2.32 ± 0.24 ^a	3.38 ± 0.53 ^b	2.68 ± 0.38 ^b
Mitral E/A ratio	1.34 ± 0.04 ^b	1.67 ± 0.10 ^{ab}	1.59 ± 0.16 ^{ab}	1.83 ± 0.14 ^a
Tricuspid E/A ratio	1.25 ± 0.03 ^{NS}	1.37 ± 0.06 ^{NS}	1.38 ± 0.09 ^{NS}	1.31 ± 0.11 ^{NS}
Main pulmonary artery Velocity(m/sec)	0.93 ± 0.03 ^{ab}	0.86 ± 0.04 ^{bc}	0.84 ± 0.05 ^{bc}	0.70 ± 0.04 ^c
Aortic velocity (m/sec)	1.28 ± 0.06 ^{NS}	1.15 ± 0.07 ^{NS}	1.29 ± 0.12 ^{NS}	1.02 ± 0.08 ^{NS}

Ejection fraction (EF) in healthy dogs fell within the expected range, consistent with findings by O’Grady and O’Sullivan [12], who also reported EF values of 50-65% in healthy dogs. Fractional shortening (FS %), along with EF, serves as a key echocardiographic indicator of ventricular performance, reflecting myocardial contractility and ventricular compliance [13].

Diastolic cardiac function investigations are crucial for understanding heart failure progression. Doppler echocardiography, particularly the E/A ratio, is widely used to diagnose diastolic dysfunction. However, interpretations may vary due to factors like heart rate, age, and loading conditions.

In our investigation, the mean peak mitral velocities varied across healthy and DCM-affected dogs, with significant differences observed between the two groups (Tab. No. 2). Left and bilateral DCM dogs showed higher velocities compared to right DCM dogs. Mitral velocities in healthy dogs were consistent with previous studies, although slight variations may be due to breed differences and recording methods. Mitral E/A ratio did not significantly differ between healthy and DCM-affected dogs, but bilateral DCM showed a higher ratio compared to left and right DCM. Normal mitral E/A ratios were consistent with established ranges from prior studies. These findings underscore the importance of assessing mitral velocities and E/A ratios in diagnosing and monitoring DCM in dogs.

Mitral regurgitation (MR) velocity showed consistent values across overall DCM, left, right, and bilateral DCM groups, without significant differences observed (Tab.No.2). This aligns with previous studies by Yuill and O’Grady [14], who did not find MR in normal dogs. Mean peak tricuspid velocities were significantly higher in all

DCM groups compared to healthy dogs, but there was no significant difference among the left, right, and bilateral DCM groups (Tab. No. 2). Tricuspid E/A ratio showed non-significant differences between healthy and DCM dogs and among the various DCM groups. Tricuspid regurgitation velocities were notably higher in overall DCM, left, and right DCM compared to bilateral DCM. The mean tricuspid velocities in healthy dogs were within the range reported by previous studies, although slightly higher. This discrepancy could be attributed to differences in Doppler techniques, sample sizes, and observer variation. Tricuspid E/A ratio reported by Ruthnéa et al. [15] in healthy German shepherd dogs was higher than the present findings [16].

Tricuspid regurgitation velocity in dogs with dilated cardiomyopathy (DCM) was comparable to previous studies. Peak pulmonary valve velocities were higher in right-sided DCM, while aortic valve velocities did not significantly differ among DCM groups or healthy dogs. Aortic velocity in DCM dogs was lower than reported previously, possibly indicating reduced cardiac output. These variations may be influenced by factors such as Doppler flow techniques, sample sizes, and breed differences [17].

The eccentricity index (EI) was significantly higher in all DCM groups (Tab. No. 3) compared to healthy dogs, indicating right ventricular pressure overload. The sphericity index (SI) was significantly lower in DCM groups, particularly in right-sided DCM, indicating left ventricular dilatation. The pulmonary artery to aorta (PA/Ao) ratio did not differ significantly between groups, suggesting no significant pulmonary hypertension. These findings align with previous studies and highlight the utility of echocardiographic parameters in assessing cardiac function and remodeling in DCM.

Table 3. D mode parameters of healthy and DCM affected dogs

Parameters	Healthy dogs	DCM	Left DCM	Right DCM	Bilateral DCM
Sphericity index	1.85 ± 0.02 ^a	1.47 ± 0.07 ^b	1.30 ± 0.04 ^b	1.96 ± 0.18 ^a	1.34 ± 0.05 ^b
Eccentricity index (end diastole)	1.01 ± 0.02 ^d	1.28 ± 0.06 ^{bc}	1.05 ± 0.03 ^{cd}	1.64 ± 0.09 ^a	1.36 ± 0.09 ^b
Eccentricity index (end systole)	1.03 ± 0.02 ^a	1.03 ± 0.04 ^a	0.99 ± 0.02 ^a	1.06 ± 0.05 ^a	1.08 ± 0.03 ^a
Pulmonary artery to aorta ratio	0.96 ± 0.02 ^{NS}	1.03 ± 0.03 ^{NS}	0.97 ± 0.04 ^{NS}	1.07 ± 0.06 ^{NS}	1.09 ± 0.04 ^{NS}

