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Case Report

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Contrast-enhanced multidetector computed tomography to diagnose pulmonary thromboembolism in an awake dog with pyothorax

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Abstract

Objectives – To (1) describe the use of contrast-enhanced multidetector computed tomography (CE-MDCT) for identifying pulmonary thromboembolism (PTE) in an awake dog with pyothorax, (2) report the first documented case of PTE associated with pyothorax in veterinary medicine, and (3) review diagnostic imaging modalities and therapeutic options for PTE.

Case Summary – A 5-year, 4-month-old female neutered Labrador Retriever was presented for respiratory distress secondary to a pyothorax. The dog underwent thoracic exploratory surgery in which no underlying etiology was identified. Aerobic bacterial culture grew *Streptococcus canis*. The patient remained hypoxemic despite thoracocentesis and surgery. CE-MDCT was performed without general anesthesia and showed luminal-filling defects in the right cranial and right and left caudal lobar primary pulmonary arteries consistent with PTE. Anticoagulant therapy using unfractionated heparin was initiated. The dog responded well and was discharged 3 days postoperatively.

New or Unique Information Provided – To the authors' knowledge, this is the first reported case of PTE diagnosed in a dog with pyothorax using CE-MDCT.

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Keywords: inflammation, nonselective angiography, thrombus, unfractionated heparin, ventilation/perfusion scintigraphy

Introduction

Pulmonary thromboembolism (PTE) is the partial or complete occlusion of a pulmonary vessel or its branches by a thrombus.[1] The pathogenesis of PTE is based on Virchow's triad: endothelial damage, altered blood flow, and an imbalance between procoagulant and anticoagulant factors.[2] Diagnosis of PTE can be very difficult with routine diagnostics such as thoracic radiography. Findings on thoracic radiographs are often nonspecific but may reveal a hyperlucent region or an alveolar pattern; however, they are frequently normal and inconclusive.[3]

Abbreviations

aPTT	activated partial thromboplastin time
CRI	constant rate infusion
CT	computed tomography
CE-MDCT	contrast-enhanced multidetector computed tomography
FOV	field of view
HU	Hounsfield units
LRS	lactated Ringer's solution
PTE	pulmonary thromboembolism
SQ	subcutaneously
UFH	unfractionated heparin
V/Q	ventilation/perfusion

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Advanced imaging modalities used to diagnose PTE include pulmonary ventilation/perfusion (V/Q) scintigraphy and a range of pulmonary angiography, including selective/nonselective angiography, digital subtraction angiography, computed tomography (CT) angiography, magnetic resonance angiography. In human medicine, contrast-enhanced multidetector computed tomography (CE-MDCT) is now the most widely used diagnostic test in patients with a high clinical suspicion for PTE.[4]

Veterinary patients traditionally require general anesthesia for these advanced imaging modalities. General anesthesia is potentially harmful in many critical patients, which has precluded the use of CT for the detection of certain diseases in veterinary medicine. Recently, CT has been performed successfully in awake or lightly sedated small animal patients to evaluate upper airway disease, intrathoracic diseases, and for acute abdomen.[5–7] CE-MDCT has several advantages over other imaging modalities such as rapid acquisition times, superior resolution, easy interpretation, visualization of vascular morphology, high lesion to normal tissue contrast, and detection of concurrent disease.[8] The MDCT is able to image faster and may preclude the need for general anesthesia in some patients. This case report, to the authors' knowledge, is the first report to document PTE secondary to pyothorax and the use of CE-MDCT to diagnose PTE in an awake dog.

