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Feline aortic thromboembolism: Presentation, diagnosis, and treatment outcomes of 15 cats

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Abstract

Background: Feline aortic thromboembolism (FATE) is a fatal disease where a blood clot gets lodged into the aortic trifurcation.

Aim: This study describes the diagnosis and treatment outcome of FATE in 15 clinical cases.

Methods: Fifteen cats with a sudden onset of hind limb paresis/paralysis, vocalization, and pain were admitted to the surgery clinic. A full case history was obtained and clinical, orthopedic, neurologic, radiographic, electrocardiographic, and echocardiographic examinations were performed for each cat. The treatment protocol included daily administration of multiple anticoagulant drugs with different mode of actions and meloxicam for 7 successive days. Prophylactic anticoagulant therapy (clopidogrel and acetylsalicylic acid) was continued for 6 months. All data were statistically analyzed and the correlation between time of admission and treatment outcome was tested using Pearson's correlation coefficient.

Results: The case history and clinical, orthopedic, and neurologic examinations revealed a sudden onset of hind limb paralysis ($n = 12$) or paresis ($n = 3$) associated with vocalization and pain, absence of trauma, cold and pale paws of hind limbs ($n = 13$, 86.7%) or cyanosed hind paws ($n = 2$, 13.3%), absence of femoral pulsation, shallow and rapid open-mouth respiration (61 ± 8 breaths/minutes), hypothermia ($37.9^\circ\text{C} \pm 0.6^\circ\text{C}$) and tachycardia (155 ± 12 beats/minutes), with a muffled heart sound in four cats (26.7%). Radiography revealed no abnormalities in the hind limbs, pelvis, and spines, cardiomegaly in five cats (33.3%), mild pleural effusion and vascular pattern of the lung in six cats (40%), and Valentine's heart shape in four cats (26.7%). Electrocardiography (ECG) revealed an R-wave < 0.9 mV, prolongation of QRS interval in five cats (33.3%), and conduction disturbance in four cats (26.7%). Echocardiography was consistent with hypertrophic cardiomyopathy (HCM) in five cats (33.3%). A statistically significant ($p = 0.023$) strong negative correlation ($r = -0.6$) was reported between time of admission and subsequent early treatment and recovery from clinical signs. The treatment was successful in nine cats (60%), while four cats (26.7%) were euthanized and two cats (13.3%) were subjected to hind limb amputation, at the owners' requests.

Conclusion: Clinical signs, radiography, ECG, and echocardiography are valuable for diagnosis of FATE. The outcome of the multiple anticoagulants therapy depends mainly upon early diagnosis and treatment within the first 6 hours from the onset of clinical signs.

Keywords: Anticoagulant, Aortic thromboembolism, Cardiomyopathy, Echocardiography, Paralysis.

Introduction

Feline aortic thromboembolism (FATE) or saddle thrombus is a catastrophic lesion where a blood clot gets embolised and lodged into the aortic trifurcation (Borgeat *et al.*, 2014). It is a fatal disease that may progress very quickly as a cat can go from being healthy to be in severe distress within few minutes (Brent *et al.*, 2006). FATE is mostly reported in male cats with no breed predisposition (Chuang *et al.*, 2001; Ferasin, 2009).

The exact etiology of FATE is unknown. It may occur as a complication of feline hypertrophic cardiomyopathy (HCM). Approximately 12%–21% of cats with HCM may develop FATE (Fox, 1999; Hogan *et al.*, 1999; Fuentes, 2012). Other diseases associated with FATE may include pulmonary neoplasia and thyroid disease

(Laste and Harpster, 1995; Ladurner *et al.*, 2005; Mackman, 2008).

The potential pathogenesis of FATE is thought to be started by cardiac disease that causes abnormal blood turbulence, leading to agitation of blood and consequently micro-clot formation in the left atrium. These micro-clots grow into large clots and either the whole blood clot or part of it can be ejected from the heart and lodged in the aortic trifurcation (Pion and Kittleson, 2008; Nakamura *et al.*, 2017).

