J Vet Clin 25(1) : 23-26 (2008)

Journal of Veterinary Clinics
www.ksvc.or.kr

Idiopathic Arterial Thromboembolism (ATE) in a Turkish Angora Cat

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(Accepted: February 5, 2008)

Abstract : A 2-year-old, female, Turkish Angora cat was presented due to hindlimb paralysis with 2-day history of vomiting and depression. Physical examination revealed weak femoral pulse and cyanotic footpads of hind limbs. Chest auscultation, thoracic radiography, and electrocardiogram were no remarkable. Abdominal ultrasonography showed suspected hyperechoic thrombus in abdominal aorta. Underlying causes of the arterial thromboembolism were not found on multiple diagnostic examinations and the cat was diagnosed as idiopathic saddle thromboembolism. Despite 4-week regimen with heparin sodium, aspirin, and diltiazem, the hind limbs were progressively and irreversibly necrotized. Therefore, coxofemoral amputation was performed. After surgery, the clinical condition of this cat returned to normal without further complications.

Key words **:** feline arterial thromboembolism, hind limb paralysis, idiopathic.

Introduction

Feline arterial thromboembolism (ATE) is commonly occurred by cardiac diseases, such as cardiomyopathies and diseases causing left atrial enlargement. A recent study showed that 69% of cats with ATE had cardiac diseases and 9% had thyroid disease (12). Rare causes of ATE include neoplasia, systemic inflammation, and endocarditis (2). Age of initial episode of thromboembolism ranged from 0.1 to 18.3 years, and a predisposition to male cat has been reported (12). Diagnostic investigations including auscultation, thoracic radiography, electrocardiography, and echocardiography are required to rule out cardiac involvements. ATE in cats induces muscle ischemia, consequently resulting in elevations of serum enzymes released from damaged muscle cells. Lower local venous glucose and higher venous lactate concentrations than those of central venous glucose and lactate may be good diagnostic indicators for ATE (8). Possible abnormalities of serum chemistry include azotemia, hypocalcemia, hyperphosphatemia, hyperkalemia, hypernatremia, and metabolic acidosis (12). Ultrasonography, angiography, and nuclear scintigraphy may be useful to locate the obstructed site.

A retrospective study of streptokinase (SK) administration in 46 cats with arterial thromboembolism indicated that there was no difference between survivors and non-survivors, based on time of administration of SK after onset of clinical signs. Hyperkalemia is a significant complication and appears to be an indicator of poor prognosis for survival (10). This case report describes acute hind limb paralysis induced by ATE without remarkable causes including cardiac disease.

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Case Report

A 2-year-old, intact female, Turkish Angora cat was presented due to acute hind limbs paralysis (Fig 1). The cat presented vomiting 2 days prior to admission and then showed lethargy, exercise intolerance, and the hind limbs paralysis. The physical and neurologic examination revealed periodically spastic hind limbs paralysis, cyanotic footpads of hind limbs, pale mucous membrane, weak or absence of femoral pulse, and swollen and painful when palpated gastrocnemius muscle. The heart rate was 160 beat per minutes without noticeable murmur. Complete blood counts revealed neutrophilia and mild to moderate thrombocytopenia. On serum chemistry profiles, mild azotemia (blood urea nitrogen: 30.9 mg/dl, reference range: 4.8-31.4 mg/dl, Creatinine: 1.6 mg/dl, reference range: 0.2-1.6 mg/dl), elevated hepatic enzymes (Alanine transaminase: 323 U/L, reference range: 13-53 U/L, Asparate transaminase: 1270 U/L, reference range: 9-69 U/L), marked elevation of lactate dehydrogenase (900 U/L, reference range: 15-277 U/L), creatine phosphokinase (2000 U/L, reference range: 10-199 U/L), hyperbilirubinemia (1.5 mg/dl, reference range: 0.3-0.9 mg/dl), hyperproteinemia (7.6 g/dl, reference range: 1.6-6.3 mg/dl), and hyperkalemia (5.5 mmol/L, reference range: 3.4-5.2 mg/dl) were observed. However, prothrombin time (PT) (6.7, reference range: 6.2-8.2 seconds), activated partial thromboplastin time (APTT) (11, reference range: 9-12 seconds), and antithrombin III (ATIII) (98%, reference range: 85-200%), fibrin degradation products (FDPs) (3, reference range: 0-10 ug/ml) measurements were normal.

