

## Case Report

# Surgical Removal of a Canine Aortic Thromboembolism Secondary to Pancreatitis

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A 7-year-old castrated male Pomeranian was evaluated on emergency for diagnostic work-up and treatment for acute nonpainful paraparesis. The neurologic examination suggested a L4-S3 myelopathy, but physical examination revealed lack of femoral pulses and rectal hypothermia, as well as a grade II/VI systolic heart murmur, so ischemic neuromyopathy was suspected. Clinicopathologic analysis revealed increased muscle enzymes and proteinuria. Abdominal ultrasonography confirmed aortic thromboembolism (ATE), and surgical histopathology diagnosed necrotizing pancreatitis. Surgical aortic thrombectomy was performed, and antithrombotic therapy was instituted. Pancreatitis was treated supportively. The dog was discharged to the owners after 10 days of hospitalization. Recheck examination 6 weeks after initial presentation revealed a normal neurologic examination and normal femoral pulses. The patient has had no further bouts of pancreatitis and remains neurologically normal 5 years after initial presentation. Canine ATE is relatively rare compared to the feline counterpart. Directed therapy for feline ATE is often not recommended, as underlying conditions are oftentimes ultimately fatal. Underlying etiologies for canine ATE include cardiovascular disease and endocrinopathies, but canine ATE secondary to pancreatitis has not yet been reported. Surgical removal of aortic thromboembolism should be considered as curative for pelvic limb dysfunction in the canine patient without a terminal underlying disease.

## 1. Introduction

Aortic thromboembolism (ATE) is a relatively rare occurrence in the dog. Compared to the syndrome in cats, it typically results in less severe clinical signs and is more enigmatic in etiology. Treatment approaches for canine ATE, including thrombolytic therapy and rheolytic thrombolysis, have been described; however, to the authors' knowledge this report is the first description of open surgical removal of an aortic thrombus [1–5]. An additional novel feature of this report is the development of canine ATE secondary to pancreatitis.

## 2. Case Presentation

A 7-year-old, 4.6 kg, castrated male Pomeranian was admitted on emergency to the Neurology/Neurosurgery Service for

a 2-day history of paraparesis. The owners reported that the dog yelped out after a failed attempt to jump into the truck and was then unable to use his pelvic limbs. The referring veterinarian prescribed unknown doses of butorphanol and prednisolone sodium succinate, but the dog remained disabled. With the concern for intervertebral disk herniation, the dog was referred.

On physical examination, the patient had rectal hypothermia at 26.4°C, with a euthermic axillary temperature of 37.7°C. The quality of femoral pulses was decreased, and the distal pelvic limbs were cool to the touch. A grade II/VI systolic heart murmur was auscultated. The remainder of the physical examination was within normal limits.

Neurologic examination revealed a nonpainful nonambulatory paraparesis. Postural reactions were decreased to absent in the pelvic limbs, and patellar areflexia was noted in both pelvic limbs. The perineal reflex was decreased, and anal

sphincter tone was decreased. Cranial nerves and thoracic limbs were within normal limits. Based on physical and neurologic examination, ATE with subsequent ischemic neuromyopathy was suspected; however, differential diagnoses also included L4-S3 myelopathies, such as spinal column trauma, fibrocartilaginous embolic myelopathy, Hansen type I intervertebral disk disease, and myelitis.