Case Summary

A 5-year, 4-month-old, 37.2 kg, female neutered Labrador Retriever was presented to a University referral hospital emergency service for evaluation of pleural effusion. Previous medical history included a recent pododermatitis treated with fluconazole, a tapering dose of prednisone and a topical miconazole spray. The owner discontinued medications 3 days prior to the initial presentation due to vomiting. Two days prior to referral, the dog was presented to her primary care veterinarian for lethargy, vomiting, and tachypnea. A serum biochemistry profile was performed and abnormalities included increased blood urea nitrogen (9.9 mmol/L [28 mg/dL], reference range, 2.5–9.6 mmol/L [7–27 mg/dL]) and globulin concentrations (49.0 g/L [4.9 g/dL], reference range, 25–45 g/L [2.5–4.5 g/dL]) and increased alkaline phosphatase activity (258 U/L, reference range, 23–212 U/L). The dog was hospitalized and treated with an antiemetic and gastroprotectants. After a day of hospitalization at the referring clinic, the dog's condition deteriorated and her respiratory effort worsened. A CBC was performed and abnormalities included increased hemoglobin (193 g/L [19.3 g/dL], reference range, 120–180 g/L [12–18 g/dL]), leukocytosis (3.28×10^9/L [$32.84 \times 10^3/\mu$L], reference range, 5.5–16.9 $\times 10^9$/L [5.5–16.9 $\times 10^3/\mu$L]), mature neutrophilia (2.79×10^9/L [$27.85 \times 10^3/\mu$L], reference range, 2–12 $\times 10^9$/L [2–12 $\times 10^3/\mu$L]), and monocytosis (3.5×10^9/L [$3.5 \times 10^3/\mu$L], reference range, 0.3–2.0 $\times 10^9$/L [0.3–2.0 $\times 10^3/\mu$L]). A review of the referring veterinarian's records notes that an oxygen saturation (SpO_2) of 94% via pulse oximetry was initially obtained. Referring thoracic radiographs showed bilaterally symmetrical retraction of the lung lobes by soft tissue opacity material consistent with moderate pleural effusion. Thoracocentesis was performed and yielded 166 mL of a serosanguinous fluid. After the procedure, the patient was noted to have an SpO_2 of 82%. Butorphanol (0.4 mg/kg subcutaneously [SQ]) was administered for sedation before the dog was referred to our emergency service.

On presentation to our hospital (day 1), the patient was dyspneic and was obtunded. Mucous membranes were slightly cyanotic and tacky with a capillary refill time of 3–4 seconds. The dog was mildly hypothermic with a temperature of 37.2°C (98.9°F). Heart rate was 140/min and respiratory rate was 60/min with marked, mixed inspiratory-expiratory effort. Due to the patient's cardiovascular instability, skin pigmentation, and motion, a consistent SpO_2 reading was not obtained. Lung sounds were increased in the dorsal lung fields but were muffled ventrally. Heart sounds were also muffled and femoral pulses were fair in quality without any pulse deficits noted. Systolic blood pressure was 110 mm Hg via indirect Doppler measurement. The patient was estimated to be 8% dehydrated based on mucous membrane moisture, decreased skin turgor, and the history of several days of anorexia. Abnormalities on a venous blood gas panel[a] included acidemia, decreased bicarbonate, hyponatremia, hypocalcemia (ionized), hypermagnesemia (ionized), hyperglycemia, hyperlactatemia, and increased blood urea nitrogen concentration (Table 1).

Treatment was initiated immediately upon presentation. An 18-Ga catheter[b] was placed in the left cephalic vein and resuscitation with IV fluid therapy was initiated with a 1 L bolus of lactated Ringer's solution (LRS)[c] administered over approximately 15–30 minutes. Supplemental oxygen was administered via flow by facemask at a flow rate of 4.5 L/min. Thoracocentesis performed on both left and right hemithoraces yielded approximately 600 mL of a serosanguinous fluid from the left and approximately 1,200 mL from the right. Samples were submitted for aerobic and anaerobic bacterial culture and sensitivity; the samples later yielded moderate growth of *Streptococcus canis*. Respiratory rate and respiratory effort did not markedly improve after thoracocentesis. SpO_2 readings were still considered unreliable at this point as heart rate on the pulse oximeter did not match with the patient's. Fluid analysis of the pleural effusion revealed a nucleated cell count of 182 ×

Table 1: Blood gas and electrolyte panel data from a dog with pyothorax and suspected development of pulmonary thromboembolism

