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RESEARCH ARTICLE

The Diagnostic Value of Soluble Suppression of Tumorigenicity 2 (sST2) in Feline Arterial Thromboembolism: A Comparative Study with Echocardiographic Findings and Cardiac Biomarkers

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A B S T R A C T

Feline arterial thromboembolism (FATE) is a serious complication of cardiomyopathies. Although various cardiac biomarkers are used clinically, the diagnostic value of soluble suppression of tumorigenicity 2 (sST2) in FATE has not yet been established. The aim of this study was to evaluate serum sST2 concentrations in cats diagnosed with FATE and to compare the clinical application of this biomarker with NT-proBNP and cTnI using radiographic and echocardiographic parameters. The study included 20 cats: 10 healthy controls, aged 1-2 years, and 10 cats diagnosed with FATE and aged 4-19 years. All animals underwent clinical, radiographic and echocardiographic examinations; blood hematological and serum biochemical parameters, sST2, cTnI and NT-proBNP levels were also analyzed. Results showed that vertebral heart score (VHS) values were higher in FATE-affected cats (9.75±0.14) than both the normal reference (7.5±0.32) and the healthy control group values (7.63±0.13; $P<0.05$). Echocardiography revealed that the left atrial size and LA/Ao ratio were increased in the FATE group compared to the healthy group ($P<0.05$). Additionally, NT-proBNP and cTnI levels were higher in the FATE group than controls ($P<0.05$). However, no difference was found in serum sST2 levels between the two groups. Blood analyses revealed that WBCs, neutrophils, basophils counts and mean platelets volume, ALT, AST, CK-MB and LDH levels were higher in the FATE group, while RBCs, hemoglobin and hematocrit values were higher in control cats. In conclusion, serum NT-proBNP and cTnI levels are important for diagnosis of FATE, while sST2 alone did not demonstrate significant diagnostic utility in this cohort; however, larger prospective studies are needed to determine the ability of sST2 to reflect cardiac remodeling and its diagnostic significance in feline cardiomyopathy.

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INTRODUCTION

Feline arterial thromboembolism (FATE) is one of the most common causes of sudden hind limb paralysis in cats. FATE is a devastating syndrome associated with high morbidity and mortality in cats; clinically, it usually affects one or more limbs, causing sudden and severe pain, paralysis, and rhabdomyolysis in the affected area, and results in serious short-term consequences (Borgeat *et al.*, 2014; Bakırel *et al.*, 2021; Guillaumin, 2024). In most cases, it is one of the common and fatal complications of cardiomyopathies that progress without clinical symptoms (Fuentes, 2012; Borgeat *et al.*, 2014). It is widely accepted that prior to the occurrence of FATE the clot typically forms on the left side of the heart (Smith and Tobias, 2004). FATE mostly lodges at the aortic bifurcation region, a condition referred to as a "saddle thrombus" (Özlek *et al.*, 2025). An intracavitary thrombus or a fragment of a thrombus that forms in the left atrium or left atrial appendage detaches and enters the systemic circulation, causing an occlusion in a peripheral artery (Pavelková, 2019). This condition leads to hypoperfusion, tissue

ischemia, pain, ischemic-reperfusion injury, systemic inflammation and tissue necrosis (Yeh *et al.*, 2025).

N-terminal pro-brain natriuretic peptide (NT-proBNP) and cardiac troponin I (cTnI) are biomarkers commonly used to assess myocardial damage and cardiac wall stress in cats. It has been reported that levels of these biomarkers are elevated, particularly in cats with hypertrophic cardiomyopathy and in cases developing FATE, and may be associated with disease prognosis (Borgeat *et al.*, 2015; Langhorn and Willesen, 2016). However, there is a need for new biomarkers that reflect the inflammatory and fibrotic mechanisms associated with thromboembolic processes.

Interleukin-33 receptor (ST2) is a member of the interleukin-1 (IL-1) receptor family. Interleukin-33 exerts its effects by binding to the ST2 receptor, which has two isoforms: transmembrane (ST2L) and soluble (sST2). The soluble isoform (sST2) binds directly to IL-33 (Tascon-Cervera *et al.*, 2025). It was first identified in 1989 by Tominaga as a receptor associated with immune and inflammatory diseases (Pascual-Figal and Januzzi, 2015), and its role in the cardiovascular system was determined by Weinberg *et al.* (2002). Memon *et al.* (2018) noted that sST2 is a potential diagnostic and prognostic tool in deep vein thrombosis. Cabrera-Garcia *et al.* (2022) highlighted the role of sST2 as a marker of hypercoagulability and inflammation in COVID-19 patients and reported that sST2 is important for patient monitoring and thrombotic risk assessment. Elevated sST2 levels in heart diseases are associated with fibrosis and adverse clinical outcomes (Uyanık *et al.*, 2024). However, the role of sST2 in arterial thromboembolism in cats has not yet been fully established.

In this study, it was hypothesized that sST2 levels in cats with FATE could serve as a biomarker for the diagnosis of the disease. Therefore, this study was planned to evaluate the additional role of sST2 to existing biomarkers (NT-proBNP, cTnI) in in the diagnosis of FATE and to investigate whether it could serve as a potential biomarker.

