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Research Article

Pulsed tissue Doppler imaging in dogs affected with hypertrophic cardiomyopathy

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Abstract

Hypertrophic cardiomyopathy (HCM) in dogs is a rare but clinically significant myocardial disorder characterized by concentric hypertrophy and diastolic dysfunction. Although echocardiography remains the diagnostic standard, early detection of impaired relaxation can be challenging. This study aimed to evaluate pulsed wave tissue Doppler imaging (PW-TDI) parameters in dogs diagnosed with HCM and compare them with healthy controls to assess its diagnostic utility. A total of 6 clinically diagnosed HCM dogs and 6 healthy controls underwent comprehensive cardiac evaluation including electrocardiography, thoracic radiography, conventional echocardiography and PW-TDI, complemented with hemato-biochemical analysis and blood pressure monitoring. Both Radial and Longitudinal myocardial velocities were analysed. Longitudinal myocardial velocities (S^{l} , E^{l} , A^{l}) were measured at the septal and lateral mitral annuli, whereas Radial motion was measured at subendocardial and subepicardial segments, and further, key diastolic indices such as E′/A′ and E/E^{l} were calculated. HCM dogs exhibited significantly reduced E′ velocities (septal: 0.069±0.014 m/s; lateral: 0.096±0.019 m/s) compared to controls (septal: 0.092±0.025 m/s; lateral: 0.129±0.035 m/s), indicating impaired early diastolic relaxation. The E/E^{l} ratios were also elevated (septal E/E^{l} : 15.3±4.6 vs. 9.4±3.0; lateral E/E′ : 11.7±3.8 vs. 6.8±2.6), suggesting increased left ventricular filling pressures. These findings affirm the potential of PW-TDI in identifying early diastolic dysfunction in dogs with HCM.

Keywords: Canine cardiology, Diastolic dysfunction, Echocardiography, Hypertrophic cardiomyopathy, Tissue Doppler Imaging

Highlights

- First study using PW-TDI in dogs with HCM vs healthy controls
- PW-TDI showed a reversed E′/A′ ratio, indicating impaired diastolic relaxation.
- Higher A^{l} , lower E′, and raised E/E′ suggest increased ventricular filling pressure and stiffness.
- PW-TDI aids early HCM detection and monitoring, improving clinical outcomes in dogs.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a myocardial disease distinguished by an unexplained thickening of the left ventricular wall, which can interfere with normal cardiac function and lead to clinical signs in affected individuals (Elliott et al., 2014). Although more frequently reported in cats, HCM has gained increasing recognition in dogs, particularly with the wider use of echocardiography in routine veterinary practice. Recent studies have highlighted potential breed predispositions and the likelihood of subclinical disease progression in canines. The primary pathological feature of HCM is diastolic dysfunction, attributed to reduced ventricular compliance and impaired relaxation (Pace, 2020). While echocardiography remains the gold standard for diagnosing HCM and distinguishing it from other cardiomyopathies, however, detecting early-stage diastolic dysfunction using conventional methods can be challenging (Matos et al., 2024).

Tissue Doppler imaging (TDI), especially in the pulsed-wave (PW-TDI) mode, offers a non-invasive approach to assess myocardial velocities and has proven to be a sensitive tool for detecting abnormalities in both systolic and diastolic heart function (Chetboul et al., 2004; Kim & Park, 2015). Its application extends to evaluating myocardial synchrony and estimating left ventricular filling pressures, offering additional diagnostic value in both veterinary and human cardiology (Hori et al., 2007; Choi et al., 2013). To the author's knowledge, this is the first study to evaluate PW-TDI parameters in dogs diagnosed with HCM and compare them with healthy controls, to establish the role of this technique in early

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diagnosis of diastolic dysfunction and its potential to guide future treatment strategies.

MATERIALS AND METHODS

Study design: The study was conducted at the Small Animal Multi-Speciality Veterinary Hospital of Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, India. A total of 6 dogs presented with clinical symptoms such as cough, exercise intolerance, dyspnoea, lethargy, syncope and cyanosis were selected for the study. Dogs meeting these criteria were subjected to hemato-biochemical analysis, thoracic radiography, electrocardiography (ECG) and echocardiography to confirm the diagnosis. Subsequently, pulsed-wave tissue Doppler imaging (PW-TDI) parameters were recorded in the affected dogs. Dogs with comparable breed andbody weights were taken as healthy controls for comparison.

