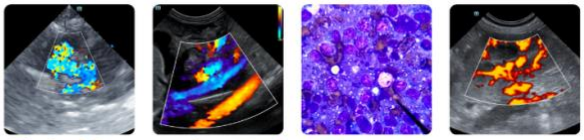

PATIENT

Jackson Burnett

SPECIES

Canine

BREED

Cattle Dog Mix

SEX

Neutered Male

AGE

9

WEIGHT

55

INTERPRETED BY

Eric Lindquist, DMV, DABVP(CFM), Cert. IVUSS

IMAGING PERFORMED BY

Cristy Fisher

HOSPITAL NAME

Pine Creek Ceterinary Hospital

REFERRING VET

Dr. Cecilia Gustafson

INVOICE

13895

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PRESENTING CLINICAL SIGNS

- ADR. P a bit wobbly coming down the stairs, moving slow, lower energy, back legs shaky.
- History of chronic pneumonia/cough (resolved as of 2/16/26 CG)
- History of acute kidney disease/glomerular disease due to anaplasma (per IM specialist) - caused significant protein losing nephropathy (UPC of 9 at one point in December of this year). Pw as originally on 1.3mg/kg clopidogrel SID but has not been on that for 2 month. P most recent UPC was 1.0 on 2/16/26.

Abnormal PE/Chem/CBC/UA Results: 2/16/26 specialist report showed UPC of 1.0; USG 1.013; creat 1.2, ALB 1.5

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra to a depth of 2.0 cm presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized, and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for his age patient. Medullary structure differed distinctly from that of the cortex. Dystrophic mineralization was noted and non-obstructive at this time. Slight pyelectasia was present in the left kidney. The left kidney measured 6.1 cm in length. The right kidney measured 6.5 cm in length. A cortical infarct was present in the caudal pole of the left kidney, appears stable with no evidence of active inflammation. A cortical infarct was present in the dorsal cortex of the right kidney as well.

Adrenal Glands

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 0.78 cm width. The right adrenal gland measured 5.0 mm width.

Spleen

The **spleen** revealed a focal hypoechoic nodule measuring 1.0 cm. the remainder of the spleen was unremarkable. The spleen revealed a thrombus extending approximately 4.0 cm from the base of the splenic vein extending approximately 2.0 cm beyond the spleen.

Liver

The **liver** presented subnormal in size with minor gallbladder sand. Increased portal markings were noted. A mild amount of remodeling was present.

Gastrointestinal

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There was some residual chyme and gas was noted in the **stomach**, yet not pathological. This is consistent with end post prandial presentation. Transit of chyme into the small intestine was normal. Curvilinear patterns were maintained throughout the GI tract. No evidence of pathology. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

The left limb of the **pancreas** was hypoechoic and irregular with enhanced surrounding mesentery.

ULTRASONOGRAPHIC FINDINGS

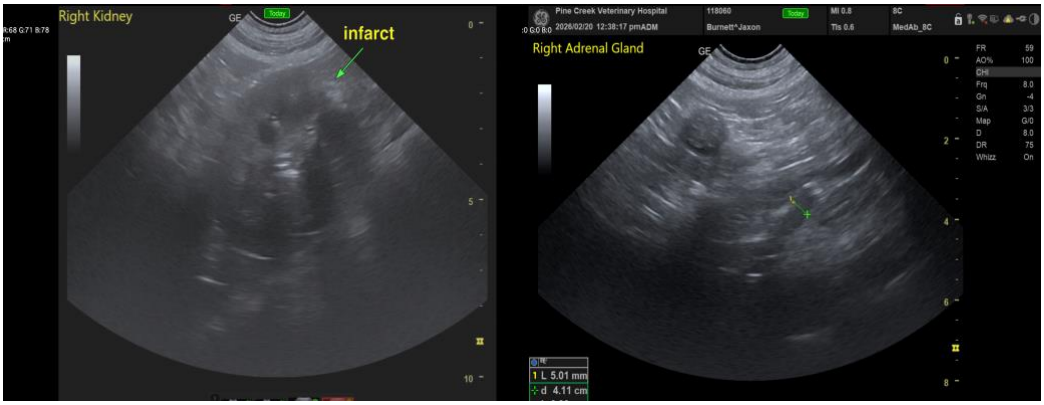
- Splenic thrombosis.
- Left limb pancreatitis.
- Renal infarcts.
- Partially full stomach.
- Subnormal liver size with remodeling.
- Minor gallbladder sand.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Obvious hypercoagulable state has been going on in this patient for some time given the renal infarcts that appear to be of prior episode and the active splenic thrombus. Loss of Antithrombin III owing to protein losing nephropathy is likely. Supportive care, GI protectants, plasma transfusion may all be appropriate. Bile acid profile is warranted given the microhepatica and moderate amount of hepatic remodeling.

