



Feline Arterial Thromboembolism: Clinical Importance and Management of Saddle Thrombus

Medine ÖZLEK^{1*}, Çağatay ESİN¹, Yücel MERAL¹

¹Department of Internal Medicine, Faculty of Veterinary Medicine, University of Ondokuz Mayıs, Samsun, Türkiye

ozlekmedine@gmail.com* (Corresponding Autor)

ORCID: 0009-0003-5139-6498

cagatay.esin@omu.edu.tr

ORCID: 0000-0002-7029-9066

ymeral@omu.edu.tr

ORCID: 0000-0001-9099-5061

Basım/Published: 25.07.2025

How to cite: Özek, M, Esin, Ç and Meral, Y (2025) Feline Arterial Thromboembolism: Clinical Importance and Management of Saddle Thrombus *VZS, 1(1), 77-94*

Atıf yapmak için: Özek, M, Esin, Ç ve Meral, Y (2025) Kedilerde Arteriyel Tromboembolizm: Eyer Trombozunun Klinik Önemi ve Yönetimi *VZS, 1(1), 77-94*.

Abstract: Feline Arterial Thromboembolism (FATE) is one of the leading causes of sudden-onset hindlimb paralysis in cats and is often the first clinical sign of an underlying, subclinical cardiac disease. This review aims to evaluate the clinical significance of thromboembolic events, particularly those involving the aortic bifurcation, commonly referred to as “saddle thrombus.” Saddle thrombus typically occurs when thrombi formed in the left atrium, usually due to conditions such as hypertrophic cardiomyopathy (HCM), enter the systemic circulation and obstruct the iliac arteries at their bifurcation. Affected cats present with dramatic clinical signs, including sudden pain, cold extremities, and paralysis. Diagnosis is primarily based on physical examination findings and is supported by Doppler ultrasonography and echocardiographic imaging. Treatment involves anticoagulant medications, analgesics, supportive therapies, and management of the underlying cardiac condition. This review provides a comprehensive overview of the pathogenesis, clinical progression, diagnostic methods, differential diagnoses, and current treatment strategies related to FATE. Rapid diagnosis and appropriate clinical intervention play a critical role in determining both the survival time and quality of life of affected patients.

Keywords: Feline arterial thromboembolism, hindlimb paralysis, hypertrophic cardiomyopathy, saddle thrombus

Kedilerde Arteriyel Tromboembolizm: Eyer Trombozunun Klinik Önemi ve Yönetimi

Özet: Feline arteriyel tromboembolizm (FATE), kedilerde ani gelişen arka bacak felçlerinin başlıca nedenlerinden biri olup, çoğu zaman altta yatan sessiz seyirli kardiyak hastalıkların ilk klinik belirtisi olabilir. Bu derleme, özellikle aort bifurkasyonuna yerleşen ve “eyer trombozu” olarak adlandırılan tromboembolik olayların klinik önemini değerlendirmek amacıyla hazırlanmıştır. Eyer trombozu, genellikle hipertrofik kardiyomiyopati (HCM) gibi kalp hastalıklarına bağlı olarak sol atriyumda oluşan trombüslerin sistemik dolaşıma katılması ve iliak arterlerin çatallanma noktasında tıkanıklığa neden olmasıyla meydana gelir. Etkilenen kedilerde ani başlayan ağrı, ekstremitelerde soğukluk ve felç gibi dramatik klinik belirtiler izlenir. Tanı, fiziksel muayene bulgularına ek olarak Doppler ultrasonografi ve ekokardiyografi gibi görüntüleme yöntemleri ile desteklenir. Tedavi sürecinde antikoagülan ilaçlar, analjezikler, destekleyici tedaviler ve altta yatan kardiyak hastalığın yönetimi büyük önem taşır. Bu derlemede, FATE’in patogenezi, klinik seyri, tanı ve ayırıcı tanı yöntemleri ile tedavi yaklaşımları güncel literatür ışığında kapsamlı bir şekilde ele alınmıştır. Saddle thrombus olgularında hızlı tanı ve uygun klinik yaklaşım, hastanın yaşam süresi ve yaşam kalitesi üzerinde belirleyici bir rol oynamaktadır.

Anahtar Kelimeler: Arka bacak felci, Eyer trombozu, feline arteriyel tromboembolizm, hipertrofik kardiyomiyopati

INTRODUCTION

Sudden paralysis of the hind limbs in cats is a serious clinical entity requiring rapid intervention by both owners and veterinarians. One of the important and fatal causes of these strokes is saddle thrombus, a thromboembolism located at the aortic bifurcation (Guillaumin, 2024; Smith et al., 2003). The term saddle thrombus literally means “saddle thrombus”. The name is an anatomical description of a rider sitting in the saddle as the thrombus is located at the bifurcation point where the abdominal aorta divides into the iliac arteries and spreads into both branches. Due to this position of the thrombus, the blood supply to both hind limbs is suddenly cut off and symptoms such as severe pain, coldness and paralysis occur (Guillaumin, 2023).

