Hypertension-driven cardiac remodeling in feline CKD: diagnostic utility of cTnI/creatinine and NT-proBNP/creatinine ratios

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ABSTRACT

The objective of this study was to evaluate cardiac remodeling and the diagnostic utility of cardiac biomarkers, cardiac troponin I (cTnI) and N-terminal pro–B-type natriuretic peptide (NT-proBNP), in cats with chronic kidney disease (CKD), with or without systemic hypertension. The study population included 11 healthy controls, 11 normotensive cats with chronic kidney disease (CKD), and 16 hypertensive cats with CKD. All cats underwent a comprehensive evaluation including blood pressure measurement, serum biochemistry, biomarker analysis, and echocardiographic assessment. CKD cats exhibited significantly elevated serum concentrations of cTnI and NT-proBNP compared to the control group, with hypertensive cats demonstrating the highest levels of these markers (P<0.001). Of notable significance is the finding that only the hypertensive CKD group demonstrated significantly elevated biomarker-to-creatinine ratios, supporting the presence of true myocardial injury rather than impaired renal clearance alone. Echocardiographic assessment revealed increased left atrial to aortic root (LA/Ao) ratios, left ventricular posterior wall thickness in diastole (LVPWd), and interventricular septal thickness in diastole (IVSd) in hypertensive CKD cats, indicating structural cardiac remodeling. Elevated serum urea and phosphorus levels in cats with CKD may also contribute to these myocardial alterations. These findings suggest that cats with hypertensive CKD experience actual myocardial injury, which is reflected in structural changes and biomarker ratios, while cats with normotensive CKD show milder and less definitive alterations. The incorporation of biomarker-to-creatinine ratios with echocardiography has the potential to enhance the early detection of subclinical cardiac injury in hypertensive CKD cats, thereby supporting the timely implementation of cardio-protective strategies.

Introduction

Chronic kidney disease (CKD) is one of the most common disorders in aging cats, characterized by progressive loss of renal function and associated systemic complications (7, 26). Although traditionally regarded as a renal-specific condition, CKD is now recognized as a systemic disease with significant cardiovascular implications (1, 15). Cardiovascular complications, particularly systemic hypertension, are frequently observed in cats with CKD and are associated with increased risks of left ventricular hypertrophy (LVH) (12), diastolic dysfunction (9), and overall cardiovascular morbidity (27).

The pathophysiology of cardiovascular involvement in CKD is multifactorial, involving complex interactions between hypertension, anemia, endothelial dysfunction, inflammation, oxidative stress, uremic toxins, and disturbances in mineral metabolism (11, 13, 27). These factors contribute to cardiac remodeling, myocardial

injury, and functional impairment, emphasizing the need for early detection and monitoring of cardiac involvement in CKD patients (21, 33).

Cardiac biomarkers, including N-terminal pro-brain natriuretic peptide (NT-proBNP) and cardiac troponin I (cTnI), have become standard in both general and referral practices for evaluating myocardial stress and injury (6, 24). NT-proBNP, secreted by ventricular myocytes in response to volume and pressure overload as an indicator of myocardial stretch and hypertrophy, has been consistently reported to be significantly elevated in cats with heart disease across all studies (19, 20, 31). Elevated NT-proBNP levels are associated with adverse cardiovascular outcomes, including heart failure and mortality (4). Similarly, cTnI, a structural component of the cardiac contractile apparatus, is released into circulation following myocardial injury or remodeling, providing a sensitive and specific marker for cardiac damage (14, 24). In cats, multiple studies have shown that circulating troponin concentrations are significantly elevated in those with hypertrophic cardiomyopathy (HCM), including asymptomatic individuals (17, 18, 29). However, in CKD, the interpretation of these biomarkers is complicated by reduced renal clearance, which can lead to elevated levels independent of overt cardiac pathology (3, 14, 28).