Internal medicine consult can be utilized through Sonopath.com. You can select the internal medicine drop down at http://spa.sonopath.com/.

One of the world's top internists & SonoPath associate Dr. Remo Lobetti BVSc, MMedVet, PhD, DECVIM can evaluate your case through SonoPath. https://sonopath.com/resources/sonopath-services/internal-medicine-teleconsultation-services



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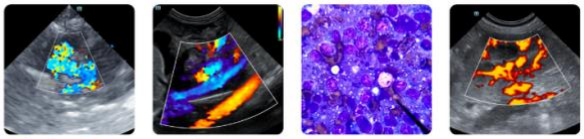

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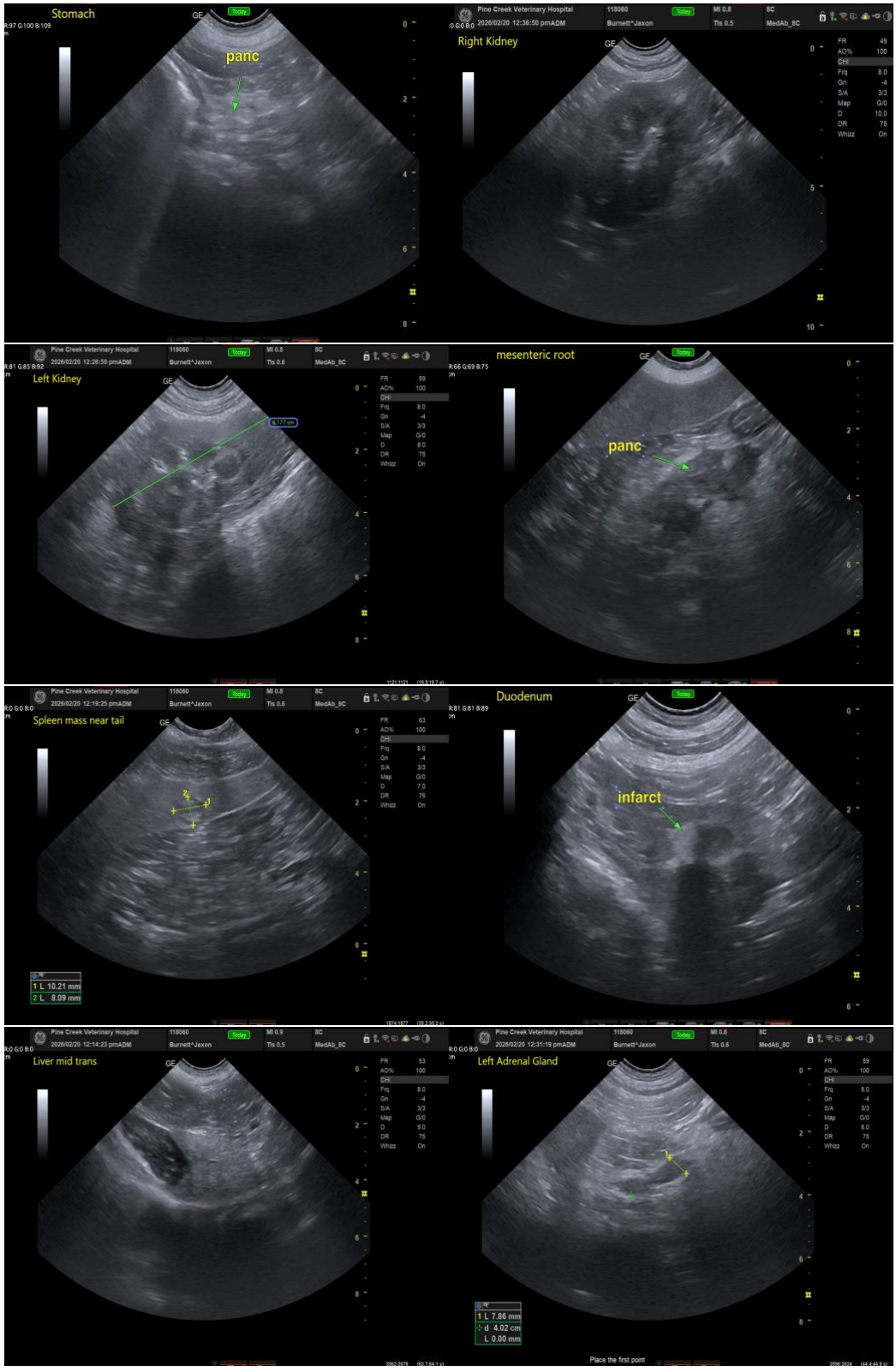