	Day 1		Day 2			Day 3			Day 4		Day 5
Venous (V)/arterial (A)	**V**	**A**	**A**	**A**	**A**	**A**	**A**	**V**	**A**	**V**	**V**
FiO_2 (%)	21	21	21	21	100	40*	40*	21	21	21	21
pH (7.39–7.49)	7.323	7.249	7.372	7.402	7.223	7.383	7.403	7.452	7.472	7.459	7.463
PCO_2 (23.11–37.4 mm Hg)	26.9	44.7	32.9	31.2	59.6	40.2	37.5	38.4	31.4	38.7	31.8
PO_2 (80–100 mm Hg)	44.4	79.9	79.4	72	127	55.9	102.9	48.2	68.6	51.9	46.3
HCO_3 (17.1–24.9 mmol/L)	14.1	19.7	19.3	19.6	24.8	24.2	23.6	27.1	30.5	27.7	22.9
Na^+ (144–151 mEq/L)	135.2	137.2	135.5	135.3	140.1	137.9	136.6	139.8	141.0	143.8	145.5
K^+ (3.7–4.9 mEq/L)	5.26	4.92	5.34	5.1	5.13	3.99	3.74	3.74	3.48	3.25	3.55
Cl^- (110–118 mEq/L)	108.4	107.9	110.6	113.4	112.6	111.3	111.2	111.2	110.5	113.3	117.7
iCa (1.17–1.37 mmol/L)	1.01	1.07	1.18	1.10	1.15	1.13	1.15	1.15	1.22	1.17	1.18
iMg (0.47–0.62 mmol/L)	0.71	0.49	0.73	0.69	0.69	0.56	0.54	0.54	0.53	0.49	0.46
Glu (4.32–7.22 mmol/L; 78–130 mg/dL)	7.1 (128)	5.5 (99)	6.8 (123)	5.7 (102)	6.3 (113)	4.5 (81)	4.1 (74)	4.1 (74)	5.8 (104)	4.8 (87)	6.8 (123)
Lac (0.44–2.93 mmol/L)	3.1	1.6	0.6	0.5	0.1	0.0	0.4	0.4	0.6	1.2	2.7
Cre (44.2–132.6 μmol/L; 0.5–1.5 mg/dL)	221 (2.5)	159 (1.8)	106 (1.2)	97.2 (1.1)	88.4 (1.0)	70.7 (0.8)	70.7 (0.8)	61.9 (0.7)	70.7 (0.8)	70.7 (0.8)	70.7 (0.8)
Blood urea nitrogen (2.14–10.7 mmol/L; 6–30 mg/dL)	34.6 (97)	23.9 (67)	15 (42)	12.9 (36)	11.4 (32)	7.1 (20)	5.4 (15)	3.9 (11)	3.2 (9)	2.5 (7)	2.5 (7)

Reference ranges in parentheses (SI units; conventional units).
Na^+, sodium; K^+, potassium; Cl^-, chloride; iCa, ionized calcium; iMg, ionized magnesium; Glu, glucose; Lac, lactate; Cre, creatinine.
*Estimated ~40% oxygen based on 134 mL/kg/min flow rate of nasal oxygen.[29]

10^9/L (182 × 10^3 cell/μL), RBC count of 56 × 10^9/L (56 × 10^3 cells/μL), with a total protein 48 g/L (4.8 g/dL), and specific gravity of 1.030. The sample consisted of a mixed inflammatory population predominated by degenerate neutrophils with few macrophages. Many neutrophils contained phagocytized cocci bacteria. Within the background were red blood cells and stippled proteinaceous material. The pleural effusion was consistent with septic suppurative inflammation.

A CBC, serum biochemistry profile, prothrombin time, and activated partial thromboplastin time (aPTT) were submitted for analysis. CBC abnormalities included a leukocytosis, neutrophilia, increased bands, and thrombocytopenia. Serum biochemistry and electrolyte abnormalities included azotemia, hypoproteinemia, hypoalbuminemia, hyperphosphatemia, hyponatremia, hypochloridemia, and increased alkaline phosphatase activity. Coagulation panel abnormalities included a slightly prolonged aPTT and increased fibrinogen concentration. The blood gas/electrolyte panel, biochemistry profile, CBC, and coagulation profile results during the dog's hospitalization are listed in Tables 1 and 2.

After stabilization with fluid resuscitation and thoracocentesis, the dog was anesthetized for bilateral thoracostomy tube placement and routine thoracic CT. The dog was premedicated with methadone[d] (2.5 mg/kg IV), induced with propofol[e] (3.5 mg/kg IV), and maintained on isoflurane[f] in oxygen. A 20-Ga catheter[g] was placed in the dorsal pedal artery for blood gas sampling purposes. Two trocar style, 28-Fr thoracostomy tubes[h] were placed as previously described.[9] An additional 500 mL of fluid and 500 mL of air was removed after thoracostomy tubes were placed. CT imaging was performed using routine CT protocols with the patient under general anesthesia and breathing passively.

