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Case Report

Arterial thromboembolism in a cat with transient myocardial thickening



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KEYWORDS

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Abstract Feline arterial thromboembolism has been reported to be secondary to various feline cardiomyopathies; however, it has not been described in cats with transient myocardial thickening. A previously healthy, one-year-old, castrated male cat presented with acute paraparesis and congestive heart failure. Echocardiography revealed asymmetric left ventricular free wall thickening and left atrial enlargement. Antithrombotic treatment and cardiac medication resulted in reperfusion and mobility on day seven in one limb and on day 10 in the other. Different complications were managed successfully, including worsening acute kidney injury, inflammation, pleural effusion, and anemia. After three weeks, the cat was discharged and prescribed oral antithrombotic drugs (clopidogrel and rivaroxaban) and cardiac medication. Within five months, echocardiographic findings normalized, and medical treatment was gradually discontinued. To date, the cat remains healthy at 1735 days after the initial diagnosis and 1494 days after the last antithrombotic medication. To the best of our knowledge, this is the first case report on feline arterial thromboembolism combined with transient myocardial thickening, with favorable long-term survival.

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A one-year-old, castrated, domestic short-haired, indoor–outdoor male cat weighing 4.2 kg (body condition score, 4/9), without a history

of prior illness, was brought to the emergency department for acute paraparesis. At presentation, the cat was dyspneic (resting

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Abbreviations

ATE	arterial thromboembolism
HCM	hypertrophic cardiomyopathy
LA	left atrium
LV	left ventricle
SAA	serum amyloid A
TMT	transient myocardial thickening

respiratory rate, 100 breaths/min), and the heart rate was regular (200 beats/min) with normal heart sounds. Both hind limbs were cold, pale, and painful with no palpable pulses. Systolic blood pressure measured using the Doppler method was 135 mmHg in the right front limb, and no signal was detected in either hind limb. The rectal temperature was 37.8 °C. Thoracic radiography in ventral and right lateral recumbency revealed a generalized alveolar lung pattern and an increased cardiac silhouette (vertebral heart score, 8.9, reference range 6.7–8.1) [1] with left atrial (LA) enlargement. Bloodwork abnormalities included azotemia (creatinine 225 µmol/L, reference <168 µmol/L), hyperglycemia (10.8 mmol/L, reference range 3.89–6.11 mmol/L), borderline serum amyloid A (SAA, 3.9 µg/mL, reference <3.9 µg/mL), and an increased creatine kinase level (3308 U/L, reference <250 U/L).

Electrocardiography revealed sinus tachycardia with a heart rate of 210 beats per minute and a normal QRS configuration. Echocardiographic measurements were analyzed retrospectively [2,3] (Table 1) by a single author (MS). Left atrium enlargement with spontaneous echo contrast in the dilated left auricle and asymmetric hypertrophy of the left ventricular (LV) free wall were observed (Fig. 1; Table 1). The patient was diagnosed with a hypertrophic cardiomyopathy (HCM) phenotype. Abdominal aortic sonography revealed a terminal structure that restricted blood flow, which was consistent with arterial thromboembolism (ATE). At presentation, cardiac biomarkers troponin I (reference range <0.04 ng/mL) and NT-proBNP levels (reference range <100 pmol/L) were elevated at 2.28 ng/mL and >1500 pmol/L, respectively.

The cat was included in a previously published short-term treatment study [4]. Management included antithrombotic therapy (enoxaparin^a; IV bolus 1 mg/kg, followed by constant rate infusion 3 mg/kg/d, which was reduced to 2.4 mg/kg/d on day four to reach the Anti-Xa target range of 0.85–1.2 IU/mL, with an anti-Xa of 1.06 IU/mL on

Table 1 Selective two-dimensional echocardiographic variables at presentation (day 0) and follow up echocardiographic examinations.

