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Feline arterial thromboembolism in cats: Clinical presentation, diagnosis, therapeutic management

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Abstract

Feline Arterial Thromboembolism (FATE), also known as saddle thrombus when the terminal aorta is involved, is a serious and potentially life-threatening vascular emergency in cats. This case report describes a 4-month-old male weighing 2kg that presented with a 5-day history of hindlimb paralysis, dragging of the hindlimbs. Clinical examination revealed cold hindlimbs, pale hind paw pads, and bilateral femoral pulse deficits. Colour Doppler ultrasonography revealed reduced aortic calibre, pulsation, and blood-flow velocity distal to the aortic bifurcation. Echocardiography showed increased left ventricular wall and interventricular septal thickness, along with increased ejection fraction and fractional shortening, consistent with hypertrophic cardiomyopathy. Electrocardiography revealed an atrial premature complex and increased P-wave width. The cat was managed with antithrombotic and cardiac-directed therapy and showed clinical improvement following treatment. This case highlights the importance of early recognition of the characteristic clinical signs of FATE and the diagnostic value of Doppler ultrasonography and echocardiography in assessing vascular obstruction and underlying cardiac disease. Prompt diagnosis and appropriate therapeutic intervention may improve the clinical outcome in cats affected with arterial thromboembolism.

Keywords: Feline arterial thromboembolism, FATE, saddle thrombus, hypertrophic cardiomyopathy, colour doppler, mixed-breed cat

Introduction

Feline arterial thromboembolism is described in the supplied conference material as one of the most common forms of thromboembolism encountered in clinical veterinary medicine. It is caused by sudden obstruction of arterial blood flow, commonly affecting the hindlimbs, and is characterized by the five cardinal signs or “5 Ps”: pain, paralysis/paresis, pulselessness, pallor and poikilothermy. The supplied material reports a prevalence of 1 in 142 new feline admissions at the Teaching Hospital, University of Pennsylvania and notes that FATE is mostly reported in tom cats.

History and presentation

A 4-month-old male (Fig. 1) weighing 2 kg was presented to RVP-IVRI with a history of trauma, hind paw pads paler than fore paw pads (Fig. 2) and sudden onset of hindlimb paralysis. Loss of appetite and lethargy had been present for approximately 5 days, accompanied by vocalization.



Fig 1: Injured cat resting on a blanket
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Fig 2: Close-up of a small animal’s paw

Clinical examination

Table 1: Clinical examination findings of the affected animal

Parameter	Finding
Temperature	100 °F
Hindlimbs	Colder than forelimbs
Mucous membrane	Pink
Hind paw pads	Paler than fore paw pads
Heart rate	160/min, arrhythmic
Respiration rate	35/min
Femoral pulse	Deficit bilaterally
Withdrawal reflex	Present in all limbs
Skeletal examination	No abnormality on palpation

Diagnostic evaluation

Haemato-biochemical findings

Table 2: Hematological and biochemical parameters with reference ranges

Parameter	Observed	Reference range
Hb	14.3 g/dL	12-18
PCV	48.8%	37-55
TEC	8.57 ×10 ⁶ /μL	5.5-8.5
TLC	15.7 ×10 ³ /μL	6-17
Platelets	200 ×10 ³ /μL	150-450
Total protein	5.97 g/dL	5.4-7.5
Albumin	2.32 g/dL	2.3-3.1
SGPT	30.48 IU/L	10-109
BUN	28.89 mg%	19-34
Creatinine	0.84 mg/dL	0.9-2.2

On X-ray, there are no changes seen in the vertebral column (Fig. 3).



Fig 3: Lateral abdominal X-ray of a small animal

On ECG R waves > 0.9mv suggestive of HCM (Fig. 4)

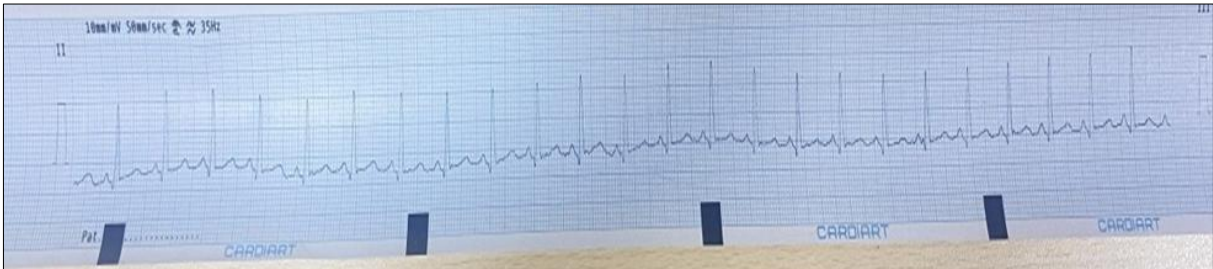


Fig 4: Atrial flutter with variable AV conduction - ECG rhythm strip

Colour Doppler examination demonstrated no blood flow and no pulse were evident in the aorta, while the observed spectral spikes were attributed to respiratory disturbances. A

hyperechoic mass was visualized within the aortic lumen (Fig. 5& 6).

