

Feline aortic thromboembolism: first case reported in Colombia[□]

Tromboembolismo aórtico felino: primer caso reportado en Colombia

Tromboembolismo aórtico felino: relato do primeiro caso na Colômbia

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Summary

Anamnesis and treatment approach: a cross-breed tomcat, 5 years old, with bilateral pelvic limb paresis was treated with saline solution hydration (20 drops/in), tramadol (2 mg/kg IV every 8 h), methylprednisolone succinate (18 mg, every 6 h), dimethyl sulphoxyde (0.36 mg diluted in sodium chloride, twice a day, IV) and ketoprofen (2 mg/kg, every 24 h) with no response to this treatment schedule. **Clinical and laboratory findings:** according to results of thorax and abdomen radiological tests, coldness in pelvic limbs and lack of bleeding after a deep nail cutting, clinical diagnosis of feline aortic thromboembolism was established and the cat was subjected to euthanasia after informed consent. A hypertrophic cardiomyopathy and thrombus in the lumbar abdominal aorta were found at necropsy. **Conclusion:** the first case of feline aortic thromboembolism in Colombia is reported and the most relevant findings and treatment schedule are discussed.

Keywords: *aorta, hypertrophic cardiomyopathy.*

Resumen

Anamnesis y aproximación terapéutica: un felino mestizo de 5 años de edad, con cuadro neurológico fue tratado con solución salina (20 gotas/min), tramadol (2 mg/kg i.v. cada 8 horas), succinato de metilprednisolona (18 mg totales cada 6 horas), dimetilsulfoxido (0.36 mg diluidos en cloruro de sodio cada 12 horas e.v.) y ketoprofeno (2 mg/kg cada 24 horas), sin responder al tratamiento. **Hallazgos clínicos y de laboratorio:** ante los resultados de las radiografías de tórax y el abdomen, las extremidades pelvianas frías y el no sangrado ante corte profundo de uña, se estableció el diagnóstico de tromboembolismo aórtico felino. El gato fue sometido a eutanasia previo

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consentimiento informado. A la necropsia fue hallada una cardiomiopatía hipertrófica y un coágulo en la porción lumbar de la aorta abdominal. **Conclusiones:** este es el primer reporte de un caso de tromboembolismo felino en Colombia, se discuten los hallazgos clínicos y el esquema de tratamiento más relevante.

Palabras claves: *arteria aorta, cardiopatía hipertrófica.*

Resumo

Anamnesis e abordagem de tratamento: um felino mestiço de 5 anos de idade com quadro neurológico foi tratado com solução salina (20 gotas / min), tramadol (2 mg / kg iv c / 8 horas), succinato de metilprednisolona (18 mg c total / 6 h), sulfóxido de dimetilo (0.36 mg de cloreto de sódio diluído em q12h ev) e cetoprofeno (2 mg / kg C/24 h), não respondendo ao tratamento. **Achados clínicos e laboratoriais:** com os resultados do exame radiológico de tórax e abdôme, membros pelvianos frios e falta de sangramento ao corte profundo da unha, foi diagnosticado com tromboembolismo aórtico felino. O gato foi eutanasiado prévio consentimento informado. Na necropsia foi encontrada cardiomiopatía hipertrófica e um coágulo na porção lumbar da aorta abdominal. **Conclusões:** Este é o primeiro reporte de tromboembolismo felino na Colômbia, neste artigo se discutem os achados clínicos e o cronograma de tratamento mais relevante.

Palavras-chave: *aorta, cardiomiopatía hipertrófica.*

Introduction

Arterial thromboembolism (ATE), or feline systemic arterial thromboembolism, is a disease generally related to a subjacent pathology of the heart, such as hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), restrictive cardiomyopathy (CMR), and non-classified ischemic cardiomyopathy (August, 2006; Baty, 2004; Bonagura, 2009; Kirk *et al.*, 2002). Due to its similarity with carcinogenic embolism in humans (Sodikoff, 1988), ATE was proposed as the name for carcinogenic arterial thromboembolism (August 2006). Thrombus formation is the consequence of vascular stasis preceding clot formation (Bonagura, 2009). Thromboembolism is the consequence of vascular obstruction caused by an intracardiac fibrin clot (August, 2006; Ettinger, 2007). ATE has a prevalence rate of 12% in the United States (Ettinger, 2007). Risk factors for clot formation in cats include blood stasis in cats suffering heart disease, endothelial injury, and hypercoagulopathy conditions (August, 2006; Ettinger, 2007).