MATERIALS AND METHODS

Experimental animals: In this study, 20 cats (10 healthy and 10 with FATE) presented to the Anipoli Veterinary Clinic, Istanbul, Türkiye between December 2022 and December 2024 were included. Age, breed, sex, and for both healthy and FATE cats were recorded. During clinical examination, cats showing acute dyspnea, pain, tachypnea, gallop rhythm, abdominal breathing, and marked numbness and cyanosis in one or both hind limbs were confirmed to be suffering from thromboembolism at the aortic bifurcation by echocardiography (Guillaumin, 2024). The control group consisted of 10 healthy cats which were selected on the basis of normal clinical examination, serum biochemistry, hematology, and echocardiographic findings related to cardiac, renal, and systemic diseases. Cats of this group included 5 females and 5 males aged 1-12 years, belonging to domestic Shorthairs (n=6), Scottish Fold (n=1), Persian (n=2), and British Shorthair (n=1) breeds. The FATE group consisted of 6 males and 4 females aged 4-19 years, including British Shorthair (n=5) and domestic Shorthair (n=5) cats. Healthy cats had an average body weight of 4.57kg (2.8-5.67kg), and cats in the FATE group had an average body weight of 4.53kg (3.0-5.7kg). Cats with renal failure, neoplasia, or other systemic diseases were excluded from the study.

Radiological and echocardiographic examinations: Radiographic examinations were performed on all cats included in the study using a Browner Beatle 05 Vet HF X-ray machine and a Mindray Vetipad M1 Plus DR Detector device; images were obtained in the right latero-lateral and dorsoventral positions. Radiographic findings and vertebral heart scores (VHS) were recorded for all cats. The normal VHS range was accepted as 7.5±0.32, with a range of 6.7-8.1 (Litster and Buchanan, 2000), and higher values indicated cardiac enlargement.

Echocardiographic examinations were performed using a 4-12MHz multi-frequency phased-array probe on a Philips Affiniti 50 (Philips Healthcare, Andover, MA, USA) and a 2-8MHz multi-frequency phased-array probe on Vetus E7 Hand-Carried Veterinary Ultrasound System (Mindray, China). M-mode echocardiography was used to measure left ventricular end-systolic (LVIDs) and end-diastolic (LVIDd) diameters, interventricular septal thickness in systole (IVSs) and diastole (IVSd), and left ventricular free wall thickness in systole (LVPWs) and diastole (LVPWd). Left atrial size was obtained from the right parasternal long-axis four-chamber view. Ejection fraction (EF) and fractional shortening (FS) were automatically calculated, and all measurements were confirmed by at least three repetitions. The left ventricular outflow tract and aorta (Ao) were evaluated in the right parasternal long-axis five-chamber view, and the LA/Ao ratio was measured using M-mode. This measurement was repeated in the right parasternal short-axis view at the aortic valve level. All findings were interpreted according to American College of Veterinary Internal Medicine (ACVIM) guidelines. An LA diameter >16mm, LA/Ao ≥1.6, and the presence of spontaneous echo-contrast or intracardiac thrombus with cardiac remodeling were accepted as inclusion criteria for the aortic thromboembolism group (Fuentes *et al.*, 2020).

Blood collection, complete blood count and biochemical analysis: From each cat of control and FATE groups, 0.5mL blood samples were taken into tubes containing K3-EDTA for complete blood count analysis and 3mL into tubes without anticoagulant for serum biochemical parameters. The FATE cats were brought to the clinic in emergency when their owners observed FATE symptoms. Blood samples from these cats were taken after they were diagnosed positive for FATE. So, it was not possible to determine the exact time of FATE development in these cats.

Hematological analyses in blood samples from cats of both groups were performed using the Veterinary Blood Count Analyzer (HASVET VH5R, China). From samples without anticoagulant, serum was separated by centrifugation at 3000rpm for 5min and stored at −20°C for further analysis.

Lactate dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate aminotransferase (AST), glucose (GLU), creatinine (CREA), blood urea nitrogen (BUN) and cardiac-derived creatine kinase isoenzyme (CK-MB) were measured using the Fujifilm

Dri-Chem Nx600V IC (Japan) fully automated analyzer in serum samples (Sandilya *et al.*, 2025; Cui *et al.*, 2026). In addition, serum concentrations of feline NT-proBNP and cTnI were measured with the Vcheck V200 immunofluorescence analyzer (Bionote®, Korea; ELK Diagnostic, Turkey), as described earlier (Javery *et al.*, 2025; Allwin *et al.*, 2026).

Serum samples were also used for measurement of sST2 levels using a commercial cat-specific sandwich ELISA kit (MyBioSource, Cat. No. MBS9350659), according to the manufacturer's instructions. Briefly, standards and samples were added to plate wells, HRP-Conjugate Reagent was added to each well and incubated at 37°C for 60 minutes. After incubation, the wells were washed, and chromogen A was added first, followed by chromogen B, and incubated at 37°C for 15 minutes. After stopping the reaction with the use of stop solution, the optical density was measured at 450nm. Analyte concentrations were calculated using the standard curve (Algibay *et al.*, 2025). All samples were evaluated in duplicate with an ELISA reader (BIO-TEK ELx800, USA) at an optical density of 450nm. The sensitivity of the sST2 assay was 10pg/ml and both intra-assay and inter-assay coefficients of variation were less than 15%. No studies regarding the reference range of sST2 for cats could be traced in the available literature.

Statistical analysis: IBM SPSS Statistics 27 was used for statistical analyses of the data generated during the study. The normality of the data was assessed using the Kolmogorov-Smirnov and Shapiro-Wilks tests, and the data was not found to follow a normal distribution. The Mann-Whitney U test was used for intergroup comparisons of quantitative data. Spearman's rho correlation analysis was used to examine the relationships between variables. The data has been shown as Median with QRL (P_{25}-P_{75}). Statistical significance was assessed at $P<0.05$ level.

