Indian J Anim Health (2025), 64(2) : 231-242
DOI: https://doi.org/10.36062/ijah.2025.09525

Review Article

A comprehensive evaluation of diagnostic modalities and contemporary therapeutic advances in canine dilated cardiomyopathy

B. Akshata[1*], P. Mosing[1], R. M. Shukla[2], A. L. Gangula[3], K. Mahendran[1], H. Saxena[1], R. Kamalla[1], N. Kumawat[1] and S. Prasad[4]

[1]Division of Medicine, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly - 243 122, Uttar Pradesh, India; [2]Department of Veterinary Pathology, Veterinary College and Research Institute, Orathanadu, TANUVAS - 614 625, Tamil Nadu, India; [3]Division of Animal Reproduction, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly - 243 122, Uttar Pradesh, India; [4]Division of Animal Nutrition, College of Veterinary Science and Animal Husbandry, DUVASU, Mathura - 281 001, Uttar Pradesh, India

Abstract

Dilated cardiomyopathy (DCM) is acquired myocardial disorder in dogs, predominantly affecting large and giant breeds, and is characterized by ventricular dilation and impaired contractility. The condition often progresses from a prolonged occult phase to overt congestive heart failure (CHF), marked by clinical signs such as dyspnea, cough, ascites and exercise intolerance. This review presents an overview of the current understanding of the pathophysiology, epidemiology, and clinical manifestations of DCM in dogs. Diagnostic advancements, including echocardiography, electrocardiography, radiographic assessment, and the use of cardiac biomarkers, in early detection and disease monitoring. Recent progress in therapeutic strategies includes the use of inodilators like Pimobendan, ACE inhibitors, and diuretics, along with emerging agents such as sacubitril/valsartan and danicamtiv. Novel therapies, including gene and stem cell treatments, as well as nutritional modulation with taurine, L-carnitine, and omega-3 fatty acids, show promising results in managing disease progression and improving survival outcomes. The integration of these diagnostic and therapeutic advancements is crucial for the effective management of DCM in the canine population.

Keywords: Cardiac biomarker, Dilated cardiomyopathy, Dogs, Echocardiography, Management

Highlights

- Dilated Cardiomyopathy (DCM) is a prevalent myocardial disorder in large and giant breed dogs, progressing from a subclinical stage to congestive heart failure (CHF), with symptoms like exercise intolerance, cough, and dyspnea
- Advanced diagnostic modalities such as echocardiography (2D, M-mode, Doppler, and Tissue Doppler), radiography, ECG, and cardiac biomarkers (NT-proBNP, cTnI, Galectin-3, ST2) enable early detection and monitoring of DCM
- Echocardiographic techniques and Tissue Doppler Imaging provide detailed functional assessment of both left and right ventricles and help evaluate diastolic dysfunction and intraventricular desynchrony
- Therapeutic Innovations include the use of Pimobendan, ACE inhibitors, diuretics, and newer agents like sacubitril/valsartan and danicamtiv, showing improved outcomes in managing CHF and DCM progression
- Emerging Therapies, such as gene therapy using VEGF-B167 and cardio sphere-derived stem cells, along with nutritional modulation (taurine, L-carnitine, omega-3 fatty acids), show promising results in improving cardiac function and longevity in affected dogs

INTRODUCTION

Cardiovascular disorders in canines are relatively common and exhibit a comparable incidence to those observed in human populations, with an estimated 10–15% of the canine population affected (Rush, 2002; MacPete, 2018; Sawhney et al., 2025). These conditions are broadly categorized into congenital and acquired forms. Epidemiological data suggest that congenital heart disease is uncommon in the general canine population, with the majority of cardiac disease burden attributable to acquired conditions such as degenerative valve disease or cardiomyopathy (Schrope, 2015; Elsharkawy et al., 2022). Breeds with a higher predisposition to valvular disease include Cavalier King Charles Spaniels, Miniature Poodles, Cocker Spaniels, Miniature Schnauzers, Dachshunds,

*Corresponding Author, E-mail: drakshatavmc@gmail.com

Pomeranians, Chihuahuas, and various mixed-breed dogs (Disatian, 2010).

Myocardial disease in dogs, particularly dilated cardiomyopathy (DCM), constitutes 8% of heart diseases (MacPete, 2018). DCM is defined as a primary myocardial disease that results in weakened contractility and dilation of one or both ventricles of the heart (O'Grady & O'Sullivan, 2004; Wess et al., 2010). Dutton and Lopez-Alvarez (2018) described dilated cardiomyopathy as a primary myocardial syndrome that may present with mechanical dysfunction resulting in ventricular dilation and circulatory congestion or electrical disturbances, leading to arrhythmias and sudden cardiac death, or a combination of both.

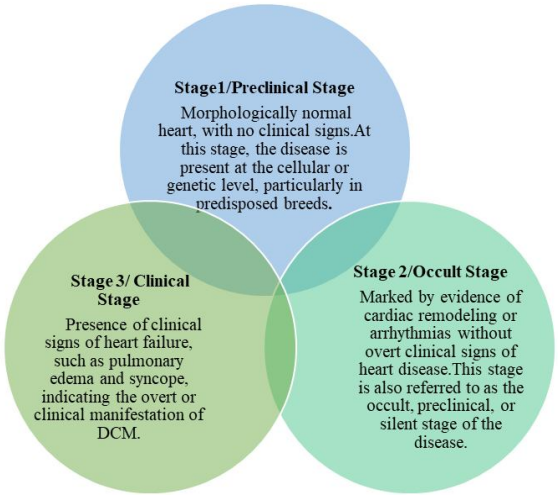

Fig. 1. Different phases of DCM

Large and giant-breed dogs are predominantly affected, and the underlying cause is often idiopathic or familial, although contributory factors such as genetic predisposition, nutritional imbalances (e.g., taurine or carnitine deficiency), persistent tachyarrhythmias, or exposure to cardiotoxic agents have been implicated (Borgarelli et al., 2006). The natural progression of DCM can be explained by three distinct phases (Fig. 1) (Wess et al., 2018; Wess, 2022). Clinically, DCM may manifest with ventricular dilation, systolic dysfunction, congestive heart failure, arrhythmias or sudden death, sometimes with minimal or no premonitory signs due to a prolonged subclinical phase. Because of the variable etiology and clinical presentation, DCM represents a complex myocardial syndrome rather than a uniform disease. Therefore, its diagnosis relies on cardiac imaging (e.g., echocardiography) and careful exclusion of secondary causes before labelling it as idiopathic DCM.

Prevalence of dilated cardiomyopathy

Prevalence of dilated cardiomyopathy has been summarized in Table 1.

Table 1. Prevalence of DCM in dogs

Study	Time Period	Sample Size	Breed(s) Most Affected	Other Observations
Dukes-McEwan et al. (2003)	1986–1991	Purdue Database	Dobermanns, Irish Wolfhounds, Great Danes, Boxers, Newfoundland	Higher incidence in pedigree dogs (0.65%) vs. crossbreds (0.16%)
Borgarelli et al. (2006)	1998–2003	76 dogs	Great Dane, Saint Bernard, Newfoundland, Doberman Pinscher, Neapolitan Mastiff	90% of affected dogs were male
Martin et al. (2009)	1993–2006	369 dogs	Doberman Pinscher, Boxer, Great Dane, Cocker Spaniel, German Shepherd, Saint Bernard, Labrador	Medium-to-large purebred dogs most commonly affected
Oyama (2015)	2015	-	Doberman Pinschers, Irish Wolfhounds	Male predominance; DCM is common in middle-aged to older dogs
Wess et al. (2018)	2018	-	Irish Wolfhounds, Boxers, Newfoundland, Great Danes, Saint Bernard's, Cocker Spaniels	Identified Dobermans as frequently affected in Europe and North America

Cont. Table 1.

