


**PATIENT**

Nephew Karney

**PRESENTING CLINICAL SIGNS**

 acute onset posterior paresis- bilateral; no murmur, no arrhythmia. R/O spinal dz /IVDD/cardiomyopathy with thrombus. Please check for saddle thrombus. On prednisolone  
 Abnormal PE/Chem/CBC/UA Results: wnl; USPG 1.057

**SPECIES**

Feline

**ULTRASONOGRAPHIC EXAMINATION OF THE HEART**
**BREED**

DSH

**SEX**

Neutered

**AGE**

14 Years

**WEIGHT**

7.4 Pounds

FELINE CARDIAC PARAMETERS	BODY WEIGHT (kg)	HR (BPM)	IVSd (cm)	LVIDd (cm)	LVWd (cm)	FS (%)	EF (%)
NORMAL PARAMETER	-----	150-240	0.3-0.6	1.0-2.1	0.25-0.6	35-67	80-100
PATIENT		NM	0.6	1.4	0.6	55	
FELINE CARDIAC PARAMETERS	LA/AO (Boon)	LA/AO HEART BASE (Sisson)	LA 2D 4-chamber long axis AS to FW (Sisson) (cm)	LVOT VEL. (m/s)	RVOT VEL. (m/s)	IVRT (m/)	
NORMAL PARAMETER	<1.5	0.88-1.79	0.7-1.7	<1.6	<1.3	40-60	
PATIENT	1.62	2.04		1.0	0.9	NM	

Adapted from June Boon, Veterinary Echocardiography, 1998  
 Sisson D et al. JVIM 1991; 5: 232, Jacobs et al. Am J Vet Res 1985; 46:1705

**INTERPRETED BY**

 Eric Lindquist, DMV  
 DABVP, Cert. IVUSS

**Cardiac Presentation**

Moderate myocardial remodeling noted. Contractility was adequate. Mild volume overload noted in the left atrium and left ventricle. Moderate left atrial dilation noted. Concentric hypertrophy noted in the ventricle with thickened mitral valve and mitral insufficiency. Minor pericardial effusion noted. No clots noted in the left atrium, however aortic thrombosis was noted and "smoke" was present in the left atrium. The thrombus measured approximately 1.0 cm in length with approximately 70-80% occlusion of the distal aorta. Right ventricular hypertrophy also noted.

**IMAGING PERFORMED BY**

Diane McFadden

**ULTRASONOGRAPHIC FINDINGS**

- Left-sided heart failure
- Hypertrophic cardiomyopathy with left atrial clot
- Aortic thrombosis

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**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**
**REFERRING VET**

Dr. Lovell

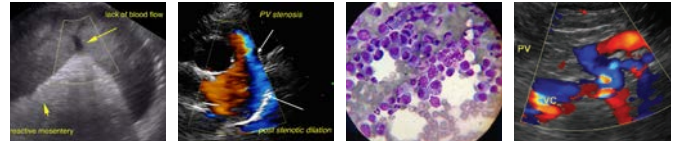
Plavix therapy, low-dose Lasix and ACE inhibitor recommended. Recheck sonogram daily upon the iliac trifurcation. Guarded prognosis.

