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ACUTE CONGESTIVE HEART FAILURE SECONDARY TO TRANSIENT MYOCARDIAL THICKENING IN A CAT: CASE REPORT

Alice-Mihaela RĂDULESCU, Elena-Teodora VULCAN, Lucian IONIȚĂ

University of Agronomic Sciences and Veterinary Medicine of Bucharest, 59 Marasti Blvd, District 1, Bucharest, Romania

Corresponding author email: alice@medicalvet.ro

Abstract

A one-year-old Scottish fold male cat was referred to the Cardiology department, presenting signs of congestive heart failure one week after anesthesia. Echocardiography revealed an enlarged left atrium with the left ventricular wall thickening and confluent "B lines". Cardiac biomarker troponin I was markedly elevated. The cat was discharged home after an intensive hospitalization treatment that led to progressive clinical improvement. It was reevaluated after two months, and a physical examination, echocardiographic measurements, and cardiac troponin were normalized. The diagnosis of transient myocardial thickening was considered based on the presence of a trigger possibly related to myocardial injury and the patient's history, cardiological, and laboratory data. This condition is relatively uncommon and closely resembles hypertrophic cardiomyopathy but involves reversions of the cardiac modifications. It usually appears in young cats with previous events. Transient myocardial thickening has a better prognosis in cats in contrast with hypertrophic cardiomyopathy. This case report describes a case of transient myocardial thickness with congestive heart failure, which returns to a case of normal cardiac features.

Key words*: feline, myocardial thickening, troponin I, echocardiography.*

INTRODUCTION

The idiom 'transient myocardial thickening' (TMT) was submitted in veterinary medicine in 2018 to describe a clinical pathology that resembles hypertrophic cardiomyopathy (HCM) and usually affects young cats (Matos et al., 2018).
Preceding articles in humans have shown that transient myocardial thickening was associated with hypertrophic cardiomyopathy secondary to acute myocarditis, atypical cases of stress-induced cardiomyopathy (Takotsubo), and storage diseases (Amyloidosis) (Kudo et al., 2011; Madias et al., 2013; Hwang et al., 2014; Elliot et al., 2014; Madias et al., 2016).
TMT was described by the increased diameter of the left ventricular walls, often with concurrent left atrial (LA) dysfunction and enlargement triggering congestive heart failure (CHF) and occasionally thromboembolism (ATE) in correlation with the increased concentration of cardiac troponin I (cTnI) in the bloodstream, suggesting myocardial injury (Matos et al., 2018; Langhorn et al., 2014; Vollmar et al., 2024).
This condition has a reversible character, improved cardiac function instead of progressive degradation of cardiac parameters associated with HCM, and a better prognosis. However, there is typically no clinical or echocardiographic evidence to differentiate the two disease processes at presentation. Cats with TMT usually had antecedent stressful events such as general anesthesia (Glaus et al., 2010).
This case report aims to describe the historical, clinical, diagnostic, therapeutic, and clinical evolution of a patient with transient myocardial thickening with secondary congestive heart failure, focusing on echocardiographic characteristics.

MATERIALS AND METHODS

Toby, an 8-month-old male British Shorthair cat, presented with dyspnea, hind leg instability, and intolerance to minimal effort, requiring emergency consultation. The patient had undergone anesthesia for orchidectomy and correction of entropion a week prior. During the physical examination, Toby weighed 5 kilograms, with a rectal body temperature of 39.0°C, capillary refilling time of 2 seconds, cardiac frequency of 140 beats per minute, Doppler front right limb systolic blood pressure