RESULTS

In our study, out of 10 cats of FATE group, hypertrophic obstructive cardiomyopathy (HOCM) was diagnosed in 5 cats, hypertrophic cardiomyopathy (HCM) in 2 cats, myocardial form (mRCM) of restrictive cardiomyopathy in 2 cats, and endomyocardial form (eRCM) of restrictive cardiomyopathy in one cat. HCM and its subtype HOCM, characterized by increased myocardial thickness, were observed in majority of the patients (70% cats).

X-rays of cats in the FATE group revealed cardiomegaly with enlarged left atrium (Fig. 1A), increased VHS (Fig. 1B) and pleural effusion (Fig. 1C). Echocardiographic examination revealed thrombus foci within the left atrium (Fig. 2A & 2C), a "smoke" appearance referred to as spontaneous echo contrast (SEC) in the left atrium (Fig. 2A & 2B), and enlarged left atrium to aorta (LA/AO) ratio (Fig. 2C & 2D). The VHS values were significantly higher in FATE-affected cats (9.75±0.14) than both the normal reference values (7.5±0.32) and the healthy control group (7.63±0.13) values ($P<0.05$).

When hematological parameter values were compared between cats of two groups, RBC, HGB and HCT values in the FATE group were significantly lower than in the control group ($P<0.05$), while the WBC, NEU, BASO, and MPV were found to be significantly higher ($P<0.05$) in FATE than the normal control group. Statistically non-significant differences between cats of two groups were recorded in other hematology parameters, including LYM, MONO, EOS, MCV, MCH, MCHC, RDWc, PLT, PDW and PCT (Table 1).

Cats in the FATE group had significantly higher ALT, AST, CK-MB, and LDH levels compared to the control group ($P<0.05$). Non-significant differences were found between the two groups in ALP, glucose, BUN and CRE. When comparing NT-proBNP, cTnI, and sST2 between the two groups, cats with FATE showed significantly higher NT-proBNP and cTnI values compared to the control group ($P<0.05$), while statistically non-significant difference was found in sST2 values between cats of the two groups (Table 2). Statistically non-significant correlation was found between sST2 and LA Diam, LA/Ao, VHS, NT-proBNP, and cTn-I in cats of FATE, as well as healthy groups (Table 3).

Table 1: Comparison of hematological parameters between cats of the two experimental groups

	FATE Group (n=10)	Healthy Group (n=10)		
	Median (P_{25}-P_{75})	Median (P_{25}-P_{75})	Reference value	P-value
WBC ($x10^9$/L)	15.97 (10.61-24.00)	5.98 (4.89-12.20)	5.50-19.50	0.010*
NEU ($x10^3/\mu L$)	14.14 (7.73-19.70)	3.56 (2.64-4.91)	2.32-12.58	0.001*
LYM ($x10^3/\mu L$)	1.44 (0.69-2.32)	2.36 (0.76-4.36)	0.73-7.86	0.290^{NS}
MONO ($x10^3/\mu L$)	0.48 (0.30-1.58)	0.41 (0.22-0.81)	0.07-1.25	0.364^{NS}
EOS ($x10^3/\mu L$)	0.32 (0.21-0.58)	0.49 (0.23-0.72)	0.06-1.93	0.650^{NS}
BASO ($x10^3/\mu L$)	0.09 (0.08-0.17)	0.04 (0.02-0.06)	0.00-0.12	0.005*
RBC ($x10^{12}$/L)	9.46 (6.48-11.08)	11.11 (10.27-11.67)	4.60-12.00	0.041*
HGB (g/dL)	13.15 (9.73-13.68)	14.8 (13.85-15.90)	9.00-15.30	0.014*
HCT (%)	43.50 (32.85-45.40)	47.55 (41.95-51.55)	26.00-49.00	0.041*
MCV (fL)	44.65 (40.78-47.43)	44.15 (40.78-47.70)	39.00-53.00	0.880^{NS}
MCH (pg)	14.15 (12.18-15.05)	13.7 (12.23-14.78)	13.00-20.00	0.677^{NS}
MCHC (g/dL)	31.15 (29.98-32.05)	30.6 (30.23-31.33)	29.00-37.00	0.256^{NS}
RDWc (%)	17.65 (15.80-18.40)	17.15 (15.60-18.40)	16.00-23.00	0.762^{NS}
PLT ($x10^3/\mu L$)	135.5 (58-243.75)	224 (120.50-294.25)	100.00-518.00	0.112^{NS}
MPV (fL)	9.65 (8.85-10.25)	8.7 (8.20-9.48)	8.10-13.90	0.041*
PDW (fL)	11.65 (8.63-14.88)	12.40 (8.33-14.38)	12.00-17.50	0.820^{NS}
PCT (mL/L)	0.13 (0.06-0.21)	0.20 (0.12-0.26)	0.90-7.00	0.198^{NS}

WBC: White Blood Cells; NEU: Neutrophils; MON: Monocytes; LYM: Lymphocytes; BASO: Basophils; EOS: Eosinophils; HGB: Hemoglobin; RBC: Erythrocytes; HCT: Hematocrit; MCH: Mean corpuscular hemoglobin; MCV: Mean corpuscular volume; MCHC: Mean corpuscular hemoglobin concentration; PLT: Platelets; RDWc: Red blood cell distribution width; PDW: Platelet distribution width; MPV: Mean platelet volume; PCT: Plateletcrit; *Significant difference ($P<0.05$); NS: Non-significant difference.