Using a 16 slice helical CT scanner,[i] the following scan and display parameters were used: 120 kV, 280 mA, 0.8 seconds rotation speed, 5 mm × 2.5 mm slice thickness, and 1.37 pitch factor. Sagittal and dorsal reformations were made from reconstructed 0.625 mm slices. Contrast was hand injected with 60 mL of a nonionic iodinated contrast medium[j] (300 mg I/mL). There was a mild volume of soft tissue attenuating, minimally contrast-enhancing material (26 Hounsfield units [HU] precontrast, 36 HU postcontrast) in the pleural space, consistent with pleural fluid. There was a rounded (4.3 mm × 8.7 mm), irregularly marginated, hypoattenuating to surrounding pleura, nonenhancing lesion with a thin enhancing soft tissue rim (58 HU precontrast, 131 HU postcontrast) along the right cranioventral thoracic wall. In the mediastinum, there was wispy soft tissue to fluid attenuating, contrast-enhancing material in the cranioventral mediastinum (or cranial and ventral-most aspect of the pleural space), which was interspersed within the mediastinal fat. The sternal lymph nodes were enlarged and rounded, measuring 1.6 cm in diameter. The cranial mediastinal lymph nodes were enlarged and rounded, measuring 1.2 cm in diameter. In the lungs, cranioventral (dependent) lung fields were characterized

Table 2: Serum biochemical profile, CBC, and coagulation data from a dog with pyothorax and suspected development of pulmonary thromboembolism

	Day 1	Day 3	Day 4	Day 5
Total protein (51–70 g/L; 5.1–7.0 g/dL)	<30 (<3.0)	<30 (<3.0)		
Albumin (25–38 g/L; 2.5–3.8 g/dL)	11 (1.1)	8 (0.8)		
Phosphorus (0.87–1.68 mmol/L; 2.7–5.2 mg/dL)	3.23 (10.0)	1.32 (4.1)		
ALP (7–92 U/L)	146	56		
WBC (5.5–16.9 $\times$ 10^3/mL)	23.3	29.7	26.2	
Neutrophils (3–11.5 $\times$ 10^3/mL)	17.7	24.1	24.1	
Bands (0–0.3 $\times$ 10^3/mL)	1.4	2.1	.265	
HCT (35–52%)	51.8	33.1	34.3	
Platelets (200–900 $\times$ 10^3)	117	106	131	
PT (6–10 sec)	9.4	10.0	9.9	9.5
aPTT (9–15 sec)	16.1	19.8	22.7	26
Fibrinogen (3.44–7.97 μmol/L;117–271 mg/dL)	17.5 (596)	9.5 (325)	10.4 (355)	11.1 (379)

Reference ranges in parentheses (SI units; conventional units).
ALP, alkaline phosphatase; PT, prothrombin time; aPTT, activated partial thromboplastin time.

by a severe alveolar pattern in all lung lobes and were decreased in volume. There was no evidence of pulmonary abscessation. Extrathoracic structures included a round, hypoattenuating nodule within the cranial aspect of the liver, measuring 4 mm in diameter (35 HU precontrast, 62 HU postcontrast). There was also evidence of mild bilateral pneumothorax with more air on the right than left.

During anesthesia for thoracostomy tube placement and CT, arterial blood gas analysis revealed a PaO_2 of 79.9 mm Hg with the SpO_2 ranging from 88% to 95% while on 100% oxygen. Posttube placement, a red rubber nasal cannula (8-Fr) was placed in the left nostril with oxygen flow set at 2 L/min. The dog was admitted to the intensive care unit for monitoring and supportive care. Continuous suction using a chest drainage system[k] was necessary due to the presence of a pneumothorax presumed to be iatrogenic. Due to the increased respiratory rate and effort (72/min) and an SpO_2 between 91% and 93%, the flow rate was increased to 4 L/min. The SpO_2 continued to fluctuate between 93% and 97% with labored breathing so an additional nasal cannula (10-Fr) was placed and combined oxygen flow rate (delivered via a Y-adapter) kept at 4 L/min. The SpO_2 increased to 97% and breathing efforts improved. Dehydration was corrected over 8 hours using LRS at 420 mL/h. She also received a constant rate infusion (CRI) of hetastarch[l] (20 mL/kg/d) to support colloid osmotic pressure in light of the hypoalbuminemia and a CRI of fentanyl,[m] lidocaine,[n] and ketamine[o] (FLK) (fentanyl 4 μg/kg/h, lidocaine 25 μg/kg/h, ketamine 3 μg/kg/min) to address analgesia. Parenteral antimicrobials included ampicillin/sulbactam[p] (30 mg/kg IV q 8 h) and clindamycin[q] (11 mg/kg IV q 12 h).