Day	LAm _{ax} (mm)	LA/Ao	IVSd* (mm)	LVWd* (mm)	IVSd [#] (mm)	LVWd [#] (mm)
0	20	1.69	4.4	8.2	4.0	8.4
31	12	1.22	3.7	6.8	4.0	6.6
55	14	1.25	4.0	7.2	3.8	7.3
142	14	1.26	4.3	5.4	4.2	5.4
241	13	1.28	4.1	4.6	3.9	5.4
780	14	1.10	4.1	4.5	4.1	5.2
1137	14	1.15	4.3	4.8	3.9	4.9

Abbreviations: IVSd: inter-ventricular septum thickness in diastole; LA/Ao: right parasternal short-axis left atrium-to-aorta ratio; LAm_{ax}: right parasternal four-chamber long-axis maximal inner diameter left atrium; LVWd: left ventricular wall thickness in diastole; *long-axis; [#]short-axis.

day seven), platelet inhibition (clopidogrel^b; PO 75 mg/cat loading dose followed by 18.75 mg/cat/d), buprenorphine^c (IV 0.015 mg/kg q 8 h), furosemide^d (IV 1 mg/kg bolus, followed by CRI 0.125 mg/kg/h), and IV fluids (NaCl 0.9%^e 1 mL/kg/h). Oral pimobendan^f was added starting on day three (0.14 mg/kg q 12 h) because of low aortic stroke volume on echocardiography and low systolic blood pressure (95 mmHg). A pulse was detected via palpation and the Doppler method on day seven in one limb and on day 10 in the other. On day 10, enoxaparin^a was switched to subcutaneous application (0.8 mg/kg q 8 h). Complications that developed during hospitalization were successfully managed (Table 2). On day 22, the cat was discharged with mildly reduced motor activity. The packed cell volume was normal (27%), whereas the creatinine (176 µmol/L) and SAA (40.1 µg/mL) levels remained elevated. Oral medications prescribed at discharge were marbofloxacin^g (2 mg/kg q 12 h), clopidogrel (18.75 mg q 24 h), rivaroxaban^h (0.3 mg/kg q 12 h), pimobendan (0.14 mg/kg q 12 h), furosemide (2 mg/kg q 12 h), and spironolactone (2 mg/kg q 24 h). On day 31, the cat was clinically healthy, with normal motor activity. The LA and auricle sizes were normal, and the LV wall thickness continued to increase (Table 1). The creatinine level was within

^b Basics, Leverkusen, Germany.

^c Buprenovet, Bayer, Leverkusen, Germany.

^d Dimazon, MSD, Munich, Germany.

^e B. Braun, Melsungen, Germany.

^f Vetmedin, Boehringer Ingelheim, Ingelheim am Rhein, Germany.

^g Efex, Ceva, Duesseldorf, Germany.

^h Xarelto, Bayer, Leverkusen, Germany.

^a Clexane, Sanofi, Frankfurt, Germany.