90 mmHg (cuff 2.0), and respiratory rate of was 60 respiration per minute with abdominal and restrictive breathing pattern. Pulmonary auscultation revealed severe, disseminated crackles on both hemithorax. Parasternal cardiac auscultation detected a high-grade systolic murmur accentuated on the left side, and the femoral pulse was strong and symmetrical. The cat was vaccinated and dewormed, feline immunedeficiency antibody /feline leukemia antigen virus (Idexx SNAP Combo) was assessed negatively. The attending physician performed blood sample tests, including a count of blood cells and quantitative pro-brain natriuretic peptide (NT- pro-BNP). The CBC showed mild neutropenia 2.92 x 10^9/L (normal range 3.12-12.58 x 10^9/L), mild monocytosis 0.05 x 10^9/L (reference range 0.07-1.36 x 10^9/L), and mild erythrocytosis 10.39 x 10^12/L (normal range 4.6-10.2 x 10^9/L). The NT-pro BNP is a hormone that belongs to the natriuretic peptide families. Cardiomyocytes secrete it in response to an abnormal extent, pressure, or volume overload (Connolly et al., 2018). The level of NT-pro BNP was mildly elevated at 63.6 pmol/L (normal values less than 50 pmol/L).
An additional blood test was performed after the clinical examination to determine if there was any ischemic myocardial injury. The results indicated that the cardiac troponin I level was significantly high, exceeding 20 ng/ml. This value is higher than the normal range, less than 0.06 ng/ml, indicating recent myocardial injury. The 6-lead electrocardiogram showed an average heart rate (140 bpm), sinus rhythm with standard QRS configuration, and axis with infrequent premature ventricular contractions (Figure 1A).

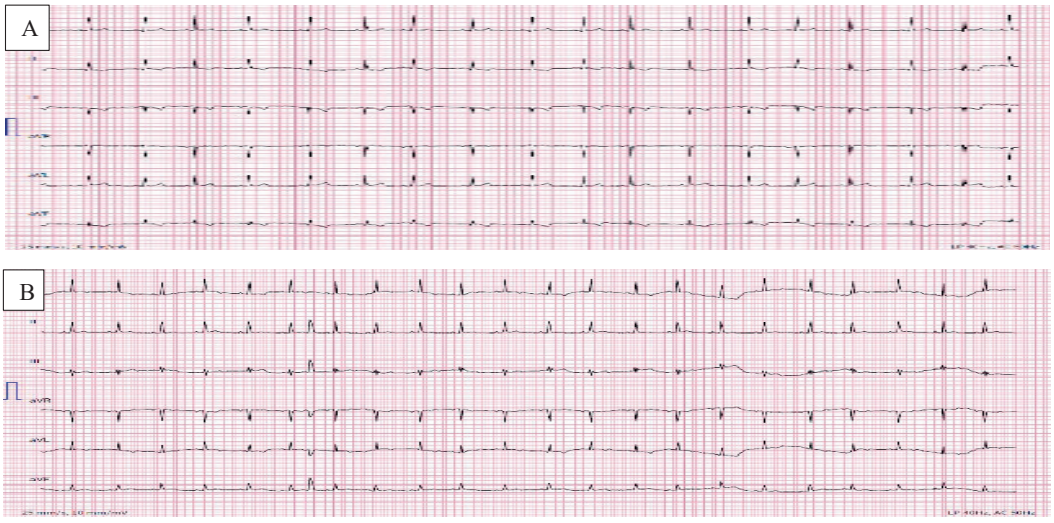

Figure 1 (A-B). The 6-lead electrocardiogram, speed 25 mm/s, amplitude 10 mm/mV. (A) performed on the first day showed 140 bpm, sinus rhythm with occasional premature ventricular contractions, and normal mean electrical axis, and (B) achieved two months after the initial presentation reviled a sinus rhythm, 110 bpm, without ventricular premature complexes

Thoracic radiographs indicated cardiac silhouette enlargement and moderate diffuse interstitial pattern in the lungs accentuated in the ventral area (Figure 3A). The Doppler ultrasound examination, which focused on the descending aorta, did not reveal any thrombus or ascites (Figure 2). A Vivid T8 ultrasonic machine (General Electric Healthcare Ultrasound) equipped with a multifrequency array transducer (2 to 7 MHz) was used for the cardiac examination. The cat was placed on the right and, afterward, on the left lateral recumbency and was examined without sedation. The same operator performed the ultrasound and the follow-up examinations using a best-practice scan of the level recommended by ACVIM to evaluate the cat's condition.