Despite the availability of echocardiography as a diagnostic tool for evaluating cardiac structure and function, its routine application in asymptomatic cats with CKD may be limited due to practical and financial constraints. This underscores the importance of identifying accessible biomarkers to detect subclinical cardiac changes early in feline CKD patients. While the prognostic utility of NT-proBNP and troponins has been well established in human CKD populations, data on their relevance in feline CKD remain scarce.

The objective of this study was to evaluate serum NT-proBNP and cTnI concentrations, alongside echocardiographic parameters, in healthy cats, normotensive cats with CKD, and hypertensive cats with CKD. The study aimed to investigate whether elevations in cardiac biomarkers are associated with systemic hypertension and cardiac structural changes in cats with CKD.

Materials and Methods

This retrospective observational study included feline patients presented to the International Veterinary Hospital, Antalya between January 2022 and October 2024. Prior to the study, a power analysis was conducted using a power of 0.80 (1-β), a type I error rate (α) of 0.05, and an effect size (f) of 0.58 (20), which indicated a requirement of 33 animals, with 11 per group. However, to account for potential subject loss, all available data were utilized. As a result, a total of 38 client-owned cats were enrolled, including 27 diagnosed with chronic kidney disease (CKD) -comprising normotensive CKD (n = 11) and hypertensive CKD (n = *16)*- and 11 clinically healthy cats serving as controls. To be eligible, cats had to be diagnosed with CKD. CKD staging was performed according to the International Renal Interest Society (IRIS) guidelines. Cats with serum creatinine levels between 2.9 and 5.0 mg/dL were classified as stage 3 CKD, while those with creatinine levels exceeding 5.0 mg/dL were categorized as stage 4. The diagnosis of CKD was based on persistently elevated serum creatinine concentrations and inadequate urine concentrating ability (urine specific gravity <1.035) documented for at least one month prior to enrollment, under IRIS guidelines.

All cats underwent a comprehensive clinical evaluation, including systolic blood pressure (SBP) measurement, transthoracic echocardiography, and routine diagnostic blood tests. Systolic blood pressure was measured non-invasively using a Doppler ultrasonic flow detector, following the ACVIM consensus guidelines (1). Five consecutive measurements were obtained, and the mean value was used for analysis. Systemic hypertension was defined as SBP >160 mmHg on at least two separate occasions.

Based on renal status and SBP values, cats were stratified into three groups: healthy controls (n = 11), normotensive CKD (n = 11), and hypertensive CKD (n = 16). Inclusion criteria for cats with CKD were as follows:1-A transthoracic echocardiographic examination performed within the previous six months, demonstrating no structural or functional cardiac abnormalities (e.g., absence of left ventricular hypertrophy, systolic dysfunction, significant valvular disease, or pericardial effusion). 2-No clinical signs suggestive of cardiac disease, such as exercise intolerance, coughing, syncope, heart murmurs, or arrhythmias, within the past six months. 3-A systolic blood pressure measurement below 160 mmHg at the time of inclusion for normotensive CKD cats. 4-Cats were excluded if they had polycystic kidney disease or concurrent systemic conditions that could independently affect cardiac or renal function (e.g., hyperthyroidism, neoplasia).

Echocardiographic Examination: Echocardiographic evaluations were performed using a Siemens Juniper ultrasound system equipped with a 7 MHz phased-array transducer (frequency range: 2,7–8 MHz). All cats were gently restrained in right and left lateral recumbency without sedation to obtain standard echocardiographic views.

Left ventricular dimensions were measured from two-dimensional right parasternal long-axis and short-axis views. The thickest regions of the ventricular walls were assessed using the leading-edge-to-leading-edge method. For each parameter, measurements were averaged over at least three cardiac cycles and represented the mean of two

independent observers (IB and ST) to ensure consistency and accuracy. Left atrial size was assessed by calculating the left atrial-to-aortic root (LA/Ao) ratio, using the inner-edge-to-inner-edge method from the right parasternal short-axis view at the level of the aortic valve.