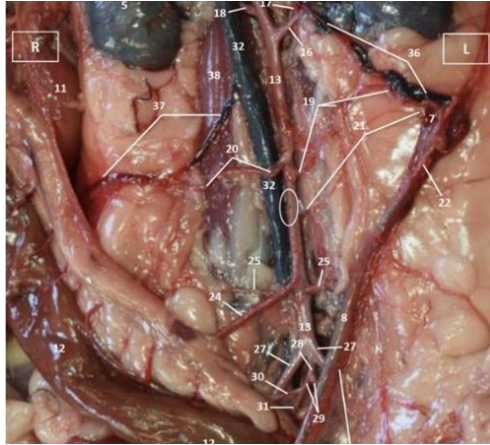


Figure 1. Bifurcation of the abdominal aorta and location of saddle thrombus in cats. Only vascular structures relevant to the study are shown in the image (adapted from Rojo Ríos et al., 2023).

Feline Arterial Thromboembolism (FATE) is a serious vascular disorder caused by the obstruction of an artery by a thrombus formed within the circulatory system. In cats, FATE usually occurs when clots formed in the heart embolize to the systemic arteries, most commonly at the bifurcation of the abdominal aorta (Hogan, 2017). The most common cause of FATE is cardiac diseases, particularly hypertrophic cardiomyopathy (HCM). In these conditions, blood flow within the heart is disrupted, endocardial damage can occur, and an environment conducive to thrombus formation is created (Yeh et al., 2025). Additionally, systemic disorders such as glomerular diseases can also increase the risk of thrombosis. In particular, glomerular lesions accompanied by proteinuria may lead to the loss of antithrombin III through urine, resulting in a hypercoagulable state that predisposes to the development of FATE (Harley & Langston, 2012). Saddle thrombus should be considered in the differential diagnosis of acute paralysis in cats, as it may mimic neurologic or traumatic causes. Early diagnosis and appropriate treatment are essential to improve survival and quality of life (Schoeman, 1999).

The aim of this review is to examine the diagnosis, treatment, and prognosis of saddle thrombus in light of current literature and to provide veterinarians with a comprehensive clinical perspective.

EPIDEMIOLOGY AND RISK FACTORS

FATE is most commonly associated with cardiomyopathies in cats and is identified as the underlying cause in approximately 90% of cases (Guillaumin, 2024). . Affected cats are typically between 8 and 12 years of age. However, at the time of diagnosis, only about 20% of cats have a previously known cardiac condition, suggesting

that FATE often represents the first clinical manifestation of cardiomyopathy . Approximately one in four cats with hypertrophic cardiomyopathy (HCM) develop FATE during their lifetime (Guillaumin, 2024). In addition, due to a higher predisposition to cardiomyopathy, the majority of FATE cases occur in male cats (Guillaumin, 2024; Smith et al., 2003).

Cardiac Causes

FATE is most commonly associated with hypertrophic cardiomyopathy (HCM). Enlargement of the left atrium, blood stasis, and the presence of spontaneous echogenic contrast (“smoke”) significantly increase the risk of thrombus formation. “Smoke” refers to an ultrasonographic finding caused by slow and turbulent intracardiac blood flow and is considered a precursor to thrombosis (Yeh et al., 2025). In addition to HCM, dilated cardiomyopathy (DCM) and unclassified cardiomyopathies (UCM) have also been linked to FATE (Pavelková, 2019; Smith et al., 2003). More than half of cats diagnosed with FATE show clinical signs of congestive heart failure, such as dyspnea, cyanosis, and pulmonary crackles (Guillaumin, 2024; Smith et al., 2003).

Non-Cardiac Causes and Genetic Risk Factors

Approximately 10% of FATE cases are associated with non-cardiac causes. The most frequently reported factor is pulmonary neoplasia, which can lead to thrombosis through direct vascular invasion or systemic hypercoagulability. In addition, infections, inflammatory conditions, hyperthyroidism, certain hormonal treatments (Guillaumin, 2024), and loss of antithrombin III due to proteinuria may also predispose to thromboembolism (Harley & Langston, 2012).

Genetic predisposition in certain cat breeds increases the risk of HCM and consequently FATE. Breeds such as Maine Coon, Ragdoll, Persian, Siamese, British Shorthair, Norwegian Forest Cat, Scottish Fold, Van, Burmese, Birman, and Abyssinian are among those at higher risk due to mutations in sarcomeric protein genes (e.g., MyBPC3) (Luis Fuentes et al., 2020; März et al., 2015).