Blood pressure monitoring, Electrocardiography, Radiographic and Echocardiographic assessment: In both group dogs, systolic Blood Pressure (BP) was measured using the Doppler blood pressure system (Vet-Dop2, Vmed Technology). Dogs were awake and manually restrained in lateral recumbency. The Doppler probe was carefully placed over the digital palmar artery to detect arterial blood flow. The average of at least five measurements from each dog was recorded as the final systolic BP.

Electrocardiography (ECG) was conducted on all dogs using the Cardiart 8108 BPL six-channel electrocardiographic machine (BPL Medical Technologies, Bangalore). Bailey's hexaxial limb lead system was used, which combined regular bipolar and unipolar enhanced limb leads as already prescribed techniques (Tilley & Smith, 2024). The recordings were made at a sensitivity of 10 mm/mV and a paper speed of 50 mm/sec.

Orthogonal Thoracic radiographic views (Lateral and Ventro-Dorsal) were taken for cardiovascular evaluation. Echocardiographic evaluation was performed on all healthy and diseased dogs using a GE Logiq P5color Doppler ultrasound machine (GE Healthcare, Chicago, United States) equipped with a 5S transducer. The assessment included standard imaging techniques such as 2D mode, M-mode, color Doppler, pulsed wave Doppler, and continuous wave Doppler to evaluate cardiac structure and function. Asymmetrical septal or left ventricular free wall (LVFW) thickening was identified by calculating the ratio of interventricular septum thickness during diastole (IVSd) to left ventricular free wall thickness at end-diastole (LVFWd). A ratio exceeding 1.30 was indicative of asymmetrical septal hypertrophy, whereas a ratio below 0.70 suggested asymmetrical thickening of the LVFW (Maron & Epstein, 1980; Schober et al., 2022).

Pulsed wave Tissue Doppler Imaging: PW-TDI was carried out in all dogs using the same ultrasound system employed for conventional echocardiography. Data acquisition was performed using a 2 mm sample volume positioned at specific myocardial sites under standardized imaging protocols. For assessment of longitudinal myocardial velocities, the left apical four-chamber view was utilized and radial myocardial motion was evaluated from the right parasternal short-axis view, specifically at the level of the papillary muscles. This dual-view approach enabled precise characterization of both directional components of myocardial function while ensuring consistency across all subjects. Longitudinal myocardial function was evaluated at two anatomically distinct locations: the lateral mitral annulus, representing the left ventricular free wall (LVFW), and the septal mitral annulus, corresponding to the interventricular septum (IVS). For assessment of radial motion, tissue Doppler measurements were obtained at the level of the papillary muscles, with the sample volume carefully positioned in both endocardial and epicardial layers of the LVFW to capture regional displacement. No angle correction was applied during acquisition. The Nyquist velocity range was set between 15–25 cm/s, optimized to enhance signal clarity while preventing aliasing. Additionally, gain and wall filter settings were adjusted to suppress background noise and enhance myocardial signals.

At each myocardial site, the peak velocities during systole (S'), early diastole (E'), and late diastole or atrial contraction (A') were measured in m/s. For accuracy, at least three cardiac cycles were recorded, and the mean value of the highest three values from these was used for analysis. The E' /A' ratio was calculated for each segment to assess diastolic function. Additionally, peak early mitral inflow velocity (E wave) and early myocardial velocity (E') at both lateral and septal mitral annular levels were measured, and the E/E' ratio was derived to estimate left ventricular filling pressures. The values were compared to those of 6 healthy control dogs of similar body weight and age, evaluated using the same protocol.

Statistical analysis: The data were stored in Microsoft Excel and analyzed using SPSS statistical software (version 16.0). Qualitative and quantitative variables were presented as means, medians, standard error of means (SEM), standard deviations (SD), ranges, counts,

Table 1. Demographic data of dogs in the healthy (control) group and the HCM group

Parameter	Control (N=6) Mean±SEM (Range)	HCM (N=6) Mean±SEM (Range)
Age (years)	4.50±1.40 (1-9)	7.00±2.00 (4-9)
Weight (kg)	13.53±2.20 (9-24)	13.77±2.94 (9-28)
Sex		
Male	2 (33.33%)	5 (83.33%)
Female	4 (66.66%)	1 (16.67%)
Breeds	Pug (3) Beagle (2) American Pitbull Terrier (1)	Pug (4) American Pitbull Terrier (2)

Table 2. Mean±SEM of haematological indices and cardiac biomarkers in healthy dogs (control) versus HCM dogs

