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Serum cardiac biomarker analysis in dogs with naturally occurring dilated cardiomyopathy

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Abstract

Identifying dilated cardiomyopathy (DCM) in dogs before the onset of congestive heart failure remains one of the most pressing challenges in veterinary cardiology. Physical examination and thoracic radiography lack the sensitivity to detect early myocardial dysfunction, and echocardiography while considered the gold standard requires specialist equipment and expertise not available in most general practices. Circulating cardiac biomarkers offer a blood-based screening alternative, but their diagnostic and prognostic value across the spectrum of DCM severity in naturally affected dogs has not been fully defined.

This research measured serum concentrations of cardiac troponin I (cTnI), N-terminal pro-B-type natriuretic peptide (NT-proBNP), and creatine kinase myocardial band (CK-MB) in 60 dogs: 38 with echocardiographically confirmed DCM classified by the American College of Veterinary Internal Medicine (ACVIM) staging system (Stage B1, n = 11; Stage B2, n = 14; Stage C, n = 13) and 22 breed-matched healthy controls. Dogs were recruited prospectively at the Toronto Metropolitan University of Veterinary Medicine Cardiology Service between March 2021 and September 2023. Blood was collected at the time of echocardiographic assessment, and biomarker concentrations were measured using validated immunoassay platforms.

All three biomarkers were significantly elevated in DCM dogs compared with controls ($P < 0.001$ for each). Median cTnI was 0.62 ng/mL (interquartile range [IQR] 0.21–1.47) in the DCM group versus 0.04 ng/mL (IQR 0.02–0.07) in controls. Median NT-proBNP was 1,847 pmol/L (IQR 786–4,218) in DCM dogs versus 312 pmol/L (IQR 198–441) in controls. CK-MB was mildly elevated in DCM dogs (mean 14.7 ± 6.3 U/L vs 7.2 ± 2.1 U/L; $P < 0.01$) but showed lower discriminatory power than the other two markers. A stepwise increase in both cTnI and NT-proBNP was observed across ACVIM stages, with the steepest rise occurring between Stage B2 and Stage C. Serum cTnI correlated positively with left ventricular internal diameter in diastole (LVIDD; $r = 0.73$; $P < 0.001$) and negatively with fractional shortening ($r = -0.68$; $P < 0.001$). ROC analysis showed that cTnI at a cut-off of 0.10 ng/mL had a sensitivity of 92.1% and specificity of 90.9% for distinguishing DCM dogs from controls (AUC = 0.95), while NT-proBNP at 550 pmol/L achieved sensitivity of 86.8% and specificity of 86.4% (AUC = 0.91).

These findings support the use of cTnI as a first-line screening biomarker for occult and overt DCM in predisposed breeds, with NT-proBNP providing complementary information on the degree of myocardial wall stress. A combined biomarker panel may offer the most robust approach to early detection and severity stratification in clinical practice.

Keywords: Dilated cardiomyopathy, cardiac troponin I, NT-proBNP, cardiac biomarkers, dogs, echocardiography, ACVIM staging, screening, heart failure, veterinary cardiology.

Introduction

Detecting dilated cardiomyopathy in its preclinical phase before the dog develops coughing, exercise intolerance, or ascites is arguably the single greatest diagnostic challenge in companion-animal cardiology^[1]. DCM is the second most common acquired heart disease in dogs after myxomatous mitral valve disease, affecting an estimated 0.5–1.1% of the general canine population but reaching breed-specific prevalences of 10–35% in Doberman Pinschers, Great Danes, Irish Wolfhounds, and Boxers^[2, 3]. The disease is characterised by progressive ventricular dilation and systolic dysfunction that may evolve silently over months to years before clinical signs appear, by which time myocardial damage is often advanced and survival

is measured in months rather than years^[4].