In cats suffering ATE, activated platelets release vasoactive substances impairing collateral blood flow around the site of obstruction (Nelson, 1998). When a clot forms inside an aortic fragment, collateral circulation is lost and clinical signs of ischemia and severe pain become evident (August,

2006; Nelson, 1998; Schaer, 2006). When the clot is located in the abdominal aorta, the cat shows unilateral or bilateral paresis, paralysis, segmental arreflexia, contracture and pain in muscles, and cold pelvic limbs without arterial pulse (August, 2006; Nelson, 1998; Schaer, 2006; Birchard, 1996), depending on the site of obstruction. Clinical signs are sudden and progressively worsen. The affected cats respond to pain with self-mutilation, and when limbs present muscle contracture and necrosis, the prognosis is poor (August, 2006; Chandler and British Small Animal Veterinary Association, 2004). However, there is a 75% probability of clinical recovery in patients presenting friable clots (August, 2006). The most frequent laboratory finding in cats affected by ATE are augmentation of aspartate amino-transferase (AST), alkaline phosphatase (ALP), and creatinin kinase (CK) serum levels (August, 2006; Sodikoff 1988). The aim of this paper was to report the first documented case of feline thromboembolism in Colombia.

Patient examination

Anamnesis

A crossbreed tomcat, 5 years old, exhibiting pelvic limbs paresis attended a consultation at the Veterinary Medicine Center (CES University, Medellín, Colombia). Vaccination, deworming, and flea control schedules were completed.

Clinical findings and diagnostic aids used

At clinical examination, a severe abdominal pain and bilateral pelvic limb paresis with tachycardia and tachypnea were found. Superficial and deep sensibility was negative. Rectal temperature was 38.8 °C. At neurological evaluation, depression was present without neural reflexes. The patient presented Babinski's signs in pelvic limbs and a severe contracture of gastrocnemius muscles. Subcutaneous emphysema and cold limbs were observed. After the clinical exam was complete, a blood sample was taken for measuring blood parameters (Table 1), blood chemistry values (Table 2), and coagulation (Table 3). The patient was given a hydration schedule. Evidences of thyroid pathologies were not found.

Table 1. Patient's blood parameters on the first day of consultation.

Red series	Value	Units	Reference value*
Red cells	7.94	Mill/ μ l	5-10
Hematocrit	33	%	24-45
Hemoglobin	11.1	g/dl	8-15
MCV	41	fl	39-55
MCHgC	34	g/dl	30-35
Platelets	480	$\times 10^3/\mu$ l	300-800
Anisocytosis	-	- to +++	Low
Polychromasia	-	- to +++	Negative
Hypochromia	-	- to +++	Negative
Howell Jowell	-	- to +++	Low
Proteins	88	g/dl	54-78
Leukocytes	13.800	μ l	5.500-19.500
Basophils	0	μ l	0-100
Eosinophils	276	μ l	100-1500
Neutrophils	10.972	μ l	3300-10000
Bands	690	μ l	0-300
Lymphocytes	1.932	μ l	1000-4500
Monocytes	0	μ l	100-700

*Reference values used at Instituto Colombiano de Medicina Tropical (ICMT) Veterinary Clinical Laboratory.

Table 2. Patient's blood chemistry.

Analyte	Value	Units	Reference value*
Creatinine	1.39	mg/dl	0.8-1.8
ALT	1.010	U/l	3-63
AP	11	U/l	15-92
BUN	29.3	mg/dl	20-30

*Reference values used at ICMT Veterinary Clinical Laboratory.

Table 3. Coagulation tests results.

Analyte	Value	Units	Reference value*
PT Ψ	18.5	Seconds	8.7-10.5
TPT ξ	25.6	Seconds	12.3-16.7

*Reference values used at ICMT Veterinary Clinical Laboratory.