Parameter	Control	HCM
Hb (g/dL)	13.38±0.28	12.21±0.45
TLC (×10³/µL)	12.36±0.56	14.67±2.37
Neutrophils (Absolute) (cells/µL)	9.97±0.51	12.10±1.93
Lymphocytes (Absolute) (cells/µL)	2.11±0.15	2.18±0.56
TEC (×10⁶/µL)	5.78±0.12	5.43±0.23
PCV (%)	39.05±0.70	36.78±2.07
Platelet count (×10³/µL)	273.42±14.20	330.00±60.72
Troponin I (ng/mL)	0.11±0.02	0.64±0.17*
NT-proBNP (pmol/L)	447.92±18.69	760.37±93.53*

HCM: Hypertrophic Cardiomyopathy; Hb: Hemoglobin; TLC: Total leucocyte count; TEC: Total erythrocytic count; PCV: Packed cell volume,*p<0.05 versus healthy

Table 3. Mean±SEM of biochemical parameters in healthy dogs (control) versus HCM group

Parameter	Control	HCM	Level of significance
Total Bilirubin	0.15±0.01	0.16±0.03	NS
AST (U/L)	37.48±2.11	46.66±6.06	NS
ALT (U/L)	44.86±3.42	77.00±12.05	NS
ALKP (U/L)	99.93±5.47	128.83±27.48	NS
GGT (U/L)	7.20±0.34	6.16±0.79	NS
Total protein (g/dL)	6.69±0.11	6.36±0.30	NS
Albumin (g/dL)	3.21±0.05	2.93±0.15	NS
BUN (mg/dL)	14.77±1.14	17.83±1.70	NS
Creatinine (mg/dL)	0.87±0.03	1.00±0.106	NS
Sodium (mEq/L)	145.69±0.92	145.50±1.72	NS
Potassium (mEq/L)	4.40±0.08	5.00±0.34	NS
Chloride (mEq/L)	111.25±1.05	105.16±3.71	NS
Phosphorus (mg/dL)	3.69±0.16	4.46±0.54	NS
Glucose (mg/dL)	101.41±2.12	107.33±7.18	NS

AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; ALKP: Alkaline Phosphatase; GGT: Gamma-Glutamyl Transferase; NS – Non-significant

and proportions. Unpaired *t*-test and one-way analysis of variance (ANOVA) were employed to examine significant differences between various parameters at $p \leq 0.05$, $p \leq 0.01$, and $p \leq 0.001$.

RESULTS

Demographic data of dogs: In our study, small-breed, geriatric dogs, especially Pugs, were diagnosed with HCM (Table 1). A notable sex difference was observed in the HCM group, which consisted predominantly of males (83.33%), whereas the control group had a female majority (66.66%).

Clinical and historical findings: Coughing (66.66%) was the most common sign, followed by dyspnoea and syncope in 50% of dogs each, in dogs affected with HCM. Exercise intolerance, lethargy and retching were less common signs observed in 33.33% of cases each. Clinical examination parameters, including rectal temperature (101.3±0.46ºF), heart rate (140±11.50 bpm) and pulse rate in diseased dogs were in normal ranges described for healthy dogs. Systolic Blood pressure measurements (137.16±5.13), ruling out hypertrophy secondary to systemic hypertension.

Hemato-biochemical findings: HCM-affected dogs demonstrated no significant differences compared to healthy dogs in hemato-biochemical parameters (Table 2, Table 3), including liver function tests, renal function tests and serum electrolytes, ruling out any systemic disease process impacting myocardial health. Total leukocyte and neutrophil counts were slightly increased ($14.67 \pm 2.37 \times 10^3/\mu L$ and $12.10 \pm 1.93\ 37 \times 10^3/\mu L$, respectively), although not reaching statistical significance, which may be due to subclinical myocardial inflammatory response. Cardiac biomarkers [canine specific Troponin I (cTnI - 0.64±0.17 ng/mL) and NT-proBNP (760.37±93.53 ng/L)] were significantly increased ($p<0.05$) relative to controls (Table 2)

Radiographic findings: In HCM-affected dogs, the mean VHS was 11.75±0.40v and ICS occupied averaged 3.83±0.10 suggesting moderate cardiac enlargement. Sternal contact was increased in 4 dogs (cardiosternal contact >3 sternebrae) (Fig. 1), while tracheal elevation was mild in 2, moderate in 3 and severe in 1 case, while thetracheal bifurcation angle showed amild to moderate increase in 3 dogs, findings that are consistent with radiographic indicators of left atrial enlargement as described by Le Roux et al. (2012), and Bahr (2018). Four dogs had a Cardio-Thoracic Index>66%, while 2 dogs each fell into=66% and <66% categories. No pleural effusion was observed. Lung patterns included alveolar (1 case), interstitial (2 cases), and mixed patterns (2 cases). Pulmonary vascular congestion was noted in 2 dogs. The cardiac apex showed mild lateral displacement in 2 dogs and remained normal in 3.