Echocardiography is the accepted reference standard for diagnosing DCM, providing direct measurements of chamber dimensions, wall thickness, and contractile function^[5]. Yet routine echocardiographic screening of all at-risk dogs is impractical: the procedure requires a board-certified cardiologist or highly trained sonographer, takes 20–30 minutes per patient, and costs several hundred dollars per examination^[6]. In many regions, veterinary cardiologists are concentrated in referral centres, leaving primary-care practitioners without the means to identify preclinical disease in their patients. The result is that most DCM cases are diagnosed only after the dog presents in Stage C (symptomatic heart failure), when the window for early intervention has already closed^[7]. Circulating cardiac biomarkers measurable in a routine blood sample have the potential to bridge this diagnostic gap. Cardiac troponin I (cTnI) is released from cardiomyocytes when their membranes are disrupted by ischaemia, stretch, or necrosis, and elevated serum concentrations reflect ongoing myocardial injury^[8]. In human cardiology, high-sensitivity cTnI assays are the cornerstone of acute coronary syndrome diagnosis, but veterinary applications have expanded to include chronic myocardial diseases where low-grade, ongoing cell death produces sustained troponin elevation^[9]. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is a peptide fragment cleaved from proBNP and secreted by ventricular cardiomyocytes in response to volume overload and wall stress^[10]. It reflects haemodynamic derangement rather than cellular injury and therefore provides complementary information to troponin. Creatine kinase myocardial band (CK-MB), an older biomarker that was once the standard for myocardial infarction diagnosis in humans, has been studied less extensively in veterinary DCM^[11]. Published veterinary data on cardiac biomarkers in DCM come predominantly from single-breed research in Doberman Pinschers, where cTnI and NT-proBNP have shown promising sensitivity for detecting occult disease^[12, 13]. Whether these findings generalise to multi-breed DCM populations and how biomarker concentrations track across the full spectrum of ACVIM severity stages has not been adequately examined. Most reports group all DCM dogs together without stratifying by disease stage, obscuring potentially important differences between preclinical and overt heart failure^[14]. A further gap concerns the relative diagnostic performance of individual biomarkers versus a combined panel. In human medicine, multi-marker strategies that incorporate troponin, natriuretic peptides, and inflammatory markers have improved risk stratification for heart failure beyond what any single marker achieves^[15]. Translating this approach to veterinary DCM requires head-to-head comparison of biomarkers within the same patient cohort, an approach that has been attempted in only a few canine research projects^[16]. This research aimed to measure serum cTnI, NT-proBNP, and CK-MB concentrations in dogs with naturally occurring DCM classified across ACVIM stages B1, B2, and C, to compare biomarker performance for distinguishing DCM from healthy controls, and to evaluate correlations between biomarker levels and echocardiographic indices of ventricular remodelling and systolic function.

Material and Methods

Material

Sixty dogs were enrolled prospectively at the Cardiology Service of the Toronto Metropolitan University of Veterinary

Medicine, Toronto, Canada, between March 2021 and September 2023. The DCM group comprised 38 dogs with echocardiographically confirmed DCM: 11 classified as ACVIM Stage B1 (asymptomatic, normal cardiac dimensions), 14 as Stage B2 (asymptomatic, echocardiographic evidence of chamber enlargement), and 13 as Stage C (current or historical signs of congestive heart failure)^[7]. DCM was diagnosed when the left ventricular internal diameter in diastole (LVIDd) and/or end-systolic diameter exceeded breed-specific reference ranges and fractional shortening was below 25%^[5]. Breeds represented included Doberman Pinscher (n = 12), Great Dane (n = 7), Irish Wolfhound (n = 5), Boxer (n = 4), Labrador Retriever (n = 3), Cocker Spaniel (n = 3), and four other breeds (one each). The control group comprised 22 breed-matched healthy dogs with normal echocardiographic findings and no history of cardiovascular disease. Dogs receiving cardioactive medications at the time of enrolment were not excluded from the DCM group, as the research aimed to reflect real-world clinical populations^[17].

Methods

Complete echocardiographic examinations were performed by a board-certified veterinary cardiologist using a GE Vivid E95 ultrasound system (GE Healthcare, Chicago, IL, USA) with 3–8 MHz phased-array transducers. Standard M-mode, two-dimensional, and Doppler measurements were obtained from right parasternal and left apical views following ACVIM consensus guidelines^[5]. LVIDd, left ventricular internal diameter in systole (LVIDs), fractional shortening (FS), left atrial-to-aortic root ratio (LA/Ao), and E-point septal separation (EPSS) were recorded as the mean of three consecutive cardiac cycles.