A blood sample was collected from a jugular vein and submitted for a CBC and serum biochemistry analysis. On presentation, there was mild dehydration evident by mild hemoconcentration (59.9%; reference interval, 37–55%) and serum protein and albumin concentration at the upper end of the reference interval (total protein 7.31 g/dL, reference interval 5.1–7.3 g/dL and albumin 3.5 g/dL, reference interval 2.6–3.5 g/dL). There was a mild to moderate inflammatory leukogram characterized by neutrophilia ( $33.3 \times 10^3/\mu\text{L}$ ; reference interval  $3\text{--}11.4 \times 10^3/\mu\text{L}$ ) and monocytosis ( $1.4 \times 10^3/\mu\text{L}$ ; reference interval  $0.15\text{--}1.35 \times 10^3/\mu\text{L}$ ). A concurrent stress leukogram associated with both endogenous and exogenous corticosteroids was also likely contributing to the mature neutrophilia, monocytosis, and low normal lymphocytes ( $1,074 \times 10^3/\mu\text{L}$ ; reference interval  $1\text{--}4 \times 10^3/\mu\text{L}$ ). Inflammation was further supported by the presence of decreased serum iron ( $47 \mu\text{g/dL}$ ; reference interval  $100\text{--}200 \mu\text{g/dL}$ ). There was mild thrombocytopenia ( $149 \times 10^3/\mu\text{L}$ ; reference interval  $164\text{--}510 \times 10^3/\mu\text{L}$ ). There was increased alkaline phosphatase activity ( $1247 \text{ U/L}$ ; reference interval  $4\text{--}95 \text{ U/L}$ ), increased alanine aminotransferase activity ( $728 \text{ U/L}$ ; reference interval  $26\text{--}200 \text{ U/L}$ ), increased aspartate aminotransferase activity ( $5608 \text{ U/L}$ ; reference interval  $15\text{--}50 \text{ U/L}$ ), and increased creatine kinase activity ( $315,200 \text{ U/L}$ ; reference interval  $92\text{--}357 \text{ U/L}$ ) indicative of damage or necrosis to both myocytes and hepatocytes. The patient was also hypoglycemic ( $66 \text{ mg/dL}$ ; reference interval  $80\text{--}100 \text{ mg/dL}$ ), hypokalemic ( $2.9 \text{ mEq/L}$ ; reference interval  $3.5\text{--}5.9 \text{ mEq/L}$ ), and azotemic (blood urea nitrogen  $47.5 \text{ mg/dL}$ , reference interval  $10\text{--}25 \text{ mg/dL}$  and creatinine  $1.4 \text{ mg/dL}$ , reference interval  $0\text{--}1.3 \text{ mg/dL}$ ). Phosphorus was at the high end of the reference interval ( $5.8 \text{ mg/dL}$ ; reference interval  $3.3\text{--}5.8 \text{ mg/dL}$ ).

Urine collected via catheterization was brown and cloudy with a specific gravity of 1.035. The urine pH was 8.0, with mild to moderate proteinuria ( $617 \text{ mg/dL}$ ), glucosuria (3+), hematuria (3+), bilirubinuria (1+), and trace ketonuria. Urine sediment examination contained 0 to 3 leukocytes, 0 to 5 erythrocytes, 0 to 3 transitional epithelial cells per high power field, rare renal tubular epithelial cells, many fat droplets, and moderate mucous threads per high power field. Additionally, 0 to 1 granular casts and moderate amorphous crystals were noted. Urine was submitted for culture and susceptibility. Some degree of renal tubular disease was suspected given the presence of glucosuria in the face of hypoglycemia. Laboratory testing supported the working diagnosis of ischemic neuromyopathy secondary to ATE.

Survey thoracic radiographs were within normal limits. Survey abdominal radiographs revealed the presence of 2 radiopaque stones (0.2 cm and 0.3 cm diameter) within the urinary bladder and faint mineralization of the left



FIGURE 1: Abdominal ultrasound at the level of the aortic bifurcation. The cranial portion of the dog is to the left of the image. The thrombus is hyperechoic compared to the aorta's anechoic appearance (arrow). Color-flow Doppler application indicates blood flow extending laterally from the area of the thrombus.

renal pelvis; otherwise, the study was within normal limits. Abdominal ultrasonography revealed the presence of a thrombus within the aorta, just proximal to the external iliac arteries. The embolism was incomplete, as flow was noted to extend laterally from the area of the thrombus (Figure 1). Hyperechoic debris and cystoliths were seen within the urinary bladder. Bilateral renal diverticular mineralization was also present. Although a grade II/VI systolic heart murmur was auscultated on physical examination, echocardiography revealed severe aortic insufficiency and severe mitral regurgitation; neither a thrombus nor spontaneous echocardiographic contrast was seen within the heart.