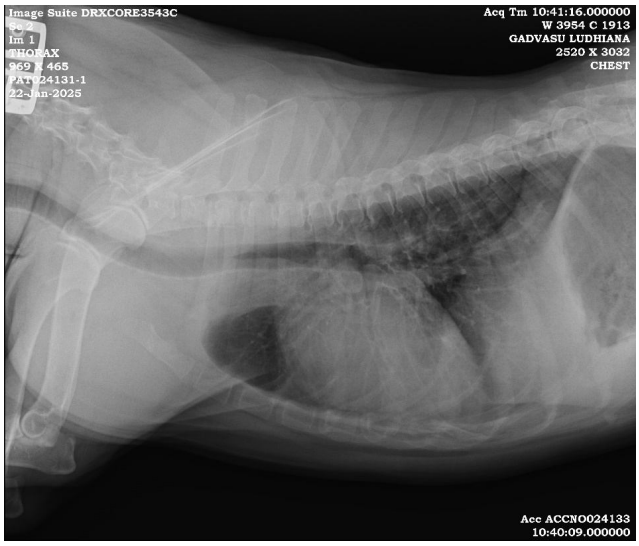


Fig. 1. Lateral chest radiograph depicting increased sternal contact and pulmonary congestion

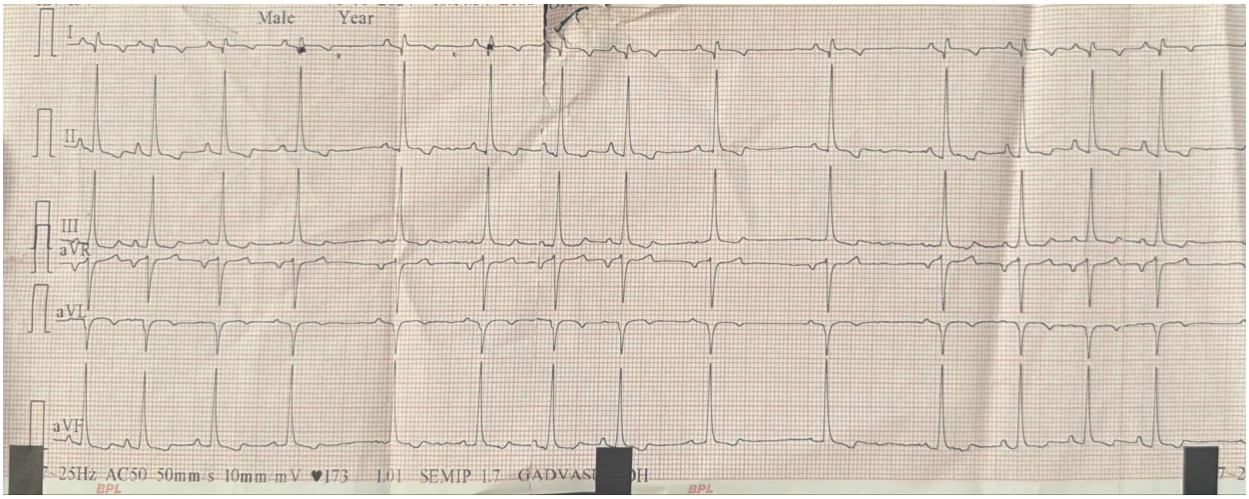


Fig. 2. ECG in an 8-year-old American Pitbull Terrier dog with hypertrophic cardiomyopathy showing Sinus Arrhythmia (Paper speed: 50 mm/s and 1 cm=1 mV)

Electrocardiographic findings: Sinus arrhythmia was the predominant finding (4 cases) (Fig. 2), supporting the autonomic influences seen in this condition. Additional findings included wandering pacemaker (1 case), peaked T waves (1 case) and ST slurring (1 case), suggesting early signs of ventricular strain and diastolic dysfunction.