The typical symptoms of FATE are acute onset of severe pain, vocalization, pulselessness, pallor, unilateral or bilateral hind limbs paralysis, poikilothermy, and sometimes dyspnea or tachypnea (Laste and Harpster, 1995; Fox, 1999; Chuang *et al.*, 2001). The survival rates of cats with unilateral occlusion (70%–93%) are

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better than that with bilateral occlusion (15%–35%) (Ramsey *et al.*, 1996; Pion and Kittleson, 2008).

The targets of treatment of FATE are enhancement of thromboembolism dissolution, inhibition of the local thrombus, and prevention of embolization of the thrombi (Rush *et al.*, 2002). Therefore, anticoagulants such as heparin and warfarin play a crucial role in the treatment of FATE with a success rate of 37%–39% (Schoeman, 1999; Smith *et al.*, 2003). Exogenous thrombolytic agents such as streptokinase or tissue plasminogen activator may have a success rate of 0%–33% (Speakman *et al.*, 1983).

This study describes the diagnosis and treatment of FATE in 15 cats, with special reference to the correlation of time of admission and treatment outcome in a cohort of cats.

Materials and Methods

Animals

Fifteen adult cats with a sudden onset of hind limb paresis/paralysis associated with pain and vocalization were admitted to the surgery clinic at the Department of Surgery, Anesthesiology and Radiology, Faculty of Veterinary Medicine, Cairo University, Egypt, between January 2017 and December 2019. The authors followed all institutional and international guidelines for animal care and use during this study.

Diagnosis

A complete case history was collected for all cats, including the exact time of onset of clinical signs, history of trauma, previous episodes or previous treatment, and presence or absence of systemic diseases. Clinical, orthopedic, and neurologic examinations were carried out in all cats.

Radiography of the spines, pelvis, hind limbs, and thorax was carried out in all cats using an X-ray machine (Fisher, Germany). The radiographic setting factors were 45–50 kVp and 10 mA.

Six limb leads electrocardiography (ECG) was recorded for each cat using a single channel ECG machine (Yasen, ECG 901A, China). All cats were gently restrained in a right lateral recumbency with the limbs positioned in a perpendicular position to the longitudinal axis of the body. Self-adhesive patch electrodes were attached to the palmar and plantar aspects of the fore limb and hind limb paws, respectively. The six standard limb leads I, II, III, aVR, aVL, and aVF were recorded at paper settings of 10 mm/mV and 50 mm/seconds with filter band settings of 0.05–150 Hz.

Echocardiography was carried out after hair shaving and application of ultrasound coupling gel. Right parasternal views (long and short axis) were performed using a 5–9 MHz microconvex probe attached to ultrasound machine (EDAN-DUS 60, China)

Treatment

The treatment protocols included the administration of Heparin sodium (Heparin[®], CID Co., Egypt) at a dose of 300 IU/kg given SC twice daily, Clexane (Enoxaparin[®],

2,000 IU/0.2 ml, Aventis Pharm, Egypt) at a dose of 1 mg/kg given SC q12h, and Cerebrolysin (Cerebrolysin[®], EVA Pharma, Egypt) at a dose of 2 mg/kg given IM once daily for 6 successive days. Acetylsalicylic acid (Baby Aspirin, 81 mg tab[®], Nile Co., Egypt) was also given orally at a dosage of 10 mg/kg q36h. The cats were also given Meloxicam (Meloxicam[®], Amiry Co., Alexandria, Egypt) at a dose of 0.1 mg/kg given SC once daily. Physiotherapy including hind limbs massage and gentle passive movement was advised for 30 minutes three times daily for 1 week.

One week following admission, a prophylactic anticoagulant oral therapy, including Clopidogrel (Thrombo[®] 18.75 mg, EPICO, Egypt) was given at a dosage of 2–3 mg/kg once daily and acetylsalicylic acid once q36h for 6 months.

Cats with HCM were put under conservative medicinal therapy to control signs of cardiac failure, including once daily oral administrations of Digoxin (Lanoxin[®], GlaxoSmithKline, Egypt) at a dose of 0.01 mg/kg q48h, Furosemide (Lasix[®], Alexandria Co., Egypt) at a dose of 1 mg/kg, and Enalapril (Ezapril[®], Multi Pharma; Egypt) at a dose of 0.25 mg/kg whenever needed. All cats were treated on outpatient basis. The cat was considered as survived when they recovered and the functioning of their hind limbs returned.