The dog was hospitalized in the critical care unit and treated for hypoglycemia with 2 bolus doses of 50% dextrose ( $1 \text{ mL/kg}$  diluted in equal parts 0.9% NaCl). The patient then received continued intravenous fluid support with 5% dextrose and KCl ( $20 \text{ mmol/L}$ ) in a balanced crystalloid solution at a constant rate of  $11.5 \text{ mL/h}$  ( $60 \text{ mL/kg/d}$ ). The dog received an intravenous bolus of heparin ( $80 \text{ U/kg}$ ) and was then placed on a constant rate infusion of heparin ( $18 \text{ U/kg/h}$ ). The dog was also started on acetylsalicylic acid ( $0.5 \text{ mg/kg per os}$  once daily). The patient was fed a mild diet (Hill's Prescription Diet i/d, Hill's Pet Nutrition Inc., Topeka, KS) and had free-choice access to water until an episode of regurgitation, at which point the food and water were removed. Overnight, it was noted that the dog's femoral pulses were decreased to absent. By use of a point-of-care assay, partial thromboplastin time was analyzed twice overnight and was found to be prolonged ( $188 \text{ s}$  and  $113 \text{ s}$ ; reference interval  $60\text{--}93 \text{ s}$ ). Blood glucose and vital parameters remained within normal limits overnight.

On the second day of hospitalization, the dog was hypoalbuminemic ( $2.5 \text{ g/dL}$ ; reference interval  $2.6\text{--}3.5 \text{ g/dL}$ ), hypocalcemic ( $8.3 \text{ mg/dL}$ ; reference interval  $9.5\text{--}11.8 \text{ mg/dL}$ ), hypophosphatemic ( $2.9 \text{ mg/dL}$ ; reference interval  $3.3\text{--}5.8 \text{ mg/dL}$ ), hyponatremic ( $144 \text{ mEq/L}$ ; reference interval  $145\text{--}160 \text{ mEq/L}$ ), and hyperglycemic ( $145 \text{ mg/dL}$ ; reference interval  $80\text{--}100 \text{ mg/dL}$ ). Alkaline phosphatase activity had decreased slightly but was still above the reference interval ( $754 \text{ U/L}$ ; reference interval  $4\text{--}95 \text{ U/L}$ ). Alanine aminotransferase activity ( $1201 \text{ U/L}$ ; reference interval  $26\text{--}200 \text{ U/L}$ ),

aspartate aminotransferase activity (7437 U/L; reference interval 15–50 U/L), and creatine kinase activity (452,500 U/L; reference interval 92–357) remained above the reference interval and were increased compared to the previous day.

A recheck urinalysis collected via cystocentesis had slightly cloudy dark yellow urine with a specific gravity of 1.012, a pH of 7.5, proteinuria (114.8 mg/dL), and 3+ blood. Sediment examination contained 0–5 erythrocytes per high power field. Urine protein to creatinine ratio was 5.5.

On evaluation of a coagulation panel, the dog was thrombocytopenic ( $146 \times 10^3/\mu\text{L}$ ; reference interval  $164\text{--}510 \times 10^3/\mu\text{L}$ ) with increased mean platelet volume (13.3 fL; reference interval 8.4–13 fL), prolonged activated partial thromboplastin time (16.0 s; reference interval 8–14.4 s), shortened thrombin time (3.3 s; reference interval 7.6–22 s), and hyperfibrinogenemia (780 mg/dL; reference interval 100–300 mg/dL). D-dimers (500–1000 ng/mL; reference interval 0–250 ng/mL) and fibrin degradation products ( $>20,000 \mu\text{g/mL}$ ; reference interval 0–5  $\mu\text{g/mL}$ ) were increased. Antithrombin activity was decreased (62%; reference interval 85–100%).