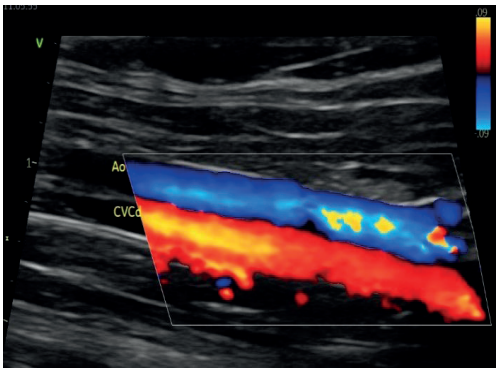

Figure 2. Abdominal ultrasound focused on the descending aorta until the trifurcation in the iliac arteries to visualize the presence of a thrombus. The Color Doppler flow was laminar and filled the lumen completely. No sign of thrombus or spontaneous contrast was visible

Echocardiography revealed moderate diffuse symmetric interventricular septal and left ventricular free wall thickening of 7.0 mm with a standard range of less than 5 mm (Figure 4A). A leading edge-to-leading edge technique was used in three cardiac cycles to determine the ventricular wall thickness during end-diastole in the long and short axes. (Boon, 2011) (Figure 4A).

The ratio between the end-systolic left atrial and end-diastolic aortic dimensions (LA/Ao) was determined in 2D in a transverse view through an inner edge-to-inner-edge technique, according to the Rishniw method. The measurement was taken from the prime diastolic frame, with the aortic valve closed, and was found to be elevated at 2.2, compared to the normal range of less than 1.6 (Rishniw et al., 2000) (Figure 5A). This ratio was obtained with a value of 2.64 in M-Mode. The left atrial fractional shortening (LAFS%) was calculated using the diastolic and systolic left atrial dimensions and was 4.5 %, below the usual 18-38% (Bussadori, 2023) (Figure 6A).

The left atrial anteroposterior diameter (LAD) was measured in a four-chambered view, inner edge to inner edge from the middle of the interatrial septum wall to the posterior free wall measured at end-systole just prior to the mitral valve leaflets opening (Smith, 2012). A value of 2.2 cm was obtained, higher than the standard measurement of less than 1.6 cm.

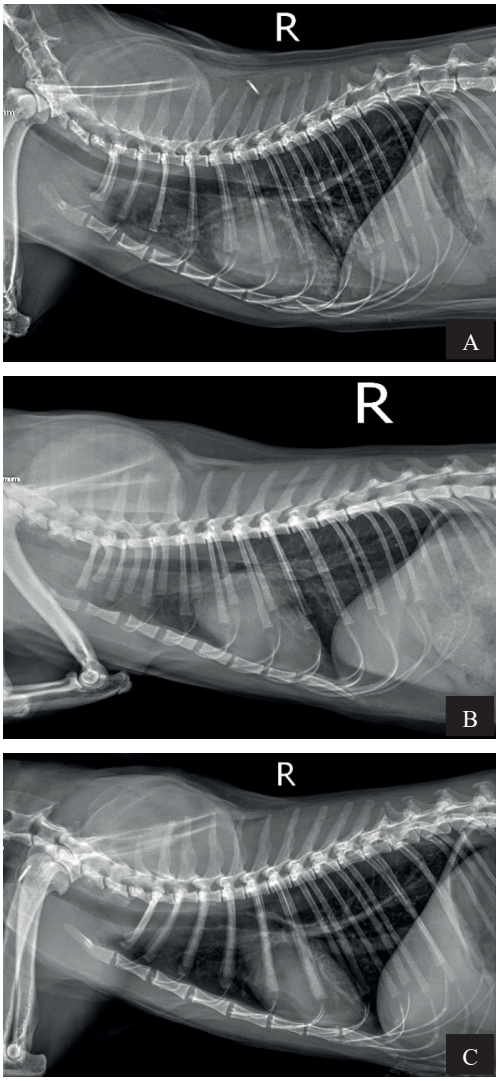

Figure 3. Thoracic radiography in right latero-lateral comparative view in day 0 (A), 48 h lateral (B), and after two months (C). (A) showed increased cardiac silhouette and moderate interstitial pulmonary pattern accentuated in the ventral field; (B) revealed the pulmonary area without infiltration, the cardiac silhouette mildly enlarged, and (C) disclosed a normal heart size and lungs without infiltrates