On day 2, the patient underwent a median sternotomy to allow exploration of the thoracic cavity and to determine the etiology of the pyothorax. Necrotic and fibrotic material was noted on the ventral aspect of the mediastinum and the pericardium. This material extended from the base of the heart to the cranial most aspect of the thoracic cavity. The necrotic and fibrotic material was removed along with 1 mediastinal lymph node and all were submitted for histopathological examination. Samples were submitted for aerobic and anaerobic bacterial culture and sensitivity; the aerobic samples again grew *S. canis*, but there was no anaerobic growth. No cause was found for the pneumothorax noted on the previous day's CT and was presumed to be secondary to thoracostomy tube placement.

Postoperatively, the dog was changed to a morphine, lidocaine, ketamine (MLK; morphine 6 μg/kg/h, lidocaine 25 μg/kg/h, ketamine 3 μg/kg/min) CRI because of supply and cost issues with fentanyl. Overnight, the patient continued to have labored breathing despite oxygen supplementation and the SpO_2 averaged 94% on 4 L/min combined oxygen flow. The flow rate was increased to 5 L/min and the SpO_2 increased to 96%. Differential diagnoses for the hypoxemia included aspiration pneumonia, cardiogenic pulmonary edema, acute respiratory distress syndrome, or PTE.

On day 3, CE-MDCT was performed without general anesthesia; other studies at our institution have demonstrated that this CT scanner can be used with minimally sedated animals.[5–7] The MLK CRI was stopped 30 minutes prior to imaging and the dog was awake but with obtunded mentation. The patient was placed in sternal recumbency in a padded trough and a cloth was placed over the eyes to minimize visual stimulation. Two Velcro straps fitted to the CT table were used to restrain the patient around the abdomen and shoulder/neck areas. Flow by oxygen at a flow rate of 5 L/min via facemask was administrated throughout imaging. Scout images of the thorax were obtained in the lateral and

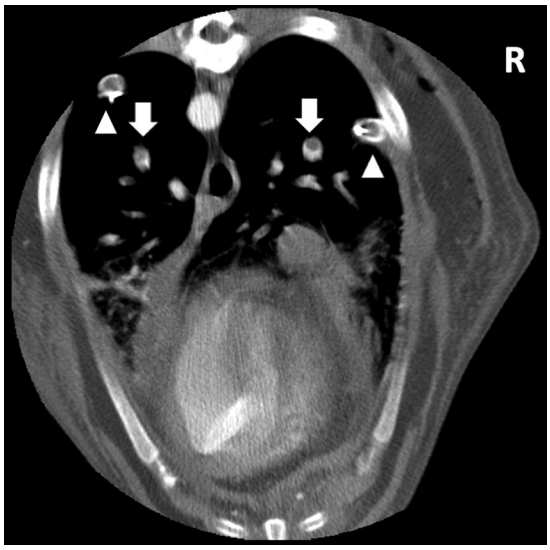

Figure 1: Transverse CT angiographic image indicating luminal-filling defects in the right and left caudal lobar primary pulmonary arteries consistent with multiple pulmonary thromboembolisms (arrows) and thoracostomy tubes (arrow heads).

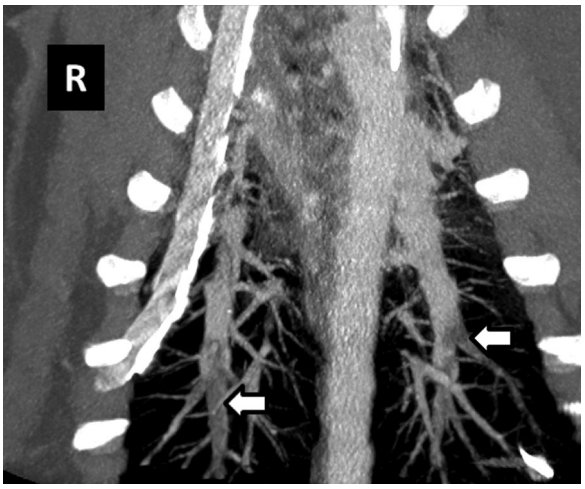

Figure 2: Dorsal plane maximum intensity projection (MIP) CT angiographic image indicating luminal-filling defects in the right and left caudal lobar primary pulmonary arteries consistent with multiple pulmonary thromboembolisms (arrows).