After one episode of vomiting, metoclopramide (0.4 mg/kg subcutaneously every 6 hours) and famotidine (1 mg/kg intravenously once daily) were added to his drug regimen. He remained on the previous doses of heparin constant rate infusion, acetylsalicylic acid, and crystalloid with KCl. He was given multiple small meals throughout the afternoon and experienced no further episodes of nausea, regurgitation, or vomiting. His food was removed overnight.

The patient remained stable the following morning and was premedicated with acepromazine (0.05 mg/kg intravenously) and hydromorphone (0.1 mg/kg intravenously). General anesthesia was induced with propofol (6 mg/kg intravenously) and maintained with isoflurane (1.5–2% in 100% O<sub>2</sub>) for an abdominal exploratory surgery. The ventral aspect of the abdomen was prepared aseptically, and a standard celiotomy was performed. White to translucent 2–3 cm long fibrin sheets were attached to the visceral and retroperitoneal surfaces of both kidneys. Portions were removed and submitted for culture. Multifocal, 3–4 mm firm, flat, white to tan plaques were visualized on the pancreas. A section of the right limb of the pancreas containing these lesions was biopsied using the guillotine method with 4-0 polydioxanone (PDS, Ethicon, Cornelia, GA). The left kidney was biopsied using a biopsy needle (TruCut, CareFusion, San Diego, CA) and the liver was biopsied using the guillotine method with 4-0 polydioxanones (PDS, Ethicon). The abdominal aorta was identified and traced caudally to the bifurcation. Umbilical tape was placed around the aorta and both external iliac arteries, and bulldog clamps were placed across the aorta immediately proximal to the thrombus and on both external iliac arteries distal to the thrombus. A full thickness, 1 cm incision was made on the ventral surface of the aorta (Figure 2). Atraumatic microforceps and a nerve hook were used to remove three to four pieces of thrombus approximately 1–2 cm in diameter (Figure 3). The bifurcation was subsequently flushed with sterile saline to ensure removal of all pieces of the thrombus. The defect was sutured using 6-0 polypropylene (Prolene, Ethicon) in

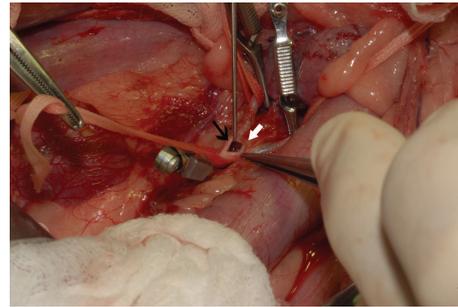


FIGURE 2: Intraoperative photograph of the aortotomy (black arrow). The cranial portion of the dog is to the left of the image. The thrombus is seen within the lumen of the aorta, just cranial to the aortic bifurcation (white arrow).

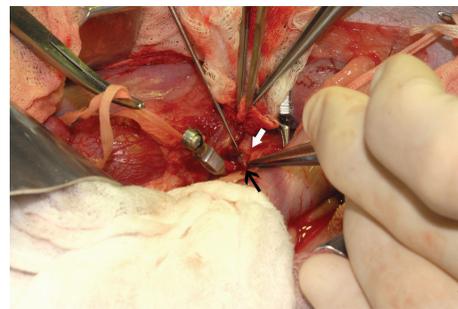


FIGURE 3: Intraoperative photograph of thrombus removal. The cranial portion of the dog is to the left of the image. The thrombus (black arrow) is removed from the aorta, at the level of the aortic bifurcation (white arrow).

a simple interrupted pattern. Prior to complete closure, the proximal vascular clamp was temporarily released to allow flooding of the operated aortic segment with blood, to prevent an air embolism. After the final sutures were placed in the aorta, the vascular clamps were carefully released and the aortotomy was observed for hemorrhage. Minor hemorrhage was controlled with gentle pressure. A Foley catheter was passed through the penis and into the urethra for retropulsion of the urethral stones into the bladder lumen. Stay sutures were placed in the urinary bladder and a cystostomy was performed. The contents of the bladder were examined and no stones were found. The bladder was closed using 4-0 polydioxanone (PDS, Ethicon), in a simple continuous pattern. The celiotomy was closed in a routine manner, and a jugular catheter was placed. All biopsy samples and the thrombus were submitted for histopathology.