Table 1., Cont. ...

Study	Time Period	Sample Size	Breed(s) Most Affected	Other Observations
Thirunavukkarasu (2019)	2019	106 dogs	Labrador Retriever	Older dogs (8–10 years) most frequently diagnosed with DCM
Jeyaraja et al. (2019)	5 years	537 dogs	Labrador Retriever	Male predominance; study focused on clinical and diagnostic findings

Clinical signs

Dogs affected with dilated cardiomyopathy (DCM) and congestive heart failure (CHF) may exhibit various clinical signs such as difficulty breathing (dyspnea), coughing, lethargy, reduced appetite, fainting (syncope), exercise intolerance, weight loss, and ascites (Martin et al., 2009; Baisan et al., 2016; Thangapandiyan et al., 2016; Sidhu et al., 2018). Canine DCM can be divided into two distinct stages based on the presence or absence of these symptoms: a prolonged pre-clinical phase, during which no signs are evident, also called as occult phase, and an overt clinical phase, characterized by the appearance of noticeable clinical symptoms (Dukes-McEwan et al., 2003). Clinical findings of canine DCM include tachypnea, dyspnea, tachycardia, arrhythmia, weak femoral arterial pulse, and a low-intensity systolic murmur (Tidholm & Jonsso, 2005; Jeyaraja et al., 2019).

Diagnosis of Dilated Cardiomyopathy

Diagnosis of DCM in dogs is primarily made through a combination of medical history, clinical signs, echocardiographic, radiographic findings, and through evaluation of cardiac biomarkers.

Radiography: In dogs with dilated cardiomyopathy (DCM), cardiac enlargement (Fig. 2) on left lateral radiograph is typically reflected by an increased vertebral heart score (VHS). The vertebral heart scale (VHS) is a method of cardiac measurement that compares the dimensions of the cardiac silhouette with the length of thoracic vertebral bodies (Buchanan & Bucheler, 1995). Oyama et al. (2008) noted that signs of congestive heart failure were more frequently observed when the VHS was 11.5 or higher. Similarly, Jeyaraja et al. (2015) reported a mean VHS of 12.51±0.14 in affected dogs, indicating significant cardiac enlargement. In DCM dogs, cardiomegaly often noticed on a dorsoventral (DV) or ventrodorsal (VD) view, this may manifest as an increased transverse

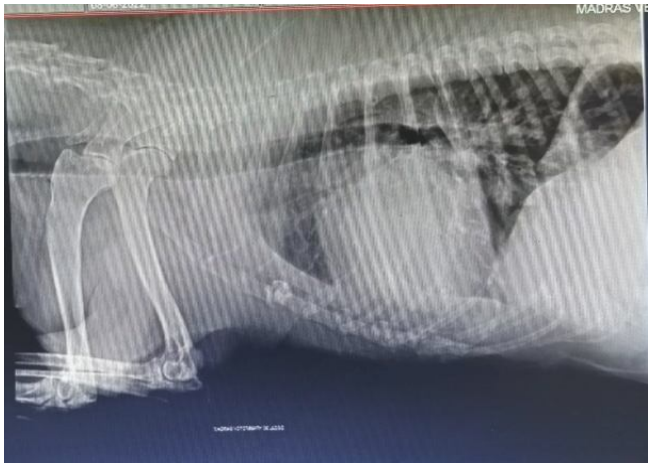

Fig. 2. Thorax left lateral radiograph of dog showing cardiomegaly with elevated trachea at the base of heart

diameter of the cardiac silhouette, displacement of mediastinal structures, and, in overt congestive cases, evidence of pulmonary congestion or edema (Saikrishna et al., 2022). Additional radiographic findings in DCM cases include pleural effusion and pulmonary edema, as documented by Jeyaraja et al. (2019). Pleural effusion is generally associated with right-sided congestive heart failure, while pulmonary edema is indicative of left-sided failure (Martin et al., 2009). Other radiographic changes may include increased sternal contact, auricular bulging, left atrial enlargement, and both pleural and pericardial effusion (Haritha et al., 2017).

The cardiothoracic index (or ratio), defined as the ratio of maximal cardiac width to maximal thoracic width on a radiograph, may also be used (on DV/VD view) as an adjunct to assess cardiac enlargement, though it is less commonly standardized than VHS. Radiographic assessments, including cardiothoracic ratio or similar heart-to-thorax width comparisons, along with observation of shape changes (e.g., more globoid heart, rounding of cardiac silhouette), tracheal

or mediastinal displacement, and pulmonary vascular/ edema changes, help support a diagnosis of DCM or other cardiomegaly-associated conditions when echocardiography is not immediately available (Saka et al., 2022).

Electrocardiography: Atrial fibrillation (Fig. 3) was the most commonly detected arrhythmia in giant and large-breed dogs with dilated cardiomyopathy (DCM), whereas ventricular premature complexes were frequently observed in Boxers and Dobermans, and supraventricular premature complexes were notably seen in Boxers and German Shepherds (Borgarelli et al., 2006; Martin et al., 2009; Baisan et al., 2016). Atrial fibrillation is a supraventricular arrhythmia characterized by excessively rapid and uncoordinated atrial electrical activity, leading to the absence of effective atrial contraction and reduced ventricular filling. Clinically, it is identified by a rapid and irregular cardiac rhythm, variable pulse intensity, and pronounced pulse deficits (Saikrishna et al., 2022). Ventricular premature complexes (VPCs) are ectopic cardiac beats that arise from spontaneous depolarization of an abnormal pacemaker focus within the ventricular myocardium. A small number of VPCs, typically fewer than 50 occurrences within 24 hours, may be observed in clinically healthy dogs. In cases of dilated cardiomyopathy (DCM), however, the diagnostic threshold for VPC frequency is breed-dependent. For most breeds, more than 100 VPCs in a 24-hour period is considered abnormal, whereas Boxers and Dobermans are regarded as positive when the count exceeds 300 VPCs over 24 hours (Saikrishna et al., 2022). Heart rate was also found to increase in correlation with the severity of heart failure (Jeyaraja et al., 2015). Electrocardiographic abnormalities associated with DCM included elevated P and R wave amplitudes, ST segment coving (Fig. 4), deep Q waves (Fig. 5), ventricular tachycardia, and wide, abnormal QRS complexes (Haritha et al., 2017). Furthermore, Wess et al. (2010), in a prospective study involving 412 Doberman Pinschers, identified ventricular premature complexes as the earliest detectable abnormality on 24-hour Holter ECG monitoring in dogs lacking echocardiographic signs of DCM.

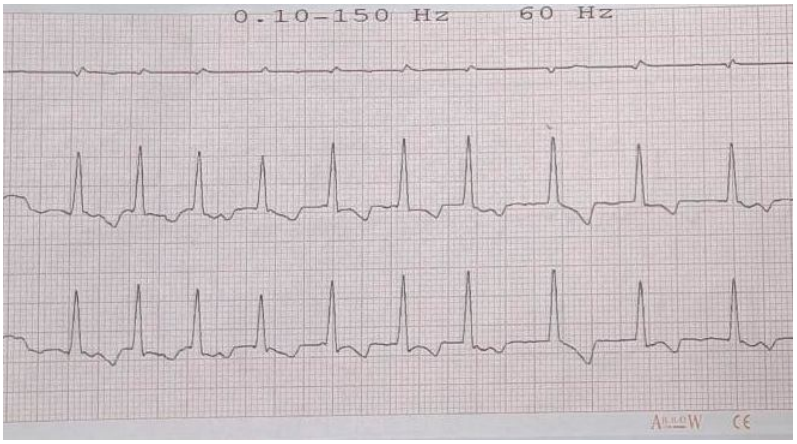

Fig. 3. No distinct P wave and irregular ventricular rhythm indicative of Atrial fibrillation