Ψ Prothrombin time. ξ Thromboplastin partial time

Therapeutic approach

First, the cat was given a hydration schedule consisting of 20 drops/min intravenous saline (0.9% NaCl). Analgesic therapy was established with tramadol (2 mg/kg, IV) every 8 h; methylprednisolone succinate (18 mg total) every 6 h; dymethylsulfoxyde (0.36 mg diluted in NaCl) every 6 h, IV; and ketoprofen (2 mg/kg) every 24 h (Abbott, 2010; August, 2006). After 24 hours the cat had not responded and cold limbs were observed. A thorax and lumbar spine radiography was taken (Figures 1 to 3). The finding of valentine's heart-like shape is indicative of dilated cardiomyopathy (Nelson, 1999) and findings in lung suggested aortic thromboembolism. After two days of hospitalization, an ATE diagnosis was established.



Figure 1. Lateral radiograph of thorax and abdominal spine, without evidence of medullar injury. A lung pattern was evidenced.



Figure 2. Ventro-dorsal radiograph of the thorax. The typical valentine's heart-like shape in feline dilated cardiomyopathy was found (Nelson, 1999). Edema was found in lungs. Lung veins were tortuous and elongated.



Figure 3. Lateral radiograph of the abdomen without evidence of medullary injuries compatible with fractures, luxation, or osteoarthritic lesions at the lumbo-sacrum segment. Coprostatics and bladder globe lesions related to neurologic injury were evidenced.

The therapeutic schedule was then completed with furosemide (5 mg/kg) given twice a day, and hydration was established with 5% dextrose. Additionally, angiotensine converting enzyme inhibitors (ACEI) antihypertensives (0.5 mg/kg) twice a day p.o., and heparine infusion (10 UI/kg/h) were administered. Cephalotine (20 mg/kg twice a day, IV) was given to prevent sepsis. The cat did not show any response to this

therapeutic schedule. On the contrary, friable and cyanotic pelvic limbs and an emphysematous process with partial skin loss and follicular fragility were found. At neurological examination, superficial or deep sensibility was not found, and a completely relaxed anal sphincter and a lack of tail motor response were observed. The owner subsequently signed the informed consent to authorize euthanasia.

Findings at Necropsy

Congestive lesions were found in caudal and intermediate lung lobes at necropsy (Figure 4). Lung congestion and/or hemorrhagic lesions suggestive of cardiogenic-type congestion were evident. Increased size of mediastinum at left ventricle was found (Figure 5), and myocardium wall was thickened (7.5 mm), compatible with HCM (Abbott, 2010). Liver and bladder were congestive (Figure 6). Petechial and hemorrhages were found in stomach mucus (Figure 7). Coprolites were the most relevant finding in colon (Figure 8). Local hemorrhagic lesions and wide congestive areas suggesting a severe ischemic infarction were found in kidneys (Figure 9).



Figure 4. Macroscopic findings in lungs. Hemorrhagic lesions and congestive areas in caudal lung lobe were found.

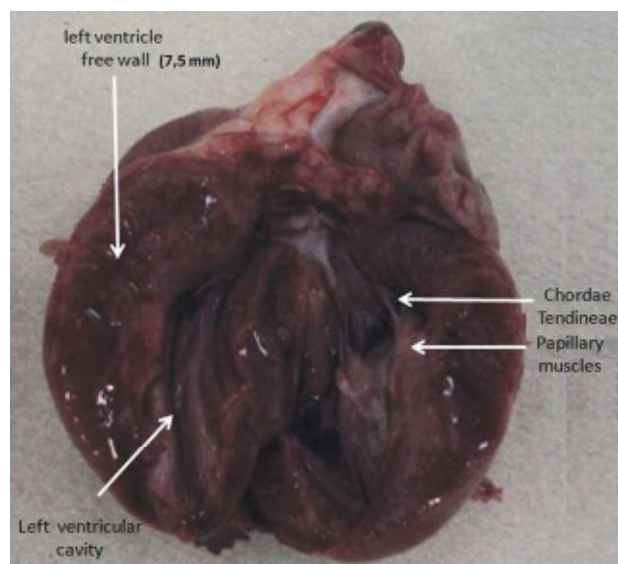


Figure 5. Macroscopic findings in the heart with evidence of left hypertrophic cardiomyopathy. Note the deformed heart in left ventricle.