The dog had an uneventful recovery from anesthesia. Postoperatively, the femoral pulse quality was immediately noted to have improved, and the distal limbs were warm to the touch. Postoperative analgesia was provided by hydromorphone (0.1 mg/kg intravenously every 6 hours) and a constant rate infusion of lidocaine (1.5 mg/kg/h intravenously). The dog experienced one mild episode of vomiting, associated with hydromorphone administration.

Histopathologic examination of the surgical biopsies confirmed the formation of a thrombus and identified changes in



FIGURE 4: Recheck abdominal ultrasound at the level of the aortic bifurcation. The cranial portion of the dog is to the left of the image. The aorta is anechoic (arrow), and Color-flow Doppler application indicates blood flow within the aorta and external iliac arteries.

the pancreas consistent with necrotizing pancreatitis. There was moderate renal tubular and hepatocellular vacuolation, suggestive of lipidosis.

Over the next several days in the hospital, the dog was weaned off of hydromorphone, lidocaine, metoclopramide, famotidine, heparin, and intravenous fluids. Tramadol (5 mg/kg *per os* every 8 hours) was instituted for continued analgesia, and acetylsalicylic acid was continued. The vomiting episodes decreased, and appetite gradually improved.

The patient was discharged on acetylsalicylic acid (0.5 mg/kg *per os* once daily), famotidine (1 mg/kg *per os* once daily for 7 days), and sucralfate (0.5 g/dog *per os* every 8 hours) after 10-day hospitalization. At the time of discharge, the dog displayed ambulatory paraparesis. His owners were instructed to continue feeding a bland diet (Hill's Prescription Diet i/d, Hill's Pet Nutrition Inc.) and restrict his activity when unsupervised.

The dog was presented 6 weeks later for a recheck examination. His physical examination revealed rectal euthermia (38.2°C) and a grade II/VI left-sided systolic heart murmur. His femoral pulses were strong bilaterally, and his distal pelvic limbs felt warm to the touch. Neurologic examination revealed no abnormalities. A recheck abdominal ultrasound showed a normal abdominal aorta (Figure 4) and pancreas. The previously noted bilateral renal diverticular mineralization and hyperechoic debris within the urinary bladder were still present; there was no evidence of cystolithiasis. The dog was discharged with instructions to wean off of oral acetylsalicylic acid over the ensuing 6 weeks. Follow-up phone call 5 years after the incident confirmed that the dog continues to do well. He ambulates normally and has not had any further bouts of pancreatitis.

### 3. Discussion

Though it frequently occurs in cats with hypertrophic cardiomyopathy, ATE is relatively uncommon in dogs. Rare cases of canine ATE have been associated with underlying conditions, such as hyperadrenocorticism, hypothyroidism,

diabetes mellitus, cardiac arrhythmias and structural cardiac disease, various neoplasms, protein-losing nephropathy, protein-losing enteropathy, gastric dilatation/volvulus, and trauma [1–8]. In contrast to feline ATE, affected dogs have a better prognosis, owing to collateral circulation [8]. Dogs also differ from cats in that they can present with acute or chronic signs. Dogs presenting with an acute onset of ATE tend to have more severe neurologic dysfunction, such as paraparesis or paraplegia, while dogs with a chronic onset tend to have milder signs, such as exercise intolerance [6]. The patient in this case had moderately to markedly increased muscle enzyme concentrations that corresponded with the acute presentation and ongoing ischemic necrosis. Some of the electrolyte changes such as the high normal phosphorous and hypocalcemia could be attributed to acute rhabdomyolysis as described in foals and people [9–12]. In addition, acute aortic thrombosis is associated with moderate to marked increase in activity of serum muscle enzymes (AST and CK), while patients with chronic lesions often have normal to mild increased activity of muscle enzymes [10, 12].