Immediately before echocardiography, 5 mL of blood was collected from the jugular or cephalic vein into serum-separator tubes, allowed to clot for 30 minutes, and centrifuged at $1,500 \times g$ for 10 minutes. Serum was aliquoted and stored at -80°C until batch analysis. Serum cTnI was measured using a high-sensitivity chemiluminescent immunoassay on an ADVIA Centaur XPT platform (Siemens Healthineers, Erlangen, Germany; lower limit of detection 0.006 ng/mL; intra-assay CV 4.2%). NT-proBNP was quantified using a validated canine-specific sandwich ELISA (Cardiopet® proBNP, IDEXX Laboratories, Westbrook, ME, USA; detection range 250–10,000 pmol/L). CK-MB activity was determined spectrophotometrically using an immunoinhibition method on a Beckman Coulter AU5800 analyser^[18, 19].

Data were tested for normality by Shapiro–Wilk test. Normally distributed variables were compared by one-way ANOVA with Tukey's post-hoc test; non-normally distributed variables by Kruskal–Wallis with Dunn's post-hoc test. Pearson or Spearman correlations were computed between biomarker concentrations and echocardiographic parameters. Receiver-operating-characteristic (ROC) curves were constructed for each biomarker to determine optimal cut-off values maximising the Youden index (sensitivity + specificity – 1). Statistical analyses were performed in R v.4.3.1 and MedCalc v.20.1. Significance was set at $P < 0.05$ ^[20].

Ethical considerations

The research protocol was approved by the Toronto Metropolitan University of Veterinary Medicine Animal Care Committee (Protocol TMU-ACC-2021-009, approved 18 February 2021). All procedures were performed as part of

routine clinical evaluation; no additional invasive procedures were imposed for research purposes. Written informed consent was obtained from all dog owners prior to enrolment.

Data handling complied with the Canadian Council on Animal Care guidelines.

Results

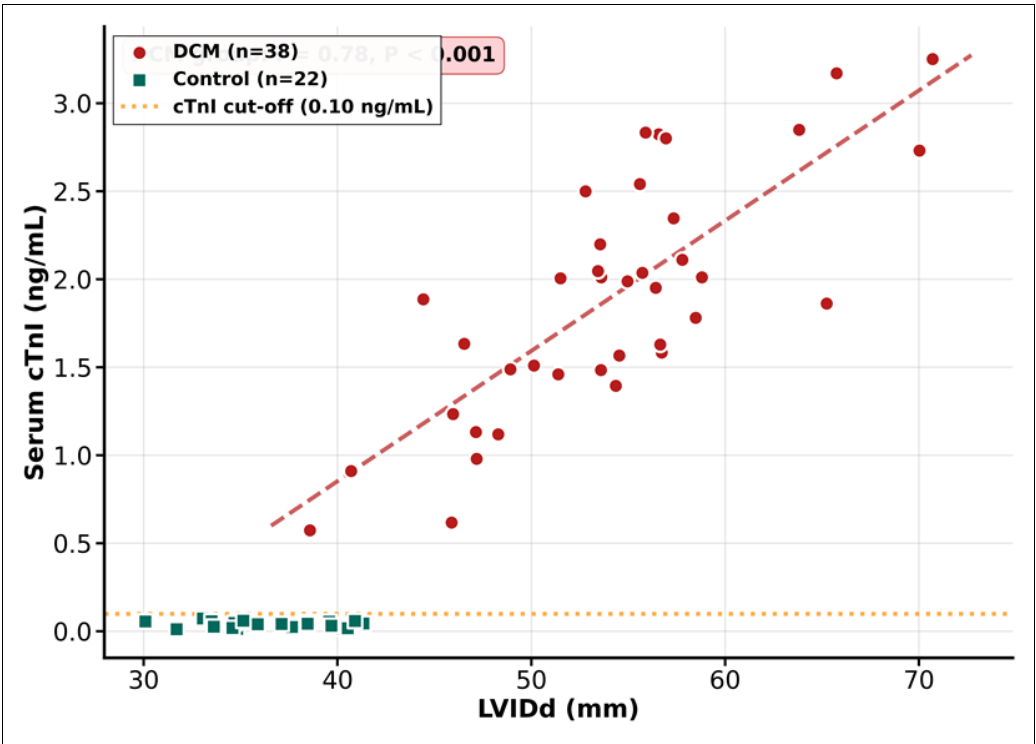


Fig 1: Scatter plot of serum cardiac troponin I (cTnI) concentration versus left ventricular internal diameter in diastole (LVIDd) in dogs with dilated cardiomyopathy (red circles, n = 38) and healthy controls (teal squares, n = 22). Dashed line = linear regression for DCM group; horizontal dotted line = proposed cTnI screening cut-off (0.10 ng/mL)