Echocardiographic findings: Significant increases in Interventricular septal thickness were observed both in diastole (IVDd: 1.23±0.15 cm vs. 0.79±0.07 cm, $p<0.05$) and in systole (IVDs:1.67±0.22 cm vs. 1.04±0.07 cm, $p<0.05$) as compared to healthy controls. Left ventricular internal dimensions remained comparable between HCM and healthy dogs, suggesting a concentric hypertrophic pattern that maintains chamber size while thickening the walls. LVFW thickness and contractile indices (FS and EF) were higher in the HCM group (FS: 51.97±5.10%; EF: 83.13±4.11%) versus controls (FS: 39.82±2.76%; EF: 71.93±3.50%) (Table 4). Two dogs in the present study with an IVSd/LVFWd ratio greater than 1.30 were diagnosed with asymmetrical hypertrophic cardiomyopathy, while the remaining four, with an IVSd/LVFWd ratio between 0.7 and 1.30, exhibited a symmetrical pattern of myocardial thickening (Fig. 3).

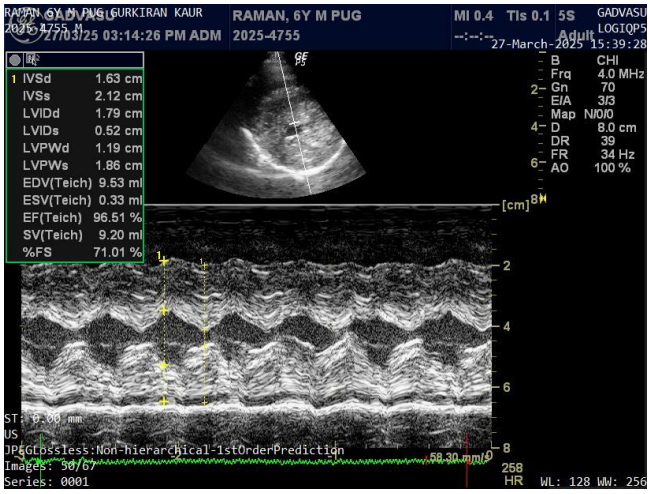


Fig. 3. M-mode echocardiographic evaluation from right parasternal short axis view at the level of papillary muscles showing severe symmetrical hypertrophy of left ventricle in a 9-year-old male Pug dog

significance and were still in the normal range of <1.6 defined for diagnosing LA enlargement. Although statistically non-significant, the mitral inflow Doppler examination revealed higher A-wave velocities in HCM dogs (0.91±0.13 m/s vs. 0.58±0.04 m/s), yielding a decreased E/A ratio (0.98±0.14 vs. 1.33±0.03) (Table 4), indicative of diastolic dysfunction with a relaxation abnormality.

PW-TDI radial myocardial velocities: In dogs with HCM, radial tissue Doppler examination revealed a significant increase in the endocardial $A^{'}$ velocity (0.088±0.016 m/s) as compared with controls (0.060±0.020 m/s, p<0.01), suggesting enhanced atrial contribution to left ventricular filling. Meanwhile, early diastolic velocities ($E^{'}$) were lower in HCM dogs in both the endocardial (0.078±0.023 vs 0.103±0.041 m/s) and epicardial (0.053±0.013 vs 0.078±0.038 m/s) layers. This shift caused a significant reduction in

Table 4. Conventional Echocardiography and Doppler parameters in the control group versus HCM group

Parameter	Control (N=6) Mean±SEM	HCM group (N=6) Mean±SEM
M-mode Echocardiographic parameters		
IVSd (cm)	0.79±0.07	1.23±0.15*
IVSs (cm)	1.04±0.07	1.67±0.22*
LVIDd (cm)	2.63±0.28	2.55±0.36
LVIDs (cm)	1.61±0.21	1.23±0.20
LVFWd (cm)	0.84±0.04	0.97±0.08
LVFWs (cm)	1.33±0.05	1.55±0.17
Fractional shortening (FS %)	39.82±2.76	51.97±5.10
Ejection Fraction (EF %)	71.93±3.50	83.13±4.11
Two dimensional Echocardiographic parameters		
Left atrium diameter (cm)	1.65±0.09	1.93±0.24
Aorta diameter (cm)	1.46±0.04	1.69±0.14
Left atrium/aorta ratio	1.12±0.06	1.16±0.10
Mitral inflow Doppler variables		
Mitral E wave (m/s)	0.77±0.06	0.83±0.09
Mitral A wave (m/s)	0.58±0.04	0.91±0.13
Mitral E/A ratio	1.33±0.03	0.98±0.14

IVSd: Interventricular septum thickness during diastole; IVSs: Interventricular septum thickness during systole; LVIDd: Left ventricular internal diameter during diastole; LVIDs: Left ventricular internal diameter during systole; LVFWd: Left ventricular free wall thickness during diastole; LVFWs: Left ventricular free wall thickness during systole; *p<0.05 versus control

In cases of HCM, the left atrial (LA) and aortic (Ao) diameters, as well as the LA/Ao ratio, were observed to be mildly elevated as compared to healthy controls; however, these differences did not reach statistical