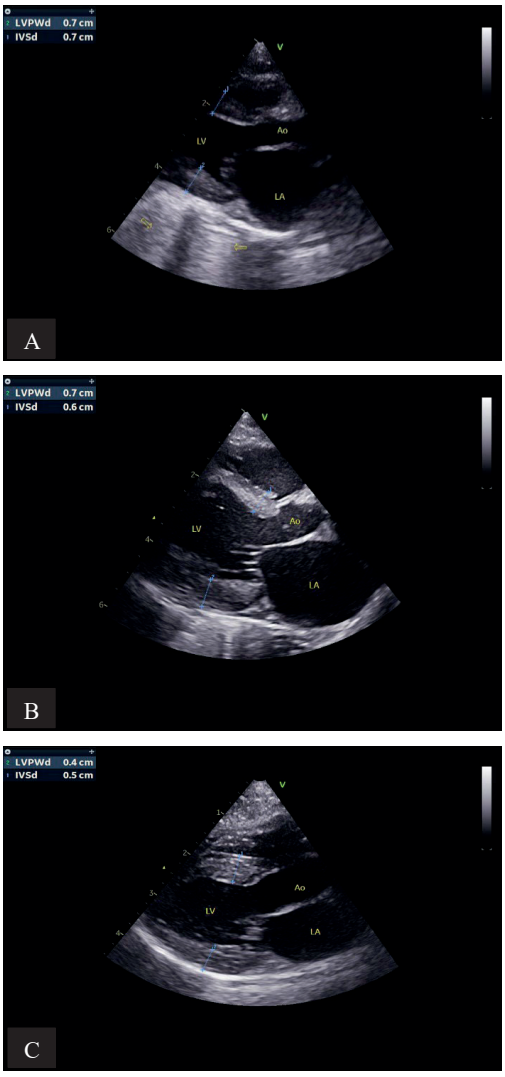

Figure 4 (A-C). Right parasternal long-axis "five-chamber view": (A) at initial presentation, (B) 48 h later, and (C) after 60 days. The initial echocardiographic image showed left atrial enlargement, increased interventricular septum wall, and left ventricular free wall thickness. Note the presence of the "B lines" (arrow) in (A), decreased after 48h (B), and normalization of myocardial thickening (left ventricular septum at end-diastole 5.0 mm, left ventricular posterior wall at end-diastole 4.0 mm, with a structural normal heart two months later (C)