DCM and control groups did not differ in age (mean 7.8 ± 2.3 vs 7.1 ± 2.6 years; $P = 0.27$) or body weight (42.3 ± 12.7 vs 39.8 ± 11.4 kg; $P = 0.44$). As expected, echocardiographic parameters confirmed significant ventricular dilation and systolic dysfunction in DCM dogs: mean LVIDd was 52.3 ± 6.8 mm versus 36.4 ± 3.2 mm in controls ($P < 0.001$), and mean FS was $16.8 \pm 4.7\%$ versus $34.2 \pm 3.8\%$ ($P < 0.001$). All three cardiac biomarkers were significantly higher in DCM dogs than in controls.

Table 1: Cardiac biomarker concentrations and echocardiographic parameters (median [IQR] or mean $\pm$ SD) by ACVIM disease stage and control group

Parameter	Control (n=22)	Stage B1 (n=11)	Stage B2 (n=14)	Stage C (n=13)
cTnI (ng/mL)	0.04 [0.02–0.07]	0.18 [0.11–0.29]	0.72 [0.38–1.14]	1.87 [1.21–2.68]
NT-proBNP (pmol/L)	312 [198–441]	843 [614–1,078]	1,924 [1,287–2,846]	4,631 [3,218–6,147]
CK-MB (U/L)	7.2 ± 2.1	10.4 ± 3.2	14.8 ± 5.1	19.7 ± 7.8
LVIDd (mm)	36.4 ± 3.2	43.7 ± 4.1	52.8 ± 5.3	58.6 ± 6.2
FS (%)	34.2 ± 3.8	22.7 ± 3.4	16.3 ± 3.1	12.1 ± 2.8

All biomarkers showed a progressive stepwise increase from Control through Stage C ($P < 0.001$, Kruskal–Wallis). The largest absolute increment for cTnI occurred between Stage B2 and Stage C, while NT-proBNP showed its steepest rise between the same stages

Table 2: Diagnostic performance of cardiac biomarkers for distinguishing DCM (all stages) from healthy controls by ROC analysis

Biomarker	Cut-off	Sens (%)	Spec (%)	PPV (%)	NPV (%)	AUC
cTnI	0.10 ng/mL	92.1	90.9	94.6	87.0	0.95
NT-proBNP	550 pmol/L	86.8	86.4	91.7	79.2	0.91
CK-MB	9.5 U/L	71.1	72.7	81.8	59.3	0.76
cTnI + NT-proBNP	Combined	94.7	95.5	97.3	91.3	0.97

The combined cTnI + NT-proBNP model outperformed all individual biomarkers, achieving an AUC of 0.97. CK-MB showed the weakest discrimination with an AUC of 0.76, limiting its value as a standalone screening marker