Clinical laboratory data in this case strongly suggested a hypercoagulable state. There are numerous underlying conditions that result in a hypercoagulable state with formation of a thrombus. These conditions affect one of the main factors of coagulation that were originally outlined by Virchow: blood flow, endothelial integrity, and the balance between coagulation and fibrinolysis [13]. The valvular disease noted on echocardiography was not thought to be the cause of the thrombus, due to the lack of spontaneous echocardiographic contrast or thrombus noted within the left atrium [14].

Clinical findings (blood work, history, and physical examination) made other causes of hypercoagulability, such as hyperadrenocorticism, hypothyroidism, immune-mediated hemolytic anemia, sepsis or other systemic infection, polycythemia vera, and lipemia, less likely; however, endocrine disease such as hypothyroidism and hyperadrenocorticism could not be ruled out without specific testing. The decreased antithrombin activity led to the consideration of hepatic or glomerular disease as etiologies for thromboembolic disease. Although the liver and kidneys were judged to be normal on abdominal ultrasound, histopathologic evaluation revealed mild to moderate vacuolation of the kidneys and liver. Pancreatitis was not originally a consideration because of the lack of history and clinical signs of pancreatitis and the lack of identifiable pancreatic lesions on ultrasound.

In this case, there were three potential factors that may have contributed to formation of the thrombus. The first and most significant contributor is local vasculitis secondary to necrotizing pancreatitis. Vasculitis mediates thrombus formation through exposure of subendothelial components such as collagen, tissue factor, and fibronectin [15]. These are all potential stimuli for platelet aggregation and coagulation [15]. Pancreatitis induces disseminated intravascular coagulation (DIC) via release of active pancreatic enzymes into circulation. These enzymes normally are inactivated by  $\alpha$ -macroglobulin [16]. Once  $\alpha$ -macroglobulin is depleted, the enzymes are capable of activating the coagulation cascade and initiating DIC [16]. Systemic inflammation induces the production of fibrinogen, an acute phase protein. In situations

where there is imbalance of fibrin formation and degradation, hyperfibrinogenemia may promote thrombosis by increasing fibrin formation and clot stabilization [17, 18]. In addition, hyperfibrinogenemia along with increased concentrations of fibrin degradation products and d-dimers are considered risk factors for thrombotic events [17]. In this case, the dog had all three risk factors, moderate to marked hyperfibrinogenemia, mild to moderate increase in d-dimers, and marked increase in fibrin degradation products.

The patient also had a protein-losing nephropathy (urine protein:creatinine ratio of 5.5). Antithrombin was likely lost in the urine and contributed to an imbalance in coagulation and fibrinolysis. Routine histopathologic examination of the kidney did not identify glomerular disease but did suggest moderate tubular lipidosis which may have inhibited protein resorption. Glucosuria in the face of hypoglycemia and trace ketonuria also support renal tubular disease. Because antithrombin was not measured until after the clot was formed, it was possibly decreased due to consumption during thrombus formation. An additional test which may have been helpful in determining this patient's global hemostatic condition would be thromboelastography (which was not available at the time).

Lastly, the patient also had underlying cardiovascular disease that altered systemic blood flow. There was no evidence of a thrombus or spontaneous echocardiographic contrast; however, even slight alternations in blood flow because of valvular disease can lead to stasis or turbulence and development of thrombi [8, 13]. While structural heart disease may have contributed to systemic alteration in blood flow, inflammation associated with pancreatitis may have also altered blood flow locally.