The cats unresponsive to treatment were either euthanized or underwent hind limb amputation according to their owners' requests. Autopsy of the euthanized cats was carried out to confirm the diagnosis.

Statistical analysis

All data were collected and presented as mean \pm standard deviation. Response of cats following medicinal therapy in relation to the time of admission was recorded for each cat. The correlation between time of admission and treatment outcome was tested using Pearson's correlation coefficient. The $r \geq 0.5$ was considered as strongly correlated and $r < 0.5$ was poorly correlated. Data were considered statistically significant when $p < 0.05$. The analysis of data was carried out by the Statistical Package for the Social Sciences (SPSS) software 21 (IMP SPSS Inc, Chicago, IL).

Ethical approval

All international and institutional guidelines for animal use and care were followed.

Results

Based on clinical, orthopedic, neurologic, radiographic, electrocardiographic, and echocardiographic examinations, aortic thromboembolism was diagnosed in all cats. HCM was confirmed in 5 out of 15 cats and no other systemic diseases were reported.

The diseased animals included six Persian, five Siamese, and four domestic shorthair cats. These cats were of different ages (4.3 ± 2.1 years), both sexes (10 male and 5 female), and different weights (3.4 ± 0.8 kg).

The cats were admitted with sudden hind limb paralysis ($n = 12$, 80%) or paresis ($n = 3$, 20%). The diseased cats exhibited either bilateral ($n = 11$, 73.3%) or unilateral ($n = 4$, 26.7%) aortic thromboembolism. In bilateral aortic thromboembolism, both hind limbs were equally affected without involvement of the forelimbs. All cats had controlled urination.

Cold and pale paws of both hind limbs were reported in 13 cats (86.7%), while 2 cats (13.3%) had cyanosed hind paws. The absence of femoral pulsation was recorded in all affected limbs. Shallow and rapid open-mouth respiration (61 ± 8 breaths/minutes) was recorded in 13 cats (86.7%), while normal respiration was seen in 2 cats (13.3%). Rectal temperature was slightly reduced ($37.9^\circ\text{C} \pm 0.6^\circ\text{C}$) in all cats. Thoracic auscultation revealed tachycardia (155 ± 12 beats/minutes) with muffled heart sound in 4 cats (26.7%).

Nine cats (60%) were presented within the first 6 hours after the onset of clinical signs, five cats (33.3%) were presented within 6–24 hours and one cat (6.7%) was admitted 1 week following the onset of signs. Out of the nine cats presented within the first 6 hours after the onset of clinical signs, five cats (33.3%) had 6 months to 1-year history of HCM. No other systemic disease was reported in any of the examined cats.

Cats admitted within the first 6 hours from the onset of clinical signs showed dragging of both hind limbs while walking. The response to painful stimuli such as skin pricks or forceps clamping was very sluggish or almost absent along the hind limbs, starting from the paws till the level of hip joints. In cats admitted within 6–24 hours from the onset of the episode, the hind limbs were relaxed with no response to pain elicited by forceps clamping. In one cat that was admitted after 1 week from episode, the mucous membranes were pale with delayed capillary refilling time (>3 seconds) and the hind limbs were atrophied and the paws were cyanotic.

Upon radiographic examination of the hind limbs, pelvis, and spines, no abnormalities were detected within the musculoskeletal system. Lateral thoracic radiography revealed cardiomegaly in five cats (33.3%) and mild pleural effusion and vascular pattern of the lung in six cats (40%). Ventrodorsal thoracic radiographs revealed a characteristic Valentine's heart shape in four cats (26.7%) (Fig. 1).

ECG revealed an R-wave < 0.9 mV with prolongation of QRS interval in five cats (33.3%) and conduction disturbance (third-degree atrio-ventricular block) in four cats (26.7%).

Echocardiographic examination was consistent with HCM in five cats (33.3%) and normal in 10 cats (66.7%), as shown in Table 1.