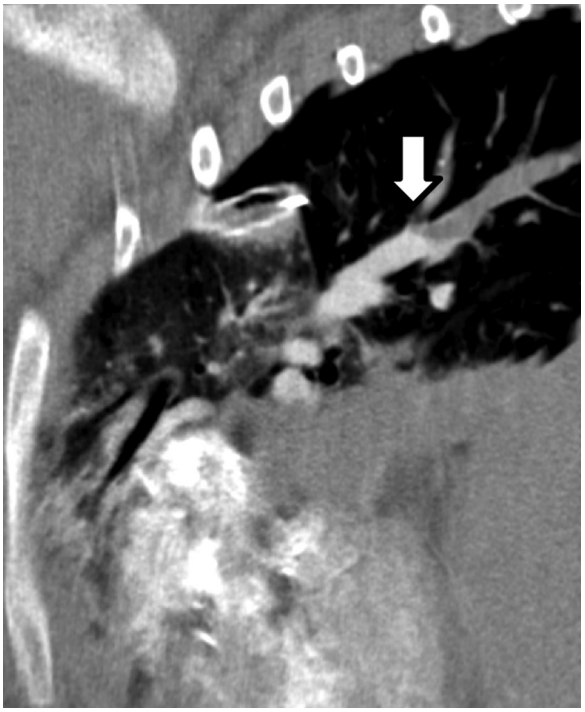

Figure 3: Sagittal plane MIP CT angiographic image indicating luminal-filling defect in the right caudal lobar primary pulmonary artery consistent with a pulmonary thromboembolism (arrow). Aspect ratios have been maintained.

dorsal-ventral projection of the entire chest. Precontrast images were obtained from the entire chest using the following helical technique: 120 kV, 200 mA, and 0.8 seconds rotation time, 0.9:1 pitch, 5 mm slice thickness with 2.5 mm overlap, large scan field of view (FOV) and 28.5 cm display FOV. A bolus of 3 mL/kg (300 mg I/mL, 900 mg I/kg body weight [BW]) iohexol was injected IV into a cephalic vein catheter at 3 mL/s using a power injector followed by a 10 mL 0.9% saline flush. Postcontrast images were obtained through a narrowed range from the cranial lung fields at a level where the pulmonary arteries measured 2 mm caudally to where the caudal lobar arteries measured 2 mm. Four contiguous imaging phases were collected starting immediately upon initiation of the contrast injection. Scan time for each phase was 5.9 seconds with 3.2 seconds delay between phases for repositioning of the couch. The following scan and display parameters were used postcontrast: 100 kVp, 250 mA, 5 × 2.5 mm slice thickness, 3-mm reconstruction index, 1.375:1 pitch, 13.75 speed (mm/rot), large scan FOV, 22 cm FOV, window center of −600 HU, and window width of 1,600 HU. CE-MDCT demonstrated hypoattenuating luminal-filling defects in the right cranial and right and left caudal lobar primary pulmonary arteries consistent with multiple PTE (Figures 1–3). There was trivial motion artifact and images were considered of excellent diagnostic quality.

Anticoagulant therapy for the PTE with unfractionated heparin[r] (UFH; 270 U/kg SQ q 8 h) was initiated after the CT. The goal of this dose was aimed to prolong the aPTT by 1–1.5 times normal. Daily treatment orders included flushing of both thoraces with 500 mL of warmed LRS. Because the dog was anorexic, a nasogastric tube[s] (8-Fr) was placed to provide enteral nutrition. A CRI of liquid diet[t] at one-quarter resting energy requirements (30 × body weight [kg] + 70) was initiated.

A CRI of metoclopramide[u] was initiated at 2 mg/kg/d to reduce regurgitation while the patient was recumbent. The blood gas/electrolyte panel performed that evening showed evidence of hypoglycemia (Table 1). Dextrose supplementation was added to the IV fluids to make a 2.5% dextrose solution (1.6 mg/kg/min) to address the hypoglycemia. The MLK CRI was discontinued due to undesirable side effects (pytalism, nausea, inappetance, and panting) and methadone (0.22 mg/kg IV q 6 h) started for analgesia. Dolasetron mesylate[v] (0.8 mg/kg IV q 24 h) was initiated to address the nausea. Aerobic culture of the fluid obtained on day 1 via thoracocentesis yielded moderate *S. canis* growth.

The thoracostomy tubes were manually aspirated every 2–4 hours and yielded significantly less fluid (363 mL/d compared to 2,500 mL/d the previous day). The next morning (day 4), the dog was clinically much improved and her increased activity led to the inadvertent removal of the thoracostomy tubes. Appetite was markedly improved, as was the dog's mentation from the previous day. The dog was transitioned to oral medications including tramadol[w] (3 mg/kg by mouth q 8–12 h) and gabapentin,[x,y] (8 mg/kg per os [PO] q 12 h) for analgesia. Oxygen supplementation was discontinued by the end of day 4.