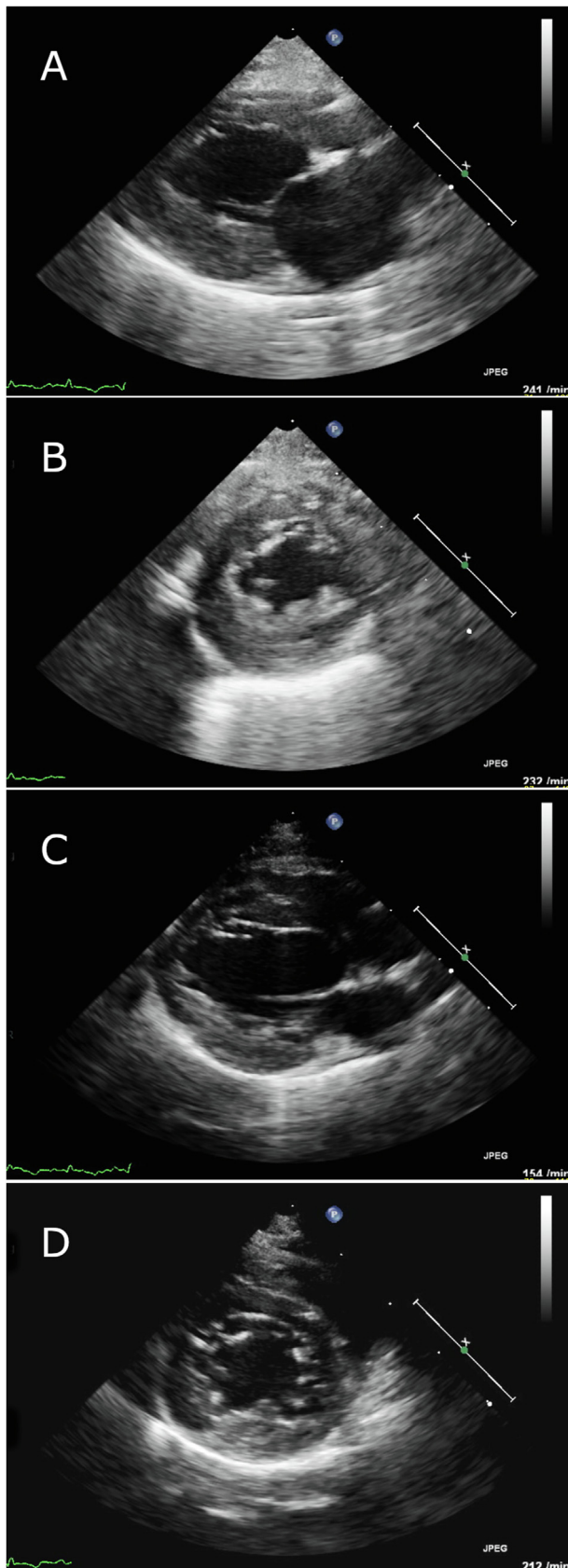


Figure 1 A–D Right parasternal long-axis and short-axis views at end-diastolic frames at initial presentation

the reference range ($160 \mu\text{mol/L}$), whereas the SAA level increased ($77.6 \mu\text{g/mL}$). Furosemide, spironolactone, and marbofloxacin treatments were discontinued.

On day 55, the cat appeared healthy, with no changes in echocardiographic measurements. The SAA level ($0.3 \mu\text{g/mL}$) was within the reference range, and clopidogrel was discontinued.

Upon re-examination on day 142, the LA size was still normal, and the LV free wall thickness decreased to 5.4 mm ($<5.5 \text{ mm}$) [2]. The cardiac biomarkers (troponin I $< 0.04 \text{ ng/mL}$; NT-proBNP 32 pmol/L) were normal. Therefore, pimobendan was discontinued, and the previous cardiac changes were attributed to transient myocardial thickening (TMT) [1]. Finally, rivaroxaban was discontinued on day 241 when the echocardiographic measurements remained normal (Table 1). Rechecks on days 780 and 1137 confirmed normal echocardiographic measurements.

To date, the cat remained healthy 1735 days after the initial diagnosis and 1494 days after the discontinuation of all medical treatments.

Discussion

Arterial thromboembolism has been described as secondary to various forms of feline cardiomyopathies with LA dilation; however, to the best of our knowledge, its association with TMT has not been previously described.

The reported prevalence of ATE in cats is approximately 0.3% in general practice and 0.6% in referral populations [5,6]. Two large studies have identified that 9%–11% of cats with HCM develop ATE [6,7]. The mean or median age at presentation with ATE is 8–12 years, with a range of 0.1–21 years [5,6]. In this case report, the cat was only one year old when paraparesis occurred. The reported incidence of ATE in young cats with HCM (<2.5 years) is only 0.7% in a large multicenter study [8].

Transient myocardial thickening is a rare condition in cats that mimics HCM and has been described in one study [2] and in some case series/reports [9–11]. Younger cats are typically affected. An elevated troponin I level is associated with mild-to-moderate LV hypertrophy and mild-to-moderate LA dilatation; however, initial

(A, B) and 142 days later (C, D). The initial left atrial enlargement (A) and severely increased left ventricular wall thickness (A, B) resolved completely, with a morphologically normal heart five months later.

Table 2 Complications during hospitalization.