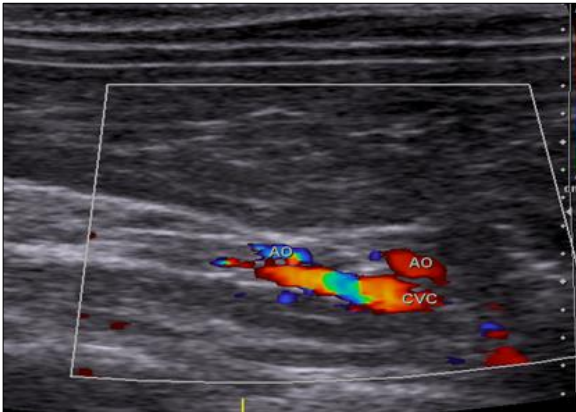


Fig 5: Color doppler ultrasound of the abdominal aorta and Inferior Vena Cava (IVC)

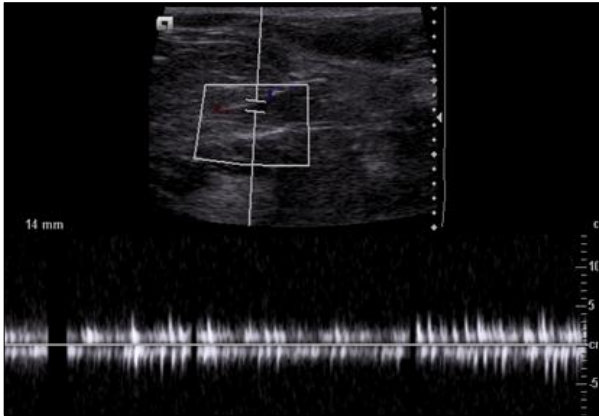


Fig 6: Spectral doppler ultrasound showing arterial blood flow

Echocardiography showed increased left ventricular wall and septal thickness and abnormal cardiac systolic function, with EF 91% and FS 51.8%. Recorded measurements were

IVSd 7.2 mm, LVWd 7.4 mm, LVIDd 8.1 mm and EDV 1.2 mL. The cardiac findings were reported as HCM (Fig. 7 & 8).



Fig 7: Echocardiographic (ultrasound) view of the heart

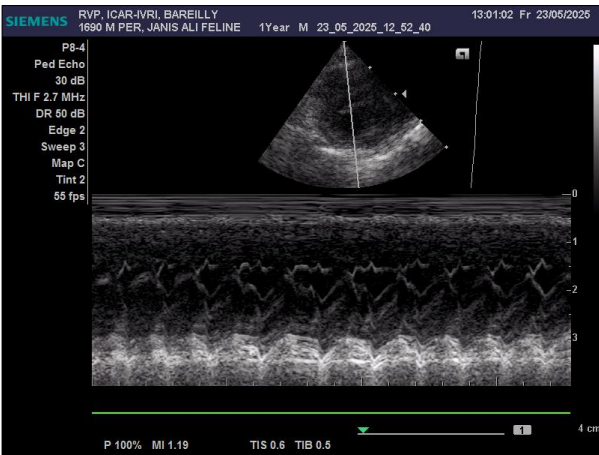


Fig 8: M-mode echocardiographic assessment of the heart

Therapeutic management

Table 3: Treatment regimen, dosage, and duration/frequency

Treatment	Dose	Duration/frequency
DNS infusion	500 mL @ 100 mL IV BID	5 days
Heparin injection	0.1 mL SC BID	10 days
Methylprednisolone succinate	4 mg IM q48h	3 doses
Clopidogrel 75 mg	¼ tablet PO OD	10 days
Ascorbic acid (Limcee)	1 tablet PO BID	7 days
Enalapril 2.5 mg	1/3 tablet PO BID	14 days
Furosemide + spironolactone	¼ tablet PO OD	14 days
Betadine ointment	Topical	7days

Imaging and follow-up

At 10 days after treatment, ultrasonography showed improvement in blood flow on colour and spectral Doppler. Cat showed ambulatory improvement and started walking. Femoral pulses became palpable. Further management consisted of clopidogrel 75 mg at ¼ tablet PO once daily

lifelong and enalapril 2.5 mg at 1/3 tablet PO once daily for 2 months. A low-cholesterol diet, physiotherapy and leg warming were advised.