Table 2: Comparison of various serum biochemical constituents between cats of two groups

	FATE Group (n=10)	Healthy Group (n=10)		
	Median (P_{25}-P_{75})	Median (P_{25}-P_{75})	Reference values	P-value
ALT (U/L)	154.00 (51.00-389.00)	46.50 (40.50-73.50)	22.00-84.00	0.011*
AST (U/L)	85.50 (59.25-238.50)	29.00 (23.75-35.75)	18.00-51.00	0.001*
ALP (U/L)	36.50 (27.25-61.50)	32.00 (24.50-41.00)	9.00-53.00	0.405
CK-MB (U/L)	181.00 (99.75-300.00)	91.00 (68.00-109.25)	5.00-25.00	0.023*
Glucose (mg/dL)	173.00 (121.25-267.25)	152.00 (115.00-181.50)	71.00-148.00	0.450
BUN (mg/dL)	34.25 (22.23-61.58)	23.80 (21.18-24.78)	17.60-32.80	0.096
CRE (mg/dL)	1.35 (0.89-1.98)	1.16 (0.94-1.31)	0.80-1.80	0.427
LDH (U/L)	501.00 (283.75-900.00)	134.50 (95.25-246.50)	35.00-187.00	0.003*
NT-proBNP (pmol/L)	1500.00 (1500.00-1500.00)	50.00 (50.00-59.18)		0.001*
cTnI (ng/mL)	6.15 (4.38-11.90)	0.01 (0.01-0.08)		0.001*
sST2 (pg/mL)	113.26 (81.83-176.10)	155.63 (103.57-205.73)		0.226

ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; ALP: Alkaline Phosphatase; CK-MB: Creatine Phosphokinase-MB; BUN: Blood Urea Nitrogen; CREA: Creatinine; LDH: Lactate Dehydrogenase; NT-proBNP: N Terminal pro-Brain Natriuretic Peptide; cTnI: Cardiac Troponin I; sST2: Soluble Suppression of Tumorigenicity 2; *Significant difference (P<0.05).

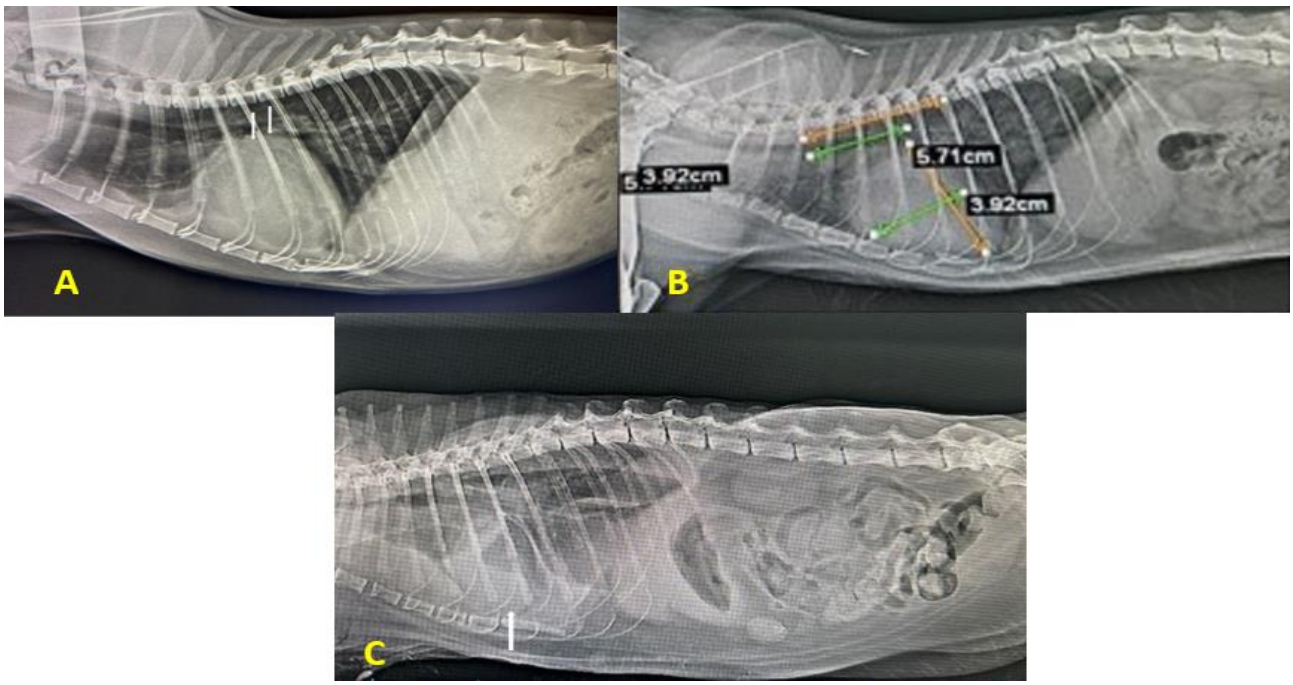

Fig. 1: Radiographs showing cardiomegaly and VHS measurement in cats of FATE group (n=10): A: Image showing cardiomegaly and a markedly dilated left atrium (white arrows) in a cat with HCM stage C on a latero-lateral view; B: VHS measurement on radiographic imaging in the LL (latero-lateral-right) position, short axis (green arrow), long axis (orange arrow): C: Pleural effusion (white arrow) on radiographic examination.