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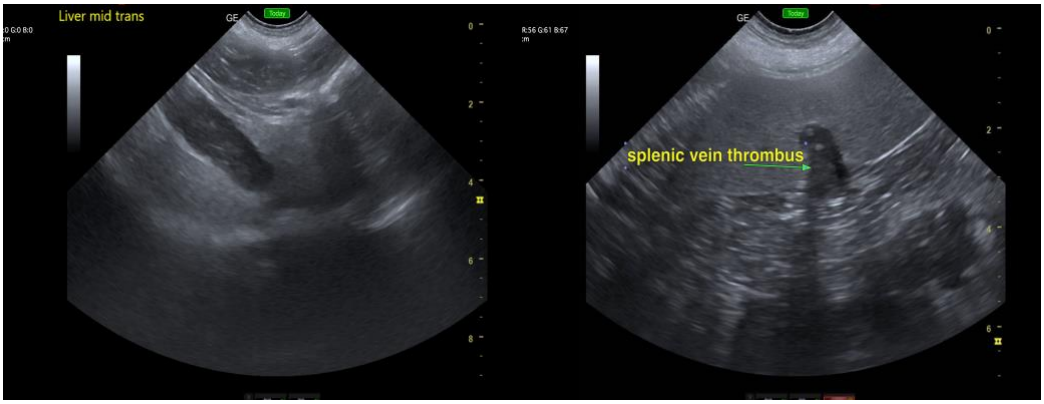

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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(CFM), Cert. IVUSS,

CEO, Owner, Founder -- SonoPath.com

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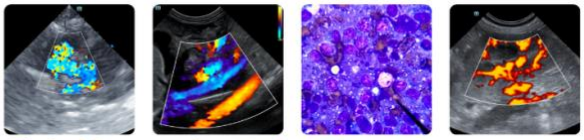
Systemic Thromboembolic Disease

http://www.sonopath.com/SystemicThrombo

Description: Thrombosis is a clot formation within a blood vessel or cardiac lumen, while embolization occurs when a foreign body or clot fragment lodges within a lumen. Thromboembolic disease (TED) can be arterial or venous (i.e., in the vena cava or portal vein). The most prominent site of arterial embolization is the distal aortic trifurcation; it is referred to as a saddle thrombus and causes ischemia, myalgia, cold extremities, and cyanotic nail beds of the hind limbs. Other frequent embolization sites include the following:

- The renal parenchyma, leading to azotemia and hematuria as a result of infarction.
- The coronary arteries, inducing myocardial ischemia and arrhythmias.
- The mesenteric arteries, causing gastrointestinal symptoms and bacterial translocation.
- The cerebral vessels, inducing neurological signs and possibly sudden death.

Caval syndrome may also occur, resulting in edema in the front parts of the body. Portal vein thrombosis can also occur due to pancreatitis, hepatitis, neoplasia, or peritonitis. Microcirculation thrombosis and reperfusion injury are additional complicating factors in cases of TED.



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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; approximately 35% of all feline patients are euthanized without attempted treatment. Yet, recent studies report a survival rate of approximately 40% with treatment. If cats survive the initial embolic event, re-embolization represents a likely cause of future morbidity and mortality (i.e., approximately 25% of patients will have recurrent thromboembolic episodes). Yet, mortality due to complications caused by underlying heart 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.

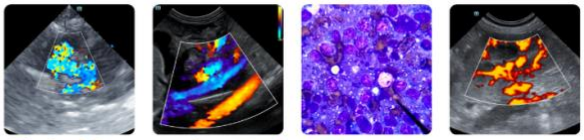
Body temperature and congestive heart failure (CHF) are two key indicators of both short- and long-term survivability. In general, less than 50% of cats will survive to discharge, despite aggressive therapy. A correlation between body temperature and prognosis has been demonstrated: a body temperature 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.

Causes of TED from an arterial standpoint include the following: CHF subsequent to endocardial damage and blood stasis (frequent in cats, occasional in dogs); protein-losing diseases (i.e., nephropathy, enteropathy); hypertension; neoplasia; hyperadrenocorticism; systemic inflammatory response syndrome (SIRS); and atherosclerosis due to hypothyroidism.

Pulmonary thromboembolism occurs as a complication to immune-mediated hemolytic anemia (IMHA), disseminated intravascular coagulation (DIC), renal disease, pancreatitis, neoplasia, sepsis, heartworm, hyperadrenocorticism, and hypothyroidism. The pathophysiology of TED is complex; however, it is essential to diagnose and treat it as adequately as possible.