PATHOGENESIS AND PATHOPHYSIOLOGY

The hemostatic system is regulated by various mechanisms to maintain blood in a fluid state within the vessels. Upon vascular injury, primary and secondary hemostatic processes are activated to initiate clot formation (Gale, 2011; Yeh et al., 2025). Feline arterial thromboembolism (FATE) has a complex pathophysiology, where commonly seen cardiomyopathies in cats contribute to thrombus formation by affecting Virchow's triad: disrupted blood flow, endothelial injury, and hypercoagulability (Yeh et al., 2025).

Systemic Hypercoagulability

The secondary hemostasis process stabilizes the clot through fibrin formation. In cats with cardiomyopathy, increased thrombin generation or insufficient anticoagulant mechanisms may lead to systemic hypercoagulability, potentially contributing to the development of FATE. However, the evidence supporting this mechanism remains inconsistent (Yeh et al., 2025). A study investigating thrombosis-related biomarkers found that cats showing signs of FATE had higher levels of TAT complexes and fibrinogen compared to healthy controls. However, these differences were not statistically significant. Increased TAT and decreased antithrombin levels may indicate active thrombin generation and consumption of antithrombin (Bédard et al., 2007; Yeh et al., 2025).

Viscoelastic tests have been used to evaluate coagulation disorders in cats with FATE. According to the cell-based model of coagulation, blood cells also play a crucial role in thrombus formation (Moses et al., 2024). Some studies using the VCM VET device have reported hypocoagulability in FATE-affected cats; however, these findings may have been influenced by secondary factors (Moses et al., 2024; Yeh et al., 2025). Other studies have not found significant evidence of systemic hypercoagulability. The sensitivity of the VCM device in detecting hypercoagulability in cats remains unclear, indicating the need for further research in this area (Yeh et al., 2025).

Platelet Activation

Systemic hypercoagulability is considered a key factor in the development of FATE. Platelets play a central role in primary hemostasis through a three-step process: adhesion, activation, and stabilization. Increased platelet activation has been demonstrated in both preclinical and clinical cases of HCM in cats. In particular, Maine

Coon cats with severe HCM have shown significantly elevated levels of platelet activation, platelet-derived microparticles, and platelet–endothelial adhesion molecules (Tablin et al., 2014; Yeh et al., 2025). Furthermore, in cats with transient myocardial thickening, platelet hyperreactivity has been observed and linked to intracardiac thrombus formation. Interestingly, in these cases, left atrial dilation was present alongside enhanced systolic atrial function. These findings suggest that platelet priming a heightened state of platelet responsiveness may contribute to the pathogenesis of FATE (Yeh et al., 2025). However, the underlying mechanisms remain unclear (Yeh et al., 2025).

Alterations in Fibrinolysis

Fibrinolysis is a tightly regulated mechanism that restores blood flow by breaking down clots after their formation. In this process, plasmin degrades fibrin to resolve the thrombus. Recent data suggest that congestive heart failure (CHF) may influence the development of FATE. Notably, the incidence of FATE is higher in cats with cardiogenic pulmonary edema compared to those with pleural effusion. This difference may be explained by the intrinsic fibrinolytic activity of pleural effusion, which can reduce thrombus formation (Busato et al., 2022; Yeh et al., 2025). Conversely, pulmonary edema may create a more proinflammatory environment, potentially triggering immunothrombosis a complex process involving both inflammation and coagulation (Yeh et al., 2025).

Immunothrombosis

Immunothrombosis, a component of innate immunity, may play a role in the development of FATE (Ryan & O’Neill, 2022; Yeh et al., 2025). Neutrophil extracellular traps (NETs), which are prothrombotic structures, can promote clot formation when excessively produced (Li & Tablin, 2018). In cats with FATE and hypertrophic cardiomyopathy (HCM), elevated levels of cell-free DNA (cfDNA) and citrullinated histones (citH), which are markers of NETs, have been detected (Li et al., 2023). These structures may contribute to platelet activation via Toll-like receptor 2 and 4 (TLR2/4) (Fuchs et al., 2011; Semeraro et al., 2011; Yeh et al., 2025). Emerging research points to a potential role for NETs and histones as both diagnostic indicators and therapeutic targets in feline arterial thromboembolism (FATE) (Yeh et al., 2025).

Hemodynamic Stasis

In cats with cardiomyopathy, structural changes in the left ventricle and atrium lead to increased left atrial pressure, dilation, and impaired contractility, resulting in blood flow stasis. This stagnant flow promotes intracardiac thrombus formation (Yeh et al., 2025). Spontaneous echocardiographic contrast (SEC), also known as “smoke,” reflects red blood cell aggregation due to low flow velocities and is considered a significant risk factor for FATE (Yeh et al., 2025). A reduced left auricular flow velocity on echocardiography has been identified as an independent predictor of both SEC and FATE risk (Yeh et al., 2025).