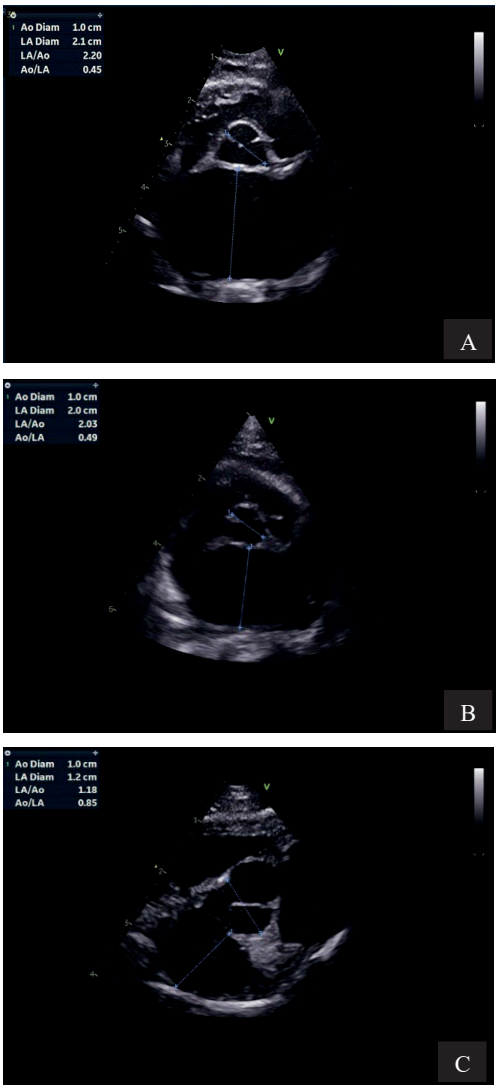

Figure 5. Right parasternal short-axis view at the aortic root level. Comparative B-Mode measurement of LA/Ao ratio in day 0 (A), day 2 (B), day 60 (C). Note the reduction of the left atrium size in (B, C)

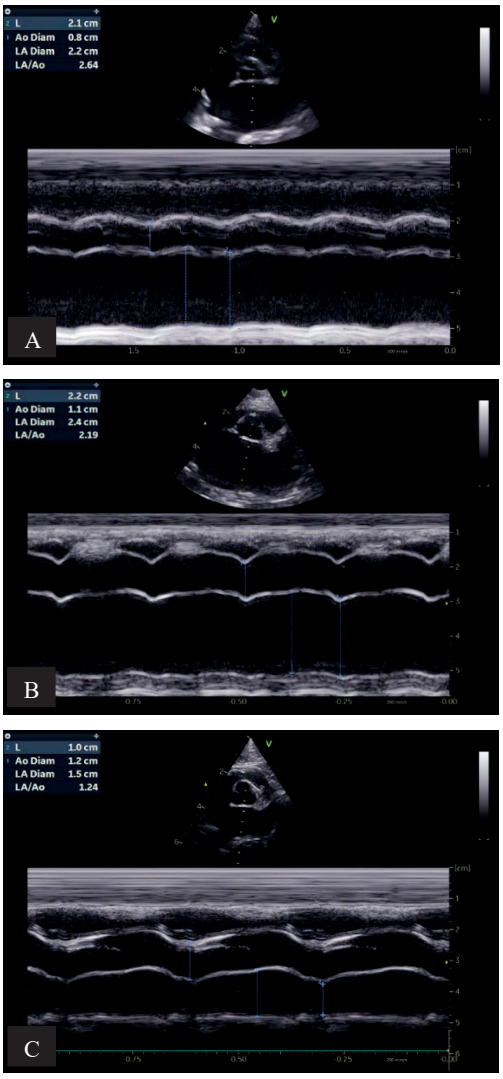

Figure 6. Right parasternal short-axis view at the aortic root level. Comparative LA/Ao ratio M-Mode measurements: day 0 (A), day 2 (B), day 60 (C). Note the reduction of the left atrium size and the improvement of the LAFS % in (B, C)

Left ventricular fractional shortening (LVFS%) was assessed using M-Mode in the "mushroom" view and found to be within the normal range (43%, standard references 30-50%) (Bonagura et al., 2000) (Figure 7A).

The systolic cranial motion of the mitral valve (SAM) is identified as the displacement of the cranial margin of the mitral valve leaflet towards the left ventricular outflow tract during systole. It causes turbulent flow, leading to obstruction and mitral regurgitation. During the examination, SAM's intermittent presence accentuated stress tachycardia was observed (Fuentes et al., 2020).