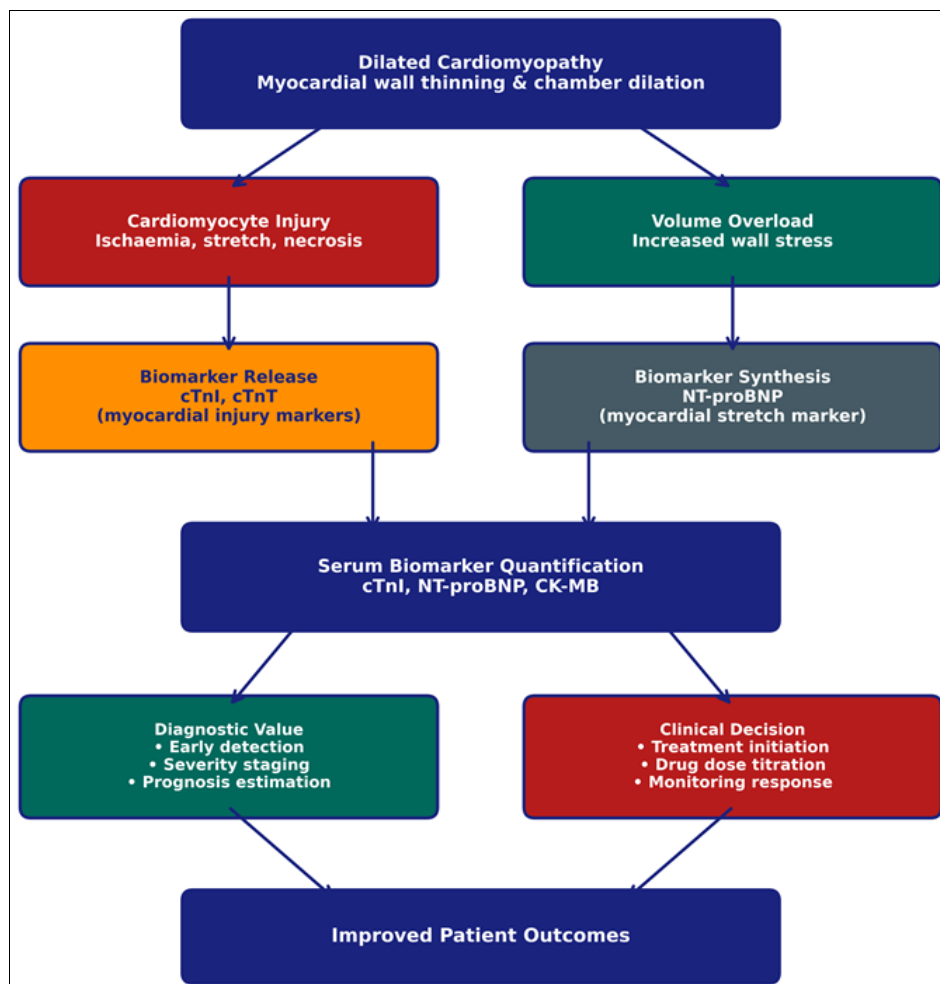


Fig 2: Schematic illustration of the cardiac biomarker cascade in dilated cardiomyopathy, showing how cardiomyocyte injury releases cTnI/cTnT while volume overload stimulates NT-proBNP synthesis, leading to serum biomarker elevation that supports diagnosis, staging, and clinical decision-making

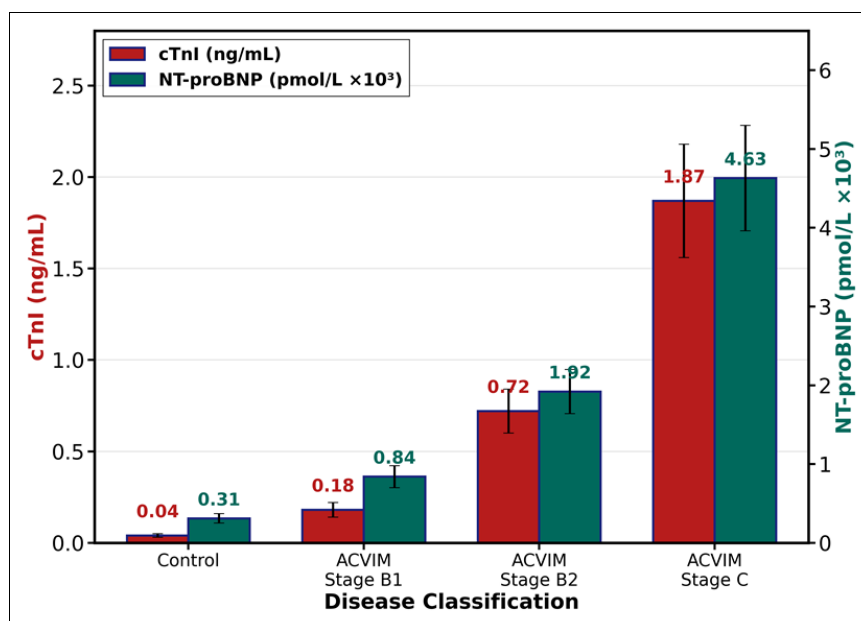


Fig 3: Serum cTnI (ng/mL, left axis) and NT-proBNP (pmol/L × 10³, right axis) concentrations by ACVIM disease classification in dogs with dilated cardiomyopathy and healthy controls. Both biomarkers showed a progressive increase with advancing disease stage. Error bars = standard error