An abdominal ultrasonography showed suspected hypere-

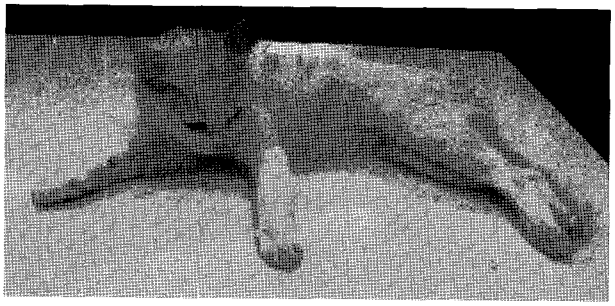

Fig 1. The cat showed hind limbs paralysis at initial examination. As the embolus was settled in the "saddle" lesion, both rear limbs were affected.

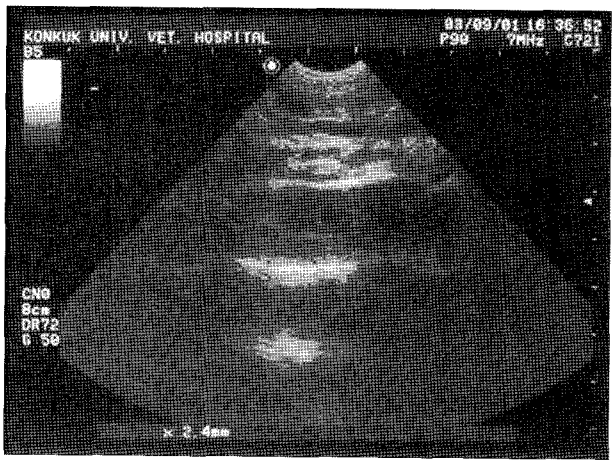

Fig 2. B-mode abdominal ultrasonography. In right lateral recumbency, thromboembolism (white arrow) within abdominal aorta was clearly visible.

choic thrombus in abdominal aorta (Fig 2). There were no remarkable findings on thoracic radiography and electrocardiogram. In addition, echocardiography revealed that there were no remarkable findings in systolic left ventricular wall diameter (6.3 mm, reference range: 5.2-10.8 mm), the size of left atrium (11.9 mm, reference range: 9.3-15.1 mm), and aorta (7.2 mm, reference range: 7.2-11.9 mm) (Fig 3).

Heartworm test was negative and Possibility of hyperthyroidism was ruled out based on T3 suppression test, basal T4 concentration, and thyroid ultrasonography. Differential diagnoses included intervertebral disc disease, trauma, hyperthyroidism, and all cardiomyopathies. Although a thickness of left ventricular wall could arouse suspicion about hypertrophic cardiomyopathy (HCM) on the echocardiography, HCM was ruled out, based on the diagnostic findings from other cardiac examinations. Because no specific etiology was identified, the cat was diagnosed as idiopathic ATE.

The cat was treated with heparin sodium (PINE® inj, Huons, Hwa-Sung, Korea, 100 IU/kg, IV, TID) and aspirin (Aspirin®, Sin Pung Pharm, Ansan, Korea, 10 mg/kg, PO, SID) at the first day of the presentation. On the second day, the cat showed hyperkalemia (8.0 mmol/L, reference range: 3.4-5.2 mmol/L),

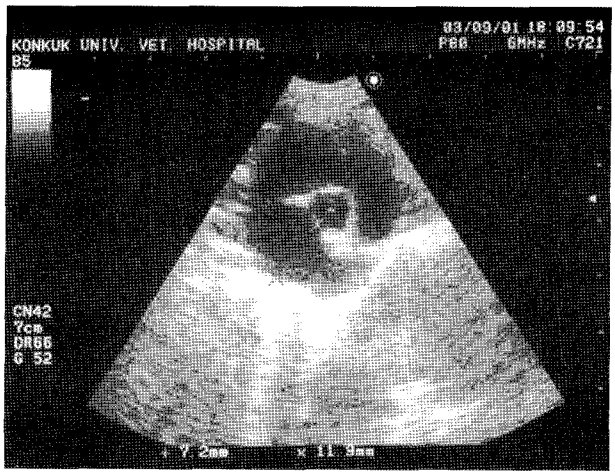

Fig 3. Two-dimensional echocardiography of the right parasternal short-axis view at aortic valve. The size of left atrium and left atrium (LA) to aorta (Ao) ratio were normal (11.9 mm, and LA/ Ao ratio : 11.9/ 7.2 = 1.65, reference range : 0.88-1.79)