Combined anticoagulant therapy of the cats admitted within 6 hours from the onset of clinical signs ($n = 9$, 60%) resulted in mild improvement in the function of their hind limbs and improved response to painful stimuli within the first 3 days. The cats completely

regained the function of their hind limbs within 1 week. No thromboembolic event was noted in all survived cats during a 6-months follow-up.

Anticoagulant therapy for 7 days could not improve the cats admitted within 6–24 hours or after 1 week of the episode ($n = 6$, 40%). These cats were either euthanized ($n = 4$, 26.7%) or underwent hind limb amputation ($n = 2$, 13.3%), according to their owners' request. At the owners' requests, four cats (26.7%) with bilateral aortic thromboembolism and unresponsive to treatment were euthanized. Autopsy of the euthanized cat confirmed the presence of blood clot completely occluding the terminal aorta at the level of aortic trifurcation (Fig. 2), while two cats (13.3%) with unilateral aortic thromboembolism and unresponsive to treatment were subjected to hind limb amputation, at the owners' requests (Fig. 3).

A statistically significant ($p = 0.023$) strong negative correlation ($r = -0.6$) was reported between time of admission and subsequent early treatment and recovery from the clinical signs (Fig. 4).

Discussion

FATE is a very serious disease which is life threatening and there is great interest and value of time from presentation to survival. In the present study, early admission and intervention of cats with FATE were very crucial for successful treatment outcomes. The



Fig. 1. An 8-year-old Siamese cat with FATE and HCM: Ventrodorsal thoracic radiograph showing severe cardiomegaly and a characteristic Valentine's heart shape.

Table 1. Detailed echocardiographic findings of 15 cats diagnosed with FATE.

Cat	LVIDd (mm)	LVIDs (mm)	RVIDd (mm)	RVIDs (mm)	IVSd (mm)	IVSs (mm)	LVWd (mm)	LVWs (mm)	LADd (mm)	AODd (mm)	LA/AO	FS (%)	EF (%)	Remarks
1	19.4	12.1	10.1	5.5	4.3	5.8	4.4	8.3	14.4	11.7	1.2	37.6	63.5	Normal echocardiographic parameters
2	19.6	12.3	10.3	6.9	4.2	6.1	4.9	9.1	15.5	11.6	1.3	37.2	63.0	
3	18.7	10.7	10.2	6.4	4.5	5.9	5.6	9.2	17.1	11.4	1.5	42.8	69.8	
4	18.1	10.1	9.6	5.9	4.6	6.1	6.2	9.4	14.2	12.1	1.2	44.2	71.5	
5	19.1	12.8	9.5	7.5	3.9	5.3	6.7	8.7	13.3	12.6	1.1	33.0	57.4	
6	23.1	9.3	16.1	10.2	3.6	5.8	3.1	6.2	14.1	12.6	1.1	59.7	85.8	
7	24.3	10.2	16.8	9.6	4.1	6.8	3.2	6.1	14.3	12.8	1.1	58.0	84.3	
8	24.2	10.5	17.1	8.5	4.1	6.8	3.1	6.3	15.6	13.1	1.2	56.6	83.2	
9	22.8	11.1	15.3	8.6	4.3	6.1	3	7.1	14.2	13.2	1.1	51.3	78.5	
10	22.9	11.1	18.1	11.1	4.2	5.3	4.1	8.1	14.3	12.6	1.1	51.5	78.7	
Mean	21.2	11.0	13.3	8.0	4.2	6.0	4.4	7.9	14.7	12.4	1.2	47.2	73.6	
SD	2.4	1.1	3.6	1.9	0.3	0.5	1.4	1.3	1.1	0.6	0.1	9.5	10.0	
11	11.6	8.3	20.1	13.2	7.1	10.6	7.2	11.2	20.7	12.4	1.7	28.4	52.1	HCM
12	14.1	10.1	20.6	12.6	8.2	10.6	9.3	12.3	20.2	12.4	1.6	28.4	51.5	
13	12.9	9.4	17.5	11.7	8.5	11.1	7.4	13.4	21.3	13.8	1.5	27.1	49.8	
14	13.9	10.1	17.9	12.8	8.7	12.2	8.1	12.5	22.7	13.3	1.7	27.3	50.0	
15	14.3	10.6	20.2	12.1	8.2	11.1	8	11.9	21.3	11.9	1.8	25.9	47.7	
Mean	13.4	9.7	19.3	12.5	8.1	11.1	8.0	12.3	21.2	12.8	1.7	27.4	50.2	
SD	1.1	0.9	1.4	0.6	0.6	0.7	0.8	0.8	0.9	0.8	0.1	1.1	1.7	