The following is a schematic outline of the phenomenon:

A) Vessel endothelial lesion → vWF(8) + fibrinogen → platelet activation/aggregation
B) Fibrinogen formation depends on:
    1) Intrinsic pathway (aPTT): factors IX, XI, and XII
    2) Extrinsic pathway (PT): factor VII
    3) Common pathway (TT): factors I, II, and X with the following end result: thrombin → fibrinogen → insoluble fibrin
C) Anticoagulation team (SAC-3T): S protein, antithrombin III (responsible for 80% of activity), C protein, TFPI-1, thrombomodulin, t-PA (from endothelium) → plasminogen → plasmin → fibrinolysis
D) Clot/thrombus breakdown markers: fibrinogen → FDPs, D-Dimers
E) Virchow's triad (i.e., the prothrombotic state):



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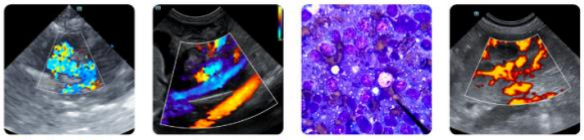
1) Altered endothelial structure/function: endothelin → activation of coagulation cascade; activation of platelet aggregation by ADP, fibrinogen, thromboxane A2
2) Blood stasis
3) Hypercoagulable state: ↓ ATIII (i.e., PLN, PLE, liver dysfunction), ↓ t-PA, ↓urokinase, ↓ plasminogen, ↓ protein C, ↓ S, ↑PAI-1, ↑ platelet aggregation

F) Thrombus → ischemia/inflammation; the effect is size dependent. Clots are firmly attached to
vessel walls, whereas postmortem clots are not attached.
Thrombus progression:
1) Fibrinolysis and dissolution
2) Organization and recanalization
3) Clot propagation
4) Dislodgement and embolization

The core marker of a hypercoagulative state is a loss or lack of ATIII production. In cases of glomerular disease and protein-losing nephropathy (PLN), ATIII moves into the urine due to its small size (65,000 Daltons). This also occurs in protein-losing enteropathy (PLE); however, in cases of PLN, other coagulation factors are additionally lost, as are larger proteins (the latter are not typically lost in the case of PLE lesions). Moreover, diseases that cause PLN also lead to increases in fibrinogen and thromboxane levels (i.e., why aspirin is recommended in PLN), further predisposing the patient to TED. Determining the ATIII levels is essential when evaluating a hypercoagulable state. When ATIII levels are 50-75% decreased, there is a moderate TED risk; when they are 75% decreased, the risk is severe.

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.

When portal vein thrombosis occurs, prehepatic portal hypertension develops, causing transudate fluid accumulation in the abdomen, which results in abdominal swelling and a palpable a fluid wave on clinical examination. Portal vein thrombosis occasionally occurs when patients are experiencing hypercoagulable states, such as autoimmune hemolytic anemia and immune-mediated thrombocytopenia, as well as paraneoplastic events. Organ infarcts develop when local thromboembolic episodes occur in organs like the kidneys and spleen. The latter can be identified sonographically, as can localized thrombosis in vessels such as the portal vein, splenic vein, aorta, vena cava, or phrenic veins. Invasive adrenal tumors often have attached thrombi that can resemble the mass sonographically, but may dissolve partially with therapy due to improved local vascular flow. High-resolution sonography with color flow and power Doppler can identify a vascular thrombosis that may have been missed on routine sonograms.



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Cattle Dog Mix

SEX

Neutered Male

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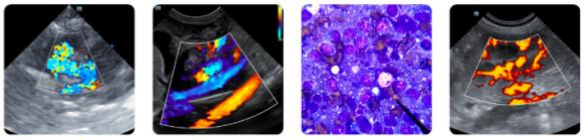
Diagnostics: The increased collection of data from thromboelastography (TEG) in human studies may provide further insight into feline cases and allow for earlier detection of hypercoagulable states; however, TEG is not widely available as of yet. Currently, clinical signs and traditional coagulation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

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.

Treatment: The goals of acute ATE management are to: 1) manage pain appropriately; 2) treat CHF, if present; 3) minimize ongoing clot formation; 4) improve blood flow; and 5) provide optimal supportive 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 the only factor one considers when determining a diagnosis. 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 for feline 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.



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- 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 the following: 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.

Treatment for canine TED:

Anticoagulant therapy (heparin 100-300 IU/kg IV or SQ every 8 hours; dalteparin 100-150 IU/kg SQ BID-TID; or enoxaparin 1 mg/kg SQ BID) can be used to prevent further thrombus formation. Antiplatelet therapy should include aspirin (0.5 mg/kg PO Q12-24hr) or clopidogrel (1-2 mg/kg PO Q24h). In early cases, thrombolytic therapy, such as streptokinase or a tissue plasminogen activator, can be used; however, there is limited experience with this type of therapy in veterinary medicine and dosing regimens vary considerably. Surgical embolectomy is feasible, but often associated with significant mortality.

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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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