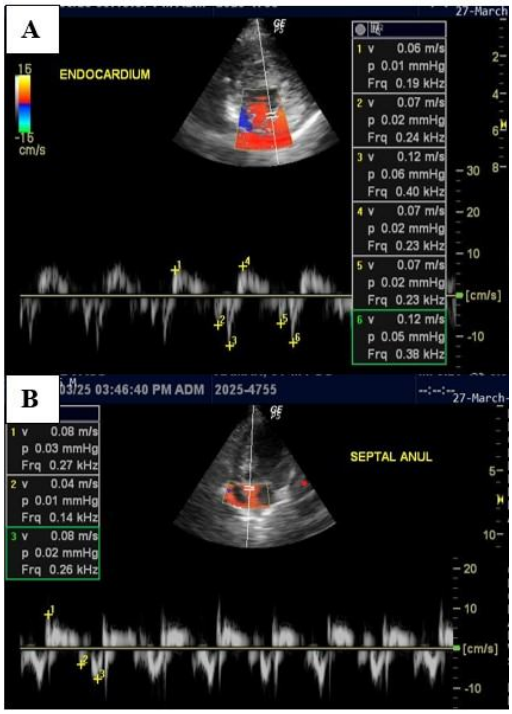


Fig. 4. Radial Pulsed Wave Tissue Doppler imaging (PW-TDI) parameters obtained from the Subendocardial segment (A) and Longitudinal PW-TDI parameters obtained from the Septal annulus segment (B) in a 9-year-old Pug showing typical $E^{'}/A^{'}$ reversal. [1 corresponds to systolic myocardial wave ($S^{'}$), 2 corresponds to early diastolic myocardial wave ($E^{'}$), and 3 corresponds to late diastolic wave ($A^{'}$)]

Table 5. Means±SD of radial myocardial velocities (m/s) obtained in the control group versus dogs affected with HCM

Parameter	Control (N=6)	HCM (N=6)
Sub endocardial segment		
S' (endo) (m/s)	0.086±0.020	0.090±0.026
E' (endo) (m/s)	0.103±0.041	0.078±0.023
A' (endo) (m/s)	0.060±0.020	0.088±0.016**
E'/A' (endo)	1.732±0.463	0.907±0.284
Sub epicardial segment		
S' (epi) (m/s)	0.068±0.017	0.073±0.023
E' (epi) (m/s)	0.078±0.038	0.053±0.013
A' (epi) (m/s)	0.051±0.019	0.063±0.017
E' /A' (epi)	1.485±0.28	0.923±0.420

HCM=hypertrophic cardiomyopathy; S' =systolic myocardial wave; E' =early diastolic myocardial wave; A' =late diastolic myocardial wave, endo=subendocardial; epi=subepicardial; **p<0.01versus control.

Table 6. Means±SD of longitudinal myocardial velocities (m/s) obtained in the control group versus dogs affected with HCM

Parameter	Control (N=6)	HCM (N=6)
Septal annulus		
S' (septal) (m/s)	0.086±0.012	0.078±0.014
E' (septal) (m/s)	0.090±0.034	0.056±0.013
A' (septal) (m/s)	0.053±0.012	0.078±0.026
E' /A' (septal)	1.648±0.349	0.750±0.159***
E/E' (septal)	9.400±2.960	15.290±4.630
Lateral annulus		
S' (lateral) (m/s)	0.111±0.043	0.086±0.017
E' (lateral) (m/s)	0.130±0.058	0.073±0.015
A' (lateral) (m/s)	0.071±0.024	0.091±0.036
E' /A' (lateral)	1.795±0.542	0.851±0.210**
E/E' (lateral)	6.790±2.630	11.730±3.780

HCM=hypertrophic cardiomyopathy; S' =systolic myocardial wave; E=peak E wave mitral early diastolic flow; E' =early diastolic myocardial wave; A' =late diastolic myocardial wave, septal=septal annulus of mitral valve; lateral=lateral annulus of mitral valve; **p<0.01 versus control; ***p<0.001 versus control.

E′/A′ ratios across both endocardial and epicardial sites, indicating a characteristic diastolic relaxation abnormality (Fig. 4). The S′ velocities remained comparable between the HCM and control groups (Table 5).