On day 5, the IV fluids were discontinued. The dog was discharged with amoxicillin/clavulanic acid[i] (14.7 mg/kg by mouth q 12 h for 21 d), clindamycin[q] (8.1 mg/kg by mouth q 12 h for 21 d), tramadol[w] (3 mg/kg by mouth q 12 h for 14 d), gabapentin[x] (8.1 mg/kg by mouth q 12 h for 14 d), and unfractionated heparin[r] (UFH) (243 units/kg SQ q 8 h for 3 d, then 189 units/kg SQ q 8 h for 3 d, followed by 189 units/kg SQ q 12 h for 3 d). The aPTT was prolonged 1.7 times normal on the day of discharge. The long-term plan included low-dose aspirin therapy (1.1 mg/kg by mouth q 24 h) to be started after the completion of UFH.

After the patient was discharged, aerobic culture results from samples obtained during surgery yielded scant *S. canis* growth, with susceptibility to ampicillin/sulbactam and clindamycin. There was no growth of any strict anaerobic bacteria. Histopathology of the mediastinal lymph node revealed subacute diffuse severe fibrinosuppurative lymphadenitis with intralesional cocci. Histopathology of the mediastinal tissue revealed subacute diffuse severe fibrinosuppurative pleuritis with thrombi. At last follow-up phone communication with the owner 4 months after discharge, the patient was doing well and was still on aspirin therapy.

Discussion

The definitive cause of the PTE in the dog described in this case report remains undetermined. There are several possibilities and one is that the dog developed pyothorax first and then subsequently developed PTE. In this scenario, proinflammatory cytokines released in response to the bacterial infection may have activated the coagulation cascade resulting in PTE.[10] Acute infection and associated systemic inflammation are risk factors for PTE in human and veterinary medicine.[1,11,12] Another possibility is that the thoracotomy procedure itself could have caused enough inflammation and cellular damage that may have resulted in the development of PTE. Major surgical procedures have been associated with increased risk of PTE.[13] Another theory is that the PTE was present first and the pyothorax occurred secondarily. Septic pulmonary emboli may have lodged in the pulmonary vasculature and subsequently caused the pyothorax. In this case, the source of the septic emboli may have been from the previously diagnosed pododermatitis. Idiopathic pododermatitis may be associated with a secondary bacterial infection.[14] In a study regarding interdigital cysts in dogs, hemolytic streptococci were cultured in 12.5% of the samples.[15] Based on the dog's clinical presentation and minimal clinical improvement after thoracocentesis, the first scenario seems most fitting and the PTE was present prior to presentation to our hospital.

Pyothorax is an accumulation of septic exudate in the pleural cavity. In 1 retrospective study evaluating both canine and feline cases, the underlying etiology of pyothorax was identified in 22% of dogs with the predominant causes being a migrating foreign body or penetrating trauma.[16] Other possible causes include esophageal perforation, parasites, bacterial pneumonia, hematogenous spread, discospondylitis, or necrotic pulmonary neoplasia.[17] One retrospective study found that 11% of aerobic bacteria isolated from pleural fluid samples from dogs with pyothorax yielded *S. canis*.[18]

CE-MDCT is the current gold standard for the diagnosis of PTE in human patients. The most specific finding of PTE on CE-MDCT is an intraluminal-filling defect in the pulmonary vasculature. CE-MDCT was used successfully to image PTE in an awake dog in this case report. Previous studies indicated the need of general anesthesia for pulmonary angiography in cats[19] and dogs.[20,21] From earlier investigations at our institution, we have successfully demonstrated CT scanning on awake or lightly sedated animals without sacrificing image quality.[5,6] This precipitated further studies investigating dogs with portosystemic shunts[22] and acute abdominal signs.[23] An ongoing study of cats in congestive heart failure using a 16 slice MDCT system indicates that adequate systemic and pulmonary arterial opacification can be obtained by performing 3 contiguous phases beginning immediately upon completion of the injected contrast bolus. These protocols did not utilize a dynamic CT sequence

and test dose to determine timing of maximum artery opacification. We modified these protocols by limiting the scanned thorax to larger pulmonary arteries (>2 mm diameter).

The CE-MDCT in this dog provided a rapid and accurate diagnosis of PTE, which led to the initiation of anticoagulant therapy. This report indicates that the diagnosis of PTE can be obtained in an awake dog with CE-MDCT using fast multiphase protocols. Further refinement of this type of protocol and a larger series of clinical cases is needed to better evaluate the clinical utility of CE-MDCT in awake dogs.