Cats with systemic hypertension were managed with amlodipine™, with the dose adjusted between 0.625 and 1.25 mg per cat per day to achieve a target systolic blood pressure (SBP) below 165 mm Hg. The hypertensive group consisted of cats whose owners were unable to implement or maintain appropriate treatment for systemic hypertension, as reported in the anamnesis.

Sample Collection and Measurement of Natriuretic Peptides: Blood samples were obtained via jugular venipuncture from all cats at the time of enrollment. Hematological and biochemical analyses were performed immediately following collection to assess renal function and other relevant parameters. Serum concentrations of feline cTnI and feline NT-proBNP were measured using commercially available assay kits (Vcheck, BIONOTE, Korea), in accordance with the manufacturers' protocols.

Statistical Analysis: Descriptive statistics were computed for each variable. Before conducting hypothesis testing, the data were assessed using the Shapiro-Wilk test for normality and the Levene test for homogeneity of variances, in accordance with the assumptions of parametric tests. One-way analysis of variance (ANOVA) was employed for data meeting parametric test assumptions to assess group differences, whereas the Kruskal-Wallis test was utilized for variables that contravene the assumptions of parametric distribution. Gabriel and Dunn-Bonferroni tests were employed as post hoc testing procedures. Pearson's Chi-square test was employed to analyze the frequency distribution of categorical data. A P value of less than 0.05 was considered statistically significant. All statistical analyses were conducted using SPSS 30.

Results

The study included 38 cats: 11 healthy controls, 11 with normotensive CKD, and 16 with hypertensive CKD. The demographic characteristics of the study population are presented in Table 1. No significant variations in age and sex have been identified across the three groups (P>0.05).

According to the IRIS classification, 3 of 27 CKD cats (11 %) were stage 3, one normotensive and two hypertensives, whereas 24 cats (89 %) were stage 4, comprising ten normotensive and fourteen hypertensive individuals.

Urine specific gravity (USG) differed significantly among the groups (P<0.001), with healthy controls exhibiting well concentrated urine (1.05 ± 0.007) while normotensive (1.021 ± 0.004) and hypertensive CKD cats (1.017 ± 0.002) had inadequately concentrated urine the two CKD subgroups did not differ in post hoc comparison (Table 2).

Clinicopathologic variables for healthy, normotensive CKD, and hypertensive CKD cats are summarized in Table 2. Both the normotensive and hypertensive CKD groups exhibited significantly higher serum urea, creatinine, and phosphorus levels compared to the control group (P<0.001).

Regarding cardiac biomarkers, serum cTnI concentrations were significantly elevated in both the normotensive CKD group (0.58 ± 0.68 ng/mL) and the hypertensive CKD group (1.96 ± 2.79 ng/mL) when compared to the control group (below detection limits) (P<0.001) (Figure 1). Similarly, NT-proBNP concentrations were significantly higher in the normotensive CKD group (215.56 ± 140.78 pg/mL) and the hypertensive CKD group (1268.79 ± 306.09 pg/mL) relative to the control group (53.18 ± 7.17 pg/mL) (P<0.001) (Figure 2). Additionally, serum cTnI and NT-proBNP levels were significantly higher in the hypertensive CKD group compared to the normotensive CKD group (P<0.001).

Table 1. Descriptive statistics regarding demographic characteristics of the study population

			Group			
			Normotensive CKD (n=11)	Hypertensive CKD (n=16)	Control (n=11)	P
Age (years)		Mean ± SD	8 ± 2.32	7.5 ± 2.16	7.64 ± 2.62	0.860 [x]
		Median (Range)	8 (7)	7.5 (7)	7 (8)	
Sex	Female	n (%)	5 (45.5%)	7 (43.8%)	6 (54.5%)	0.849 [y]
	Male	n (%)	6 (54.5%)	9 (56.3%)	5 (45.5%)	

x: One way ANOVA, y: Pearson Chi Square Test

Table 2. Clinicopathologic variables for healthy (control), Normotensive CKD and Hypertensive CKD cats