PW-TDI longitudinal myocardial velocities: Longitudinal Tissue Doppler imaging revealed significant changes when compared with healthy controls. At the septal annulus, early diastolic velocity (E′) was markedly reduced in the HCM group (0.056±0.013 m/s) compared to controls (0.090±0.034 m/s), indicative of impaired early diastolic relaxation. Conversely, the late diastolic velocity (A') was higher in HCM dogs (0.078±0.026 m/s) versus controls (0.053±0.012 m/s). This shift caused a significant decrease in the septal E′ /A′ ratio (0.750±0.159) versus the controls (1.648±0.349, p<0.001). Similarly, in the lateral wall, HCM dogs exhibited decreased E′ (0.073±0.015 m/s) and increased A′ (0.091±0.036 m/s), yielding a lower E′/A′ ratio (0.851±0.210) as compared with controls (1.795±0.542, p<0.01) (Table 6).

E/E' in HCM dogs: Transmitral flow assessment revealed that HCM dogs had slightly higher E wave velocities (0.836±0.228 m/s) versus controls (0.776±0.157 m/s), suggesting elevated filling pressures. More critically, the E/E' ratio, a key marker of left ventricular filling pressure, was increased in HCM dogs across both septal (15.290±4.630 vs. 9.400±2.960) and lateral (11.730±3.780 vs. 6.790±2.630) annuli (p<0.001) (Table 6).

DISCUSSION

Owing to the scarcity of research on hypertrophic cardiomyopathy (HCM) in dogs (Pang et al., 2005; Pace, 2020; Schober et al., 2022; Matos et al., 2024), detailed knowledge about its clinical manifestations and symptoms in canines remains limited when compared to the well-documented presentations in cats and humans (Gersh et al., 2011; Elliott et al., 2014; Maron & Fox, 2015). The results of this study add on to the findings of previous studies on the clinical, hemato-biochemical, electrocardiographic, radiographic and echocardiographic findings in HCM-affected dogs and are the first to our knowledge about the importance and use of PW-TDI as an additional diagnostic aid in canine HCM patients.

Several studies have most frequently documented the condition in large-breed male dogs, including German Shepherds, Rottweilers, Dalmatians, German Shorthaired Pointers, Weimaraners, Boxers, and Golden Retrievers (Liu et al., 1993; Jyothsna et al., 2024). However, other reports indicate that HCM also occurs in small-breed dogs, particularly among terrier breeds (Schober et al., 2022). There are variable reports of sex and age predilection to this condition (Liu et al., 1993; Schober et al., 2022; Matos et al., 2024). The findings from our study broaden the spectrum of predisposed breeds and highlight the need for further investigation into breed and sex-related predilections for HCM in dogs.

Previous studies have reported that syncope, dyspnoea, sudden cardiac death and thrombosis as clinical signs in HCM in cats (Maron & Fox, 2015), whereas dyspnoea and syncope were the most commonly observed signs in dogs diagnosed with HCM (Ando et al., 2023), which are in concordance with our findings. Hemato-biochemical profiles were found to be non-significant,as also observed in previous studies (Kumar et al., 2010), while cardiac biomarkers like cardiac troponin I (cTnI) have been investigated more extensively. The release of cardiac troponin I (cTnI) into the bloodstream is primarily associated with myocardial injury and necrosis (Maron & Fox, 2015). Markedly elevated serum cTnI concentrations (>15 ng/mL) have been linked to significant myocardial ischemia, injury, and necrosis, particularly in dogs experiencing acute decompensation of end-stage HCM-induced heart failure (Ando et al., 2023). In contrast, milder increases in cTnI levels are often associated with transient myocardial stress or injury without extensive cardiomyocyte necrosis (Kubo et al., 2011). The relatively lower elevation in cTnI observed in our study may suggest myocardial ischemia or increased hemodynamic load rather than severe cardiomyocyte damage (Ando et al., 2023).

Increased VHS obtained from lateral chest radiographs, indicating cardiomegaly is usually reported in HCM in dogs. Additionally, radiographic findings such as left atrial enlargement, tracheal elevation, and pulmonary edema have been reported in approximately 33–66% of cases, and our observations further are consistent with a similar pattern, in line with previous reports (Yadav et al., 2021; Saini et al., 2023). In humans, HCM has been associated with the development of atrial fibrillation and ventricular arrhythmias, as evidenced by electrocardiographic findings (Segev et al., 2023) while ECG alterations reported in dogs were increased amplitude and duration of P and T waves, pronounced ST segment slurring, deep Q wave and Ventricular Premature Complexes (Jyothsna et al., 2024) but in our limited number of cases, we did not observe any life-threatening cardiac arrhythmias. Continuous 24-hour Holter monitoring may provide additional diagnostic value and help in the detection of potentially fatal arrhythmias.