Endothelial Injury

The endothelium normally produces various anticoagulant and fibrinolytic substances to prevent unwanted blood clotting (Neubauer & Zieger, 2022; Yeh et al., 2025). However, in cats with hypertrophic cardiomyopathy (HCM), these protective mechanisms can become impaired (Ciaramella et al., 2006; Yeh et al., 2025). Specifically, decreased levels of thrombomodulin may promote easier clot formation (Ciaramella et al., 2006; Yeh et al., 2025). Additionally, severe enlargement of the left atrium causes endothelial damage, leading to increased levels of von Willebrand factor (vWF) (Yeh et al., 2025). Elevated vWF has been found both in the blood plasma and the heart’s inner lining (endocardium) and is associated with the development of FATE (Stokol et al., 2008; Yeh et al., 2025). This suggests that endothelial dysfunction in cats with cardiomyopathy increases the risk of thrombosis (Yeh et al., 2025).

CLINICAL FINDINGS

Clinical signs of arterial thromboembolism (FATE) in cats vary depending on the location of the occluding thrombus. The most common form, the “saddle thrombus” at the aortic bifurcation, presents with sudden paralysis, pain, pulselessness, poikilothermy, pallor, and cyanotic nail beds in the pelvic limbs; these signs are collectively referred to clinically as the “5P rule” (Guillaumin, 2024). Embolism can also cause serious complications in various organs such as the mesenteric infarction, renal infarction, or cerebral embolism, which may manifest as abdominal pain, vomiting, kidney dysfunction, and neurological symptoms (Hogan, 2017). Heart murmurs or arrhythmias may be auscultated, but most cases have normal heart sounds on examination (Fuentes,

2012a). The majority of cats with FATE have previously undiagnosed cardiomyopathy and often concurrent congestive heart failure (Fuentes, 2012a).



Figure 2. A cat diagnosed with FATE and presented to Ondokuz Mayıs University Animal Hospital shows prominent cyanosis of the nail beds in the pelvic limbs. This finding is a clinical indicator of ischemic damage and impaired peripheral circulation due to arterial thromboembolism and is one of the characteristic symptoms evaluated within the scope of the “5P rule”

DIAGNOSTIC METHODS

Laboratory tests

Feline arterial thromboembolism (FATE) diagnosis relies heavily on laboratory tests to both identify the disease and investigate underlying causes. Common blood biochemistry findings include stress-induced hyperglycemia, azotemia, hyperphosphatemia, and electrolyte imbalances such as hypocalcemia, hyponatremia, and hyperkalemia (Fuentes, 2012a). Elevated creatine kinase (CK) indicates muscle damage, while increased ALT and AST suggest liver or muscle inflammation (Pavelková, 2019). Venous blood glucose levels in the affected limb are lower than systemic levels, whereas lactate levels are elevated both aiding diagnosis (Guillaumin, 2024). Coagulation test results are often within normal limits, but elevated D-dimer levels can be observed (Fuentes, 2012a). NT-proBNP is a biomarker reflecting cardiac muscle stress or strain and helps differentiate cardiac causes in cats with respiratory distress, though it may be normal in early-stage hypertrophic cardiomyopathy (HCM) (Hsu et al., 2009; Yeh et al., 2025). Troponin I indicates cardiac injury and is especially useful in distinguishing asymptomatic HCM cats from healthy ones. However, levels may also rise due to comorbidities like kidney disease or hyperthyroidism. Significant Troponin I increases

are often seen in FATE cases (Hertzsch et al., 2019; Yeh et al., 2025). These biomarkers should be interpreted alongside other diagnostic tests for accurate assessment (Yeh et al., 2025).

Radiography

Thoracic radiographs are considered the most effective method for detecting pulmonary edema and can also help identify other thoracic or lung abnormalities, such as tumors (Pavelková, 2019). However, cardiomegaly may not always be apparent (Pavelková, 2019).

Blood Pressure Measurement

Blood pressure measurement can be performed using Doppler or oscillometric methods. The Doppler method is also used to assess blood flow, but pulses may still be palpable in cases of partial arterial blockage (Côté et al., 2011; Pavelková, 2019). Most cats with ATE present with low blood pressure, which is often associated with congestive heart failure (Pavelková, 2019). Hypertension usually occurs due to pain, and regular blood pressure monitoring is necessary for managing both high and low blood pressure cases (Pavelková, 2019).