Traits	Normotensive CKD (n=11)		Hypertensive CKD (n=16)		Control (n=11)		
	Mean ± SD	Median (Range)	Mean ± SD	Median (Range)	Mean ± SD	Median (Range)	P
Urea (mg/dl)	253.7 ± 125.87	250.52 (474.19) [a]	335.64 ± 190.31	285.3 (596.98) [a]	50.11 ± 7.89	52 (22) [b]	<0.001 [y]
Creatinine (mg/dl)	5.84 ± 1.67	5.2 (5.7) [a]	7.93 ± 4.24	6.58 (17.38) [a]	1.19 ± 0.17	1.27 (0.5) [b]	<0.001 [y]
Phosphorus	11.48 ± 5.78	9 (17.66) [a]	12.42 ± 7.86	10.65 (29.95) [a]	5.15 ± 0.87	5.1 (3.36) [b]	<0.001 [y]
Albumin (mg/dl)	3.21 ± 0.53	3.2 (1.8)	2.89 ± 0.61	2.67 (2.4)	3.18 ± 0.51	3.1 (1.6)	0.268 [x]
Nt-pro bnp (pmol/L)	215.56 ± 140.78	192.7 (394) [b]	1268.79 ± 306.09	1500 (785.7) [a]	53.18 ± 7.17	50 (20) [c]	<0.001 [y]
Troponin-I (ng/mL)	0.58 ± 0.68	0.4 (1.89) [b]	1.96 ± 2.79	1.1 (11.98) [a]	0.03 ± 0.02	0.03 (0.07) [c]	<0.001 [y]
LVPWd (mm)	4.84 ± 0.68 [b]	5 (2.4)	6.04 ± 1.42 [a]	6 (6.2)	4.02 ± 0.31 [b]	4 (1)	<0.001 [x]
IVSd (mm)	4.57 ± 0.65 [b]	4.8 (2)	5.92 ± 1.38 [a]	6.05 (5.5)	4.29 ± 0.35 [b]	4.2 (1.1)	<0.001 [x]
FS (%)	55.09 ± 8.8 [b]	53 (26)	62.75 ± 6.78 [a]	65 (25)	51 ± 6.07 [b]	52 (16)	0.001 [x]
SBP (mm Hg)	140.07 ± 9.97 [b]	139 (32.2)	175.73 ± 6.9 [a]	177.7 (20.4)	135.27 ± 4.47 [b]	136 (14)	<0.001 [x]
LA : Ao	1.22 ± 0.15 [b]	1.2 (0.4)	1.55 ± 0.2 [a]	1.5 (0.7)	1.26 ± 0.08 [b]	1.2 (0.2)	<0.001
RBC	6.86 ± 2.65 [ab]	6.73 (8.59)	6.1 ± 1.82 [b]	5.97 (6.8)	8.9 ± 1.62 [a]	9.22 (5.3)	0.005 [x]
HGB	9.4 ± 2.63 [b]	10 (8.7)	9.09 ± 3.14 [b]	8.4 (10.1)	12.53 ± 2.31 [a]	12.3 (5.9)	0.008 [x]
HCT	26.2 ± 7.66 [b]	23.6 (27.4)	26.41 ± 9.35 [b]	24.65 (31)	36.9 ± 6.39 [a]	37 (15.9)	0.004 [x]
NT-proBNP/ Creatinine ratio	36.65 ± 23.21	29.38 (73.57) [b]	193.03 ± 90.48	181.71 (291.48) [a]	45.33 ± 7.86	48.54 (19.84) [b]	<0.001 [y]
Troponin-I/ Creatinine ratio	0.11 ± 0.14	0.06 (0.38) [b]	0.28 ± 0.34	0.18 (1.44) [a]	0.03 ± 0.02	0.02 (0.05) [b]	<0.001 [y]
USG	1.021 ± 0.004	1.02 (0.01) [b]	1.017 ± 0.002	1.018 (0.008) [b]	1.05 ± 0.007	1.05 (0.022)[a]	<0.001 [y]