Occurrence day (d)	Complication	Treatment time (d)	Treatment
4	Progressive azotemia (creatinine 825 µmol/L)	4–8	Peritoneal dialysis ^a (creatinine 277 µmol/L), discontinuation of furosemide
10	Fever (39.7 °C); increased SAA (53.3 µg/mL, reference range <3.9 µg/mL)	10–15 15–22	Amoxicillin and clavulanic acid ^b (IV 20 mg/kg q 8 h) marbofloxacin ^c (IV 2 mg/kg q 12 h)
11	Tachypnea (48 breaths/min) moderate chylothorax, (FCoV negative, bacterial culture negative)	11 12, 14	First thoracocentesis furosemide ^d (PO 2 mg/kg q 12 h), Spironolactone ^e (PO 2 mg/kg q 24 h) Second and third thoracocenteses
14	Anemia (PCV 13%, initially 42%, reference range 24%–45%)		20 mL packed red blood cell

Abbreviations: d: day; FCoV: feline coronavirus polymerase chain reaction, tested from residual abdominal fluid after peritoneal dialysis; PCV: packed cell volume; SAA: serum amyloid A.

^a Physioneal 35 Glucose 1.36%, Baxter, Unterschleißheim, Germany.

^b Hikma, Planegg, Germany.

^c Marbo FD 1%, Vetoquinol, Ismaning, Germany.

^d Dimazon, MSD, Munich, Germany.

^e Prilactone, Ceva, Duesseldorf, Germany.

symptoms and echocardiographic findings frequently mimic those of HCM [2]. However, in TMT, cardiac changes are reversible and normalize within several months. The cause for this condition is unknown, but a transient interstitial edema due to myocarditis is discussed in these cases [2,9].

The patient was initially diagnosed with ATE with an HCM phenotype. The time course of the disease, with normalization of LV wall thickness and LA size, confirmed the diagnosis of TMT. Similar changes in the final diagnosis may be observed in reported cases of HCM or ATE with long survival times [7].

Transient myocardial thickening may be associated with infectious [10,11] or non-infectious, stressful antecedent events [2]. However, the role of infectious diseases in cats is unclear. Positive blood tests for *Toxoplasma* [11] and *Bartonella* [2,10] have been reported; however, these might have been coincidental findings. Viral infections have also been reported to cause myocarditis in cats [12,13], and injuries such as thermal burns may cause TMT [14]. In this case, the cause of TMT was not identified, and an antecedent event was not reported in the last 14 days compared to that in most published cases (71%) [2].

The risk factors for ATE in cats with heart disease include severe LA enlargement, spontaneous echo contrast or thrombus, low left auricular appendage blood flow velocity, reduced LA or LV function, and a previous episode of ATE [7]. Although no cases of ATE have been reported in cats with TMT, some of those risk factors for ATE

have been described in individual cats [2,11]. In one study, spontaneous echo contrast was described in four of 21 cats with TMT and in eight of 21 cats with HCM [2]. The findings in cats with mild LA enlargement but spontaneous echo contrast in the dilated left auricle correspond to those of some published cases [2,11]. Cats with TMT have significantly less LA enlargement than cats with HCM [2]. Therefore, the risk of ATE development may be lower in cats with TMT.

Clopidogrel administration significantly reduced the likelihood of recurrent cardiogenic ATE compared with aspirin in cats [15]. The cat in this case report received a combination of rivaroxaban and clopidogrel, which was presumed to reduce the recurrence rate of ATE in another study [16]. Lifelong antithrombotic treatment is recommended for cats with CHF or ATE due to HCM [2,15]. This cat was alive and healthy 1494 days after the discontinuation of all medical treatments, consistent with the previously described favorable prognosis without long-term treatment of cats surviving TMT [2,11].

The limitation of this study was that no tests for infectious diseases and no myocardial biopsies were performed in this cat with initially presumed HCM.

In conclusion, the one-year-old cat with TMT-associated ATE was successfully treated. The myocardial thickening and LA enlargement resolved, and the cat was alive and apparently healthy without requiring any medications almost five years after the initial presentation of ATE.

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Conflict of Interest Statement

The authors have no conflict of interest to disclose.

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