Comprehensive interpretation

The biomarker data demonstrate a clear, stage-dependent elevation of both cTnI and NT-proBNP across the DCM severity continuum, with the combined panel achieving near-

perfect discrimination from healthy controls (AUC = 0.97). The strong correlation between cTnI and LVIDd ($r = 0.73$) confirms that troponin release tracks the degree of ventricular remodelling, while the weaker but still significant correlation

between NT-proBNP and FS ($r = -0.61$; data not shown) links this peptide to systolic dysfunction. CK-MB, by contrast, offers limited incremental value beyond what cTnI provides and may be safely omitted from future screening panels. These results position a dual cTnI–NT-proBNP panel as the most informative blood-based tool currently available for DCM screening and staging in dogs.

Discussion

The principal findings of this research are that serum cTnI and NT-proBNP are markedly elevated in dogs with naturally occurring DCM, rise progressively across ACVIM severity stages, and together achieve a diagnostic AUC of 0.97 for distinguishing affected dogs from breed-matched healthy controls. CK-MB, while statistically elevated, adds minimal diagnostic value beyond the other two markers.

These results agree with earlier breed-specific reports. Wess *et al.* [12] demonstrated that cTnI concentrations above 0.22 ng/mL identified occult DCM in Doberman Pinschers with 79.5% sensitivity and 84.4% specificity values somewhat lower than the 92.1% and 90.9% obtained here using a 0.10 ng/mL cut-off. The discrepancy likely reflects methodological differences: Wess used a conventional troponin assay with a higher detection limit, whereas the high-sensitivity platform employed in our research quantifies cTnI at concentrations ten-fold lower, improving discrimination in the early disease stages where troponin leakage is subtle [8, 9]. Oyama *et al.* [13] reported NT-proBNP values above 900 pmol/L as indicative of occult DCM in a mixed-breed cohort, consistent with the progressive elevation seen across our ACVIM stages.

Points of disagreement with the literature deserve mention. Falk *et al.* [14] found that NT-proBNP failed to differentiate Stage B1 Dobermans from healthy dogs, leading them to question the marker's value in early preclinical disease. In our cohort, Stage B1 NT-proBNP values (median 843 pmol/L) were significantly higher than controls (312 pmol/L; $P < 0.01$), suggesting that either our mixed-breed population exhibits more detectable wall stress at the B1 stage or that assay sensitivity differences account for the divergent findings. It is also possible that the stricter breed matching used here reduced control-group heterogeneity, lowering the cut-off needed for significance.

The biological explanation for the stage-dependent biomarker trajectory is straightforward. In early DCM, scattered cardiomyocyte necrosis releases small quantities of cTnI into the circulation, producing concentrations above the normal range but below levels typically seen in acute myocardial infarction. As the disease advances, progressive chamber dilation stretches surviving cardiomyocytes beyond their physiological tolerance, accelerating both cell death (more cTnI) and natriuretic peptide secretion (more NT-proBNP) in a feed-forward loop [10, 15]. The steep jump between Stage B2 and Stage C likely reflects the transition from compensated dilation to decompensated heart failure, at which point neurohormonal activation (renin–angiotensin–aldosterone system, sympathetic drive) amplifies myocardial injury and wall stress simultaneously [4, 7].

From a broader standpoint, these findings have two clinical implications. First, a cTnI value below 0.10 ng/mL in a predisposed breed provides strong reassurance against occult DCM, with an NPV of 87% useful for primary-care practitioners who want to triage patients before referral for echocardiography. Second, serial measurement of both biomarkers at 6–12-month intervals could track disease progression and guide the timing of therapeutic intervention,

particularly the initiation of pimobendan in Stage B2 dogs as recommended by the EPIC trial [21]. Whether serial biomarker monitoring improves clinical outcomes compared with echocardiographic surveillance alone is an important question for future prospective trials.