Follow-up colour Doppler demonstrated restoration of blood-flow velocity to the external iliac arteries of the hindlimbs. (Fig. 9 & 10).



Fig 9: Color doppler ultrasound demonstrating blood flow

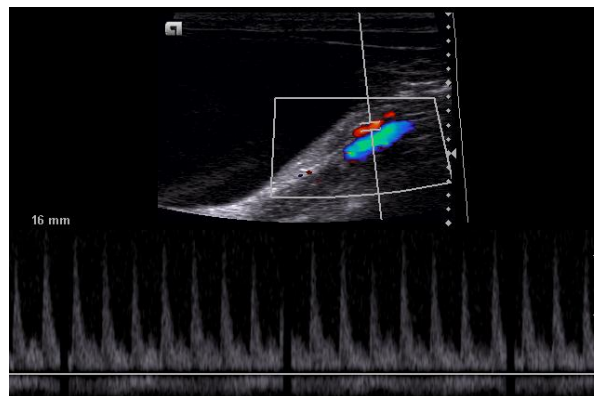


Fig 10: Doppler ultrasound with color flow imaging

Discussion

The present case demonstrated the characteristic clinical features of feline arterial thromboembolism (FATE), including hindlimb paralysis, cold extremities, pallor and bilateral femoral pulse deficits. These findings correspond to the classical “5 Ps” pain, paresis/paralysis, pulselessness, pallor and poikilothermy described in feline arterial thromboembolism (Smith *et al.*, 2003; Smith and Tobias, 2004) [4, 5]. The combination of acute hindlimb dysfunction with absent or diminished peripheral pulses should prompt immediate consideration of FATE, as timely diagnosis and treatment are critical for limb recovery (Hogan, 2017) [1].

Colour Doppler ultrasonography provided important evidence of vascular compromise, revealing reduced aortic calibre, pulsation and blood-flow velocity distal to the aortic bifurcation. Restoration of blood flow to the external iliac arteries on follow-up examination further supported improvement in arterial perfusion following therapy. Doppler ultrasonography is therefore valuable for both confirming vascular obstruction and monitoring treatment response in cats with FATE (Hogan, 2017; Yeh *et al.*, 2025) [1, 6].

A clinically important finding in the present case was the concurrent Hypertrophic Cardiomyopathy (HCM),

characterized by increased left ventricular wall and interventricular septal thickness, together with increased ejection fraction and fractional shortening. Electrocardiography additionally revealed an atrial premature complex and increased P-wave width. HCM is one of the major cardiac disorders associated with cardiogenic arterial thromboembolism in cats, and the development of FATE is closely linked to intracardiac thrombus formation and subsequent systemic arterial embolization (Luis Fuentes *et al.*, 2020; Hogan, 2017) [3, 1]. The echocardiographic findings in this case therefore provide a plausible underlying cardiac substrate for thromboembolism. Assessment for underlying cardiomyopathy is consequently important in cats presenting with FATE, as identification and management of the primary cardiac disease form an integral part of long-term case management (Luis Fuentes *et al.*, 2020) [3].

The cat was treated with clopidogrel along with cardiovascular-directed therapy and showed clinical improvement, accompanied by restoration of arterial blood flow on follow-up Doppler examination. Clopidogrel is an established antiplatelet agent for prevention of recurrent cardiogenic arterial thromboembolism, with the FAT CAT study providing evidence supporting its use in cats with

FATE (Hogan *et al.*, 2015) ^[2]. Management should also address the underlying cardiac disease to reduce the risk of recurrent thromboembolic events (Hogan, 2017) ^[1].

7. Conclusion

This case highlights the close clinical association between FATE and underlying HCM in cats. Recognition of the characteristic clinical signs, supported by colour Doppler ultrasonography and echocardiographic evaluation, facilitates early diagnosis and identification of an underlying cardiac cause. In cats with FATE, timely antithrombotic therapy combined with appropriate management of concurrent cardiomyopathy may improve vascular perfusion and clinical outcome.

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