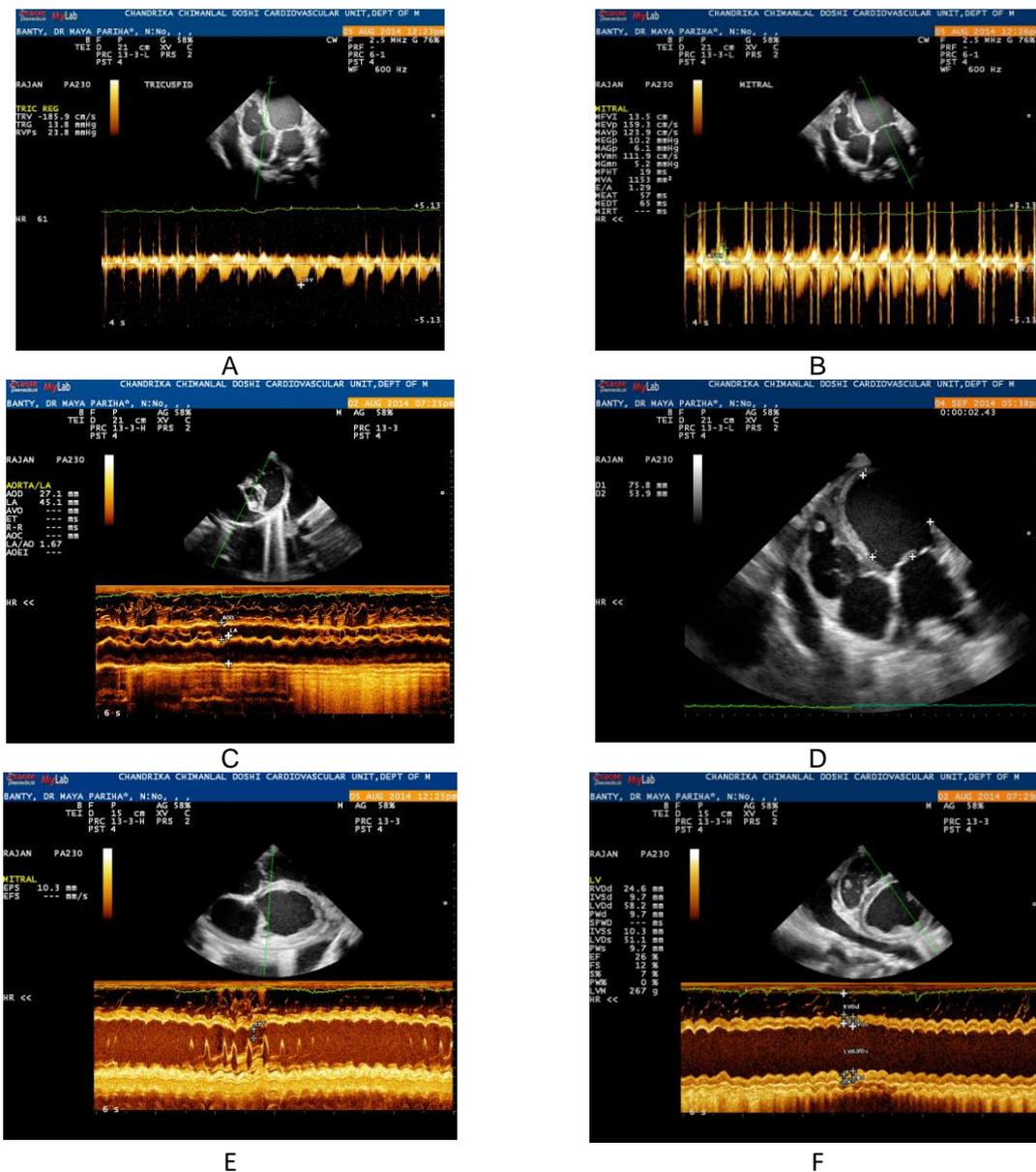


Fig. 7. Echocardiographic analysis of bilaterally DCM affected dog (case-1)

A. Tricuspid, B. Mitral regurgitation, C. Increased LA/Ao ratio
 D. Sphericity index E. E point septal separation F. Left ventricle dimension, EF and FS

4. CONCLUSION

Echocardiographic measurements revealed substantial alterations in left atrial dimensions (LA/AO ratio) and E point septal separation (EPSS), highlighting progressive cardiac remodeling and impaired myocardial function in DCM. Specifically, dogs with DCM exhibited significantly enlarged left ventricular dimensions (LVDD and LVDs), indicative of chamber dilation and reduced systolic function. Posterior wall thickness (PWD and PWs) showed variable

changes, reflecting myocardial remodeling and potential thinning in advanced stages of DCM.