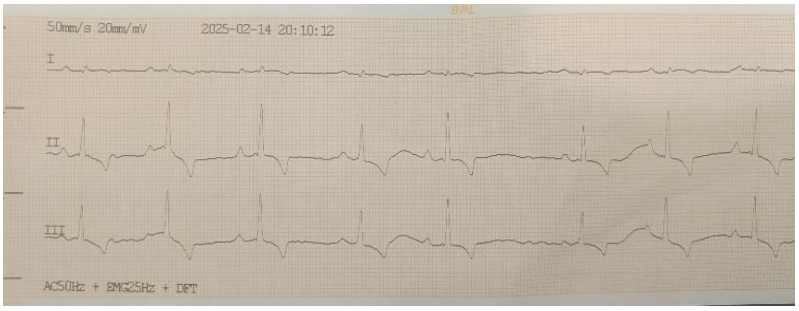

Fig. 4. ST coving

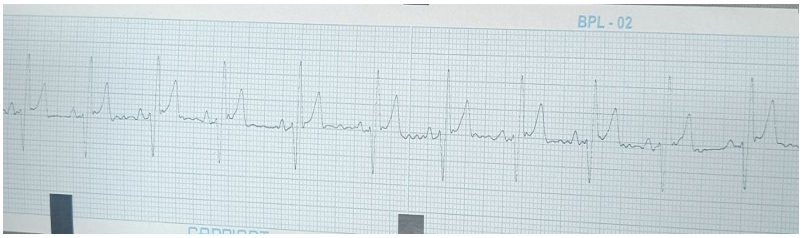

Fig. 5. Deepening of Q wave with elevated T wave

Cardiac biomarkers: A promising method of diagnosing cardiac diseases may be the identification and monitoring of blood-based cardiac biomarkers, either alone or in conjunction with other diagnostic tools. Canine cardiac biomarkers, including cardiac troponin I (cTnI) and brain natriuretic peptide (BNP), have been extensively studied and are associated with myocardial injury (Oyama & Sisson, 2004). Cardiac troponin I (cTnI) is a protein bound to the actin filament that plays a crucial role in cardiac muscle contraction and relaxation (Gavazza et al., 2020). It

becomes detectable in circulation within 2–3 hours following myocardial injury and reaches peak levels between 18–24 hours post-injury (Eggers et al., 2009). Natriuretic peptides, a group of hormones released by the myocardium in response to volume overload, contribute to the suppression of the renin–angiotensin–aldosterone system (RAAS) and play crucial role in counteracting cardiac remodeling (Oyama, 2015; De Lima & Ferreira, 2017). The prohormones pro-BNP and pro-ANP are enzymatically cleaved into NT-proBNP, NT-proANP, and their respective biologically active C-terminal fragments, BNP and ANP. Elevations in NT-proBNP and BNP levels are commonly observed in various cardiac pathologies (Oyama, 2015).

Several recent Indian studies have investigated the utility of cardiac biomarkers for detecting and characterizing cardiomyopathy in dogs. For example, in a North Indian study on 2,582 registered dogs (41 suspected cases), Cardiac Troponin-I (cTnI) was significantly elevated in dogs across different cardiac disease categories (including DCM), using a cut-off value > 92 ng/L to indicate cardiac affection (Yadav et al., 2023). Another study comparing B-type Natriuretic Peptide (BNP), Cardiac Troponin T (cTnT), and Atrial Natriuretic Peptide (ANP) in dogs with cardiac disease (confirmed by echocardiography) reported that BNP had the highest sensitivity (88.5%) and specificity (78.6%) among the biomarkers tested suggesting BNP may serve as a useful diagnostic tool under field conditions where imaging is not available (Mahendran et al., 2022).

Creatine kinase (CK) is a cytosolic enzyme found in skeletal muscle, cardiac tissue, and the brain, existing in three isoforms: CK-MM, CK-MB, and CK-BB. Among these, CK-MB is specific to cardiac muscle (Sagar et al., 2021). Lactate dehydrogenase (LDH), a widely distributed enzyme responsible for the conversion of lactate to pyruvate during glycolysis, exists in five isoforms (LDH1–LDH5). Of these, LDH5 has cardiac specificity and shows elevated levels under hemodynamic stress (Gavazza et al., 2020).

Emerging biomarkers such as Galectin-3 and Interleukin-1 receptor-like 1 protein (ST2) have shown diagnostic value in human cardiology, although their application in veterinary medicine remains limited due to a paucity of research. Galectin-3, a member of the β-galactoside-binding lectin family, is implicated in fibrosis and inflammatory processes, promoting the differentiation of fibroblasts into myofibroblasts and enhancing collagen production (Gehlken et al., 2018; Ho et al., 2012). In dogs, circulating plasma Galectin-3 levels are reported to be 0.64±0.15 ng/mL under normal conditions, with elevations up to 1.12±0.83 ng/mL observed in cardiac disease cases (Lee et al., 2021). Increased Galectin-3 levels have also been documented in dogs with degenerative mitral valve disease (DMVD) and patent ductus arteriosus (PDA) (Sakarin et al., 2016). ST2, another novel cardiac biomarker, is associated with CD4+T-cell activation and the production of Th2 cytokines. Elevated levels of ST2 have been linked to myocardial fibrosis in humans (Kakkar & Lee, 2008). Canine cardiac biomarkers have been summarized in the Table. 2.

Table. 2. Summary of canine cardiac biomarkers

Biomarkers	Role	Source	Clinical Relevance	Reference(s)
Cardiac Troponin I (cTnI)	Regulates cardiac muscle contraction & relaxation; binds to actin filament	Cardiac muscle	Detectable 2–3 h post-injury, peaks at 18–24 h; marker of myocardial injury	Eggers et al., 2009; Gavazza et al., 2020; Mukherjee, 2024; Rebez and Ninan, 2024
BNP/NT -proBNP	Released due to myocardial stretch; suppresses RAAS; aids in cardiac remodelling	Myocardium	Elevated in volume overload and various cardiac diseases	Oyama, 2015; Lima and Ferreira, 2017; Morey et al., 2023
Creatine Kinase (CK-MB)	Enzyme involved in energy metabolism	Cardiac, skeletal muscle, brain	CK-MB is heart-specific; elevated in myocardial damage	Sagar et al., 2021
Lactate Dehydrogenase	Catalyzes lactate ↓ pyruvate in	Widely distributed;	LDH5 levels increase during hemodynamic	Gavazza et al., 2020

Cont. Table 2.

Table 2., Cont. ...

Biomarkers	Role	Source	Clinical Relevance	Reference(s)
(LDH5)	glycolysis	LDH5 heart-specific	stress	
Galectin-3	Inflammation & fibrosis; activates fibroblasts, increases collagen	β-galactoside-binding lectin family	Elevated in dogs with cardiac disease (e.g., DMVD, PDA); Normal: 0.64 ng/mL, Diseased: up to 1.12 ng/mL	Gehlken et al., 2018; Ho et al., 2012; Lee et al., 2021; Sakarin et al., 2016
ST2	Immune modulation; activates CD4+ T-cells & Th2 cytokine production	Interleukin-1 receptor-like protein	Linked to myocardial fibrosis in humans; limited veterinary application due to lack of studies	Kakkar and Lee, 2008

Echocardiography

Transthoracic echocardiography (Fig. 6a and 6b) has been a standard diagnostic tool in veterinary medicine since the early 1980s for evaluating the structural and functional dynamics of the canine heart (Boon, 2011; De Lima, 2022). It is a diagnostic technique that heavily depends on the operator's skill in accurately acquiring and interpreting the data. Initially, A-mode (amplitude mode) echocardiography was employed, where returning echoes were visualized as peaks on an oscilloscope, enabling the identification of cardiac structures based on the timing, amplitude, and intensity of these peaks. Subsequently, B-mode (brightness mode) was introduced, which displayed echoes as stationary dots, with the brightness corresponding to the strength of the reflected signals. The integration of a temporal component into B-mode imaging led to the development of motion mode (M-mode) echocardiography, wherein echoes appear as vertical lines arranged sequentially along a time axis, facilitating the assessment of cardiac structures intersected by the ultrasound beam (Carerj et al., 2003).