[a,b,c]: Different letters in the same row represents statistically significant difference (P<0.05). [x]: One-way analysis of variance (ANOVA), [y]: Kruskal Wallis test. FS, fractional shortening; IVSd, interventricular septal thickness in diastole; LVPWd, left ventricular free-wall thickness in diastole; SBP, systolic blood pressure, USG, urine specific gravity

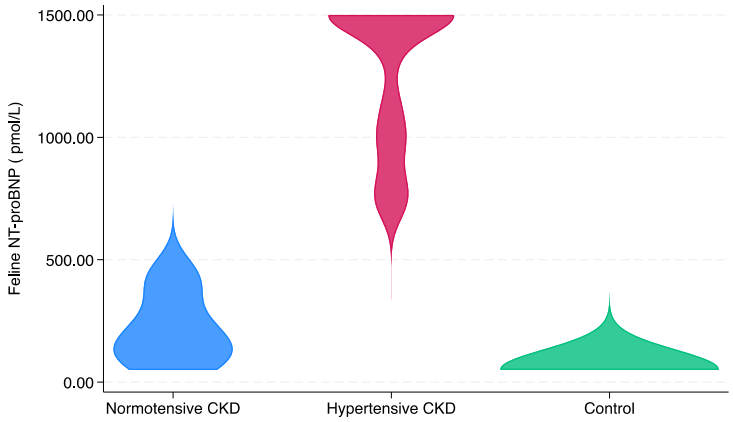

Figure 1. A Joy-plot showing density of serum cardiac troponin I (cTnI) concentrations in healthy cats, (n = 11), cats with normotensive chronic kidney disease (n = 11) and hypertensive chronic kidney disease (n = 16).

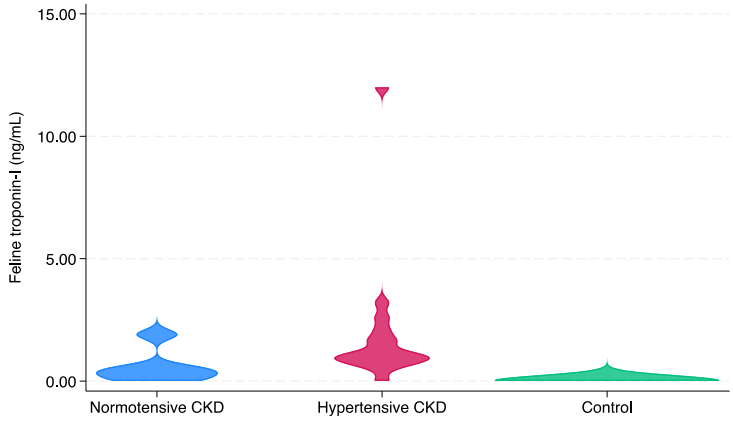

Figure 2. A Joy-plot showing density of serum N-terminal probrain natriuretic peptide (NT-proBNP) concentrations in healthy cats, (n = 11), cats with normotensive chronic kidney disease (n = 11) and hypertensive chronic kidney disease (n = 16).

Both the NT-proBNP/creatinine and cTnI/creatinine ratios were evaluated to assess cardiac biomarker levels relative to renal function in the study groups. The NT-proBNP/creatinine ratio was significantly higher in hypertensive CKD cats (181.71 ± 291.48) compared to normotensive CKD cats (29.38 ± 73.57) and healthy controls (48.54 ± 19.84) ($P<0.05$), while no significant difference was found between the normotensive CKD and control groups. Similarly, the cTnI/creatinine ratio was significantly elevated in hypertensive CKD cats (0.18 ± 1.44) relative to both normotensive CKD cats (0.06 ± 0.38) and healthy controls (0.02 ± 0.05) ($P<0.05$). No significant difference was observed between the normotensive CKD and control groups for the cTnI/creatinine ratio.