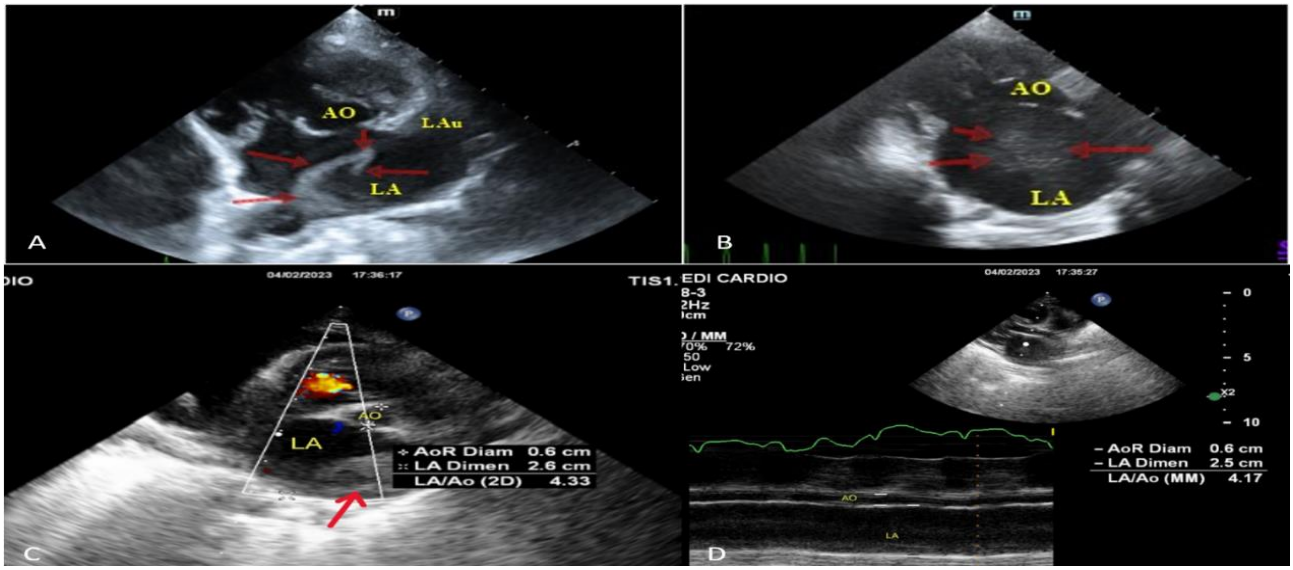

Fig. 2: Echocardiographic examination in FATE cats: A: Marked left atrial (LA) dilatation and a thrombus within the LA appearing as a "smoke" pattern (red arrows); AO: Aorta; LAu: Left Atrial Appendage; B): A spontaneous echo contrast (SEC, smoke-like) appearance within the LA (red arrows); C: Thrombus (red arrow) and enlarged LA/Ao ratio (4.33) in the right parasternal short-axis view; D: Right parasternal long axis enlarged LA/Ao ratio (LA/Ao: 4.17).

Table 3: Correlation between sST2 and LA Diam, LA/Ao, VHS, NT-proBNP, and Tn-I in the FATE and the healthy groups

Group		sST2	
		r	P
FATE Group (n=10)	LA Diam (cm)	0.348	0.325
	LA/Ao	0.127	0.726
	VHS	0.182	0.615
	NT-proBNP (mmol/L)	0.522	0.122
	cTnI (ng/mL)	-0.334	0.345
Healthy Group (n=10)	LA Diam (cm)	0.316	0.374
	LA/Ao	0.105	0.774
	VHS	0.385	0.271
	NT-proBNP (mmol/L)	0.055	0.881
	cTnI (ng/mL)	-0.478	0.162

LA Diam: Left Atrial Diameter; LA/Ao: Left Atrium to Aorta ratio; VHS: Vertebral Heart Score; NT-proBNP: N Terminal pro-Brain Natriuretic Peptide; cTnI: Cardiac Troponin I; sST2: Soluble Suppression of Tumorigenicity 2.

When the experimental groups were evaluated based on 2D-M mode echocardiographic measurements, cats in the FATE group exhibited significantly lower LVOT Diam, LVEF and LVFS measurements than the control group cats (P<0.05), while IVSd, IVPWd, IVSs, LVIDs, LVPWs, LA Diam and LA/Ao ratio measurements were found to be significantly higher (P<0.05) in FATE compared to healthy control cats. However, the differences in LVIDd and Ao Diam measurements between cats of the two groups were statistically non-significant (Table 4).

Table 4: Evaluation of groups in terms of 2D-M mode echocardiographic measurements

	FATE Group (n=10)	Healthy Group (n=10)	
	Median (P_{25}-P_{75})	Median (P_{25}-P_{75})	P-value
IVSd (cm)	0.64 (0.53-0.79)	0.52 (0.46-0.55)	0.007*
LVIDd (cm)	1.48 (1.40-1.70)	1.35 (1.22-1.65)	0.256
LVPWd (cm)	0.68 (0.55-0.83)	0.42 (0.38-0.48)	0.001*
IVSs (cm)	0.83 (0.72-0.93)	0.71 (0.64-0.73)	0.021*
LVIDs (cm)	1.05 (0.87-1.19)	0.76 (0.59-0.98)	0.028*
LVPWs (cm)	0.80 (0.67-0.91)	0.62 (0.55-0.69)	0.014*
LA Diam (cm)	2.35 (2.08-2.50)	1.30 (1.15-1.40)	0.001*
Ao Diam (cm)	0.75 (0.68-0.90)	0.9 (0.80-1.00)	0.073
LVOT Diam (cm)	0.60 (0.50-0.73)	0.80 (0.68-0.80)	0.044*
LA/Ao	3.36 (2.66-3.90)	1.41 (1.30-1.56)	0.001*
LV EF (%)	65.80 (54.45-72.00)	78.30 (68.90-87.68)	0.034*
LV FS (%)	33.20 (25.73-38.25)	44.00 (35.15-53.35)	0.026*

IVSd: Interventricular Septal thickness in diastole; LVIDd: Left Ventricular Internal Diameter in diastole; LVPWd: Left Ventricular Posterior Wall thickness in diastole; IVSs: Interventricular Septal thickness in systole; LVIDs: Left Ventricular Internal Diameter in systole; LVPWs: Left Ventricular Posterior Wall thickness in systole; LA Diam: Left Atrial Diameter; Ao Diam: Aortic Diameter; LVOT Diam: Left Ventricular Outflow Tract Diameter; LA/Ao: Left Atrium to Aorta ratio; LV EF: Left Ventricular Ejection Fraction; LV FS: Left Ventricular Fractional Shortening; *Significant difference (P<0.05).