Cardiac dimensions are typically assessed using either M-mode or two-dimensional (2D) echocardiography, both of which facilitate measurement of left ventricular (LV) parameters (Boon, 2011). 2-Dimensional echocardiography was performed with the patient positioned in right lateral recumbency. These techniques are primarily employed in cardiology to evaluate interventricular septal thickness (IVS), left ventricular internal diameter (LVD), and left ventricular posterior wall thickness (LVPW) during both systole and diastole (Fig. 7). Measurements are generally obtained from two standard imaging planes: (1) the right parasternal long-axis view at the level of the mitral valve, and (2) the right parasternal short-axis view at the level of the chordae tendineae (Noviana et al., 2011).

Measures of overall left ventricular (LV) systolic function, including fractional shortening (FS) and ejection fraction (EF), can be derived from the acquired echocardiographic data. Fractional shortening (FS) serves as a primary marker for assessing systolic function in veterinary echocardiography. In human medicine, ejection fraction (EF) is considered a more reliable measure of systolic function, as fractional shortening (FS) can be misleading in the presence of severe mitral regurgitation (Petric & Tomsic, 2008). End-systolic volume, end-diastolic volume, and stroke volume will be calculated using the Teicholz formula. Furthermore, ejection fraction and fractional shortening will also be derived using this same method, as outlined below:

End diastolic volume (EDV in mL) = 7 (Õ) $LVDd^3$/ (2.4+ LVDd)
End systolic volume (ESV in mL) = 7 (Õ) $LVDs^3$/(2.4 +LVDs)
Stroke volume (SV in mL) = EDV - ESV
EF (%) = EDV-ESV/EDV× 100
FS (%) = LVIDd – LVIDS/LVIDd× 100

The European Society of Veterinary Cardiology (ESVC) task force has established guidelines for diagnosing dilated cardiomyopathy (DCM), which are mainly based on M-mode and 2D echocardiography. A diagnosis of DCM is confirmed when the following three conditions are present: (i) left ventricular dilation, (ii) reduced systolic function, and (iii) an increase in left ventricular sphericity (Dukes-McEwan et al., 2003). In the diagnostic scoring system for dilated cardiomyopathy (DCM), certain findings are assigned

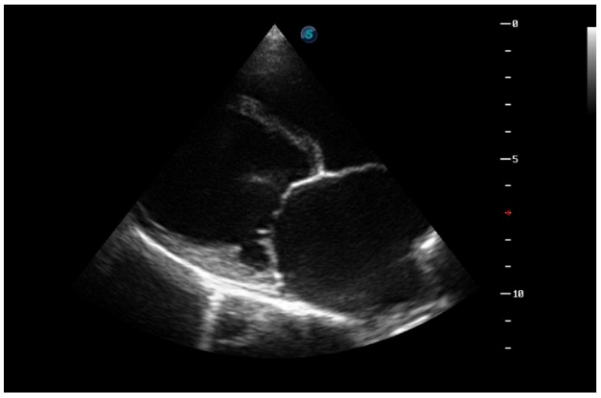

Fig. 6a. Dilatation of left atrium and left ventricle in right parasternal long axis four chamber view

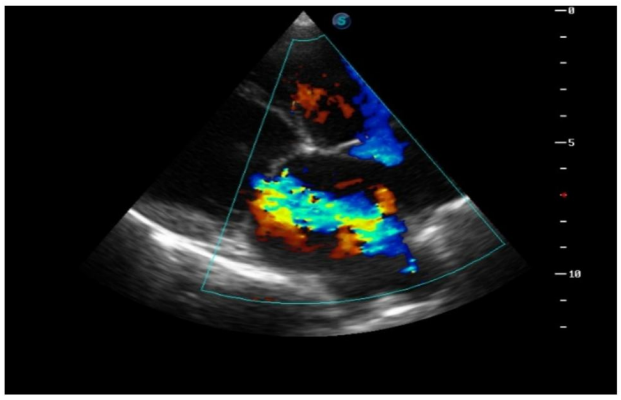

Fig. 6b. Turbulent blood flow visible on colour Doppler across mitral valve and inside the atrium

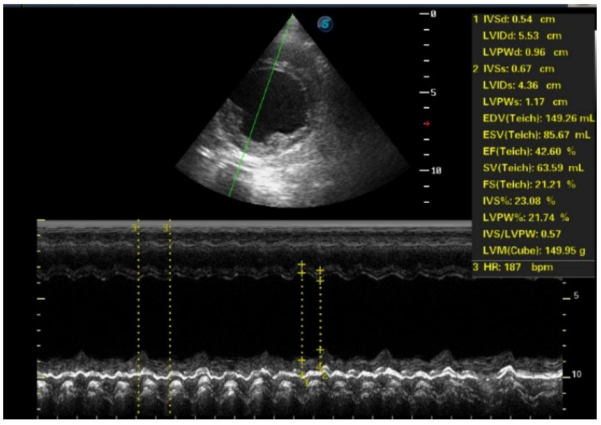

Fig. 7. Right parasternal short axis view for M-mode measurement

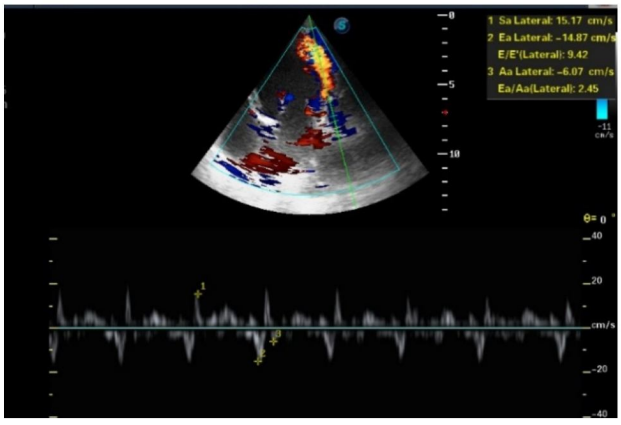

Fig. 8. Left parasternal apical four chamber view: Pulse wave doppler for Trans mitral velocity (E & A velocity)

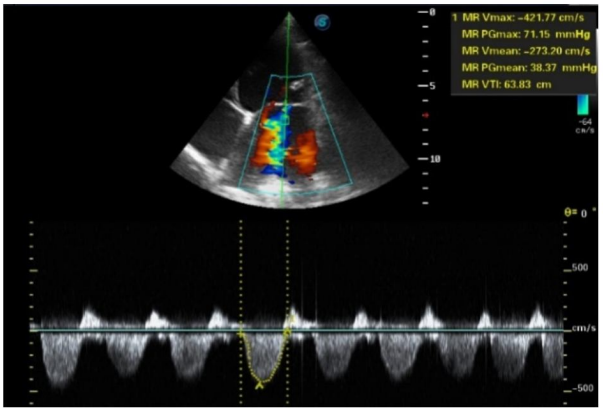

Fig. 9. Left parasternal apical four chamber view: Continuous wave doppler for mitral regurgitation velocity (MRVmax)

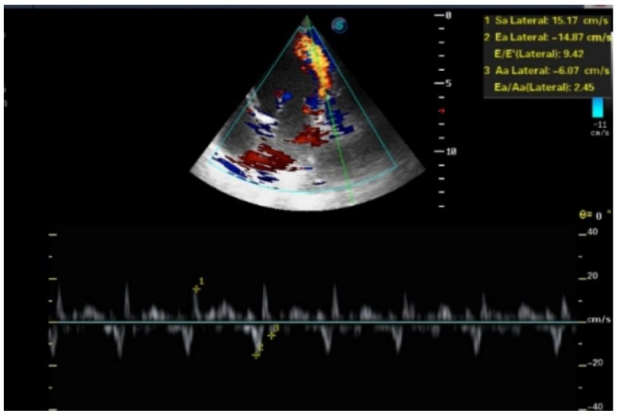

Fig. 10. Left parasternal apical four chamber view: Tissue doppler for mitral valve annulus velocity (E', A' & S' velocity)

a minor weight of one point each. These include: (a) arrhythmias specific to certain breeds; (b) atrial fibrillation (AF); (c) increased E-point septal separation (EPSS); (d) elevated pre-ejection period to ejection time ratio; (e) left ventricular (LV) fractional shortening within an equivocal range; and (f) enlargement of the left atrium or both atria. A cumulative score of six or more points suggests the presence of DCM, while a total between one and five points indicates the need for ongoing monitoring to detect potential disease progression (McNally et al., 2013; Simpson et al., 2021).