LVIDd = left ventricular internal diameter in diastole; LVIDs = left ventricular internal diameter in systole; RVIDd = right ventricular internal diameter in diastole; RVIDs = right ventricular internal diameter in systole; IVSd = interventricular septal thickening in diastole; IVSs = interventricular septal thickening in systole; LVWd = left ventricular wall thickness in diastole; LVWs = left ventricular wall thickness in systole; LADd = left atrial diameter in diastole; AoDd = aortic root diameter in diastole; LA/AO = left atrial diameter to aortic root ratio; FS = fractional shortening; EF = ejection fraction; HCM = Hypertrophic cardiomyopathy.

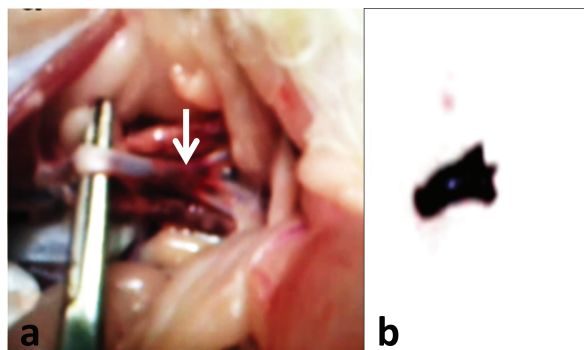


Fig. 2. FATE in a 4-year-old Persian cat: (a) Post-mortem examination of a cat with FATE showing a blood clot lodged in the aortic trifurcation. (b) The removed blood clot showing trifurcations.

first 6 hours from the onset of the episode are very critical for survival. With regard to the etiology of FATE, it may be a complication of HCM. In the present study, 30% of cats with FATE had HCM. This is in agreement with previous studies that recorded FATE in 12%–21%

of cats with HCM (Fox, 1999; Hogan *et al.*, 1999; Fuentes, 2012).

In this study, the male cats were more commonly affected with FATE than females. Also, three breeds of cats were recorded in this study with no breed predisposition. Similar findings were reported by earlier studies (Chuang *et al.*, 2001; Ferasin, 2009).

The characteristic sudden clinical signs associated with FATE should be considered in differentiating FATE from other orthopedic conditions. The cold and pale paws as well as weak or absence of femoral pulsation are characteristic clinical signs associated with FATE. Also, a controlled urination is helpful in exclusion of spinal neurological injuries. All of the clinical symptoms could be attributed to the lodgment of a blood clot into the aortic trifurcation and stoppage of arterial supply and damage of the affected hind limbs. Thoracic radiographs of five cats with FATE revealed characteristic changes in the cardiac size and shape. Similar findings were previously recorded, particularly in the late stages of HCM (Trehieu-Sechi *et al.*, 2012). Therefore, the authors recommend thoracic radiography during diagnosis of FATE. Also, it was noted that the cats with HCM were admitted within 6 hours from the



Fig. 3. A 3-year-old Persian cat with unilateral aortic thromboembolism showing unilateral hind limb gangrene.

onset of clinical signs because the owners had increased knowledge about what signs and risk to watch for.

The electrocardiographic findings, including tall R-wave, prolonged QRS interval, and third-degree atrio-ventricular block, indicated feline HCM (Moore *et al.*, 2007). In previous studies, echocardiographic diagnosis of HCM in 10 cats favored the correlation between HCM and FATE (Pion and Kittleson, 2008; Nakamura *et al.*, 2017). Similarly, HCM was recorded in 30% of cats with FATE studied here.

Although surgical embolectomy appears to be the most logical therapeutic approach in managing thromboembolism, it was a difficult option in cats with FATE due to the small-sized blood vessels and the anesthetic risk encountered in cardiac patients (Brent *et al.*, 2006).