In pulse wave Doppler transmitral flow and mitral annulus Doppler echocardiographic examinations, MV Dec Time, E' medial, A' medial, E' lateral and A' lateral measurement values were significantly lower in cats of the FATE group compared to those in the control group (P<0.05). However, MV E vel, E'/E' medial and E'/E' lateral measurement values were significantly lower in control cats than those of the FATE group (P<0.05). Statistically non-significant differences between the two groups in terms of MV A vel, E/A, E'/A' medial and E'/A' lateral measurements were observed (Table 5).

Table 5: Pulse Wave Doppler transmitral flow and mitral annulus Doppler echocardiographic measurements

	FATE Group (n=10)	Healthy Group (n=10)	
	Median (P_{25}-P_{75})	Median (P_{25}-P_{75})	P-value
MV E vel (cm/s)	90.15 (71.05-139.00)	61.55 (59.65-68.68)	0.002*
MV A vel (cm/s)	39.20 (28.60-82.33)	41.05 (33.73-43.13)	0.705
E/A	2.40 (1.33-3.60)	1.55 (1.38-1.80)	0.211
MV Dec. Time	108.50 (81.50-133.50)	169.50 (162.50-176.25)	0.002*
E' medial	4.63 (4.03-5.93)	8.90 (8.08-9.71)	0.001*
A'medial	5.08 (3.70-6.47)	10.75 (7.41-12.13)	0.001*
E' lateral	4.09 (3.86-6.65)	7.36 (6.74-8.43)	0.021*
A'lateral	5.20 (3.61-6.22)	6.96 (5.87-9.04)	0.023*
E'/A' medial	0.75 (0.68-1.57)	1.00 (0.68-1.15)	0.970
E'/E' medial	21.05 (14.28-30.15)	7.20 (6.48-8.30)	0.001*
E'/A' lateral	0.80 (0.60-2.23)	1.30 (0.78-1.60)	0.544
E'/E' lateral	20.55 (15.80-26.65)	8.50 (7.33-9.03)	0.001*

MV E vel: Mitral Valve Early diastolic filling velocity (E wave velocity); MV A vel: Mitral Valve Late diastolic filling velocity (A wave velocity); E/A: Ratio of early to late ventricular filling velocities; MV Dec. Time: Mitral Valve Deceleration Time; E' medial: Medial (septal) mitral annular early diastolic velocity; A' medial: Medial (septal) mitral annular late diastolic velocity; E' lateral: Lateral mitral annular early diastolic velocity; A' lateral: Lateral mitral annular late diastolic velocity; E'/A' medial: Ratio of medial early to late diastolic velocities; E'/E' medial: Ratio of mitral inflow E velocity to medial E' velocity; E'/A' lateral: Ratio of lateral early to late diastolic velocities; E'/E' lateral: Ratio of mitral inflow E velocity to lateral E' velocity; * Significant difference (P<0.05).

DISCUSSION

Feline arterial thromboembolism is a serious complication of cardiomyopathies, characterized by sudden onset and high morbidity and mortality rates (Borgeat *et al.*, 2014). Left atrial enlargement in cardiomyopathy slows atrial blood flow, promoting blood stasis and thrombus formation (Fuentes, 2012). Clinically, the LA/Ao ratio is commonly used to assess left atrial enlargement, with values >1.5–1.6 are considered as indicative of the problem (Schober and Maerz, 2006; Payne *et al.*, 2015). Busato *et al.* (2022) reported that the LA/Ao ratio was higher in cats that developed arterial thromboembolism compared to clinically healthy cates. Similarly, Payne *et al.* (2015) demonstrated that an enlarged left atrium in cats with hypertrophic cardiomyopathy is associated with conditions such as congestive heart failure, arterial thromboembolism, and cardiac death. In addition, a study by Borgeat *et al.* (2014) reported that left atrial enlargement is associated with the development of arterial thromboembolism and long-term prognosis. In the present study, it was observed that the LA Diam and LA/Ao ratio, which reflect left atrial enlargement, were significantly higher in cats having cardiomyopathy with embolism compared to the control group. The high LA/Ao ratio and increased left atrial diameter, indicating left atrial remodeling, are associated with the development of thromboembolism; this finding is consistent with previous studies indicating that an increase in LA to Ao ratio is significantly associated with a higher risk of developing FATE (Payne *et al.*, 2015; Busato *et al.*, 2022). An increase in the size of the left atrium plays a significant role in the development of FATE by increasing the risk of thrombus formation, along with changes in atrial hemodynamics and blood stasis.

In this study, spontaneous echo contrast (SEC) was detected in all cats in the FATE group. Spontaneous echo

contrast results from erythrocyte aggregation due to low blood flow velocity and characterized by a "smoke-like" appearance on echocardiography; it is considered a precursor to thrombus formation (Schober and Maerz, 2006). Payne *et al.* (2015) and Yeh *et al.* (2025) reported that the SEC and intracardiac thrombi within left atrium (LA) are high-risk factors for the development of FATE. These findings support the detection of SEC in all cats in the FATE group in our study.