Doppler Echocardiography: Doppler echocardiography (DE) encompasses several modalities, including pulse wave Doppler (PW-D), continuous wave Doppler (CW-D), and color Doppler (CD) (Fig. 6b). Doppler echocardiography (DE) offers essential insights into the heart's hemodynamics, enabling early detection of cardiac disorders before irreversible structural or functional changes occur. It aids in identifying both the nature and severity of heart disease. Color Doppler (CD) is utilized to visualize the direction of blood flow, pulse wave Doppler (PW-D) measures the velocity of blood flow, and continuous wave Doppler (CW-D) is employed to assess high-velocity or turbulent blood flow. Pulse wave Doppler (PWD), initially validated through invasive measurement techniques, is currently the most commonly used method for directly assessing diastolic function and indirectly evaluating atrial function.

Spectral Doppler evaluation is performed by positioning the animal in left lateral recumbency and acquiring left parasternal apical four-chamber view. Mitral and tricuspid valve regurgitation produces a turbulent jet, which appears as a mosaic pattern within the atria during systole (De Madron et al., 2015). Pulse wave Doppler imaging is employed to evaluate trans-mitral flow velocities (Fig. 8), specifically the E and A wave velocities and the E/A ratio, in accordance with the standard procedure described by Boon (2011). In the same view, parameters such as Mitral Regurgitation velocity (MR Vmax) (Fig. 9), Tricuspid Regurgitation velocity (TR Vmax), Pulmonary Regurgitation velocity (PR Vmax), and the percentage of left atrial (LA) area affected by regurgitation were measured using Boon's (2011) established method.

Tissue Doppler imaging: Tissue Doppler Imaging (TDI) is an advanced echocardiographic method that utilizes the Doppler principle to measure the velocity of myocardial segments and other cardiac structures. It is particularly effective for evaluating long-axis ventricular function. Since reduced longitudinal myocardial fiber motion is an early indicator of myocardial dysfunction and ischemia, TDI holds promise as a valuable tool in routine cardiac assessment. This technique enables accurate, quantitative evaluation of regional myocardial performance both at rest and during stress testing. TDI is especially useful for diagnosing diastolic left ventricular dysfunction, as it minimizes the influence of preload conditions common to conventional Doppler methods. Additionally, it can assess right ventricular function, estimate intracardiac and pulmonary artery pressures, detect transplant rejection, and evaluate intraventricular desynchrony (Nikitin & Witte, 2004).

Tissue Doppler imaging (Fig. 10) was utilized to assess lateral myocardial velocities by placing the sample volume at the mitral annulus of the left ventricular free wall. The systolic (Sa), early diastolic (Ea), and late diastolic (Aa) velocities were recorded as described by Thirunavukkarasu (2014). From these measurements, isovolumic relaxation time (IVRT), isovolumic contraction time (IVCT), and ejection time (ET) were determined. Subsequently, the TEI index was calculated using the formula: Tei index = (IVCT + IVRT) / ET.

Management of dilated cardiomyopathy

The conventional approach to managing the clinical signs of congestive heart failure (CHF) includes the use of inotropic agents, diuretics, vasodilators, and antiarrhythmic drugs. The therapeutic strategy for cardiac insufficiency is based on two key principles: decreasing the workload of the heart and enhancing its contractile function (Caro et al., 2009). Pimobendan, a novel drug with both phosphodiesterase-inhibitory and calcium-sensitizing effects, enhances myocardial contractility and induces arterial and venous dilation, making it highly effective in treating CHF in dogs (Smith et al., 2005; Gordon et al., 2006). Additionally, angiotensin-converting enzyme (ACE) inhibitors, which suppress the renin-angiotensin-aldosterone system, have been shown to improve clinical outcomes and prolong survival in dogs with CHF when used alongside other therapeutic agents (Kvart et al., 2002).

Various angiotensin-converting enzyme (ACE) inhibitors are used in clinical practice, including enalapril, benazepril, captopril, zofenopril, and alacepril. ACE inhibitors containing a sulfhydryl group (e.g., captopril) may offer additional benefits, such as antioxidant effects and enhanced nitric oxide production, which contribute to improved vascular

endothelial function and remodelling (Hori et al., 2018). These drugs have also been shown to reduce myocardial fibrosis in animal models of heart failure. According to Bernay et al. (2010), incorporating spironolactone into standard therapy significantly decreases the risk by 55% of cardiac-related mortality, euthanasia, or substantial clinical deterioration in heart failure cases. Loop diuretics remain a cornerstone in the treatment of CHF in both human and veterinary medicine due to their effectiveness in reducing intravascular hydrostatic pressure and alleviating edema-related symptoms (Peddle et al., 2012). Among these, furosemide is the only loop diuretic currently recommended by the American College of Veterinary Internal Medicine (ACVIM) for managing CHF resulting from myxomatous mitral valve disease (MVD) in dogs (Chetboul et al., 2017), while research on the use of torsemide in veterinary patients is still underway.

Sacubitril/valsartan (S/V), an angiotensin receptor-neprilysin inhibitor (ARNI), has shown promising results in treating canine heart failure (Saikrishna et al., 2025). Studies indicate that S/V can reverse myocardial remodelling, evidenced by significant reductions in echocardiographic parameters such as the left atrium to aortic root ratio (LA/Ao) and left ventricular internal diameter in diastole normalized to body weight (LVIDDN) in dogs with myxomatous mitral valve disease (Saengklub et al., 2021). Advancements in pharmacotherapy include the exploration of agents like danicamtiv, a cardiac myosin activator. This drug enhances myocardial contractility by increasing the efficiency of the heart's contractile proteins. In vitro and in vivo trials in dogs have indicated that danicamtiv can improve systolic function without adversely affecting diastolic performance, offering a potential therapeutic option for DCM management (Grillo et al., 2021).

Gene therapy has emerged as a promising avenue for treating DCM by targeting molecular abnormalities within the myocardium. One notable approach involves the intracoronary administration of adeno-associated viral vectors carrying the gene for vascular endothelial growth factor-B167 (VEGF-B167). This cytoprotective factor has demonstrated anti-apoptotic properties and the ability to improve cardiac function (Paradies et al., 2019). In a case study, a St. Bernard dog treated with this gene therapy exhibited a stable ejection fraction over a 36-month follow-up period and survived for nearly four years post-treatment, suggesting a potential for slowing disease progression (Paradies et al., 2025). The use of cardio sphere-derived stem cells (CDCs) represents another innovative strategy in DCM treatment. These cells, derived from adult dog hearts, have the capacity to differentiate into cardiomyocytes (Simpson et al., 2021). In studies involving Doberman Pinschers with DCM, intracoronary infusion of CDCs was well-tolerated, with no adverse reactions reported. Preliminary findings suggest that CDC therapy may slow ventricular enlargement and mitigate systolic dysfunction, although larger-scale studies are needed to confirm these effects (Hensley et al., 2017).