Figure 6. Macroscopic findings in the liver and bladder.

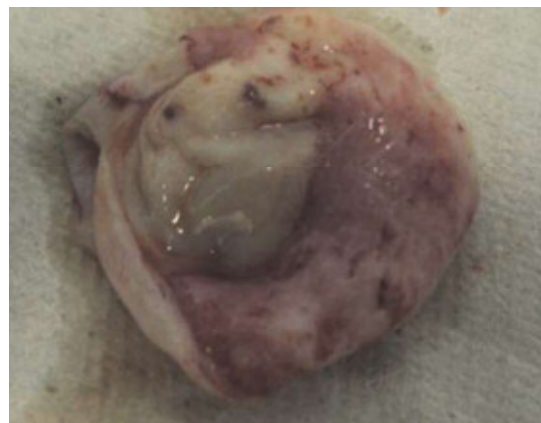


Figure 7. Macroscopic findings in the stomach. Ecchymosis lesions suggestive of inflammatory coagulopathy processes were found in mucus.

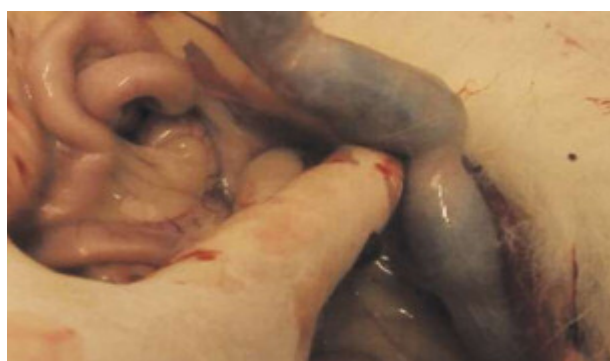


Figure 8. Macroscopic findings in colon. Coprolites indicative of coprostasis were found, probably related to neurologic injury or ischemic alteration of the abdominal aorta.

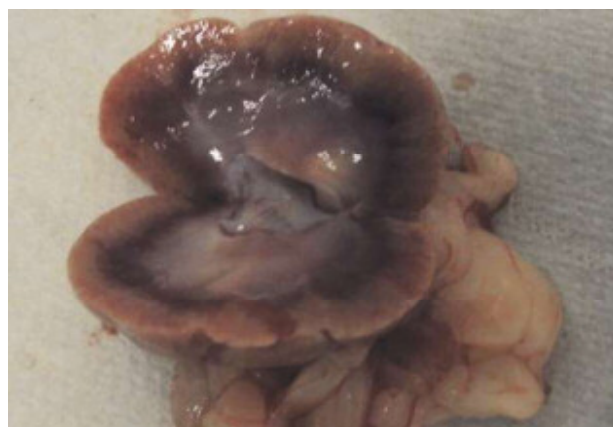


Figure 9. Macroscopic findings in kidney. Severe congestion at cortico-medullary region suggesting kidney hypoperfusion shock or prerenal ischemia.

Several ecchymosis lesions were found at the sub lumbar abdominal muscles. After dissection of the abdominal aorta, a 2 cm long clot was found at the bifurcation of the common iliac arteries (Figures 10 and 11). This finding confirmed the diagnosis of feline aortic thromboembolism.

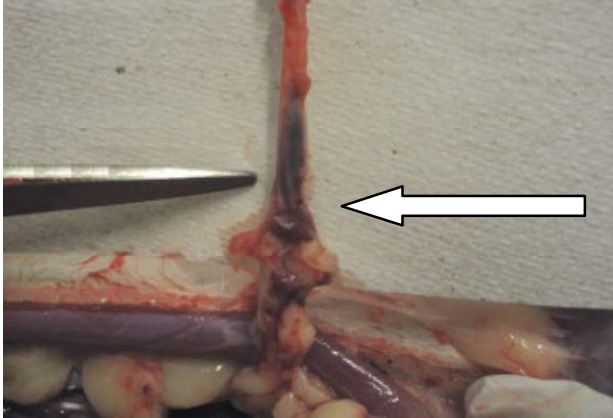


Figure 10. Macroscopic aspect of the abdominal aorta. When the artery was dissected, a clot was found causing a complete obstruction of the lumen. The complete clot longitude can be observed (indicated by arrow).