In our study, NT-proBNP values were higher in the FATE group than in healthy cats, indicating that FATE is closely linked to advanced cardiac stress, particularly left atrial remodeling. According to Ironside *et al.* (2021), cats with an LA/Ao ratio ≥1.5 had a fourfold higher risk of congestive heart failure and FATE, with a similar increase in risk for NT-proBNP ≥700pmol/L. Oranges *et al.* (2026) found NT-proBNP levels to be higher in cats with FATE than in those with cardiomyopathy without thromboembolism. They reported that NT-proBNP >491pmol/L predicted thromboembolism with 96.0% sensitivity and 93.5% specificity, correlating with larger left atrial size, spontaneous echocardiographic contrast, and higher prevalence of left atrial thrombus. The mean NT-proBNP level recorded in this study was 1489.09±10.91pmol/L, which is within the risk threshold value. However, when compared with control, elevated NT-proBNP levels in cats with thromboembolism suggest not only the presence of cardiomyopathy but also significant atrial remodeling and susceptibility to thrombus formation. It may also be noted that NT-proBNP levels alone do not help diagnose thromboembolism; these values should be evaluated in relation to clinical findings, echocardiographic assessment, and other diagnostic methods.

Cardiac troponin I (cTnI), a specific biomarker of myocardial cell damage, is associated with the severity of cardiomyopathy in cats (Hori *et al.*, 2018). Hertzsch *et al.* (2019) reported that cTnI levels above 0.06ng/ml can be used as a screening test to identify cats with asymptomatic HCM. In this study, cTnI concentrations were higher in the FATE group compared to healthy cats. Previous studies have reported that high cTnI levels in cats with hypertrophic cardiomyopathy show a positive correlation with disease severity and left atrial enlargement and may serve as an indicator of left atrial enlargement and poor prognosis (Borgeat *et al.*, 2014; Langhorn and Willesen, 2016; Hori *et al.*, 2018; Hanas *et al.*, 2022). In FATE, increased cTnI may indicate not only underlying cardiomyopathy but also myocardial stress and ischemic injury. Based on these data, this study suggests that the marked increase in cTnI observed in the FATE group is associated with severe myocardial damage and hemodynamic stress related to the development of thromboembolism, and supports the finding of more severe cardiac involvement in these cases.

In the present study, VHS values were higher in the FATE group than in the healthy group. In addition, VHS values in cats in the FATE group were above the reference range of 6.7–8.1 (Litster and Buchanan, 2000). VHS is a parameter commonly used in the radiographic assessment of cardiac dimensions in cats and has been found to be associated specifically with left atrial enlargement and general cardiac dilation (Guglielmini *et al.*, 2014). However, VHS has limited sensitivity and offers lower diagnostic value than echocardiography in detecting early cardiac changes. When evaluating FATE, the elevated VHS values may be interpreted not as a direct indicator of thromboembolism development, but rather as a radiographic reflection of concomitant advanced cardiac enlargement.

In our study, an evaluation of WBCs and neutrophils revealed a distinct inflammatory response in the FATE group. Similarly, Esin and Uzun (2025) reported higher neutrophil and leukocyte counts in cats with FATE compared to healthy cats. Same researchers associated the increase in the neutrophil/lymphocyte ratio, an indicator of systemic inflammation, with a shorter survival time. These results indicate that arterial thromboembolism is not merely a mechanical vascular occlusion but is also associated with a systemic inflammatory response. In the presented study, although the hemogram RBC, HGB, and HCT values were within the reference range, they were significantly lower in the FATE group compared to the healthy group. This may be associated with tissue perfusion disturbances and systemic effects, resulting from thromboembolism.

Although there is no specific biochemical marker for cardiac injury in human and veterinary medicine, significant increase in certain enzyme activities have been reported following severe damage to myocardial tissue (Oyama, 2013; Aydin *et al.,* 2019). Among the biochemical parameters frequently used to assess myocardial damage in the heart, CK-MB, LDH, AST and ALT provide important information (Özkanlar and Akçay, 2014). In the current study, ALT, AST, CK-MB, and LDH levels were higher in the FATE group compared to the healthy group, which may be associated with cellular damage resulting from tissue ischemia and hypoxia, caused by thromboembolism. Similarly, Borgeat *et al.* (2014) reported that biochemical changes associated with systemic effects and tissue damage may be observed in cases of FATE. In their study involving a total of 20 cats (10 diagnosed with HCM and 10 with aortic thrombosis), Bakırel *et al.* (2021) detected elevated CK-MB levels in the group with aortic thromboembolism. Vatnikov *et al.* (2024) found that in cats with HCM-related cardiorenal syndrome, LDH activity increased 2.69-fold and CK-MB levels increased 2.02-fold. Aroch *et al.* (2010), in a study including 601 cats, found that CK levels were significantly elevated in various types of heart disease; in different heart conditions such as cardiomyopathy, congestive heart failure associated with degenerative valve disease, myocarditis, and congenital heart defects, serum CK levels were found to be above the reference range in 7.6% of the cats. Elevated CK levels were observed in 6.7% of cases, particularly in those with hypertrophic, restrictive, dilated, and hypertrophic-obstructive cardiomyopathy. In the present study, higher concentrations of ALT, AST, CK-MB and LDH were detected in cats in the FATE group. We believe that rather than using these enzymes as diagnostic markers on their own, it would be more accurate to evaluate them in conjunction with other biomarkers and clinical data.