Echocardiographic assessments revealed that the LA/Ao ratio was significantly increased in the hypertensive CKD group compared to both the normotensive CKD and control groups ($P<0.001$). Similarly, left ventricular posterior wall thickness in diastole (LVPWd) and interventricular septal thickness in diastole (IVSd) were significantly greater in the hypertensive CKD group (6.04 ± 1.42 mm) compared to the normotensive CKD (4.84 ± 0.68 mm) and control groups (4.02 ± 0.31 mm) ($P<0.001$ for both comparisons).

Discussion and Conclusion

In the present study, both normotensive and hypertensive CKD groups exhibited significantly elevated serum cTnI and NT-proBNP levels compared to healthy controls, with the hypertensive CKD group showing markedly higher values. Additionally, echocardiographic evaluation revealed that LA/Ao ratio, LVPWd, and IVSd were significantly increased in the hypertensive CKD group, indicating more severe structural cardiac remodeling in the presence of systemic hypertension.

The findings of the present study align with those of Lalor et al. (23), who documented significantly increased NT-proBNP levels in hypertensive CKD cats relative to both normotensive CKD and healthy controls. Similarly, our data demonstrated markedly higher serum concentrations of NT-proBNP in hypertensive CKD cats, reflecting increased myocardial stress and injury in association with systemic hypertension. Although NT-CKD cats also exhibited elevated biomarker levels compared to healthy cats, the magnitude of increase was less pronounced than in the hypertensive group, suggesting that hypertension may act as a key driver of cardiac biomarker elevation in feline CKD.

Our findings corroborate and extend the work of Bijsmans et al. (5), who demonstrated that plasma cTnI levels were markedly elevated in hypertensive cats, irrespective of the presence of ocular target organ damage (TOD), as well as in cats with CKD, in comparison to healthy controls. In their study, log-transformed cTnI was a significant positive predictor of systemic hypertension, and ROC analysis suggested a diagnostic cut-off of >0.045 ng/mL for identifying hypertensive cats, with moderate sensitivity and specificity. Notably, cTnI was also predictive of ocular-TOD, reinforcing its utility as a biomarker of hypertensive target organ involvement (5). In contrast to Bijsmans et al. (5), who did not specifically distinguish between hypertensive and normotensive CKD cats in their cTnI analysis, our study revealed a significant difference in cTnI concentrations between these two groups. Hypertensive CKD cats in our cohort exhibited markedly higher cTnI levels compared to their normotensive counterparts, suggesting a stronger association between systemic hypertension and subclinical myocardial injury. Furthermore, echocardiographic findings in the hypertensive CKD group, including increased LA/Ao ratio, LVPWd, and IVSd, indicate more advanced structural cardiac remodeling, likely driven by pressure overload. These results support the hypothesis that systemic hypertension exacerbates cardiac strain in cats with CKD and highlight the value of combining biomarker analysis with echocardiography to improve early identification of cardiovascular involvement in this population.

NT-proBNP is released by cardiac myocytes in reaction to myocardial distension resulting from pressure or volume overload (32). Troponin I, a key regulator of cardiac muscle contraction, is released into the circulation with myocardial injury or structural remodeling (2). Both NT-proBNP and cTnI are widely recognized as reliable biomarkers for the diagnosis and monitoring of left ventricular dysfunction in clinical practice (30). However, in patients with chronic kidney disease, impaired renal clearance may lead to increased circulating levels of NT-proBNP and cTnI even in the absence of overt cardiac pathology. Consequently, the interpretation of these biomarkers in individuals with CKD becomes challenging, as elevations may reflect reduced renal elimination rather than true myocardial injury (10, 30). Although our findings mirror this phenomenon, as both normotensive and hypertensive CKD cats showed elevated cTnI concentrations, the underlying cause of cardiac biomarker elevation in normotensive CKD cats remains unclear. Several mechanisms have been proposed, including uremic cardiomyopathy characterized by LV hypertrophy, fibrosis, and diastolic dysfunction (25) and elevated serum phosphate concentrations have been associated with cardiac remodeling in both human (8, 16) and several animal models (22). In our study, both normotensive and hypertensive CKD cats had increased cTnI concentrations, but the cause in normotensive cats remains unclear. Potential mechanisms include uremic cardiomyopathy and cardiac remodeling associated with