Nutritional factors have been implicated in the development and progression of DCM. Supplementation with taurine and L-carnitine has shown benefits in dogs with suspected nutritional deficiencies, leading to improvements in clinical signs and echocardiographic parameters. Additionally, omega-3 fatty acids have been associated with reduced muscle loss and increased survival rates in DCM-affected dogs (McCauley et al., 2020).

Conclusion

Dilated cardiomyopathy (DCM) remains one of the most critical myocardial disorders in dogs, particularly affecting large and giant breeds, with a progressive course that leads to congestive heart failure and sudden cardiac death if left undiagnosed or poorly managed. The integration of advanced diagnostic techniques, including echocardiography, electrocardiography, radiographic assessment, and the evaluation of novel cardiac biomarkers, has significantly improved the ability to detect early-stage disease and monitor progression more accurately. The development of newer therapeutic agents such as sacubitril/valsartan and danicamtiv, along with innovative approaches like gene therapy, stem cell therapy, and targeted nutritional support, has opened promising avenues for better disease control and improved survival outcomes. Despite these advances, breed-specific predispositions, diagnostic challenges in the subclinical phase, and limited accessibility to advanced therapies in routine practice remain concerns. Therefore, continued research, coupled with a multidisciplinary approach involving early diagnosis, tailored therapeutics, and regular monitoring, is essential to optimize the clinical management of canine DCM and enhance the quality of life in affected dogs.

Conflict of interest: The authors declare that they have no conflict of interest.

Author's contributions: BA: Conceptualized the topic and conducted the literature review; PM, RS, AG: Involved in data curation, writing, and preparation of the original draft; KM, HS: Contributed to the writing and editing of the manuscript; RK, NK, SP: Critically reviewed and revised the manuscript for important intellectual content. All authors have read and approved the final version of the manuscript. All authors contributed significantly to the preparation of this manuscript.

Data availability statement: This is a review article and does not involve the generation of primary data. All data presented in this manuscript are derived from previously published studies, which have been duly cited. Additional details or materials can be made available by the corresponding author upon reasonable request.

Use of artificial intelligence tools: 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 this manuscript. The author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

ACKNOWLEDGEMENT

We acknowledge the Head, Division of Medicine and Director of IVRI, Izatnagar, for providing the necessary facilities for disease diagnostics and treatment.

REFERENCES

Baisan, R. A., Bîrsan, O., & Vulpe, V. (2016). Electrocardiographic changes in chronic valvular disease and dilated cardiomyopathy in dog. *Human and Veterinary Medicine*, *8*(2), 98-102.

Bernay, F., Bland, J., Häggström, J., Baduel, L., Combes, B., Lopez, A., & Kaltsatos, V. (2010). Efficacy of spironolactone on survival in dogs with naturally occurring mitral regurgitation caused by myxomatous mitral valve disease. *Journal of Veterinary Internal Medicine*, *24*(2), 331–341. https://doi.org/10.1111/j.1939-1676.2009.0467.x

Boon, J. A. (2011). *Veterinary Echocardiography* (2nd ed.). John Wiley & Sons.

Borgarelli, M., Santilli, R. A., Chiavegato, D., D'Agnolo, G., Zanatta, R., Mannelli, A., & Tarducci, A. (2006). Prognostic indicators for dogs with dilated cardiomyopathy. *Journal of Veterinary Internal Medicine*. https://doi.org/10.1111/j.1939-1676.2006.tb02829.x

Buchanan, J. W., & Bücheler, J. (1995). Vertebral scale system to measure canine heart size in radiographs. *Journal of the American Veterinary Medical Association*, *206*(2), 194–199. https://doi.org/10.2460/javma.1995.206.02.19

Carerj, S. S., Micari, A., Trono, A., Giordano, G., Cerrito, M., Zito, C., Luzza, F., Coglitore, S., Arrigo, F., & Oreto, G. (2003). Anatomical M mode: an old-new technique. *Echocardiography*, *20*(4), 357-361. https://doi.org/10.1046/j.1540-8175.2003.03041.x

Caro, A., Ynaraja, E., & Montoya, J. A. (2009). Effects of short-term treatment with pimobendan in dogs with myxomatous valve disease. *Journal of Applied Animal Research*, *35*(2), 113-117. https://doi.org/10.1080/09712119.2009.9706999

Chetboul, V., Pouchelon, J., Menard, J., Blanc, J., Desquilbet, L., Petit, A., Rougier, S., Lucats, L., & Woehrle, F. (2017). Short term efficacy and safety of torasemide and furosemide in 366 dogs with degenerative mitral valve disease: the TEST study. *Journal of Veterinary Internal Medicine*, *31*(6), 1629-1642. https://doi.org/10.1111/jvim.14841

De Lima, A. M., Moreira, R. M., Gomes, M. S., Ramos, M. T., dos Santos-Sousa, C. A., Souza-Júnior, P., & Abidu-Figueiredo, M. (2022). Echocardiographic evaluation of working dogs of the military police of Rio de Janeiro: effects of the breed and body weight. *Brazilian Journal of Veterinary Medicine*, *44*, e001322. https://doi.org/10.29374/2527-2179.bjvm001322

De Lima, G. V., & Da Silveira Ferreira, F. (2017). N-terminal-pro brain natriuretic peptides in dogs and cats: a technical and clinical review. *Veterinary world*, *10*(9), 1072. https://doi.org/10.14202/vetworld.2017.1072-1082

De Madron, E., Chetboul, V., & Bussadori, C. (2015). *Clinical Echocardiography of the Dog and Cat*. Elsevier Health Sciences.

Disatian, S. (2010). Myxomatous degenerative mitral valve disease: an update. *The Thai Journal of Veterinary Medicine*, *40*(2), 151-157. https://doi.org/10.56808/2985-1130.2218

Dukes-McEwan, J., Borgarelli, M., Tidholm, A., Vollmar, A. C., & Häggström, J. (2003). Proposed guidelines for the diagnosis of canine idiopathic dilated cardiomyopathy. *Journal of Veterinary Cardiology*, *5*(2), 7–19. https://doi.org/10.1016/s1760-2734(06)70047-9

Dutton, E., & López Alvarez, J. (2018). An update on canine cardiomyopathies–is it all in the genes?. *Journal of Small Animal Practice*, *59*(8), 455-464. https://doi.org/10.1111/jsap.12841

Eggers, K. M., Jaffe, A. S., Lind, L., Venge, P., & Lindahl, B. (2009). Value of cardiac troponin I cutoff concentrations below the 99th percentile for clinical decision-making. *Clinical chemistry*, *55*(1), 85-92. https://doi.org/10.1373/clinchem.2007.101683

Elsharkawy, S. H., Torad, F. A. S., Abu-Aita, N. A., Abdel-Ghany, A. K., & El-Husseiny, I. N. (2022). Acquired cardiac diseases in 72 dogs: A prospective study [2017-2020]. *International Journal of Veterinary Science*, *4*(11), 427- 434. https://doi.org/10.47278/journal.ijvs/2022.132

Gavazza, A., Fruganti, A., Turinelli, V., Marchegiani, A., Spaterna, A., Tesei, B., Rossi, G., & Cerquetella, M. (2020). Canine traditional laboratory tests and cardiac biomarkers. *Frontiers in Veterinary Science, 7*,320. https://doi.org/10.3389/fvets.2020.00320

Gehlken, C., Suthahar, N., Meijers, W. C., & de Boer, R. A.