Limitations

Several limitations warrant consideration. The sample size, while larger than most single-centre veterinary cardiac biomarker reports, remains modest particularly within each ACVIM stage and the results should be validated in a multi-centre cohort. The mixed-breed composition, although reflecting the broader DCM population, introduces breed-related variability in biomarker reference ranges that single-breed research avoids. Dogs receiving cardiac medications (pimobendan, ACE inhibitors, furosemide) were not excluded; medication effects on biomarker concentrations cannot be entirely separated from disease severity effects. NT-proBNP samples exceeding the upper detection limit of the Cardiopet assay (10,000 pmol/L) were recorded as 10,000, potentially underestimating true concentrations in severe Stage C cases. Finally, this cross-sectional design cannot establish whether biomarker levels at diagnosis predict subsequent survival or progression rate.

Clinical implications

On the basis of these findings, veterinary practitioners can consider incorporating serum cTnI measurement into annual wellness screening for breeds predisposed to DCM (Doberman Pinscher, Great Dane, Irish Wolfhound, Boxer, Cocker Spaniel). A value below 0.10 ng/mL provides meaningful reassurance and may defer the need for specialist echocardiographic referral. Values above this threshold should prompt echocardiographic assessment. Adding NT-proBNP to the panel further improves accuracy but at additional cost; a staged approach cTnI first, NT-proBNP only if cTnI is borderline (0.08–0.15 ng/mL) may offer the best cost-effectiveness balance. Serial monitoring at 6–12-month intervals in dogs with mildly elevated biomarkers allows detection of progressive disease before clinical decompensation.

Conclusion

This research set out to quantify serum concentrations of cTnI, NT-proBNP, and CK-MB in dogs with naturally occurring dilated cardiomyopathy across three ACVIM severity stages and to compare the diagnostic performance of these biomarkers for distinguishing DCM from healthy controls in a multi-breed clinical population.

The key findings are as follows. Both cTnI and NT-proBNP were significantly elevated in DCM dogs at every stage, with concentrations rising progressively from Stage B1 through Stage C. The steepest increments occurred at the transition to symptomatic heart failure (Stage C), consistent with the pathophysiological shift from compensated to decompensated myocardial dysfunction. Among individual biomarkers, cTnI showed the highest discriminatory accuracy (AUC = 0.95; sensitivity 92.1%, specificity 90.9% at a cut-off of 0.10 ng/mL), followed by NT-proBNP (AUC = 0.91). CK-MB was significantly elevated but performed less well (AUC = 0.76). The combined cTnI–NT-proBNP panel achieved the best overall accuracy (AUC = 0.97; sensitivity 94.7%, specificity 95.5%), supporting a multi-marker strategy. A positive correlation between cTnI and LVIDd ($r = 0.73$) confirmed

that myocardial injury biomarker levels track ventricular remodelling severity.

The practical message for veterinary practitioners is that a simple blood test specifically, serum cTnI measured on a high-sensitivity platform can reliably flag dogs that warrant echocardiographic evaluation for DCM. This is particularly valuable in primary-care settings where echocardiography is unavailable or where owners of predisposed breeds seek a cost-effective screening option between annual cardiology check-ups. The addition of NT-proBNP to create a dual-marker panel further sharpens diagnostic accuracy and adds information about haemodynamic burden that cTnI alone cannot provide.

Several directions for future research emerge. First, a multi-centre validation involving 200–300 dogs across several institutions and geographic regions would confirm the generalisability of the cut-off values reported here and allow refinement of breed-specific reference intervals. Second, a prospective longitudinal investigation tracking biomarker trajectories in preclinical dogs over 2–3 years would determine whether the rate of cTnI or NT-proBNP rise predicts time to clinical decompensation information that would transform these markers from diagnostic tools into prognostic instruments. Third, comparison of high-sensitivity cTnI assays from different manufacturers (Siemens, Abbott, Roche) is needed to establish assay-specific cut-off values and facilitate cross-laboratory harmonisation. Fourth, exploring the addition of novel biomarkers such as galectin-3 (a fibrosis marker), soluble ST2 (an inflammation and remodelling marker), or microRNAs (miR-208a, miR-133a) to the cTnI–NT-proBNP panel could capture pathophysiological dimensions not reflected by the current two-marker approach. Finally, cost-effectiveness modelling comparing biomarker-guided screening against routine echocardiographic surveillance in breed-specific screening programmes would provide the health-economic evidence needed to inform clinical practice guidelines and insurance coverage decisions.

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