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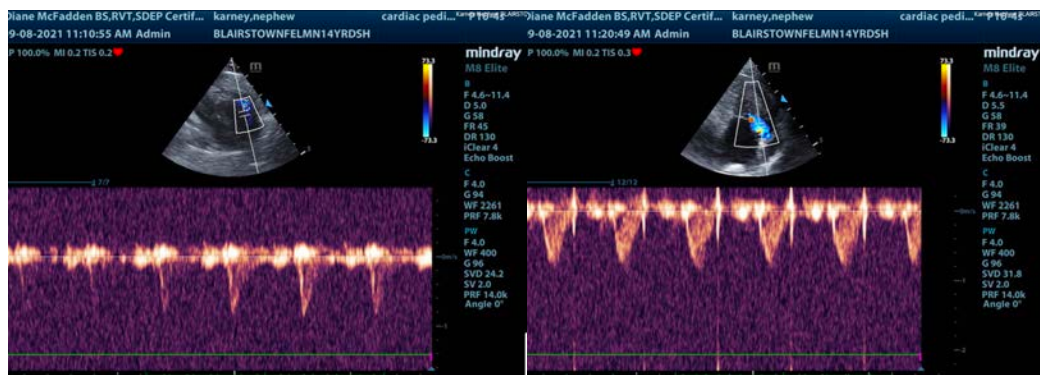
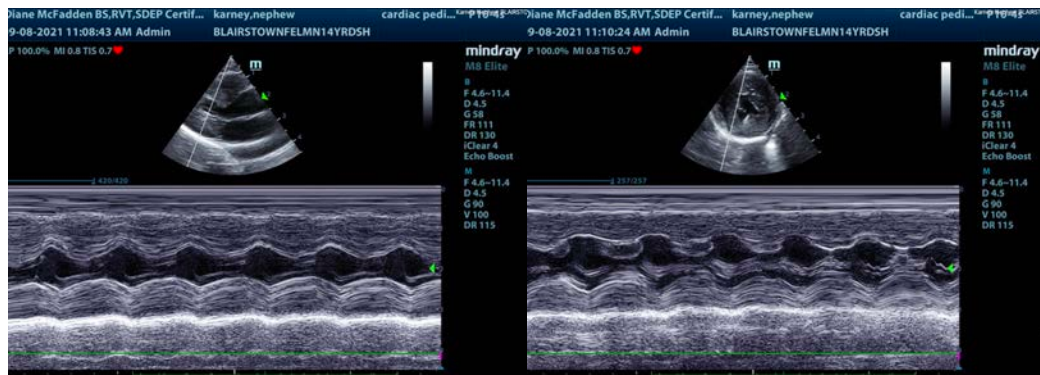
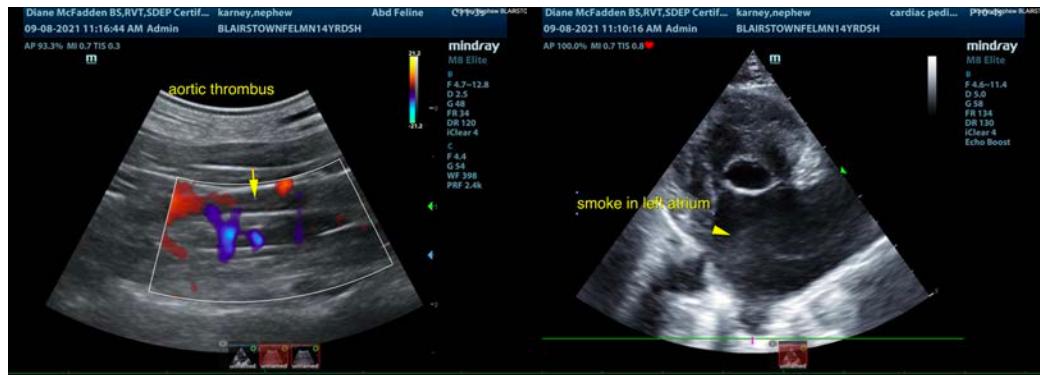
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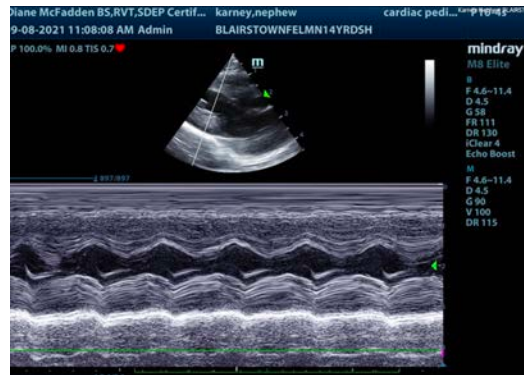
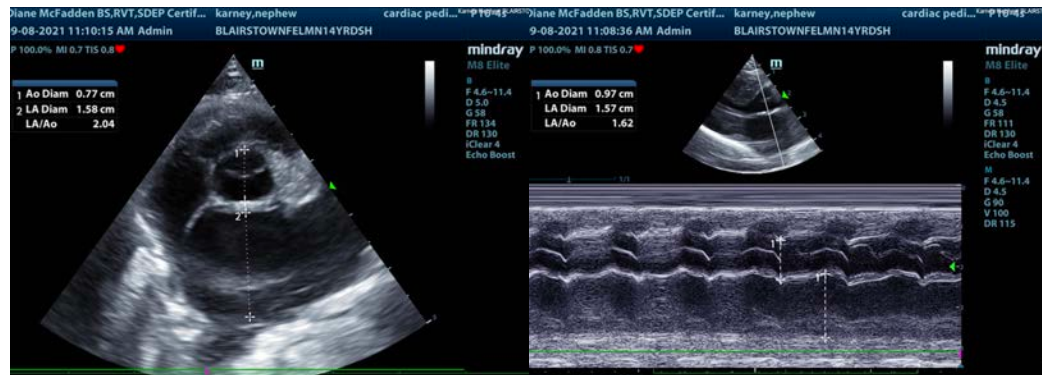
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

**Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com**  
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**Feline Arterial Thromboembolism**

<http://www.sonopath.com/FelineATE>

**Description:** Arterial thromboembolism (ATE) occurs when a thrombus develops in the left atrium and then moves to a distant site. In cases where pulmonary neoplastic cells have been found in the thrombus, those particular emboli may have originated in the lungs. Traditionally, this disease has been considered to carry a grave prognosis in all instances; however, recent studies report a survival rate of approximately 40% with treatment (approximately 35% of all feline patients are euthanized without attempted treatment). If cats survive the initial embolic event, re-embolization represents a likely cause of future morbidity and mortality (approximately 25% of patients will have recurrent thromboembolic episodes). Yet, mortality due to complications caused by underlying heart



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disease is three times more likely to result in morbidity than is recurrent ATE. Prognosis is determined by assessing the underlying cardiac disease, degree of vascular obstruction, and any underlying conditions of comorbidity. Paralysis of one limb carries a much better prognosis than if two limbs are affected.

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Body temperature and congestive heart failure (CHF) are two key indicators of both short- and long-term survivability. In general, less than 50% of cats will survive to discharge despite aggressive therapy. A correlation between body temperature and prognosis has been demonstrated: a body temperature of 100°F indicates a 73% survival rate; a core temperature of 99°F suggests a 50% survival rate; and at 97°F it is reasonable to expect 25% survival. The presence of CHF upon initial presentation does not affect survival to discharge but does make a significant difference in the long-term prognosis. The median survival time (MST) in a patient with CHF is 77 days, regardless of thromboembolism. Recent data comparisons indicate that no cat with CHF has ever lived longer than 254 days.

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**Clinical Signs:** Loss of limb function is common. Cats most often display hind limb paresis or paralysis, which indicates a thrombus located at the aortic trifurcation. The classic clinical sign is the absence of a pulse; however, there can be other reasons why one may not detect a pulse: it can be difficult to palpate in an overweight cat; the patient may be hypotensive, which can result in poor peripheral pulses; or the cat may have a partially obstructed artery, which can result in the loss of a pulse. The presence of a heart murmur potentially supports a diagnosis, but the lack of one does not rule out either ATE or CHF. Other presentations may include: tachypnea; hypothermia; loss of function in a forelimb; neurologic signs attributable to the occlusion of a local artery; and abdominal pain and/or vomiting due to a mesenteric arterial thrombus and intestinal necrosis. Firm, stiff muscles may be seen acutely. Acute renal failure can also occur with renal artery thrombosis.

**IMAGING PERFORMED BY**

Diane McFadden

**Diagnostics:** The increased collection of data from thromboelastography (TEG) in human studies may provide further insight into feline cases and allow for earlier detection of hypercoagulable states; however, TEG is not widely available as of yet. Currently, clinical signs and traditional coagulation panel alterations are used to arrive at the presumptive diagnosis. If serum muscle enzymes, such as AST and creatine kinase, are within normal limits, arterial thromboembolism is not likely to be the cause of clinical signs. Common biochemical abnormalities include hyperglycemia, azotemia, and acid-base disturbances.

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It is important to note that in the overwhelming majority of cases, cardiac disease or neoplasia is an underlying disorder. Commonly associated neoplasias include, but are not limited to: pulmonary carcinoma, hepatocellular carcinoma, vaccine-associated fibrosarcoma, and squamous cell carcinoma. (Very few patients do not exhibit underlying abnormalities that fall into these categories.) Therefore, routine diagnostics should include radiographs, serum chemistry, urinalysis, CBC, total T4, ECG, and echocardiogram. Concurrent underlying pathologies affect long-term survival rates, so the early identification of such disorders can help provide a more accurate prognosis.