Echocardiography and Ultrasonography

Since the type of cardiomyopathy is of little importance, emergency echocardiography is generally not required (Fuentes, 2012a). Most cats show left atrial enlargement and some have left ventricular dysfunction. Spontaneous echo contrast is an important finding that increases the risk of FATE. Left atrial enlargement alone may not indicate heart failure, as it is present in most FATE cases. Left atrial dilation is assessed by the ratio of left atrial to aortic diameter and is a risk factor for FATE. The size of the left ventricle and left atrium can be measured using point-of-care ultrasound. Low flow velocity (<20 cm/s) in the left atrial appendage indicates severe dysfunction and increased thrombus risk (Yeh et al., 2025). In newly symptomatic cats, thrombus may be visible in the terminal aorta by ultrasound; however, in cases with pelvic limb paralysis, diagnosis is usually based on clinical findings. The absence of a visible thrombus does not rule out

ATE. Vascular imaging is more useful in cases without detectable heart disease (Fuentes, 2012a).

Electrocardiography (ECG)

Electrocardiography (ECG) is useful for detecting arrhythmias but is not the primary diagnostic tool for cardiomyopathy or congestive heart failure. Common findings include left ventricular enlargement patterns, sinus tachycardia, premature ventricular and supraventricular beats, prolonged QRS duration, and left atrial enlargement (Pavelková, 2019). Severe hyperkalemia may cause bradycardia and atrial standstill (Côté et al., 2011; Pavelková, 2019).

Infrared Thermography

Infrared thermography can be used not only to directly visualize thrombi in the affected limbs but also as a useful method to assess reperfusion (Guillaumin, 2024).

DIFFERENTIAL DIAGNOSES

Aortic thromboembolism (ATE) may present with symptoms similar to traumatic paraplegia but is differentiated by physical examination findings. In traumatic paraplegia, the femoral pulse is usually preserved and cyanosis is not seen, whereas pulse loss and cyanosis are common in ATE. The most important differential diagnoses of ATE are spinal cord diseases (acute trauma, disc disease, neoplasia, fibrocartilagenous embolism) (Schoeman, 1999). Definitive diagnosis is based on imaging or palpation of arterial occlusion and identification of the underlying cause.

TREATMENT METHODS AND MANAGEMENT

The emergency management of FATE includes pain control, treatment of the underlying disease, supportive care, thromboprophylaxis, and, if necessary, thrombolytic therapy and support of collateral circulation (Guillaumin, 2024).

Analgesia

Severe pain is common in FATE cases, and it should be assumed that all cats with FATE experience clinically significant pain; therefore, pain management is crucial. Methadone is the first choice, followed by a continuous fentanyl infusion (1–5 µg/kg/hour

IV) with titratable effect. Oxymorphone, hydromorphone, and buprenorphine are also alternative opioids. Pain assessment should use objective methods such as the Feline Grimace Scale; based on this, additional sedation, analgesia, and anxiolytics may be required (Adami et al., 2023; Evangelista et al., 2019).

Treatment of Primary Disease

For emergency stabilization in FATE, cardiac disease and congestive heart failure should be evaluated. Most patients have hypertrophic cardiomyopathy and CHF, and treatment includes oxygen support, furosemide (1–4 mg/kg), and ACE inhibitors (0.5 mg/kg). Diuretics should be used cautiously, and kidney function must be closely monitored (Guillaumin, 2024; Smith & Tobias, 2004).

Thromboprophylaxis

Anticoagulant therapy is recommended in FATE cats to reduce the risk of thrombus progression and may include unfractionated heparin, low molecular weight heparin, aspirin, clopidogrel, rivaroxaban, or apixaban (Guillaumin, 2024). Clopidogrel is commonly preferred but its effectiveness can vary due to genetic variants in cats, making personalized treatment with molecular testing possible (Ueda et al., 2021; Yeh et al., 2025). CURATiVE guidelines recommend clopidogrel combined with LMWH, while newer oral anticoagulants like rivaroxaban and apixaban have also been found safe and effective (Goggs et al., 2019). Additionally, emerging treatments like rapamycin show cardioprotective and antithrombotic potential, especially in cats with hypertrophic cardiomyopathy, though further research is needed (Saxton & Sabatini, 2017; Yeh et al., 2025).

Thrombolysis

According to recent expert opinion, thrombolytic therapy (e.g. TPA, alteplase, reteplase) may be considered in acute (<6 hours) cases of FATE after individual risk-benefit assessment (Guillaumin, 2024). Studies have shown that pulse return and limb function improved in some cases, but the overall survival rate ranged from 27-45%. In a study using reteplase, this rate was as high as 90%. Although TPA has a targeted effect due to its fibrin specificity, it carries the risk of systemic thrombolysis at high doses (Guillaumin, 2024; Guillaumin et al., 2019). Commonly used protocol: 1 mg/kg dose, 10% bolus, remaining 90% infusion over 1 hour; CRI of 0.1-0.5 mg/kg/hour is

recommended for pediatric/small animals (Guillaumin, 2024; Mulcaire-Jones et al., 2020).