Acute therapeutic options described for ATE in the short term include thrombolytic drug therapy and rheolytic thrombectomy. Rheolytic thrombectomy for ATE has been mentioned in the veterinary literature, but its use has not been fully described in dogs [3, 19, 20]. In a recent feline study, thrombi were successfully dissolved in 5/6 cats, using a commercially available rheolytic thrombectomy system, but only 3/6 cats survived till discharge [19]. There are few reports regarding the use of thrombolytic drug therapy, such as tissue plasminogen activator (t-PA), for dissolution of an ATE in cats and dogs [2, 4, 5, 21–23]. Though t-PA can reportedly improve functional neurologic outcome in cats, it is also associated with severe adverse side effects, including fatal hyperkalemia and hemorrhage, and its use is not widely recommended [19, 21]. Catheter-directed thrombolysis, in which thrombolytic drugs are applied directly to the thrombus, has been described [22, 24]. Side effects include hemorrhage, pulmonary embolism, and reperfusion injury. While catheter-directed thrombolysis has been shown to improve outcome and have fewer complications in people, such studies in animals are lacking. Balloon dilation and angioplasty present problems, such as pulmonary embolism and reocclusion, although this technique has shown some promise when combined with endovascular stents in people [22]. Studies on this technique are lacking in veterinary patients.

Long-term treatment options include anticoagulant and antiplatelet medications, such as heparin, acetylsalicylic acid, and clopidogrel [25]. In this case, heparin was given immediately not to lyse the thrombus but to reduce its expansion/extension [26]. Heparin interrupts coagulation in both the intrinsic and extrinsic pathways. At a low dose, heparin prevents further thrombosis by inactivating factor Xa and preventing the conversion of prothrombin to thrombin through the enhancement of antithrombin activity [25, 26]. Given the decreased antithrombin activity reported in this patient, as well as the presence of pancreatitis, the overall effect of the heparin therapy cannot be determined. Acetylsalicylic acid reduces platelet aggregation by inhibiting cyclooxygenase-1, which decreases synthesis of prostaglandins and thromboxanes (TXA<sub>2</sub>) [25]. However, antiplatelet activity of acetylsalicylic acid in diseased canine patients has not been fully elucidated [25].

Thrombectomy for canine ATE has been mentioned in the literature, but methods for open aortic thrombus removal have not been previously described [2]. Surgical removal of thrombi has reportedly been met with mixed results and is considered to be a risky procedure in cats with ATE [21, 22], but to the authors' knowledge there are no reports describing open surgical thrombectomy in dogs. Feline ATE is usually caused by underlying cardiac disease, specifically hypertrophic or restrictive cardiomyopathy. The long-term prognosis for cats presenting with ATE and underlying cardiac disease is poor, with mean and median survival times reported at 51 and 350 days [27, 28]. Even if the ATE can be removed or dissolved, recurrence rates range within 17–50% with treatment of the cardiac disease [27, 28]. Aortic thromboembolism in dogs is more rarely seen than in cats, and it may not be due to cardiac disease. The dog in this report had evidence of structural cardiac disease, but there was no evidence that this was directly contributing to the development of thromboembolism [14]. The cause for this dog's ATE was unknown at the time of work-up. Due to the significant neurologic dysfunction and the lack of information about rheolytic thrombectomy in dogs, the decision to perform a surgical thrombectomy was made. It was also speculated that this patient's ATE was an isolated incident, decreasing the risk of recurrence after thrombectomy that often occurs in feline patients. Surgical thrombectomy may be warranted in dogs that do not have the underlying risk factors for ATE recurrence, primarily cardiac disease, and that are considered good anesthetic candidates.

Ischemia-reperfusion injury is a concern after ATE [29]. After a period of decreased or absent tissue oxygenation, reactive oxygen species (ROS), such as the hydroxyl radical, are formed [29, 30]. Reactive oxygen species interact with cells in many deleterious ways, including protein, DNA, and RNA damage, lipid peroxidation of cell membranes, and loss of cell membrane permeability [29, 30]. Neutrophils are recruited into the area of tissue damage, become activated, and release their own ROS. After addressing the occluded vessel, reperfusion of the damaged tissue allows release of these reactive oxygen species into systemic circulation, which may cause myocardial and lung damage [29].