hyperphosphatemia (16, 22, 25). Notably, both serum urea and phosphorus levels were significantly elevated in our CKD groups, suggesting a possible role in biomarker elevation. Further research is needed to differentiate between biomarker accumulation due to renal dysfunction and subclinical myocardial injury.

To further delineate the impact of renal dysfunction from true cardiac pathology, this study evaluated the cTnI/creatinine and NT-proBNP/creatinine ratios—an approach not previously reported in feline studies. These ratios revealed no significant differences between the normotensive CKD and control groups, whereas cats with hypertensive CKD exhibited significantly elevated ratios. This may suggest that hypertension in CKD may drive myocardial injury independent of renal impairment. Supporting this interpretation, echocardiographic findings demonstrated structural cardiac remodeling in hypertensive CKD cats, evidenced by increased LA/Ao ratios, left ventricular posterior wall thickness (LVPWd), and interventricular septal thickness (IVSd). These changes are indicative of hypertensive heart disease and reinforce the relationship between systemic hypertension and adverse cardiac remodeling in cats with CKD.

Despite these important findings, the study has several limitations. Although the minimum sample size required for the study to reach sufficient power was determined prior to the study, it would be beneficial to use a larger sample size to control for additional potential confounding variables and to improve the generalizability of the findings. Furthermore, the relatively small sample size limited our ability to perform a robust stratified analysis of cardiac biomarkers across different CKD stages. Future studies with larger cohorts are warranted to confirm these findings, assess their prognostic significance, and better elucidate the relationship between CKD stage, hypertension, and cardiac biomarkers. While normalization of cTnI to creatinine helps adjust for renal impairment, this approach may not fully account for all potential confounding factors. Moreover, other CKD-related systemic influences, such as uremia or anemia, were not independently evaluated. The cross-sectional nature of the study precludes conclusions regarding causality between biomarker elevations and myocardial injury. Additionally, the absence of longitudinal follow-up limits our understanding of biomarker dynamics and the progression of cardiac remodeling over time.

In conclusion, serum cTnI and NT-proBNP levels were elevated in CKD cats, with the highest values observed in hypertensive individuals. After normalization to creatinine, only hypertensive cats showed significantly increased biomarker ratios, indicating a stronger association with underlying cardiac pathology. These results provide a baseline for the potential use of cTnI/creatinine and NT-proBNP/ creatinine ratios, interpreted alongside cardiac imaging, to discriminate true myocardial injury from elevations driven primarily by reduced renal clearance. Future investigations with larger sample sizes should employ multi-class ROC methodology to define clinically actionable cut-off points and to validate the prognostic value of these ratios.

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Ethical Statement

Ethical approval for the study protocol was obtained from the Animal Research Ethics Committee of Ankara University (2024-19-164).

Conflicts of Interest

The authors declared that there is no conflict of interest

Author Contributions

This study was designed as a retrospective analysis. İB performed the echocardiographic examinations of all included cases. ED and ST collected and organized the clinical data. DÖ conducted the statistical analysis. İB was responsible for echocardiographic data interpretation and contributed to data evaluation. All authors contributed to the writing, critically revised the manuscript, and approved the final version.

Data Availability Statement

The data supporting this study's findings are available from the corresponding author upon reasonable request.

Animal Welfare

The authors confirm that they have adhered to ARRIVE Guidelines to protect animals used for scientific purposes.

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