Figure 11. Mechanical extraction of the clot from the abdominal aorta. The size and aspect of the extracted material was evident.

Discussion

In this study, the first case of feline thromboembolism is reported in Colombia. Clinical signs of our patient can overlap with those of medullar compression. Survival rate of cats with partial or complete episodes of feline thromboembolism is 15% and 93%, respectively. Global survival rate is 36% to 39% (Baty, 2004). Mortality rate of cats at 36 hours after the beginning of the episode can reach 28%. However, cats could receive palliative treatment for pain amelioration (Baty, 2004). Hypertrophic cardiomyopathy (HCM) was considered the cause of thromboembolic disease in our patient because the normal value of the ventricular septal thickness in cats is 6 mm (Abbott, 2010; August, 2006).

The results of blood parameter in our patient did show slight leukocytosis with bands and increased coagulation times (PT and PTT). These findings could be associated to deficiency of extrinsic and common coagulation factors, acquired vitamin K deficiency, intravascular disseminated coagulopathy (IDC) (a clinical entity that could end in coagulopathy by consumption), and with liver/biliary insufficiencies. Once liver dysfunction and toxic entities were discarded, three probable entities must be considered in our patient (Stockham and Scott, 2008): IDC, local consumption coagulopathy, and increased PTT of unknown cause.

ALT and creatinine plasma values were increased in our patient probably as a consequence of muscle injuries caused by ischemia and necrosis associated to the thrombus (Bonagura, 2009). These findings are in agreement with the results of a retrospective study carried out with 127 cats with ATE, where more than 70% of the patients had increased ALT, AST, and creatinine plasma levels (Smith *et al.*, 2003).

The prophylaxis for thrombus formation in cats at risk of ATE has not been defined (Smith and Tobias, 2004). In our patient, clinical signs of heart or previous cardiogenic anomalies were not previously reported. For this reason, no treatment for avoiding thrombus formation had been defined. The recommended treatment for ATE in cats does not include thrombolytic therapy or omega-3 fatty acid supplementation. On the contrary, aspirin (5mg/kg/72 h) and non-fractionated L-heparin (NFH) (250 to 300 U/kg/8 h s.c.) are prescribed (Lunsford and Mackin, 2007). Clopidogrel could be used as an alternative to aspirin for long-term control of the disease (Lunsford and Mackin, 2007).

ATE with subjacent HCM was confirmed in our case by macroscopic findings in abdominal aorta. Hypertrophy could be symmetric or asymmetric, located in the subaortic region (Haggstrom, 2003; Cesta *et al.*, 2005). Primary HCM includes a genetic cause, but—could be secondary to hyperthyroidism, systemic arterial hypertension, acromegaly, and myocardial tumor inflammatory infiltration (Chetboul and Biourge, 2009). Prognosis of cats affected by ATE is reserved and the starting time for

therapy depends on speed of diagnosis. The lesions found in the pelvic limbs in our patient at day three after consultation are in agreement with a previous report (Smith *et al.*, 2003).

The radiographic finding compatible with lung edema (Figure 1) and tortuous lung veins are frequent in HCM (Nelson, 1999). Tsujino *et al.* (2005) reported chronic suppurative pyelo-nephrosis and liver and spleen congestion supporting the preventive use of antibiotics in early ATE diagnosis (Tsujino *et al.*, 2005).

Conclusion

In conclusion, the acute phase of the signs in our patient, the severe pain he exhibited, the cold temperature of his pelvic limbs, and the lack of bleeding when a deep nail cut (including nail chorion) was performed, are clinical signs highly suggestive of feline thromboembolism.

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