Massive administration of anticoagulant drugs as quickly as possible was crucial in preventing the irreversible damage of the limbs. Heparin was mainly used for the prevention of extension of the existing thrombus and prevention of further clot formation. Heparin does not breakdown the already formed blood clots, but it enhances the normal body's natural thrombolytic mechanisms (Uppe *et al.*, 2013).

Clexane is a low molecular weight heparin used for treatment and prevention of deep vein thrombosis. It binds to and accelerates the anti-thrombin III (Van De Wiele *et al.*, 2010). In addition, cerebrolysin is a natural neuropeptide having several neurotrophic effects, such

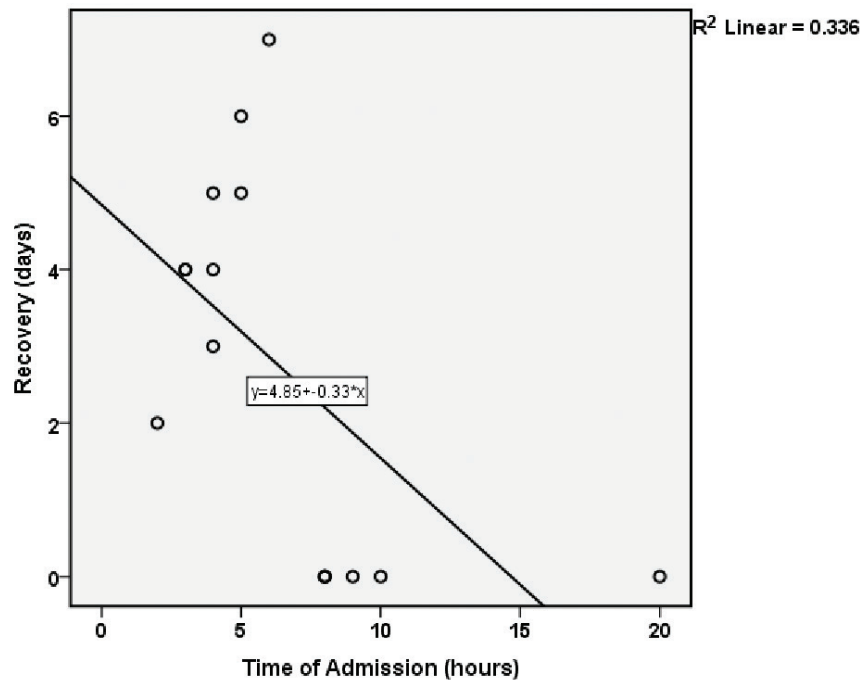


Fig. 4. The correlation between time of admission and treatment outcome of cats with FATE: Scatter plot demonstrating the correlation between time of admission and subsequent early treatment (h) of cats with aortic thromboembolism and recovery from clinical signs (days) ($r = -0.6$; $p = 0.023$; $y = 4.85 - 0.33 * x$).

as stimulation of new neuron growth, maintenance of neuron, and slows down the neural damage (Whigham *et al.*, 1998).

Early (within first 6 hours) and massive administration of anticoagulant drugs with different mode of actions resulted in rapid improvement in clinical signs and lysis of the small, recently formed, thrombus before being organized. On the other hand, delayed treatment (after 6 hours) resulted in poor prognosis. This poor prognosis may be attributed to the lodgment and adhesion of the organized thrombus within the blood vessel, as well as the ischemia and irreversible damage of the hind limbs. Like many studies, the main limitation of this study is the small population of 15 cats. Therefore, we could not study the exact effect of the age, sex, and breed of the cats studied on the survival rate.

Conclusion

Clinical signs, radiography, ECG, and echocardiography are valuable for diagnosis of FATE. Time of admission has a detrimental influence on the survival of cats with FATE. Early admission, diagnosis, and treatment with multiple anticoagulant drugs within the first 6 hours from the onset of clinical signs have a favorable survival rate of cats with FATE.

Conflict of interest

The authors declare no conflict of interests

Authors' contributions

All authors were involved in the diagnosis and treatment of the cases. All authors prepared the manuscript and approved the final version of the manuscript.

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