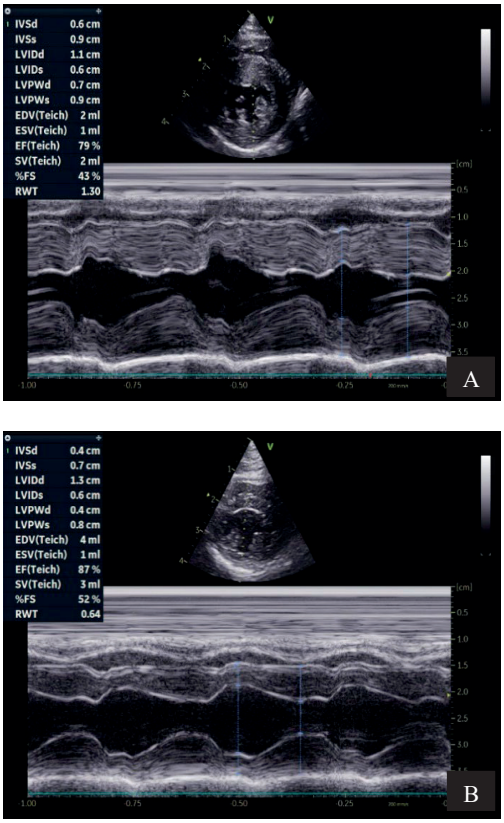

Figure 7. M-Mode measurement in right parasternal short axis view at the level of papillary muscles in diastole. (A) image obtained at the presentation and (B) obtained approximately two months after the case presentation. (B) Compared to (A), note the reduction of the interventricular septum thickness and the left ventricular free wall

The left ventricular outflow tract peak velocity was 0.8 m/s, increased on stress and visible on pulse-wave Doppler.

During the examination, spontaneous echocardiographic contrast ("smoke") or a thrombus was evaluated in the right and left parasternal four-chamber view, short-axis view at the heart base, and oblique long-axis view optimized for the left auricle. The last view is suitable for assessing the risk of thromboembolism, measuring the blood flow velocity in the left appendage. In our case, the emptying velocity was 0.16 m/s, the filling velocity was 0.18 m/s and a velocity below 20

cm/s indicated an increased risk of thromboembolic disease (Schober et al., 2006).
The diastolic function was assessed using trans-mitral flow velocities, isovolumic relaxation time, and tissue Doppler imaging.
In the left standard four chambers, the peak velocity of early diastolic trans-mitral flow showing a restrictive profile on the pulsed wave was 1.58 m/s with a deceleration time of 120 msec, and atrial contraction A-wave velocity was 0.46 m/s (Figure 8A).
The isovolumic relaxation time (IVRT) was less than 19 msec in the left apical five chambers, shorter than the standard 34-56 msec range. It was assessed with the gate volume positioned between the left ventricle inflow to record the onset of the E wave and the outflow tract to record the closure of the aortic valve (Schober et al., 2015) (Figure 8A).

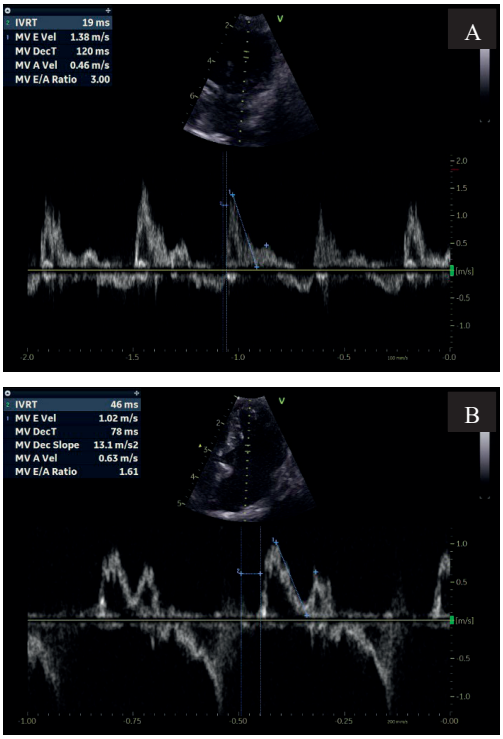

Figure 8. Echocardiographic images from initial presentation (A) revealed diastolic disfunction with a restrictive pattern. Revaluation after 6 weeks (B) revealed normal diastolic function with decreasing in E wave and increased IVRT