Supportive Care and Adjunctive Therapies

Supportive care and adjunctive therapies in FATE cases should be addressed in an integrated approach. Hospitalization is usually 2-5 days, but full recovery of neuromuscular function may take 2-6 weeks. During this period, physical therapy (e.g. passive joint movements and heat applications) should be applied according to the patient's tolerance (Guillaumin, 2024; Smith & Tobias, 2004). Acute kidney injury and reperfusion injury, which are common complications, may develop even without thrombolytic therapy (Guillaumin, 2024). Therefore, the use of drugs to support collateral circulation has gained importance. Antioxidant and vasodilator agents such as pentoxifylline may increase circulation (Guillaumin, 2024), while cyproheptadine may prevent paralysis by protecting collateral circulation (Agnew & Korman, 2014; Guillaumin, 2024). In addition, drugs such as cilostazol and flunarizine are among the supportive treatment options, offering potential benefit in the management of complications related to FATE (Guillaumin, 2024).

SURGICAL AND INTERVENTIONAL THROMBECTOMY APPROACHES

Surgical or interventional removal or dissolution of the thrombus in the treatment of feline aortic thromboembolism (FATE) is theoretically feasible, but is generally not recommended due to high risks and complications. Surgical thrombus removal is associated with serious side effects and high mortality, while interventional thrombectomy methods are associated with technical difficulties and anesthesia risks (Fuentes, 2012b; Pavelková, 2019). Alternative rheolytic thrombectomy also has limited

success, with survival and recurrence rates similar to medical therapy (Reimer et al., 2006).

PROGNOSIS AND COMPLICATIONS

Care after feline arterial thromboembolism, especially in bilateral cases, should be performed in multidisciplinary veterinary centers with 24/7 support due to the high risk of complications. In aortic occlusion, platelet-derived vasoconstrictors such as serotonin and thromboxane trigger reperfusion injury, which occurs in 20-50% of cases and acute kidney injury (30%) is a common serious complication (Guillaumin, 2024). Sudden death is rare (10-15%) and usually occurs in the first 12 hours (Pavelková, 2019). Body temperature, number of affected limbs and limb lactate levels are important for prognosis (Guillaumin, 2024). Renal function and potassium should be monitored in the early period and support should be provided with diuretic adjustment and intravenous fluid therapy if necessary (Côté et al., 2011). Preservation of collateral circulation with vasodilator and antioxidant agents (pentoxifylline, cyproheptadine, etc.) may be potentially beneficial in reducing future complications. With supportive care, 27-35% of patients were discharged, with a median survival of 117-345 days (Pavelková, 2019).

Feline arterial thromboembolism (FATE) is a serious condition with a high risk of recurrence, particularly in relation to hypertrophic cardiomyopathy. Long-term thromboprophylaxis plays a key role in management. The FAT CAT study showed that clopidogrel is more effective than aspirin (Guillaumin, 2024). The curative guidelines recommend clopidogrel, UFH and LMWH (Goggs et al., 2019; Guillaumin, 2024). Rivaroxaban has also been found safe. The SUPER-CAT trial found no significant difference between clopidogrel and rivaroxaban, while combination therapies (16.7% relapse rate) show promise (Lo et al., 2022).

CONCLUSION

In the future, a focus on individualized and combination-based therapeutic approaches is recommended to improve outcomes in feline arterial thromboembolism. There are still significant knowledge gaps in the pathogenesis of FATE in areas such as procoagulant platelets, immunothrombosis, impaired fibrinolysis and endothelial

damage. A better understanding of these processes is critical for the development of targeted treatment and prevention strategies.

REFERENCES

Adami, C., Filipas, M., John, C., Skews, K., & Dobson, E. (2023). Inter-observer reliability of three feline pain scales used in clinical practice. *Journal of Feline Medicine and Surgery*, 25(9), <https://doi.org/1098612X231194423>.

Agnew, W., & Korman, R. (2014). Pharmacological appetite stimulation. *Journal of Feline Medicine and Surgery*, 16(9), 749–756. <https://doi.org/10.1177/1098612X14545273>

Bédard, C., Lanevski-Pietersma, A., & Dunn, M. (2007). Evaluation of coagulation markers in the plasma of healthy cats and cats with asymptomatic hypertrophic cardiomyopathy. *Veterinary Clinical Pathology*, 36(2), 167–172. <https://doi.org/10.1111/j.1939-165X.2007.tb00203.x>

Ciaramella, P., Piantedosi, D., Lindquist, E., Loria, A. Di, Cortese, L., Skeels, M., & Persechino, A. (2006). Plasma Thrombomodulin (TM) Concentration in Cats with Cardiomyopathies. *Veterinary Research Communications*, 30(S1), 289–291. <https://doi.org/10.1007/s11259-006-0063-3>

Côté, E., MacDonald, K.A., Meurs, K.M., & Sleeper, M.M. (2011). *Feline Cardiology*. Wiley. <https://doi.org/10.1002/9781118785782>