(2018). Galectin-3 in heart failure: an update of the last 3 years. *Heart Failure Clinics*, *14*(1), 75-92. https://doi.org/10.1016/j.hfc.2017.08.009

Gordon, S. G., Miller, M. W., & Saunders, A. B. (2006). Pimobendan in heart failure therapy – a silver bullet? *Journal of the American Animal Hospital Association*, *42*(2), 90-93. https://doi.org/10.5326/0420090

Grillo, M. P., Markova, S., Evanchik, M., Trellu, M., Moliner, P., Brun, P., Perreard-Dumaine, A., Vicat, P., Yang, C., Driscoll, J. P., & Carlson, T. J. (2021). Preclinical in vitro and in vivo pharmacokinetic properties of danicamtiv, a new targeted myosin activator for the treatment of dilated cardiomyopathy. *Xenobiotica*, *51*(2), 222–238. https://doi.org/10.1080/00498254.2020.1839982

Haritha, G. S., Kumar, K. S., Ayodhya, S., & Kumar, V. V. V. (2017). Prevalence of cardiac disorders in canines-a clinical study. *Intas Polivet*, *18*(1), 148-152.

Hensley, M. T., Tang, J., Woodruff, K., Defrancesco, T., Tou, S., Williams, C. M., Breen, M., Meurs, K., Keene, B., & Cheng, K. (2017). Intracoronary allogeneic cardiosphere derived stem cells are safe for use in dogs with dilated cardiomyopathy. *Journal of Cellular and Molecular Medicine*, *21*(8), 1503-1512. https://doi.org/10.1111/jcmm.13077

Ho, J. E., Liu, C., Lyass, A., Courchesne, P., Pencina, M. J., Vasan, R. S., Larson, M. G., & Levy, D. (2012). Galectin-3, a marker of cardiac fibrosis, predicts incident heart failure in the community. *Journal of the American College of Cardiology*, *60*(14), 1249–1256. https://doi.org/10.1016/j.jacc.2012.04.053

Hori, Y., Nakamura, K., Kanno, N., Hitomi, M., Yamashita, Y., Hosaka, S., Isayama, N., & Mimura, T. (2018). Effects of the angiotensin-converting enzyme inhibitor alacepril in dogs with mitral valve disease. *Journal of Veterinary Medical Science*, *80*(8), 1212–1218. https://doi.org/10.1292/jvms.17-0557

Jeyaraja, K., Arun, A., Hamsa Yamini, S., Sesh, P. S. L., & Nambi A. P. (2015). Diagnostic evaluation of dilated cardiomyopathy in Labrador retrievers. *International Journal of Advanced Research*, *3*(12), 628–655.

Jeyaraja, K., Yamini, S. H., & Chandrasekaran, D. (2019). A retrospective analysis of dilated cardiomyopathy in Labrador Retrievers. *Journal of Animal Research*, *9*(5), 707-714.https://doi.org/10.30954/2277-940X.05.2019.12

Kakkar, R., & Lee, R. T. (2008). The IL-33/ST2 pathway: therapeutic target and novel biomarker. *Nature Reviews Drug Discovery*, *7*(10), 827-840. https://doi.org/10.1038/nrd2660

Kvart, C., Häggström, J., Pedersen, H. D., Hansson, K., Eriksson, A., Järvinen, A., Tidholm, A., Bsenko, K., Ahlgren, E., Lives, M., Åblad, B., Falk, T., Bjerkås, E., Gundler, S., Lord, P., Wegeland, G., Adolfsson, E., & Corfitzen, J. (2002). Efficacy of enalapril for prevention of congestive heart failure in dogs with myxomatous valve disease and asymptomatic mitral regurgitation. *Journal of Veterinary Internal Medicine*, *16*(1), 80–88. https://doi.org/10.1111/j.1939-1676.2002.tb01610.x

Lee, G., Kang, M., Ro, W., Song, D., & Park, H. (2021). Circulating galectin-3 evaluation in dogs with cardiac and non-cardiac diseases. *Frontiers in Veterinary Science*, *8*, 741210. https://doi.org/10.3389/fvets.2021.741210

MacPete, R. (2018). *Dogs and heart disease: An overview*. IDEXX Laboratories Inc.

Mahendran, K., Thakur, N., Chethan, G., Priyanka, Choudhary, S. S., Dey, S., Saxena, A., Mahajan, S., & Kavitha, K. (2022). Comparative assessment of troponin T, atrial natriuretic peptide, B-type natriuretic peptide and echocardiography in the diagnosis of cardiac and renal disorders in canine. *Indian Journal of Animal Research*, *56*(2), 234-238. https://doi.org/10.18805/ijar.b-4159

Martin, M. W. S., Stafford Johnson, M. J., & Celona, B. (2009). Canine dilated cardiomyopathy: a retrospective study of signalment, presentation and clinical findings in 369 cases. *Journal of Small Animal Practice*, *50*(1), 23-29. https://doi.org/10.1111/j.1748-5827.2008.00659.x

McCauley, McCauley, S. R., Clark, S. D., Quest, B. W., Streeter, R. M., & Oxford, E. M. (2020). Review of canine dilated cardiomyopathy in the wake of diet-associated concerns. *Journal of Animal Science*, *98*(6), skaa155. https://doi.org/10.1093/jas/skaa155

McNally, E. M., Golbus, J. R., & Puckelwartz, M. J. (2013). Genetic mutations and mechanisms in dilated cardiomyopathy. *The Journal of clinical investigation*, *123*(1), 19-26. https://doi.org/10.1172/JCI62862

Morey, A. G., Lamb, K. E., Karnia, J. J., Wiggen, K. E., Lyons, B. M., Nafe, L. A., & Leach, S. B. (2023). N terminal brain natriuretic peptide, cardiac troponin I, and point of care ultrasound in dogs with cardiac and noncardiac causes of nonhemorrhagic ascites. *Journal of Veterinary Internal Medicine*, *37*(3), 900-909. https://doi.org/10.1111/jvim.16702

Mukherjee, J. (2024). Canine cardiac biomarkers: nature, activity and use. *Exploratory Animal and Medical Research*, *14*(1), 06–13. https://doi.org/10.52635/eamr/14.1.6-13

Nikitin, N. P., & Witte, K. K. (2004). Application of tissue Doppler imaging in cardiology. *Cardiology*, *101*(4), 170-184. https://doi.org/10.1159/000076694

Noviana, D., Paramitha, D., & Wulansari, R. (2011). Motion mode and two dimensional echocardiographic measurements of cardiac dimensions of Indonesian mongrel dogs. *HAYATI Journal of Biosciences*, *18*(1), 1-5. https://doi.org/10.4308/hjb.18.1.1

O'Grady, M. R., & O'Sullivan, M. (2004). Dilated cardiomyopathy: an update. *Veterinary Clinics of North America Small Animal Practice*, *34*(5), 1187–1207. https://doi.org/10.1016/j.cvsm.2004.05.009

Oyama, M. A. (2015). Using cardiac biomarkers in veterinary practice. *Clinics in Laboratory Medicine*, *35*(3), 555-566. https://doi.org/10.1016/j.cll.2015.05.005

Oyama, M. A., & Sisson, D. D. (2004). Cardiac troponin I concentration in dogs with cardiac disease. *Journal of Veterinary Internal Medicine*, *18*(6), 831-839. https://doi.org/10.1111/j.1939-1676.2004.tb02629.x

Oyama, M. A., Fox, P. R., Rush, J. E., Rozanski, E. A., & Lesser, M. (2008). Clinical utility of serum N-terminal pro-B-type natriuretic peptide concentration for identifying cardiac disease in dogs and assessing

disease severity. *Journal of the American Veterinary Medical Association*, *232*(10), 1496-1503. https://doi.org/10.2460/javma.232.10.1496

Paradies, P., Carlucci, L., Digiaro, S., Recchia, A., Colella, A., Woitek, F., Lacitignola, L., Staffieri, F., Giacca, M., & Recchia, F. A. (2025). Intracoronary cytoprotective gene therapy in a dog with dilated cardiomyopathy: long term follow up. *Veterinary Research Communications*, *49*(2), 117. https://doi.org/10.1007/s11259-025-10691-2