It should be noted that the PTE was not found on retrospective examination of the first CT. The initial postcontrast CT was a "delayed study," not timed for pulmonary arteries. The acquisition was initiated approximately 15 seconds after completion of a 600 mg I/kg BW injection of nonionic iodinated contrast medium. The intent of the study was to determine parenchymal pulmonary, pleural or mediastinal lesions. There was insufficient contrast on the pulmonary arteries for determination of PTE lesions.

Therapy for PTE can be difficult and the use of anticoagulants, antiplatelet therapy, and thrombolytics has been described. Clot resolution may take weeks to months, and the goal of therapy is to limit thrombus growth and prevent recurrence.[24] Heparins are a common anticoagulant and enhance the inhibitory function of antithrombin. Antithrombin functions as an anticoagulant by inactivating certain proteases such as factor Xa and thrombin. Heparins consist of unfractionated and low molecular weight formulations. UFH has a high variable dose-response behavior necessitating close monitoring by coagulation testing. Low molecular weight heparins tend to enhance antithrombin's inhibition of factor Xa more so than its inhibition of thrombin. Although low molecular weight heparins have better bioavailability, fewer adverse effects and has more predictable pharmacokinetics than UFH, their high cost often prohibits their use in veterinary medicine.[25] UFH was the most economical choice for this dog and was used for the acute treatment phase. One suggested dose of UFH in dogs is 200–500 U/kg administered SQ every 8 hours with a target aPTT of 1 and one-half to 2 times baseline values.[25]

Aspirin was selected for chronic treatment because it may reduce the recurrence of PTE. Aspirin has a wider safety margin than other anticoagulants[26] and is not costly. One human study found that aspirin therapy reduced the rate of recurrence by approximately 40% compared to the placebo group; this study supported the benefit of aspirin in recurrent venous thromboembolism prevention with no apparent increase in the risk for major bleeding.[27] Clopidogrel, an oral antiplatelet medication with a different mechanism than that of aspirin, has been evaluated in dogs. In a population of dogs with immune-mediated hemolytic anemia, clopidogrel was found to be safe and had similar short-term survival rates to dogs receiving ultra-low-dose aspirin.[28] As with UFH and low molecular weight heparin, aspirin was chosen over clopidogrel due to cost.

Prognosis for recovery from PTE is guarded but improves with early detection and treatment. The use of CE-MDCT without general anesthesia allowed for rapid diagnosis and reduced any potential morbidity associated with anesthesia. Unfractionated heparin followed by low-dose aspirin therapy was successfully used in this patient. In the future, the use of CE-MDCT for the detection of PTE as described in this report may be used to build imaging protocols at other institutions with similar equipment, eliminating the need for general anesthesia in this critically ill population.

Footnotes

[a] Critical Care Xpress, Nova Biomedical, Waltham, MA.
[b] BD Insyte™, Becton Dickinson, Sandy, UT.
[c] Veterinary Lactated Ringer's Injection USP, Abbott Laboratories, North Chicago, IL.
[d] Methadone hydrochloride, Mylan Institutional LLC, Rockford, IL.
[e] Propoflo™, Abbott Laboratories.
[f] VetOne Fluriso, MWI, Boise, ID.
[g] BD Insyte™.
[h] Thoracostomy tubes, Tyco Healthcare Group LP, Mansfield, MA.
[i] GE Lightspeed 16 Slice CT, Milwaukee, WI.
[j] Omnipaque 300, GE Healthcare Inc, Princeton, NJ.
[k] Pleur-evac, Deknatel, Inc, Falls River, MA.
[l] Six percent hetastarch in 0.9% sodium chloride injectable, Hospira Inc.
[m] Fentanyl Citrate, Hospira Inc.
[n] Two percent lidocaine injection, Hospira Inc.
[o] Ketaset®, Pfizer, New York, NY.
[p] Unasyn, Pfizer Animal Health, New York, NY.
[q] Clindamycin hydrochloride injection, Hospira Inc.
[r] Heparin sodium injection, Fresenius Kabi USA LLC, Schaumburg, IL.
[s] Nasogastric feeding tube, Mila International, Erlanger, KY.
[t] Clinicare, Abbott Laboratories.
[u] Metoclopramide injection, Hospira Inc.
[v] Anzemet, Sanofi-aventis U.S., Bridgewater, NJ.
[w] Tramadol hydrochloride, Northstar Rx LLC, Memphis, TN.
[x] Gabapentin, Northstar Rx LLC.
[y] Clavamox, Pfizer Animal Health.

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