Although above mentioned modalities can help to screen the dogs suspected of HCM, Echocardiography remains the gold standard for confirmatory diagnosis (Anwar & Tencate, 2021; Schober et al., 2022). In cats, a left ventricular wall thickness of 6 mm is considered the diagnostic threshold for HCM (Payne et al., 2013), while in humans, the cutoff is typically 15 mm (Gersh et al., 2011; Enriquez & Goldman, 2014). However, applying this approach to dogs presents challenges, as no universally accepted cutoff value exists due to the wide range of body sizes across different breeds (Schober et al., 2022). Furthermore, echocardiographic reference data for canines remain limited (Ando et al., 2023). As a result, identifying concentric hypertrophy in dogs requires a more comprehensive evaluation using breed and size-specific echocardiographic parameters and standardized diagnostic criteria (Schober et al., 2022). LA enlargement is commonly observed in cats with HCM (Linney et al., 2014) and is considered an indicator of disease severity (Payne et al., 2013). However, some studies have noted that only about 34% of affected cats show LA enlargement (Duler et al., 2019). In contrast, LA enlargement appears to be less prevalent in dogs with HCM as recorded in our study, with other reports indicating its presence in only 22% of cases (Schober et al., 2022).

The diagnostic utility of tissue Doppler imaging for the detection of systolic and diastolic dysfunctions has already been established in dogs for dilated cardiomyopathy (Pereira et al., 2016) and valvular diseases (Kim & Park, 2015). Decreased maximum early diastolic velocities at the level of mitral valve annulus (septal E′ : 4.12±0.85 vs 6.42±1.83 cm/s and lateral E′ : 5.24±1.22 vs 8.39±2.58) have been reported in cats with HCM (Koffas et al., 2006) and in human patients (Loureiro et al., 2008; Butz et al., 2010).

The increased A^{l} velocity combined with reduced E^{l} and reversal of E^{l}/A^{l} ratios (Fig. 4) was found in all dogs along longitudinal myocardial segments at the level of mitral valve annulus in our present study in HCM-affected dogs, which is a peculiar finding of diastolic dysfunction seen in human HCM patients (Butz et al., 2010). Similar findings of altered myocardial diastolic velocities have been reported in feline HCM studies (Koffas et al., 2006) aligning with the pathophysiology of impaired early diastolic relaxation and a compensatory reliance on atrial contraction for left ventricular filling.

The overall results of our study indicate that HCM-affected dogs experience increased diastolic stiffness and filling pressures, which are consistent with the reported findings of changes in TDI parameters across different studies on feline (Koffas et al., 2006; Sugimoto et al., 2015) and human patients (Loureiro et al., 2008; Butz et al., 2010), and PW-TDI can prove to be a sensitive tool for early detection of diastolic dysfunction in HCM.

Our study suggests that PW-TDI provides valuable diagnostic insight into myocardial function in dogs with hypertrophic cardiomyopathy. This study demonstrates that E^{l} , E^{l}/A^{l} and E/E^{l} ratios are sensitive markers of diastolic dysfunction in affected dogs. All dogs with hypertrophic cardiomyopathy (HCM) demonstrated a characteristic reversal of the E^{l}/A^{l} ratio in the longitudinal (septal and lateral) segments. It also contributes to our better understanding of canine HCM, which is still a poorly understood condition. Incorporating PW-TDI into routine echocardiographic evaluations may improve early detection and therapeutic outcomes in canine HCM. The ability of PW-TDI to detect early diastolic abnormalities, even in dogs with preserved fractional shortening and ejection fraction, highlights its value as a diagnostic adjunct to conventional echocardiography. However, further research involving larger cohorts is warranted to optimize its use, refine reference intervals and further clarify prognostic implications of PW-TDI in canine HCM.

Conflict of interest: The authors do not have any conflict of interest to declare.

Author's contribution: RS: Data collection and formulation, conducted research, writing original draft; NS: Supervision, data curation, reviewing and editing the manuscript; ST: Reviewing manuscript and methodology validation; RSS: Statistical analysis of data

Data availability statement: All the data supporting the research findings have been presented in this paper. The corresponding author is willing to provide the raw data upon reasonable request.

Ethical approval: Ethical clearance was obtained from the Institutional Animal Ethics Committee (IAEC No. GADVASU/2024/IAEC/74/10) before commencing the experiment, and humane animal handling procedures were followed throughout the experiment.

Use of artificial intelligence tools: During the preparation of this work, the author(s) used no generative AI tool such as ChatGPT, COPILOT, etc or other text-to-image generators.

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