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**Treatment:** The goals of acute ATE management are: 1) to manage pain appropriately; 2) to treat CHF if present; 3) to minimize ongoing clot formation; 4) to improve blood flow; and 5) to provide optimal supportive



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care. Nearly all ATE patients will demonstrate dyspnea and tachypnea, but only 50% of patients will present with CHF. Therefore, the respiratory rate or pattern is not a reliable indicator of CHF and should not be used alone. One should not administer diuretics without confirming CHF. At minimum, radiographs should be obtained prior to the use of diuretics. Analgesia is of primary therapeutic importance, as the negative effects of inadequate analgesia on recovery are well documented. Initial stabilization and supportive care for at least 48-72 hours is key before electing euthanasia.

**Treatment Steps for ATE:**

- Analgesia: Buprenorphine (0.01-0.03 mg/kg given orally or IV BID) may be effective, but is often inadequate. Other opiates, such as methadone (0.6 mg/kg slow IV Q4-6hr) or CRI fentanyl (3-5 mcg/kg slow IV, followed by 2-5 mcg/kg/hr as a CRI), are likely to be more effective.
- If there is radiographic or other evidence of CHF, then furosemide may be administered at 1-2 mg/kg IV.
- Unfractionated heparin (UH) (250-300 IU/kg given IV initially, followed by 150-250 IU/kg SQ every 8 hours) should be administered as an anticoagulant therapy to reduce the formation of additional clots. Low molecular weight (LMW) heparin offers no advantage over UH in short-term management; however, there is no conclusive evidence-based medicine that indicates whether UH or LMW heparin has any therapeutic value in cats. LMW heparins, such as enoxaparin and dalteparin, are expensive, and dosing ranges have not been established. Warfarin should not be used in cats.
- Aspirin (5mg/kg orally every 48-72 hours) should be given once the patient has resumed eating. Discontinue heparin after the patient is stable and receiving aspirin by decreasing the dose gradually over several days.
- Clopidogrel (Plavix), an antiplatelet drug, can be administered (18.75 mg/cat) in conjunction with aspirin and should be initiated as soon as possible; however, it should not be given concurrently with UH. A recent study showed a significant improvement in cats receiving clopidogrel. These cats survived 8 months longer than those receiving aspirin.
- One should administer IV fluids for those patients not experiencing CHF. Ongoing nursing care should include attending to patient comfort and warmth, as well as monitoring for signs of improvement, such as pulse quality, limb temperature, and motor function. Patients should also be monitored for signs of reperfusion injury, such as depression, cardiac arrhythmias, conduction disturbances, hyperkalemia, and acid-base disturbances.
- Treatment of hypothermia should not be a priority until shock and systemic perfusion are adequately addressed.
- Follow-up diagnostics should include: echocardiogram; ECG; serum chemistries that include electrolytes, acid-base status, and thyroid levels; and urinalysis. Additional testing for neoplasia, such as three-view thoracic radiographs and abdominal ultrasound, should be conducted if primary cardiac disease is not confirmed.

**References:**



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Hogan DF. Feline cardiogenic embolism: what do we know and where are we going? Proceedings from the American College of Veterinary Internal Medicine Forum, Louisville, KY, May 31-June 3, 2006.

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Feline

Hogan D, Fox P, Jacob K, Keene B, Laste N, Rosenthal S. Analysis of the feline arterial thromboembolism: clopidogrel vs aspirin trial (fat cat). Proceedings from the American College of Veterinary Internal Medicine Forum, Seattle, WA, June 12-15, 2013.

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Luis Fuentes V. Arterial thromboembolism: risks, realities and a rational first-line approach. *J Feline Med Surg* 2012;14(7):459-70.

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Smith SA, Tobias AH. Feline arterial thromboembolism: an update. *Vet Clin North Am Small Anim Pract* 2004;34(5):1245-71.

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Smith SA, Tobias AH, Jacob KA, Fine DM, Grumbles PL. Arterial thromboembolism in cats: acute crisis in 127 cases (1992-2001) and long-term management with low-dose aspirin in 24 cases. *J Vet Intern Med* 2003;17(1):73-83.

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