The echocardiographic evaluation of the study revealed significant structural and functional cardiac changes in the FATE group. Left ventricular wall

thicknesses (IVSd, LVPWd, IVSs, LVPWs) and left atrial diameter, as well as the LA/Ao ratio, were found to be significantly higher, whereas EF and FS values, which are indicators of left ventricular systolic function, were found to be reduced in the FATE group. These findings suggest that systolic function may also be affected alongside hypertrophic remodeling in cats with FATE. Indeed, it has been reported that ventricular wall thickening and the accompanying left atrial enlargement in cats with hypertrophic cardiomyopathy are closely associated with the development of thromboembolism (Borgeat *et al.*, 2014; Payne *et al.*, 2015). In Doppler echocardiographic evaluation, the MV E velocity and E/E' ratios were higher in the FATE group, whereas E', A', and deceleration time were decreased. These findings suggest diastolic dysfunction and increased filling pressures. It has been reported that diastolic dysfunction in cats is associated with left atrial enlargement and the development of thromboembolism (Schober and Maerz, 2006). Although the FATE group comprises different cardiomyopathy phenotypes (HCM, HOCM, and restrictive types), the observation of similar left atrial enlargement and elevated filling pressures in all cases suggests that the primary determinant of thromboembolism is shared hemodynamic abnormalities rather than a specific phenotype. The echocardiographic changes identified in this study are consistent with structural remodeling, atrial stasis, and diastolic dysfunction, which contribute to the development of thromboembolism.

The biological effects of sST2 are mainly based on its interaction with interleukin-33 (IL-33). The IL-33/sST2 signaling pathway exerts antihypertrophic and antifibrotic effects on cardiomyocytes, playing a cardioprotective role, while the circulating soluble form, sST2, inhibits this interaction, reflecting cardiovascular stress and fibrotic remodeling processes (Sanada *et al.*, 2007; Seki *et al.*, 2009). Therefore, sST2 has been identified as an important biomarker associated with morbidity and mortality, particularly in heart failure and other cardiovascular diseases (Kakkar and Lee, 2008; Mueller *et al.*, 2008; Januzzi *et al.*, 2015). Furthermore, it has been demonstrated that elevated sST2 concentrations increase in parallel with disease severity and serve as an independent prognostic marker (Boswood, 2009).

In this study, there was no difference between the FATE group and the healthy control group in terms of serum sST2 levels. Similarly, Kaya and Bakırel (2022) found no difference in sST2 levels when comparing cats with cardiomyopathy to healthy controls in their study. This suggests that sST2 biomarker may be more closely associated with chronic myocardial stress, inflammation, and fibrotic remodeling rather than acute hemodynamic and thrombotic processes involved in the development of thromboembolism. Indeed, it has been reported that sST2 is particularly associated with long-term cardiac remodeling and fibrosis, while its capacity to reflect acute events may be limited (Pascual-Figal and Januzzi, 2015). Therefore, while sST2 plays an indirect role in this process, it is thought to have lower sensitivity in distinguishing the development of thromboembolism compared to biomarkers such as NT-proBNP and cTnI, which more directly reflect cardiac stress and myocardial damage. In addition, it has been reported that the prognostic value of sST2 is more pronounced in serial measurements in some studies (Pascual-Figal and Januzzi, 2015).

The inability of single-time measurements to adequately reflect biomarker changes in acute clinical conditions may be another reason for non-significant differences in sST2 levels between cats of two groups observed in the present study. Moreover, the absence of statistical significance for sST2 between cats of the two groups may reflect insufficient statistical power due to the limited sample size (n=10 per group). Furthermore, the fact that the FATE group consisted of different cardiomyopathy phenotypes may also have contributed to this outcome. Thus, sST2 may be a more appropriate marker of chronic cardiac remodeling rather than acute thromboembolic events. Future studies involving large sample groups and serial measurements will provide a clearer picture of the clinical utility of sST2 in cats.

Conclusions: In the current study, NT-proBNP and cTnI levels were found to be higher in the FATE group compared to the control group; however, no difference was observed in serum sST2 levels between cats of the two groups. Radiographic evaluation revealed statistically significant differences in vertebral heart scores between the two groups. Echocardiographic examination demonstrated that left atrial size and the LA/Ao ratio were significantly increased in the FATE group compared to the healthy group. Hematological analyses indicated that the increase in white blood cells and neutrophils in the FATE group reflected a distinct inflammatory response. Additionally, higher concentrations of ALT, AST, CK-MB, and LDH, consistent with cardiomyopathy, were detected in cats in the FATE group. In conclusion, serum NT-proBNP and cTnI levels are important for diagnosis of FATE, while sST2 alone is not useful for FATE diagnosis. However, the absence of statistical significance for sST2 between cats of the two groups may be due to the limited sample size. Further studies involving larger sample groups are needed to determine the ability of sST2 to reflect cardiac remodeling and its diagnostic significance in feline cardiomyopathy.

Ethical approval: The experimental procedure followed in this study was approved by the Erciyes University Animal Experimentation Ethics Committee on November 3, 2022 (No. 22/232) and was conducted in accordance with all guidelines on animal ethics and welfare.

Authors contribution: ÖŞ and ÖA designed and conceptualized the study. ÖŞ, ÖA, MAR, İR, İKB and İK collected and analyzed blood samples. ÖŞ, ÖA and MAR analyzed the data. İKB, MAR, ÖŞ, İR and ÖA prepared the manuscript. All authors interpreted the data, critically reviewed the manuscript for significant intellectual contents and approved the final version.

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