Functional indices such as ejection fraction (EF) and fractional shortening (FS) were significantly lower in DCM-affected dogs, underscoring compromised myocardial contractility and ventricular performance. Doppler assessments revealed altered mitral velocities and E/A ratios, indicating diastolic dysfunction and hemodynamic disturbances associated with DCM progression.

Furthermore, intergroup comparisons within the DCM cohort demonstrated distinctive echocardiographic profiles between left-sided, right-sided, and bilateral DCM. While some parameters like EPSS and EF differed significantly across these groups, others such as interventricular septum thickness (IVS) and tricuspid regurgitation velocities (TVrg) showed comparable values, suggesting common pathophysiological mechanisms despite varying clinical presentations.

In conclusion, echocardiography emerges as a pivotal tool for the comprehensive evaluation and characterization of DCM in canine patients. The precise assessment of cardiac structure, function, and hemodynamics provided by echocardiography enables early detection, accurate diagnosis, and informed management strategies.

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. Bonagura D, Visser LC, Echocardiographic assessment of dilated cardiomyopathy in dogs, *Journal of Veterinary Cardiology*. 2022;40:15-50.
2. Fox PR, Sisson D, Moise NS. In text book of canine and feline myocardial diseases of dogs. WB saunders company, Philadelphia Chapter. 1988;18:230,356-378. Chapter 27. pp: 582, 585–587, 589–593, 597–599,600-601,746,892-93.
3. Nyland JS, Mattoon TG. Small animal diagnostic ultrasound, 2nd Edition, Saunders, Philadelphia, PA. 1995;354-379, 380, 393-397, 406-408.
4. Wayne W, Marc R. Cardiomyopathy. The veterinary ICU book. Teton NewMedia, LACKSON, USA. 2002;559.
5. Deni Noviana, Devi, Paramitha, Retno, Wulansari. Motion mode and two dimensional echocardiographic measurements of cardiac dimensions of Indonesian mongrel dogs. *Hayati J. Biosciences*. 2011;18(1):1-5.
6. Lee BH, Dukes-McEwan J, French A, French AT, Corcoran BM. Evaluation of a novel doppler index of combined systolic and diastolic myocardial performance in Newfoundland dogs with familial prevalence of dilated cardiomyopathy. *Vet. Radiol. Ultrasound*. 2002;43:154-165.
7. Domanjko-Petric A, Stabejand P, Zemva A. Dilated cardiomyopathy in the dobermann dog: Survival, causes of death and a pedigree review in a related line. *J. Vet. Cardio*. 2002;4:17-24.
8. Rush JE, Lisa MF, Donald JB, Barbara PB, James N, Ross Jr, Peter JM. Clinical, echocardiographic, and neurohormonal effects of a sodium-restricted diet in dogs with heart failure. *J. Vet. Intern. Med*. 2000;14:513–520.
9. Kumar K, Satish DS, Tirumala Rao, Nagaraj P. Dilated cardiomyopathy in dogs: A study for 3 years (2006 To 2009). *Vet Scan*. 2011;6:2(93):1-10.
10. 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.
11. Rosângela OA, Andréa CT, Matheus MM, Severiana CMCC, Luiz HS, Hugo CMP, Vivian CM, Aline BC. Dilated cardiomyopathy In boxer puppy of 8 months of age: A case report. *Biosci. J. Uberlândia*. 2012;28(5):842-845.
12. O'Grady MR, O'Sullivan ML. Clinical cardiology concept for the dog and cat. (An educational website for veterinary students and practitioners); 2011. Available:www.vetgo.com/cardio/concepts/concindx.php
13. Voros K, Hetyey C, Reiczegel J, Czirok GN. M-mode and two dimensional echocardiographic reference values for three Hungarian dog breeds. *Acta Veterinaria Hungarica*. 2009;57:217-227.
14. Yuill CDM, O'Grady MR. Doppler-derived velocity of blood flow across the cardiac valves in the normal dog. *Can. J. Vet. Res*. 1991;55:185-192.
15. Ruthnéa ALM, Leonardo ALM, Roberto BA, Marcos C. Echocardiographic indices

- in normal German shepherd dogs. J. Vet. Sci. 2006;7(2):193-198
16. Wess G. Screening for dilated cardiomyopathy in dogs. Journal of Veterinary Cardiology. 2022 Apr 1;40:51-68.
17. Borgarelli M, Santilli RA, Chiavegato D, D'Agnolo G, Zanatta R, Mannelli A, Tarducci A. Prognostic indicators for dogs with dilated cardiomyopathy. Journal of veterinary internal medicine. 2006 Jan;20(1):104-10.

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