Tissue Doppler Imaging (TDI) was used to measure the velocity in early (E' wave) and late diastole (A' wave) by placing a pulsed wave gate at the annulus of the lateral mitral valve. In this case, the TDI showed decreased velocity in both waves, indicating a restrictive diastolic pattern (Gavaghan et al.,1999).
The echocardiographic findings were compatible with a diagnosis of hypertrophic cardiomyopathy and secondary congestive heart failure.
The cat was hospitalized and given oxygen therapy to stabilize its condition. The medications administered were Furosemide (2 mg/kg/6 h), Butorphanol (IV 0.2 mg/kg every 8 hours), Clopidogrel (18.75 mg/cat orally at 24 hours), and Pimobendane (0.125 mg/kg orally at 12 hours) due to low systolic blood pressure (90 mmHg).
This approach resulted in progressive clinical improvement within 48 hours. The patient became clinically stable; radiography showed no sign of alveolar or interstitial pattern (Figure 3B), and echocardiography measurements were ameliorated (Figures 4-8B). The cTnI level was also notably reduced (1.7 ng/ml).
The patient was dismissed home with oral medication: Clopidogrel 18.75 mg/day and Torsemide 0.1 mg/kg q24 h. The owner was recommended to monitor the cat's resting respiratory rate daily and schedule a follow-up appointment for 60 days.
The patient was examined two months later, and no abnormalities were found. Electrocardiographic, radiological, and cardiac ultrasound aspects and cTnI concentration were normalized (Figure 1B, Figures 3-8C). Clopidogrel and Torsemide was discontinued.
The owner informed us of the cat's progress via phone call six months later, and it was found that the cat showed no symptoms of illness, and its resting respiratory rate was less than 20 rpm.
After conducting a series of tests, including physical examination, laboratory investigations, chest radiographs, electrocardiogram, Doppler-focused point of care abdominal ultrasound, and echocardiogram, and following the patient's evolution, the primary diagnosis was transitory myocardial thickness.

Table 1. Evolution of the echocardiographic measurements at presentation (day 0) and follow up echocardiographic examinations (Selective two-dimensional measurement)

Day	IVSd	LVFWd	LAD	LA/Ao M-Mode	LAFS %
0	7	7	2.2	2.64	4.5%
2	6	7	18	2.22	8.3%
60	5	4	12	1.24	33%

Abbreviations: IVSd: inter-ventricular septum thickness in diastole in long axis; LVFWd: left ventricular wall thickness in diastole in long-axis. LA/Ao: right parasternal short-axis left atrium-to-aorta ratio; LAD: right parasternal four-chamber long-axis maximal inner diameter left atrium; LAFS%: left atrial fractional calculated in shortening in short axis left atrium-to-aorta.

Table 2. Evolution of the echocardiographic measurements at presentation (day 0) and follow up echocardiographic examinations (Selective Doppler measurement)

Day	E	A	E/A	IVRT
0	1.58	0.46	3.0	19
2	1.3	0.52	2.5	26
60	1.02	0.63	1.61	46

Abbreviations: E: early diastolic filling; E/A early diastolic filling to atrial contraction ratio; E': early diastole in TDI; IVRT: isovolumic relaxation time.

RESULTS AND DISCUSSIONS

Transient myocardial thickening (TMT) is a condition that initially appears similar to HCM. However, cats with TMT experience a reverse remodeling of the heart that leads to normal cardiac morphology and functionality, comparatively to familial/genetic HCM, which is typically associated with a poor long-term prognosis.

In cats, an HCM phenotype has been associated with hypertension, endocrine disorders (hyperthyroidism and acromegaly), dehydration (pseudo-hypertrophy), and infiltrative disease (lymphoma) and especially in young cats with transient myocardial thickness and myocarditis (Bond et al., 1988; Campell et al., 2007; Carter et al., 2008; Myers et al., 2014).

In humans, TMT was associated with myocarditis, Takotsubo cardiomyopathy, and amyloidosis. In the first two conditions, the increased ventricular wall thickness correlates with myocardial edema; in the last one, it is secondary to the intracellular accumulation of metabolic products. The number and dimension of myocytes and the interstitial space volume influence the myocardial wall's thickness (Fujiwara et al., 1983; Kaltenbach et al., 2008).

In hypertrophic cardiomyopathy, left ventricular wall thickness is mediated to myocyte hypertrophy and interstitial fibrous connective tissue comparatively to transient myocardial thickening caused by interstitial infiltration of proteins, cells, or fluid (myocardial edema) (Matos et al., 2018).