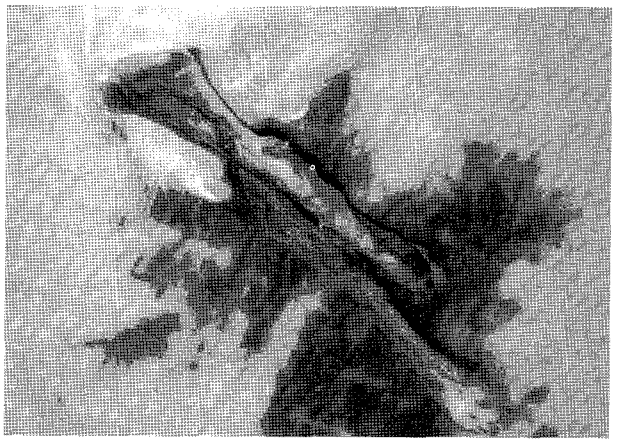

Fig 4. Self-mutation of the hindlimbs. Although generalized condition of the cat was improved after treatment, the hindlimbs were progressively and irreversibly necrotized. The hindlimbs of this cat were surgically amputated.

therefore we prescribed dextrose (20% Glucose®, Dai Han Pharm, Ansan, Korea, 3 ml/kg, IV), insulin (Novolin N®, Green Cross, Yong-In, Korea, 0.2 IU/kg, SC), and sodium bicarbonate (Sodium bicarbonate®, Dai Han Pharm, Ansan, Korea, 1.5 mEq/kg, IV bolus for 25 min followed by 1.5 mEq/kg CRI for 6 hours). On the third day, the concentration of the potassium was corrected and we added diltiazem HCl (Diltam®, Sam Chun Dang Pharm, Hwa-Sung, Korea, 1.5 mg/kg, PO, BID), since we could not fully rule out HCM. When the patient was discharged, aspirin (Aspirin®, Sin Pung Pharm, Ansan, Korea, 10 mg/kg, PO, every other day) and diltiazem were prescribed for reduced hypercoagularity and heart rate, respectively. Although generalized condition was improved, the hindlimbs were progressively and irreversibly necrotized (Fig 4). Amputation of the coxofemoral lesion was performed 4 weeks after

therapy due to necrosis of the hindlimb. For 2 weeks after the operation, we administered L-carnitine (Rexal®, Boca Raton, USA, 100 mg/cat, PO, SID), since its supplementation could be beneficial by improving myocardial energy production, and the dosage was gradually increased to 250 mg/cat. After surgery, the clinical condition of this cat returned to normal without further complications. The cat has survived more than 30 months until recently.

Discussion

Although more than 70% of cases of ATE seen in veterinary small animal practice occur in cat with cardiomyopathy, ATE can occur in cats that do not have cardiac diseases (6,12). Thyroid diseases, neoplasia in thorax or abdomen, systemic inflammation or endocarditis can be rarely occurred with ATE (2). However, in this case, our diagnostic investigation failed to find any abnormalities for these diseases.

Generally, it is thought that the thrombus is formed within the left side of the heart prior to development of ATE in cats with HCM (13). This assumption is supported by the report that 21% of cats with HCM identified to have left atrial thrombi at necropsy (7). However, the exact mechanism for formation of intracardiac thrombi is under investigation. For the formation of thrombosis, one or more of three conditions, known as *Virchow's triad*, such as alterations of the endothelial surface, blood flow, or composition of blood are required essentially. The association between cardiac disease and thrombus is explained by the fact that atrial enlargement associated with cardiomyopathy leads to blood stasis and turbulence (13).