Evangelista, M.C., Watanabe, R., Leung, V.S.Y., Monteiro, B.P., O’Toole, E., Pang, D.S.J., & Steagall, P. V. (2019). Facial expressions of pain in cats: the development and validation of a Feline Grimace Scale. *Scientific Reports*, 9(1), 19128. <https://doi.org/10.1038/s41598-019-55693-8>

Fuchs, T.A., Bhandari, A.A., & Wagner, D.D. (2011). Histones induce rapid and profound thrombocytopenia in mice. *Blood*, 118(13), 3708–3714. <https://doi.org/10.1182/blood-2011-01-332676>

Fuentes, V.L. (2012a). Arterial Thromboembolism. *Journal of Feline Medicine and Surgery*, 14(7), 459–470. <https://doi.org/10.1177/1098612X12451547>

- Fuentes, V.L. (2012b). Arterial Thromboembolism. *Journal of Feline Medicine and Surgery*, 14(7), 459–470. <https://doi.org/10.1177/1098612X12451547>
- Gale, A.J. (2011). Continuing Education Course #2: Current Understanding of Hemostasis. *Toxicologic Pathology*, 39(1), 273–280. <https://doi.org/10.1177/0192623310389474>
- Goggs, R., Blais, M., Brainard, B.M., Chan, D.L., deLaforcade, A.M., Rozanski, E., & Sharp, C.R. (2019). American College of Veterinary Emergency and Critical Care (ACVECC) Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (Curative) guidelines: Small animal. *Journal of Veterinary Emergency and Critical Care*, 29(1), 12–36. <https://doi.org/10.1111/vec.12801>
- Guillaumin, J. (2023). Why could thrombolysis be an option for cats with acute aortic thromboembolism? *Companion Animal*, 28(11), 2–6. <https://doi.org/10.12968/coan.2023.0028>
- Guillaumin, J. (2024). Feline aortic thromboembolism: recent advances and future prospects. *Journal of Feline Medicine and Surgery*, 26(6). <https://doi.org/10.1177/1098612X241257878>
- Guillaumin, J., Gibson, R.M., Goy-Thollot, I., & Bonagura, J.D. (2019). Thrombolysis with tissue plasminogen activator (TPA) in feline acute aortic thromboembolism: a retrospective study of 16 cases. *Journal of Feline Medicine and Surgery*, 21(4), 340–346. <https://doi.org/10.1177/1098612X18778157>
- Harley, L., & Langston, C. (2012). Review Article Compte rendu Proteinuria in dogs and cats. *In CVJ* (Vol. 53).
- Hertzsch, S., Roos, A., & Wess, G. (2019). Evaluation of a sensitive cardiac troponin I assay as a screening test for the diagnosis of hypertrophic cardiomyopathy in cats. *Journal of Veterinary Internal Medicine*, 33(3), 1242–1250. <https://doi.org/10.1111/jvim.15498>
- Hogan, D.F. (2017). Feline cardiogenic arterial thromboembolism. *Veterinary Clinics of North America: Small Animal Practice*, 47(5), 1065–1082. <https://doi.org/10.1016/j.cvsm.2017.05.001>

- Hsu, A., Kittleson, M.D., & Paling, A. (2009). Investigation into the use of plasma NT-proBNP concentration to screen for feline hypertrophic cardiomyopathy. *Journal of Veterinary Cardiology*, 11, S63–S70. <https://doi.org/10.1016/j.jvc.2009.02.005>
- Li, R.H.L., Fabella, A., Nguyen, N., Kaplan, J.L., Ontiveros, E., & Stern, J.A. (2023). Circulating neutrophil extracellular traps in cats with hypertrophic cardiomyopathy and cardiogenic arterial thromboembolism. *Journal of Veterinary Internal Medicine*, 37(2), 490–502. <https://doi.org/10.1111/jvim.16676>
- Li, R. H. L., & Tablin, F. (2018). A comparative review of neutrophil extracellular traps in sepsis. *Frontiers in Veterinary Science*, 5. <https://doi.org/10.3389/fvets.2018.00291>
- Lo, S.T., Walker, A.L., Georges, C.J., Li, R.H., & Stern, J.A. (2022). Dual therapy with clopidogrel and rivaroxaban in cats with thromboembolic disease. *Journal of Feline Medicine and Surgery*, 24(4), 277–283. <https://doi.org/10.1177/1098612X211013736>
- Luis Fuentes, V., Abbott, J., Chetboul, V., Côté, E., Fox, P.R., Häggström, J., Kittleson, M.D., Schober, K., & Stern, J.A. (2020). ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. *Journal of Veterinary Internal Medicine*, 34(3), 1062–1077. <https://doi.org/10.1111/jvim.15745>
- März, I., Wilkie, L.J., Harrington, N., Payne, J.R., Muzzi, R.A.L., Häggström, J., Smith, K., & Luis Fuentes, V. (2015). Familial cardiomyopathy in Norwegian Forest cats. *Journal of Feline Medicine and Surgery*, 17(8), 681–691. <https://doi.org/10.1177/1098612X14553686>
- Moses, I.A., Hallowell, T.C., & Johnson, J.A. (2024). Feline aortic thromboembolism with and without congestive heart failure did not exhibit hypercoagulability using a novel viscoelastic coagulation monitor. *American Journal of Veterinary Research*, 85(8). <https://doi.org/10.2460/ajvr.24.03.0065>
- Mulcaire-Jones, J.P., Bailly, D.K., Frank, D.U., Verma, A.R., Barney, B.J., & Siefkes, H.M. (2020). Spontaneous aortic thrombosis in neonates: a case report and review of literature. *Cardiology in the Young*, 30(1), 95–99. <https://doi.org/10.1017/S1047951119003093>
- Neubauer, K., & Zieger, B. (2022). Endothelial cells and coagulation. *Cell and Tissue Research*, 387(3), 391–398. <https://doi.org/10.1007/s00441-021-03471-2>