Paradies, P., Carlucci, L., Woitek, F., Staffieri, F., Lacitignola, L., Ceci, L., Romano, D., Sasanelli, M., Zentilin, L., Giacca, M., Salvadori, S., Crovace, A., & Recchia, F. A. (2019). Intracoronary gene delivery of the cytoprotective factor vascular endothelial growth factor-B167 in canine patients with dilated cardiomyopathy: a short-term feasibility study. *Veterinary Sciences*, *6*(1), 23. https://doi.org/10.3390/vetsci6010023

Peddle, G. D., Singletary, G. E., Reynolds, C. A., Trafny, D. J., Machen, M. C., & Oyama, M. A. (2012). Effect of torsemide and furosemide on clinical, laboratory, radiographic and quality of life variables in dogs with heart failure secondary to mitral valve disease. *Journal of Veterinary Cardiology*, *14*(1), 253-259. https://doi.org/10.1016/j.jvc.2012.01.003

Petric, A. D., & Tomsiè, K. (2008). Diagnostic methods of cardiomyopathy in dogs-old and new perspectives and methods. *Slovenian Veterinary Research*, *45*(1), 5-14.

Rebez, E. B., & Ninan, J. (2024). Biomarkers in the cardiovascular system of animals: A review. *Indian J Anim Health*, *63*(1), 22-28. https://doi.org/10.36062/ijah.2024.08023

Rush, J. E. (2002, October). Chronic valvular heart disease in dogs. In *Proceedings of 26th Annual Waltham Diets/OSU Symposium for the Treatment of Small Animal Cardiology*.

Saengklub, N., Pirintr, P., Nampimoon, T., Kijtawornrat, A., & Chaiyabutr, N. (2021). Short-Term Effects of Sacubitril/valsartan on Echocardiographic Parameters in Dogs With Symptomatic Myxomatous Mitral Valve Disease. *Frontiers in Veterinary Science*, *8*, 700230. https://doi.org/10.3389/fvets.2021.700230

Sagar, R. S., Mudraje, N. B., Chikmagalur, A. K., Siddaraju, N. K., Appaiah, K. M., Sahadev, A., Rao, S., & Panchaxarayya, K. G. (2021). Creatine Kinase-MB as a cardiac biomarker in canine cardiac disorders. *Indian Journal of Veterinary Sciences & Biotechnology*, *17* (3), 27-30.

Saikrishna, K. S., Jeyaraja, K., Vairamuthu, S., Shafiuzama, M., & Selvaraj, P. (2022). Electrocardiographic abnormalities in canine dilated cardiomyopathy and their management. *Indian Journal of Veterinary Sciences & Biotechnology*, *18*(5), 63-67. https://doi.org/10.48165/ijvsbt.18.5.13

Saikrishna, K., Jeyaraja, K., Selvaraj, P., Vairamuthu, S., & Shafiuzama, M. (2025). Therapeutic evaluation of sacubitril-valsartan (ARNI) in canine dilated cardiomyopathy. *Indian Journal of Animal Research*. https://doi.org/10.18805/ijar.b-5588

Saikrishna, K.S., Jeyaraja, K., Vairamuthu, S., Shafi, M., Selvaraj, P. (2022). Canine Dilated Cardiomyopathy: A Clinical and Diagnostic Study. *Journal of Animal Research*, *12*(5), 783-788.

Saka, S., Ýskefli, O., Or, M., & Bakýrel, U. (2022). The objectivity of vertebral heart scale: comparison of detailed radiographic findings with vertebral heart scale in symptomatic dogs with cardiac failure. *German Journal of Veterinary Research*, *2*(3), 1–7. https://doi.org/10.51585/gjvr.2022.3.0036

Sakarin, S., Rungsipipat, A., & Surachetpong, S. D. (2016). Galectin-3 in cardiac muscle and circulation of dogs with degenerative mitral valve disease. *Journal of Veterinary Cardiology*, *18*(1), 34-46. https://doi.org/10.1016/j.jvc.2015.10.007

Sawhney, A., Shahi, A., Das, B., & Vaish, R. (2025). Study on incidence of canine cardiac disorders in Jabalpur city. *Indian Journal of Animal Research*, *59*(9), 1570. https://doi.org/10.18805/ijar.b-4866

Schrope, D. P. (2015). Prevalence of congenital heart disease in 76, 301 mixed-breed dogs and 57,025 mixed-breed cats. *Journal of Veterinary Cardiology*, *17*(3), 192-202. https://doi.org/10.1016/j.jvc.2015.06.001

Sidhu, S., Uppal, S. K., Saini, N., Randhawa, C. S., & Chhabra, S. (2018). Diagnosis of cardiomyopathies in dogs-a clinical study of twenty three patients. *Intas Polivet*, *19*(1), 121-125.

Simpson, S., Kordtomeikel, K. Z., Wong, S., Bennison, S., El-Gendy, S. A., Cobb, M., & Rutland, C. S. (2021). Diagnosis, prognosis, management, treatment, research and advances in canine dilated cardiomyopathy. In *Intech Open eBooks*. https://doi.org/10.5772/intechopen.97682

Smith, P. J., French, A. T., Van IsrarL, N., Smith, S. G. W., Swift, S. T., Lee, A. J., Corcoran, B. M., & Dukes McEwan, J. (2005). Efficacy and safety of pimobendan in canine heart failure caused by myxomatous mitral valve disease. *Journal of Small Animal Practice*, *46*(3), 121-130. https://doi.org/10.1111/j.1748-5827.2005.tb00302.x

Thangapandiyan, M., Mohanapriya, T., Balachandran, C., Jeyaraja, K., Arulanandam, K., & Sridhar, R. (2016). Study on pathomorphological changes in 21 cases of canine dilated cardiomyopathy. *International Journal of Science, Environment and Technology*, *5*(6), 4612-4617.

Thirunavukkarasu, P. (2014). *Doppler echocardiographic assessment of acquired heart diseases in dogs* (PhD Thesis), Tamil Nadu Veterinary and Animal Sciences University.

Thirunavukkarasu, P. (2019). Prevalence of canine acquired heart diseases in Chennai, India. *International Journal of Livestock Research*, *9*(3), 331-339. https://doi.org/10.5455/ijlr.20181213035610

Tidholm, A., & Jönsson, L. (2005). Histologic characterization of canine dilated cardiomyopathy. *Veterinary Pathology*, *42*(1), 1-8. https://doi.org/10.1354/vp.42-1-1

Wess, G. (2022). Screening for dilated cardiomyopathy in dogs. *Journal of Veterinary Cardiology*, *40*, 51–68. https://doi.org/10.1016/j.jvc.2021.09.004

Wess, G., & Torti, M. (2018). Arrhythmias in canine cardiomyopathies and valvular heart disease. *Guide to Canine and Feline Electrocardiography*, 285-299.

Wess, G., Mäurer, J., Simak, J., & Hartmann, K. (2010). Use of Simpson's method of disc to detect early echocardiographic changes in Doberman Pinschers with dilated cardiomyopathy. *Journal of Veterinary Internal Medicine*, *24*(5), 1069–1076. https://doi.org/10.1111/j.1939-1676.2010.0575.x

Yadav, A., Kumar, T., Sindhu, N., Agnihotri, D., Jajoria, C., & Sharma, M. (2023). Characterization of Cardiac Diseases in Dogs Prevalent in Indian Conditions. *Indian Journal of Animal Research*, *57*(5). https://doi.org/10.18805/ijar.b-4544

Received - 31.05.2025, *Accepted - 22.11.2025*, *Published - 27.11.2025 (Online), 01.12.2025 (Print)*
Section Editor: Dr. Joydip Mukherjee, Editorial Board Member