Additionally, during ischemia there is lack of aerobic cellular respiration, leading to decreased adenosine triphosphate (ATP). This causes failure of ATP-dependent cell membrane pumps, such as  $\text{Na}^+/\text{K}^+$  and  $\text{Na}^+/\text{Ca}^{++}$ . As a result of pump failure and direct damage to cell membranes, sodium and calcium are allowed to accumulate within cells, leading to cytoplasmic swelling and lysis and the initiation of the proapoptotic caspase and calpain cascades [29]. Massive cell death can be the end result. Additionally, potassium is allowed to accumulate extracellularly, leading to systemic hyperkalemia. This, in turn, can have deleterious effects on the patient, causing cardiac arrhythmias and further neuromuscular dysfunction [31, 32]. In this case, the dog was not hyperkalemic. A possible explanation for this finding may be associated with decreased dietary intake secondary to the pancreatitis or increased renal excretion. Fortunately, the thrombus in this report caused an incomplete blockage of the arterial supply to the patient's pelvic limbs, and the ischemic damage was mild and incomplete. Postoperative blood work and monitoring did not reveal evidence of clinically significant ischemia/reperfusion damage. Lidocaine has been purported to be helpful in prevention of ischemia/reperfusion injury. Its mechanisms of action are inhibiting intracellular calcium, decreasing neutrophil accumulation, activation and release of ROS, and scavenging the hydroxyl radical [30].

Though pancreatitis has been associated with splenic vein thrombosis, it has not yet been documented as an inciting cause of ATE in the dog [33]. Arterial thromboembolism is a known complication of pancreatitis in humans [34], and rare cases of ATE are documented in the human medical literature [35, 36]. Pancreatitis was an unexpected finding in this case, as the dog had no historical findings which would indicate that diagnosis, such as nausea, vomiting, or abdominal pain, though he did begin regurgitating/vomiting once hospitalized. Abdominal ultrasound did not show evidence of pancreatitis, though it has been shown that ultrasound is not a sensitive indicator of pancreatitis, nor hepatic or presumably renal histologic disease [37, 38].

Development of thromboembolic disease is a common complication of other primary disease processes. Prognosis for acute thromboembolism in cats is poor to grave when seen with underlying cardiac conditions; however, in dogs, the prognosis is often more favorable, dependent upon the underlying disease processes. A proposed explanation for the more favorable prognosis is better potential for the development of collateral circulation in dogs [8]. In cats, there is inhibition of collateral vessel development with experimental thrombotic occlusion [39]. This finding is thought to be associated with effects of vasoactive agents such as serotonin and prostaglandins that are released from platelets. To the authors' knowledge, these findings have not been evaluated in dogs.

This case presents new and interesting findings for canine ATE. Pancreatitis, alone or in combination with other systemic causes for hypercoagulability, should be considered as an etiology for canine ATE. Additionally, surgical aortic thrombectomy can have an excellent functional outcome and should be considered for patients without a terminal primary diagnosis.

## Disclosure

This paper was presented in abstract form at the Auburn University College of Veterinary Medicine Annual Conference, Auburn, AL, April 2009.

## Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

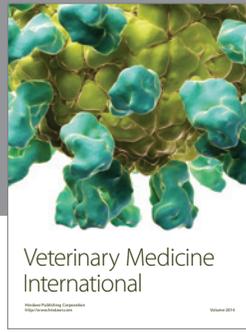
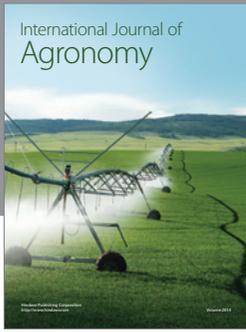
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