- Pavelková, E. (2019). Feline arterial thromboembolism. *Companion Animal*, 24(8), 426–430. <https://doi.org/10.12968/coan.2019.0021>
- Reimer, S.B., Kittleson, M.D., & Kyles, A.E. (2006). Use of rheolytic thrombectomy in the treatment of feline distal aortic thromboembolism. *Journal of Veterinary Internal Medicine*, 20(2), 290–296. <https://doi.org/10.1111/j.1939-1676.2006.tb02859.x>
- Ryan, T.A.J., & O'Neill, L.A.J. (2022). Innate immune signaling and immunothrombosis: New insights and therapeutic opportunities. *European Journal of Immunology*, 52(7), 1024–1034. <https://doi.org/10.1002/eji.202149410>
- Saxton, R.A., & Sabatini, D.M. (2017). mTOR signaling in growth, metabolism, and disease. *Cell*, 168(6), 960–976. <https://doi.org/10.1016/j.cell.2017.02.004>
- Schoeman, J.P. (1999). Feline distal aortic thromboembolism: A review of 44 Cases (1990–1998). *Journal of Feline Medicine and Surgery*, 1(4), 221–231. <https://doi.org/10.1053/jfms.1999.0049>
- Semeraro, F., Ammollo, C.T., Morrissey, J.H., Dale, G.L., Friese, P., Esmon, N.L., & Esmon, C.T. (2011). Extracellular histones promote thrombin generation through platelet-dependent mechanisms: involvement of platelet TLR2 and TLR4. *Blood*, 118(7), 1952–1961. <https://doi.org/10.1182/blood-2011-03-343061>
- Smith, S.A., & Tobias, A.H. (2004). Feline arterial thromboembolism: an update. *Veterinary Clinics of North America: Small Animal Practice*, 34(5), 1245–1271. <https://doi.org/10.1016/j.cvsm.2004.05.006>
- Smith, S.A., Tobias, A.H., Jacob, K.A., Fine, D.M., & Grumbles, P.L. (2003). Arterial thromboembolism in cats: Acute crisis in 127 cases (1992–2001) and long-term management with low-dose aspirin in 24 cases. *Journal of Veterinary Internal Medicine*, 17(1), 73. <https://doi.org/10.1892/0891-6640>
- Stokol, T., Brooks, M., Rush, J.E., Rishniw, M., Erb, H., Rozanski, E., Kraus, M.S., & Gelzer, A.L. (2008). Hypercoagulability in cats with cardiomyopathy. *Journal of Veterinary Internal Medicine*, 22(3), 546–552. <https://doi.org/10.1111/j.1939-1676.2008.0098.x>
- Tablin, F., Schumacher, T., Pombo, M., Marion, C.T., Huang, K., Norris, J.W., Jandrey, K.E., & Kittleson, M.D. (2014). Platelet Activation in Cats with Hypertrophic

Cardiomyopathy. *Journal of Veterinary Internal Medicine*, 28(2), 411–418.
<https://doi.org/10.1111/jvim.12325>

Ueda, Y., Li, R.H.L., Nguyen, N., Ontiveros, E.S., Kovacs, S.L., Oldach, M.S., Vernau, K.M., Court, M. H., & Stern, J. A. (2021). A genetic polymorphism in P2RY1 impacts response to clopidogrel in cats with hypertrophic cardiomyopathy. *Scientific Reports*, 11(1), 12522. <https://doi.org/10.1038/s41598-021-91372-3>

Yeh, N.S., Shaverdian, M., & Li, R.H.L. (2025). Evolving FATE: A new lens on the pathogenesis and management of feline cardiogenic arterial thromboembolism. *Animals*, 15(11), 1630. <https://doi.org/10.3390/ani15111630>