In reference to composition of blood for coagulation in the cat with cardiomyopathy, one study reported that these cats had higher antithrombin (AT) and lower plasminogen activity than normal cat (15), and another study reported that plasma arginine and vitamin B12 concentrations were significantly low in cats with cardiomyopathy and ATE (9). In one study, platelets from cats with cardiomyopathy required less adenosine diphosphate to induce aggregation than platelet from normal cat (3). However, since well-balanced diets including adequate vitamins have been fed prior to presentation and the concentration of AT-III was within reference range, the possibility that ATE will be related to nutritional deficiency in this case is likely to be low.

Reportedly, pure breeds included Abyssinian, Himalayan, Persian, Siamese, Manx, and Maine Coon may have the predisposition for ATE (6,10,14). Smith *et al* (13) demonstrated the possibility that some cats can have genetic abnormality predisposed to hypercoagulability. In recent retrospective study for 127 cats with ATE, 3 cats had not been identified as any disease. In these 3 cats, the cardiac evaluation was normal and no disease was identified the underlying etiology that would have predisposed to ATE (12). This means apparent idiopathic thrombosis can present and then it may explain some genetic abnormality of coagulopathy. Unidentified genetic abnormality for hypercoagulability may be present. This condition could produce thrombus without overt underlying etiology.

We examined whether or not hypercoagulable disorders including diabetes mellitus, hyperadrenocorticism, and polycythemia are present based on CBC, serum biochemistry, and coagulation profiles (PT, APTT, FDPs, ATIII). But, no remarkable findings were observed in this cat.

In the cat with ATE, thrombolytic therapy was thought to have no additional benefit, compared with anticoagulant therapy. In human medicine, the efficacy and safety of tissue plasminogen activator, streptokinase, and urokinase were evaluated as thrombolytic agents for the treatment of coronary artery occlusion. However, in cats with ATE, the studies for thrombolytic therapy are limited until recently (12). In contrast to human medicine, cats treated streptokinase showed much more adverse effects including hemorrhage (10,13). Therefore, since there is little information regarding thrombolytic therapy in naturally occurring small animal diseases, it is impossible to make specific recommendations with regard to thrombolytic therapy.

In this study, we used heparin and aspirin which was used commonly for the cat with ATE to prevent additional thrombus formation and reduce thrombus extension. At initial day, unfractionated heparin was administered as low dose (100 IU/kg, TID), and aspirin was administered 10 mg/kg q 48 hours. However, in cats with ATE, additional studies are required to determine the optional dosage of heparin and aspirin. The standard dose of plasma heparin concentration in human has been applied to studies of the cat. Most studies suggested the dosages of heparin were based on studies for human being (5,11). Recent study (12) showed no difference of potency between cats receiving high-dose aspirin (>40 mg/cat q 72 hours) and low-dose aspirin (5 mg/cat, q 72 hours). But, adverse effects, such as gastrointestinal irritation are less frequent and milder for the lower dosage.

According to the several previous studies for ATE, the presence of concurrent congestive heart failure did not have significant effect on clinical signs during acute ATE episode, although it had significant harmful effect on long term survival (12,14).

In conclusion, this case report describes that clinical features and therapeutic management of acute hind limbs paralysis caused by idiopathic etiology.

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터키산 앙고라 (Turkish Angora) 고양이에서 발생한 특발성 동맥 색전혈전증 (arterial thromboembolism: ATE) 1례

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요 약 : 2일간의 구토와 침울을 동반한 후지마비를 주증상으로 내원한 2년령의 암컷 터키 앙고라 고양이에서 약한 대퇴 동맥 맥박, 후지의 청색증이 관찰되었으나 흉부 청진, 흉부 방사선, 심초음파 상에서 이상소견을 보이지 않았으며 복부 초음파상에서 복부 대동맥내의 색전으로 판단되는 고에코성 물질이 확인되었다. 대동맥 혈전의 원인이 될 수 있는 다양한 진단 검사를 시행한 결과, 특발성 안장 색전혈전증으로 진단하였다. Heparin sodium, aspirin 및 diltiazem으로 4주간 치료하였으나 양쪽 후지의 병변은 진행적이고 비가역적으로 괴사되어 대퇴부를 절단한 결과, 30개월 이상 임상적으로 건강한 생활을 하고 있다.

주요어 : 고양이 동맥 색전혈전증 (ATE), 후지 마비, 특발성