A retrospective study showed that cats diagnosed with transitory myocardial thickness were younger than those diagnosed with hypertrophic cardiomyopathy, with a median age of 1.7 versus eight years (Matos et al., 2017).

Certain criteria need to be met to consider a cat affected by TMT. These included undergoing at least two cardiac ultrasounds, with the initial one revealing an increase of the interventricular septum and left ventricular free wall diameter in diastole and left atrial dilatation. Follow-up echocardiographic tests were also required after a period to demonstrate normalization of these parameters.

It is recommended that the circulating levels of cTnI be tested in these cases, as they tend to be severely elevated, and then retested during the follow-up appointment to observe any reduction in cTnI levels compared to the initial evaluation. Troponins are released in the bloodstream when there is damage to cardiomyocytes, but without differentiate between primary cardiac and non-cardiac diseases involving heart damage, such as renal disease, hyperthyroidism, and anemia (Porciello et al., 2008; Sangster et al., 2014; Lalor et al., 2014).

Troponin I is veterinary medicine's most sensitive and commonly used troponin (Borgeat et al., 2015). There is a correlation between cTnI concentrations and the severity of heart disorders, and it has helped estimate the prognosis (Langhorn et al., 2014). Troponins can help differentiate between hypertrophic cardiomyopathy and myocarditis-induced thickening despite possible overlap in troponin values (Matos et al., 2018).

After a cardiac event, TnI plasma concentrations increase and peak within 24 hours. They may remain elevated for up to a week after the causative factor has been triggered.

In our case, on day 0, the level of cardiac troponin I in the blood was more than 20 ng/ml. After 48 hours, it decreased to 1.7 ng/ml and

continued to decrease to less than 0.06 ng/ml at two months control.

Another criterion that could be considered is the occurrence of antecedent stressful events, such as exposure to general anesthesia. A retrospective study suggests that 71% of cats confirmed with TMT had experienced stressful events before presenting with congestive heart failure. In comparison, only 29% of HCM cases had a history of such events (Matos et al., 2017). Another retrospective study on cats during necropsy described stressful events before the development of CHF in 75% of young cats with endomyocarditis (Stalis et al., 1995). Toby experienced dyspnea that worsened 48 hours before the emergency consultation, which occurred one week after their general anesthesia. Hypersensitivity drug reactions have been described as a cause of myocarditis in humans, which could be a potential explanation for cats since they also receive certain drugs during anesthesia induction (Kuchynka et al., 2016).

In literature, infective agents described in association with feline myocarditis and TMT were bacteria *(Bartonella henselae, Streptococcus canis),* viruses (feline coronavirus, feline immunodeficiency virus, panleukopenia virus), and parasites (*Toxoplasma gondii, Sarcocystis felis, Hepatozoon silvestris)* (Elsheikha et al., 2006; Matsuu et al., 2007; Simpson et al., 2005; Rolim et al., 2016; Kegler et al., 2018; Joseph et al., 2018; Ernandes et al., 2019; Romito et al., 2022). In the case presented, the cat was vaccinated against panleukopenia and tested negative for FIV antibodies. No further tests were done.

Although the exact cause of this condition in this species is not yet understood, it is believed that myocardial edema with inflammatory cells could be the most likely explanation. TMT may be considered a form of myocarditis, similar to what is observed in humans, and the primary cause of reverse remodeling of TMT could be transient interstitial infiltration, which is also a common characteristic of myocarditis (Hauser et al., 1983; Hiramitsu et al., 2001; Zagrosek et al., 2008; Radovanovic et al., 2022). Another possibility is that the myocardial changes could be a result of a catecholamine stream caused by emotional or physical triggers like "stress-induced cardiomyopathy", "broken-heart syndrome", or "Takotsubo cardiomyopathy".

CONCLUSIONS

Currently, limited research is available on this medical condition in cats, with only a few published studies consisting of small case series. As a result, our current knowledge of its etiology, epidemiology, clinical features, therapeutic options, and prognosis is narrow.

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